RISK ASSESSMENT

Hexabromocyclododecane

CAS-No.: 25637-99-4

EINECS-No.: 247-148-4

Final report
May 2008

FINAL APPROVED VERSION

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Foreword

We are pleased to present this Risk Assessment Report which is the result of in-depth work carried out by experts in one Member State, working in co-operation with their counterparts in the other Member States, the Commission Services, Industry and public interest groups.

The Risk Assessment was carried out in accordance with Council Regulation (EEC) 793/93[1] on the evaluation and control of the risks of "existing" substances. "Existing" substances are chemical substances in use within the European Community before September 1981 and listed in the European Inventory of Existing Commercial Chemical Substances. Regulation 793/93 provides a systematic framework for the evaluation of the risks to human health and the environment of these substances if they are produced or imported into the Community in volumes above 10 tonnes per year.

There are four overall stages in the Regulation for reducing the risks: data collection, priority setting, risk assessment and risk reduction. Data provided by Industry are used by Member States and the Commission services to determine the priority of the substances which need to be assessed. For each substance on a priority list, a Member State volunteers to act as "Rapporteur", undertaking the in-depth Risk Assessment and recommending a strategy to limit the risks of exposure to the substance, if necessary.

The methods for carrying out an in-depth Risk Assessment at Community level are laid down in Commission Regulation (EC) 1488/94[2], which is supported by a technical guidance document[3]. Normally, the "Rapporteur" and individual companies producing, importing and/or using the chemicals work closely together to develop a draft Risk Assessment Report, which is then presented at a meeting of Member State technical experts for endorsement. The Risk Assessment Report is then peer-reviewed by the Scientific Committee on Health and Environmental Risks (SCHER) which gives its opinion to the European Commission on the quality of the risk assessment.

If a Risk Assessment Report concludes that measures to reduce the risks of exposure to the substances are needed, beyond any measures which may already be in place, the next step in the process is for the "Rapporteur" to develop a proposal for a strategy to limit those risks.

The Risk Assessment Report is also presented to the Organisation for Economic Co-operation and Development as a contribution to the Chapter 19, Agenda 21 goals for evaluating chemicals, agreed at the United Nations Conference on Environment and Development, held in Rio de Janeiro in 1992 and confirmed in the Johannesburg Declaration on Sustainable Development at the World Summit on Sustainable Development, held in Johannesburg, South Africa in 2002.

This Risk Assessment improves our knowledge about the risks to human health and the environment from exposure to chemicals. We hope you will agree that the results of this in-depth study and intensive co-operation will make a worthwhile contribution to the Community objective of reducing the overall risks from exposure to chemicals.

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[1] O.J. No L 084, 05/04/199 p.0001 – 0075

[2] O.J. No L 161, 29/06/1994 p. 0003 – 0011

[3] Technical Guidance Document, Part I – V, ISBN 92-827-801 [1234]

Information on the rapporteur

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0 CONCLUSIONS AND OVERALL RESULTS OF THE RISK ASSESSMENT

CAS No.: 25637-99-4

EINECS No.: 247-148-4

Substance Name: Hexabromocyclododecane (HBCDD)

Environment

Aquatic compartment

(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 (ii) applies to production and micronising, industrial use of EPS and HIPS and for individual sites in the other use areas.

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

Conclusion (iii) applies to some sites involved in EPS formulation including the generic scenario, one site involved in XPS formulation including the generic scenario, the generic local scenario for formulation of polymer dispersions for textiles, sites involved in industrial use of XPS including the generic local scenario for industrial use of XPS compound and sites involved in textile backcoating including the generic scenario. A general conclusion (iii) is drawn for textile backcoating based on measured concentrations in sediment downstream three different locations giving RCRs >1. There is no concern at the regional level.

STP

(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 (ii) applies to production, micronising, EPS formulation, XPS formulation, Textile formulation, Industrial use of EPS, industrial use of HIPS, individual sites involved in industrial use of XPS compound, sites involved in industrial use of HBCCD powder for XPS and individual sites involved in textile backcoating.

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

Conclusion (iii) applies to some sites with industrial use of XPS having intermittent releases to waste water and for 1 textile backcoating site including the generic textile backcoating.

Terrestrial compartment

(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 (ii) applies to production, micronising, EPS-formulation, XPS-formulation, textile backcoating formulation, industrial use of EPS and HIPS, all sites involved in industrial use of XPS compound (based on site specific risk characterisation), most of the sites involved in industrial use of HBCDD powder for XPS production and for most textile backcoating sites. There is no concern at the regional level.

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

Conclusion (iii) applies to the generic local scenario for the industrial use of XPS compound, three sites involved in industrial use of HBCDD powder in the production of XPS and one site involved in textile backcoating includingthe generic textile backcoating scenario.

Atmosphere

(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.

This conclusion applies to all use areas of HBCDD.

Non-compartment specific effects relevant for the food chain (secondary poisoning)

In the light of HBCDD being a PBT substance (c.f. 3.4.6) and considering the large uncertainties both in the derivation of PECs and in the derivation of PNEC it is not considered appropriate to draw conclusions for the individual sites. Since for PBT-substances the major concern is that accumulation of such substances in the foodchain may result in unpredictable effects in the long term it is appropriate to draw an overall conclusion (iii) for secondary poisoning.

Marine compartment

(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 (ii) applies to production and micronising, industrial use of EPS and HIPS and for individual sites in the other use areas. There is no concern at the regional level.

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

Conclusion (iii) applies to some sites involved in EPS formulation including the generic scenario, one site involved in XPS formulation including the generic scenario, one site involved in formulation of polymer dispersions for textiles including the generic scenario, some sites involved in industrial use of XPS including the generic local scenario for industrial use of XPS compound and sites involved in textile backcoating including the generic scenario. A general conclusion (iii) is drawn for textile backcoating. based on measured concentrations in sediment downstream three different locations giving RCRs >1.

PBT-assessment

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

HBCDD does not unequivocally fulfil the specific P-criterion, with some reliable studies indicating that biodegradation can occur. It does however not degrade rapidly and monitoring data indicate a significant degree of environmental transport and overall stability. The BCF of HBCDD is 18100 and thus the vB criterion is fulfilled. Also the T-criterion is fulfilled according to available data. HBCDD is ubiquitous in the environment, being also found in remote areas far away from point sources. The highest concentrations of HBCDD are detected in marine top-predators such as porpoise and seals showing that HBCDD bioaccumulates up the food chain. Based on an overall assessment the TCNES subgroup on identification of PBT and vPvB substances have concluded that HBCDD has PBT properties according to the PBT criteria of the TGD.

Human health effects assessment

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

There are indications of developmental neurotoxicity in adult mice exposed to HBCDD as pups. However, this study by Eriksson et al (2006) is not performed according to current guideline and GLP and therefore this potential developmental neurotoxicity needs to be examined further and conclusion (i) is reached for all exposure scenarios.

However, similar results on developmental neurotoxicity have been published for decabromodiphenylether by the same authors using the same method. For decabromodiphenylether it has been agreed to perform a new toxicokinetics/developmental neurotoxicity study according to a modified OECD guideline and GLP. The results from this new decabromodiphenylether study will serve as guidance on how to interpret the data from the Eriksson study, and may also serve as a basis on how to proceed with further testing of

neurotoxicity. While awaiting these results a **conclusion (i) on hold** with regard to a developmental neurotoxicity study applies.

Workers assessment

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

Conclusion (iii) applies for repeated dose toxicity for workers during filling of HBCDD fine grade powder in production.

Conclusion (iii) also applies for toxicity on reproductive toxicity/fertility for workers during filling of HBCDD fine powder and powder in the production and adding of HBCDD fine powder and powder in industrial use.

Consumers

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

Conclusion (ii) applies to all scenarios for consumers.

Human exposed via the environment

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

Conclusion (ii) applies to all scenarios for humans exposed via the environment.

Human health (physico-chemical properties)

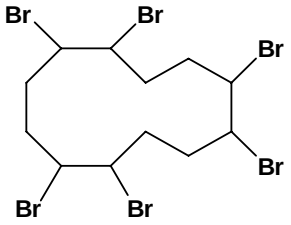
(ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those, which are being applied already

This conclusion applies to flammability, explosive and oxidising properties since they are not considered to be of concern.

1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION

CAS No.:	25637-99-4 (mixture of mainly three diastereomers)
EINECS No.:	247-148-4
IUPAC name:	HEXABROMOCYCLODODECANE
Molecular formula:	$C_{12}H_{18}Br_6$
Structural formula:	



see further Figure 1-1

Molecular weight:	641.7
Synonyms:	cyclododecane, hexabromo-

Two different CAS Numbers can describe Hexabromocyclododecane (HBCDD):

CAS No.	**EINECS**	**Name**
25637-99-4	2471484	Hexabromocyclododecane, and
3194-55-6	2216959	1,2,5,6,9,10- Hexabromocyclododecane

The CAS-No. 3194-55-6 has also been taken into account in this risk assessment report. There are no differences in molecular structure or properties between the chemicals represented by these CAS-numbers.

Separate CAS-numbers have been reported for the three diastereomers, termed α- β- and γ-HBCDD (Janák *et al.*, 2004).

CAS No.	**Name**
134237-50-6	α-Hexabromocyclododecane
134237-51-7	β-Hexabromocyclododecane
134237-52-8	γ-Hexabromocyclododecane

Technical grade HBCDD is generally produced from *cis trans, trans*-1,5,9-cyclododecatriene (CDT), one of four CDT isomers, (CAS No. 27070-59-3). The reaction, *trans*-addition of bromine to the double bounds of CDT, results in the three diastereomers α-, β- and γ-HBCDD. The final distribution of the diastereomers in technical HBCDD varies with a range of about 70-95 % γ-HBCDD and 5-30 % α- and β-HBCDD.

Independently of the isomer composition, the end-product HBCDD could be described by both CAS-No. 2563799-4 and 3194-55-6, where the latter is the most specific due to the included numbering.

1.1.1 Stereochemistry of HBCDD

The occurrence of three chiral diastereomers (pairs of enantiomers) in technical HBCDD complicates the risk assessment. A brief introduction to the stereochemistry of HBCDD and some explanations of the terminology is given below, a more detailed information can be found in articles by Heeb et al., 2005, and Becher, 2005, (Becher, 2005; Heeb *et al.*, 2005).

The stereochemistry of HBCDD is complex and still under evaluation. Theoretically, 16 HBCDD stereoisomers can be formed by bromination of all four CDT isomers and so far eight of those 16 stereoisomers have been found in a technical HBCDD product (Heeb *et al.*, 2005).

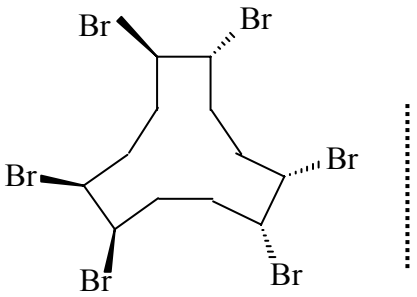
Stereoisomers may be further sub-categorized and have a relation as either enantiomers or diastereomers.

Enantiomers are a pair of molecules that are non-congruent mirror images of one another. They have identical chemical and physical properties except that they rotate plane-polarised light in different directions. An enantiomer with a rotation to the right is denoted (+) while an enantiomer with a rotation to the left is denoted (-). Generally in the production of chiral compounds (i.e. substances existing as enantiomers), the enantiomers are formed in about equal proportions. A 50:50 mixture of enantiomers is termed a racemate. After release to the environment, the enantiomers may interact differentially with other chiral molecules, like in biological systems, and be enriched to various extent in different compartments. Thus, chiral substances will only be distinguishable, or may behave, as two different molecules in a chiral environment. In an achiral environment only one molecule will be evident.

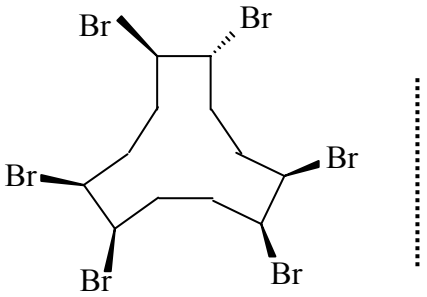
Stereoisomers not related as enantiomers are related as diastereomers. Unlike enantiomers, diastereomers may have different chemical and physical properties.

The three diastereomers α-, β- and γ-HBCDD (Figure 1-1) are all chiral and exist as pairs of enantiomers in technical HBCDD, altogether six stereoisomers (+/-)α-, (+/-)β- and (+/-)γ-HBCDD). The two other stereoisomers found in a technical HBCDD product, termed as δ- and ε -HBCDD (0.5 and 0.3 %, respectively, of total HBCDD product), are tentatively regarded as achiral, i.e. meso forms. The presence of δ- and ε-HBCDD in the technical product is probably due to impurities in the starting material, likely the presence of another CDT isomer (Heeb *et al.*, 2005).

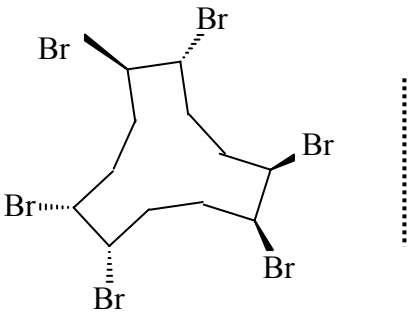
The 16 stereoisomers of HBCDD can also be termed as 10 diastereomers of which six are chiral and exist as pair of enantiomers (altogether 12 stereoisomers) and four are meso forms (achiral).



α-HBCDD, the line indicates a mirror plane



β-HBCDD, the line indicates a mirror plane



γ-HBCDD, the line indicates a mirror plane

Figure 1-1 Main diastereomers (pairs of enantiomers) in technical product of HBCDD.

Data from (Heeb *et al.*, 2005).

1.1.2 Analytical methods

Gas chromatography mass spectrometry (GC/MS) operated in the electron-capture negative-ion (ECNI) mode is a sensitive and commonly used method for analysis of brominated

lipophilic compounds. This method has also been used for detection and quantification of HBCDD in environmental samples (Sternbeck *et al.*, 2001; Sellström *et al.*, 2003).
However, the decomposition and thermal rearrangement of HBCDD at higher temperatures (>160 ºC), see section 1.3.3 on boiling point and thermal rearrangement, cause GC/MS methods to have limitations for HBCDD determinations. Since the high temperatures used in the GC system leads to thermal conversion of HBCDD isomers, isomer specific data cannot be obtained. Thus, GC/MS analysis can so far only give the total HBCDD concentration in a sample. Another limitation has been the lack of analytical standards, to compensate for possible decomposition of HBCDD in the GC system (Law *et al.*, 2005).

The disadvantages with GC/MS methods and an increased interest in determination of the isomer composition of HBCDD lead to development of methods applying liquid chromatography (LC)/MS. Industry has recommended an analytical method with the use of high performance liquid chromatography (HPLC) with negative ion atmospheric pressure chemical ionisation (APCI) mass spectrometry (Ranken, 1999). For identification and quantification selected ion monitoring was recommended.

The use of LC/MS methods makes it possible to achieve both diastereo- and enatioselective separations of HBCDD (Janák *et al.*, 2004; Heeb *et al.*, 2005). A drawback is lower sensitivity compared to GC/ECNI/MS methods, often due to matrix-related problems disturbing the analysis. However, a sensitive and isomer specific method has been developed using HPLC with electrospray ionisation and tandem mass spectrometry (LC/ESI-MS/MS) (Budakowski and Tomy, 2003). Further refinements of LC/MS methods may be done by using labelled standards that now are commercially available (d_{18}-α-, -β- and -γ–HBCDD and $^{13}C_{12}$–α-, -β- and -γ–HBCDD) (Tomy *et al.*, 2004).

For enantioselective separation methods applying chiral-phase HPLC/MS/MS have been developed ((Janák *et al.*, 2004; Heeb *et al.*, 2005; Becher, 2005)).
Determination of the enantiomeric fractions of (+/-)-α-,-β- and -γ-HBCDD in environmental samples have been performed, in liver of three fish species and in muscle of two fish species (Janák *et al.*, 2005). However, enantioselective separations require samples with rather high contamination levels of HBCDD as the method are less sensitive.

An interlaboratory study with the aim to come to a standardised method for HBCDD a determination has been performed (de Boer *et al.*, 2002a). The study included a standard solution, fish (lake trout) and sediment samples. Three laboratories were involved of which two produced both GC/MS and LC/MS data while one laboratory only produced LC/MS data. The results showed variation, which are explained by differences in analytical methodology between the laboratories.
Also, the methods were still under development and not optimized. However the comparability between the GC/MS and LC/MS data was reported to be generally good, although relatively strong deviations were occasionally found between the two methods. In the final BSEF/QUASIMEME interlaboratory study on brominated flame retardants, it was concluded that the main sources of error concerning HBCDD determinations was in the extraction and clean-up procedures (de Boer *et al.*, 2002b).

In another study were the comparability between GC/MS and LC/MS determinations of HBCDD were tested, five soil and five water samples showing a wide range of HBCDD concentrations were analysed by using both techniques (Petersen *et al.*, 2004). The results show small differences between total HBCDD concentrations determined by GC/MS and LC/MS, below 24 %, and in the range of deviations found for duplicate GC/MS determinations within the study (up to 27 %). Two of the soil samples were below the limit of quantification of the LC/MS system.

1.2 PURITY/IMPURITY, ADDITIVES

According to IUCLID the impurities in HBCDD are less than 4 % w/w. The stated impurities are tetrabromocyclododecane and other brominated cyclododecanes.

Technical products with a reported lower purification grade are present in the literature and have been used in some studies, e.g. Hexabromid S (92 % purity). Further information on those products is not available.

The occurrence of polybrominated dibenzofurans (PBDFs) and polybrominated dibenzodioxins (PBDDs), in a technical HBCDD product has been measured (mono- to octa-bromo congeners) (Brenner, 1993). The result, presented in Table 1-4 in the EHC document on dioxins and furans (International Programme on Chemical Safety, 1998) show low amounts of tetra- and penta-BDFs, 20 ppb and 30 ppb, respectively, and no detectable PBDDs (the detection limit was >10 ppb for both PBDFs and PBDDs).
Technical HBCDD is manufactured in two forms, high-melting (HM) and low-melting (LM). The low-melting HBCDD consists of 70-80 % γ-, 20-30 % of α- and β-HBCDD. The high-melting HBCDD consists of 90 % or more of γ-HBCDD.

According to Material Safety Data Sheets from Great Lakes there are three technical products of hexabromocyclododecane (Great Lakes Chemical Corporation, 2002a-c). One product is 100 % pure, another product contains an inorganic stabilizer, and a third product contains 40-60 % HBCDD, water and a component A. The Dead Sea Bromine Group has two technical products, one which is 99.5 % pure and another which is heat stabilized grade (Dead Sea Bromine Group, 2000, 2002). Albemarle also has two technical products, one of high-purity grade available in powder or granular form, the other one of high-purity grade is ground to a fine particle size (Albemarle Corporation, 2000a-b).

All producers supply a stabilised grade of HBCDD. The nature of the particular stabiliser may vary between companies.

1.3 PHYSICO-CHEMICAL PROPERTIES

A summary of the physico-chemical properties of technical HBCDD are shown in Table 1-1. Information on the properties for the individual diastereomers, γ-, α- and β-HBCDD are scarce, for melting points (1.3.2) and indications on water solubility (1.3.6.1) see respective chapter and Table 1-1.

Table 1-1 Physico-chemical properties of technical HBCDD

Property	Value	Reference
Chemical formula	$C_{12}H_{18}Br_6$	
Molecular weight	641.7	
Physical state	White odourless solid	
Melting point	Ranges from approximately: 172-184 °C to 201-205 °C 190 °C , as an average value, is used as input data in EUSES. 179-181 °C α-HBCDD 170-172 °C β-HBCDD 207-209 °C γ-HBCDD	(Smith *et al.*, 2005). (Smith *et al.*, 2005).
Boiling point	Decomposes at >190 °C	(Peled *et al.*, 1995).
Density	2.38 g/cm^3 2.24 g/cm^3	(Albemarle Corporation, 1994) (Great Lakes Chemical Corporation, 1994)
Vapour pressure	6.3·10-5 Pa (21 °C)	(Stenzel and Nixon, 1997)
Water solubility (20 ^{O}C)	66 µg/l (sum of α-, β- and HBCDD) 48.8 µg/l* α-HBCDD 14.7 µg/l* β-HBCDD 2.1 µg/l* γ-HBCDD	(MacGregor and Nixon, 2004).
Partition coefficient n-octanlol/water	Log Kow = 5.62 (technical product) 5.07 ± 0.09 α-HBCDD 5.12 ± 0.09, β-HBCDD 5.47 ± 0.10γ-HBCDD	(MacGregor and Nixon, 1997) (Hayward *et al.*, 2006).
Henry's Law constant	0.75 Pa×m^3/mol Calculated from the vapour pressure and the water solubility (66µg/l)	
Flash point	Not applicable	
Auto flammability	Decomposes at >190 °C	
Flammability	Not applicable-flame retardant!	
Explosive properties	Not applicable	

Oxidizing properties	Not applicable	
Conversion factor	1 ppm = 26.6 mg/m^3 1 mg/m^3 = 0.037 ppm	

*Determined for the isomers present as a mixture not for the pure isomers.

1.3.1 Physical state

HBCDD is a white odourless solid.

1.3.2 Melting point

A variety of melting point ranges are documented in IUCLID and in the open literature. The melting range varies from approximately 172-184 °C for a crude product to 201-205 °C for the highest melting version following crystallisation (Smith *et al.*, 2005).

The following melting intervals have been reported for two products from Albemarle Corporation: 175-183 °C (Saytex HBCDD-LM) and 187-195 °C (Saytex HBCDD-HM) (Albemarle Corporation, 1994).

HBCDD from several unknown suppliers have been tested (Larsen and Ecker, 1988):

HBCDD-1, 202-204 °C (>90 % γ-HBCDD) designated as HBCDD-HM

HBCDD-2, 177-188 °C (13 % α-HBCDD, 10 % β-HBCDD, 73 % γ-HBCDD and 3 % unidentified)

HBCDD-3, 170-186 °C (13 % α-HBCDD, 10 % β-HBCDD, 73 % γ-HBCDD and 3 % unidentified) designated as HBCDD-LM

Melting points for the individual diastereomers have been determined by Smith et al, 2005; γ-HBCDD 207-209 °C, α-HBCDD 179-181 °C and β-HBCDD 170-172 °C (Smith *et al.*, 2005) and by Peled et al., 1995; γ-HBCDD 208-210 °C, α-HBCDD 171-173 °C and β-HBCDD 169-170 °C (Peled *et al.*, 1995):

A melting point of 190 °C, as an average value, is used as input data in EUSES.

1.3.3 Boiling point, thermal rearrangement and decomposition

No boiling point has been measured due to decomposition at higher temperatures.

The thermal rearrangement and decomposition of pure α-, β- and γ-HBCDD was studied at temperatures of 160-200 °C (Peled *et al.*, 1995).

At 190 °C C, the diastereomers started to rearrange already within 10 minutes and it was found that, irrespective of diastereomer studied, all three diastereomers rearranged to give approximately the same final diastereomer distribution within ~30-60 minutes. The thermal equilibrium composition was approximately 78 % α-HBCDD, 13 % β-HBCDD and 9 % γ-HBCDD.

The decomposition was studied at 170-200 °C and was defined as the time taken for weight loss greater than 5 %. According to Peled et al., no substantial differences between the three diastereomers were discerned from the results. Up to 190 °C, α-HBCDD and β-HBCDD were more resistant to thermal decomposition than γ-HBCDD. At 190 °C, it took 2 hours for α-HBCDD to reach a 5 % weight loss while β- and γ-HBCDD needed less time (60 min+). However at 200 °C the results show the opposite, with β- and γ-HBCDD being somewhat more resistant (30 min.) compared to α-HBCDD (20 min+).

According to Albemarle the decomposition of HBCDD starts at around 200 °C and at 215 °C there is 1 % weight loss and at 274 °C the weight loss is 90 % (Albemarle Corporation, 2000a). There is no information on the time period for the degradation.

1.3.4 Density

Two values determined by non-GLP methods were given by industry:

2.38 g/cm^3 at 20 °C (Albemarle Corporation, 1994)

2.24 g/cm^3 (Great Lakes Chemical Corporation, 1994)

In a letter from Albemarle 2003 the following information was given:

- The granular form has a density of 1.3 g/cm^3 as packed bulk density and 1.2 g/cm^3 as aerated density
- The powder form has a density of 1.4 g/cm^3 as packed bulk density and 1.0 g/cm^3 as aerated density
- The finely ground form has a density of 7.1 g/cm^3 as packed bulk density and 5.8 g/cm^3 as aerated density

1.3.5 Vapour pressure

For the temperature range 10-50 °C the vapour pressure was calculated to between 6.4×10^{-6} Pa (10 °C) and 1.7×10^{-4} Pa (50 °C). Calculations were based on regression analysis data from determinations at 50-100 °C (Dimmler, 1992b). The method used was an effusion method but it was not described further. Neither of the effusion methods described in OECD guidelines is recommended for substances with vapour pressures below 10^{-3} Pa. At 20 °C the vapour pressure was calculated to 1.6×10^{-5} Pa (Dimmler, 1992b). In one further study using a spinning rotor gauge method the vapour pressure of a composite of HBCDD samples from three manufacturers was determined to be 6.27×10^{-5} Pa at 21 °C (Stenzel and Nixon, 1997). The study was conducted under GLP. The composite sample consisting of equal parts of each HBCDD manufacturers material (300 g) contained the following percentage diastereomer s: α-HBCDD 6.0 %; β-HBCDD 8.5 %; and γ-HBCDD 79.1 %. The spinning rotor method used is not recommended for substances with vapour pressure below 10^{-4} Pa. According to OECD guideline 104 it is only the gas saturation method that is recommended for substances with as low vapour pressure as 10^{-5} Pa.

Both vapour pressures (20-21 °C) determined in the studies are in the same range. Therefore, the methods used are considered acceptable. The technical product of HBCDD contains isobutanol 0.1 %. The amount of isobutanol is considered to be too low to have an influence

on the vapour pressure. A vapour pressure of 6.3×10^{-5} Pa at 21 °C will be used in the assessment.

1.3.6 Solubility

1.3.6.1 Water solubility

1.3.6.1.1 Studies using the generator column method

There are three studies available where the generator column method has been used to determine the water solubility. Two studies are performed with pure water and the third is an algal growth inhibition study (see section 3.3.1.2.3) where the water solubility in salt-water medium was determined.

Study 1

The water solubility of the three diastereomers α-, β- and γ- of HBCDD were determined in general accordance with OECD guideline 105 and US EPA OPPTS 830.1760 and in compliance with GLP (MacGregor and Nixon, 2004). The test was performed using the generator column method with high-purity non-buffered reagent water. The test substance was a composite sample from three manufacturers, assigned Wildlife International, Ltd. identification number 5850, and consisted of 8 % α-, 5.37 % β- and 86.63 % γ-HBCDD. Tetrahydrofuran was used to prepare stock solutions. A single generator column was coated with the test substance. The column temperature was maintained at 20.0±0.1 °C and the flow rate and sampling time were adjusted to get enough volume. Standards were prepared with the respective diastereomers. Dichloromethane was used to extract the target analytes from the saturated aqueous solutions eluted from the column. The analyses were performed with HPLC/MS. Limit of quantitation was set to 0.5 µg/l for each diastereomer of HBCDD. The results are shown in Table 1-2.

Table 1-2 Diastereomers of HBCDD in samples collected from the generator column.

Diastereomer of HBCDD	Flow rate (ml/min) and sampling time (min)	Mean* measured concentration ± S.D. µg/l	Overall mean measured concentration ± S.D. µg/l
α	1.0/50	49.9±2.09	48.8±1.87
	0.5/100	47.7±0.679	C.V.** 2.83 %
β	1.0/50	15.0±0.530	14.7±0.499
	0.5/100	14.5±0.306	C.V.** 3.39 %
γ	1.0/50	2.21±0.0714	2.08±0.219
	0.5/100	1.96±0.253	C.V.** 10.5 %

*n = 5

**C.V. = coefficient of variation

Study 2

The generator column method was used to determine the water solubility limit of the test substance and was done in compliance with GLP and in general accordance with the OECD guideline 105 and EPA 40 CFR Ch. 1, 796.1860 (Stenzel and Markley, 1997). The test substance consisted of a composite of HBCDD samples from the three manufacturers containing 6.0 % of α-, 8.5 % of β-, and 79.1 % γ-HBCDD. According to Industry (M. Hardy, Albemarle; personal communication) the remaining 6.4 % were 0.1 % isobutanol and 6.3 % unknowns. Tetrahydrofuran was used for preparation of stock solution. In this study the flow rate and sampling time were either ~2 ml/min and ~500 min or ~1 ml/min and ~950 min to get enough volume. The water solubility was determined to 3.4 µg/l at 25 °C for γ-HBCDD. Neither α- nor β-HBCDD could be quantified with the HPLC method used because of interfering UV-active substances. Thus, the solubility of the commercial product determined in 1997 only represented the solubility of γ-HBCDD, whereas later studies, due to improved analytical methodology, have been able to measure all three diastereomers. The results from this study and Study 4 below using ^{14}C-labelled compound are in the same order of magnitude.

Study 3

In the algal growth inhibition study (section 3.3.1.2.3) (Desjardins *et al.*, 2004)the generator column method was used to generate salt-water medium containing HBCDD at the level of water solubility of the respective diastereomers. The flow rate and sampling time were 1 ml/min and 30 min. The solubility of the three diastereomers was determined to 34.3, 10.2 and 1.76 µg/l for α-, β- and γ-HBCDD, respectively. Resulting in a nominal water soluble concentration of HBCDD in saltwater medium of 46.3 µg/l.

1.3.6.1.2 Other studies

Study 4

The solubility of Firemaster-100 (HBCDD, CAS 3194-55-6) was determined to approximately 8 µg/l at 25 °C using ^{14}C-labelled compound (Yu, 1979b). HBCDD was diluted in distilled water, placed in 15, 25 or 35 °C and shaken in the dark overnight. After centrifugation the radioactivity in the solution was determined. However, the exact identity of the compound actually determined is unclear based on the study description. The study is not considered valid.

Study 5

The solubility of HBCDD was determined to 0.12 mg/l at approximately 23 °C (Dimmler, 1992a). This study was performed at BASF according to OECD guideline, it is however very shortly described. There is no information on the composition of HBCDD. This study is not considered reliable.

1.3.6.1.3 Summary of water solubility studies

The results from valid water solubility studies are compiled in Table 1-3.

Table 1-3 Water solubility (generator column method) of HBCDD in water and in salt-water medium

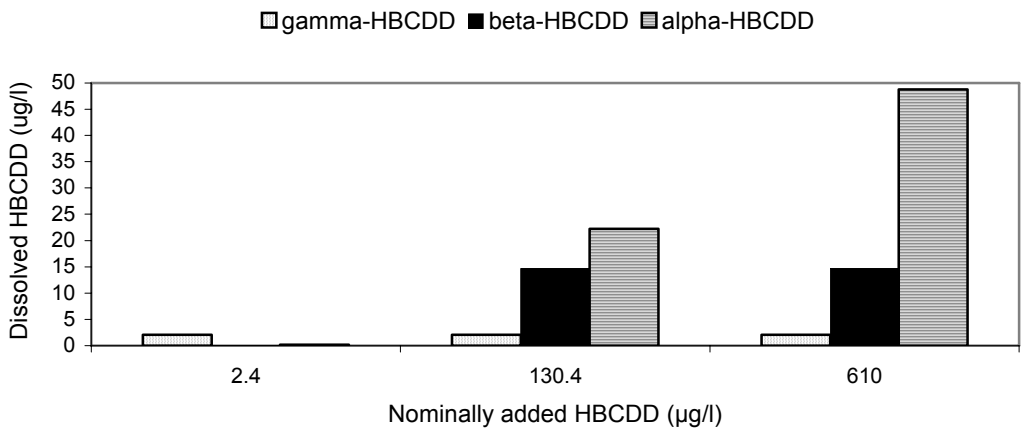
Test substance	Water	Water solubility (µg/l)*	Reference
α -HBCDD	Water	48.8±1.9	Study 1
β -HBCDD		14.7±0.5	(MacGregor and Nixon, 2004)
γ -HBCDD		2.1±0.2	
HBCDD technical product, sum of above		65.6	
α -HBCDD	Salt-water medium	34.3	Study 3
β -HBCDD		10.2	(Desjardins *et al.*, 2004)
γ -HBCDD		1.76	
HBCDD technical product, sum of above		46.3	
γ -HBCDD	Water	3.4±2.3**	Study 2
			(Stenzel and Markley, 1997)

*20 °C, **25 °C

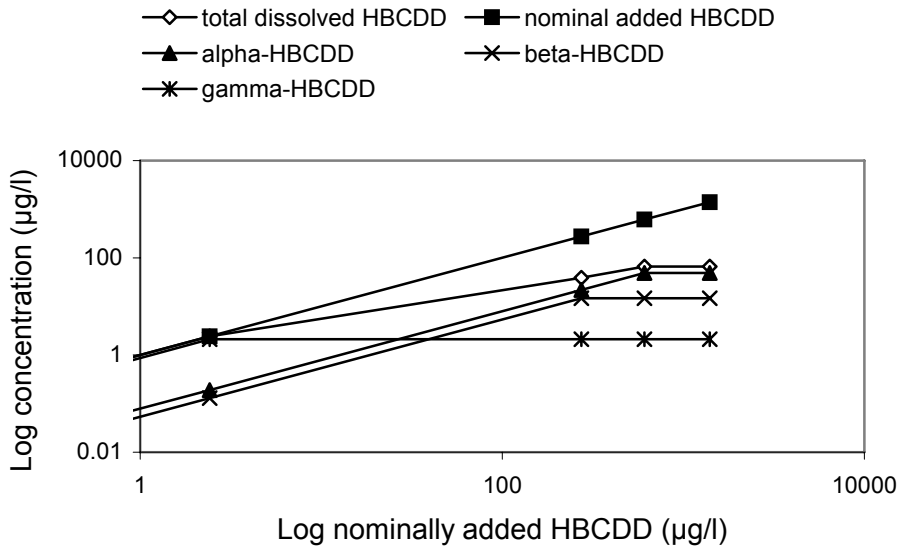
The water solubility was determined for all three diastereomers in study 1 and 3, (Desjardins *et al.*, 2004; MacGregor and Nixon, 2004), given is also the total solubility of HBCDD taken as the sum of the diastereomer s. In study 2 (Stenzel and Markley, 1997), only the water solubility for γ-HBCDD was determined.

The solubility of total HBCDD is slightly lower in salt-water medium (study 3) than in high-purity water (study 1) but the relative amount of respective diastereomer are similar.

The Generator column method will show the maximum solubility of each diastereomer in the mixture. Thus, when technical HBCDD is added to water, all three diastereomer will start to dissolve and the dissolved concentration will increase in a manner as shown in Figure 1-2 . γ-HBCDD will reach its maximum solubility at 2.1 µg/l (with a total HBCDD dissolved concentration of 2.4 µg/l), β-HBCDD at 14.7 µg/l (with a total HBCDD dissolved concentration of 39 µg/l) and α-HBCDD at 48.8 µg/l (with a total dissolved HBCDD concentration of 65.6 µg/l). At this level, 65.6 µg/l dissolved HBCDD, the total content of HBCDD in the water is 610 µg/l, with 544.4 µg/l representing non-dissolved γ- and β-HBCDD. The technical HBCDD used in study 1 contained 8 % of α-HBCDD. To dissolve 48.8 µg/l of α-HBCDD it is therefore necessary to add 610 µg technical HBCDD per l water.



A



B

Figure 1-2 A: Dissolved amount of respective diastereomer of HBCDD depending on added amount of technical product. B: Total added amount of HBCDD and total dissolved amount of HBCDD on a log-log-scale. Figures calculated with data from Study 1.

Although at a first glance, a water solubility of 66 μg/l may not seem to represent a realistic situation considering a solubility of 2.1 μg/l for the major component, γ-HBCDD. However, it is likely that HBCDD concentrations in water, such as in saturated waste water or when increasing amounts of HBCDD is added as a solution (e.g. in acetone) to aquaria-water, can with time reach levels close to 66 μg/l. It should be noted that non-dissolved HBCDD would also be present, although, the non-dissolved fraction may not be visible at these low concentrations of substance. In addition, in many cases, the composition of the HBCDD released is not known, as high temperature is known to convert γ- to α-HBCDD (with a water solubility of 49 μg/l). A value of 66 μg/l for the water solubility is therefore considered realistic.

It has been argued that, for substances containing components differing in their water solubility, the Generator column method is not appropriate for producing aqueous solutions used in toxicity testing (Organisation for Economic Co-operation and Development. Environment Directorate., 2000), as the composition will change relative the technical mixture. However, from the reasons presented above, it is clear that the composition will change also when the substance is added from an acetone solution because of the different solubilities of the diastereomers.

Thus depending on how a solution of HBCDD is prepared the level of dissolved HBCDD in the final solution will differ, as will the relative distribution between the diastereomers:

- *Saturation:* In a solution made by adding enough HBCDD until the maximum water solubility for all three diastereomers has been reached, the total water solubility for the three diastereomers is around 66 μg/l).
- *First precipitation/crystallization:* In a solution made by adding technical HBCDD (with γ-diastereomer constituting 85-90%) until precipitation starts to occur, γ-HBCDD will start to precipitate first. Thus, for the technical product, including α-, β- and γ- HBCDD, apparent water solubility will be slightly above the solubility for γ-HBCDD (*i.e.* slightly above 2 μg/l). However, it can be noted that the precipitation is not visible at these low concentrations, and that the value of 2 μg/l is obtained from the generator column experiments where also α- and β-HBCDD is present. To which extent the simultaneous presence of the other diastereomers affect the solubility of γ-HBCDD is not known.

The water solubility ranges from 0.0024 to 0.066 mg/l at 20 °C depending on how the water solubility is measured. Dealing with three diastereomers having different chem./phys. properties (e.g. water solubility) makes modelling difficult. Ideally, EUSES should be run separately for each diastereomer. However, there are currently not enough data to do that. Therefore, for calculations in the EUSES, the value 0.066 mg/l will be used as a worst case estimate for the technical product.

1.3.6.2 Solubility in other solvents

Data on the solubility of HBCDD in different organic solvents are compiled in Table 1-4.

Table 1-4 Solubility of HBCDD in organic solvents at 25 °C.

Solvent	HBCDD weight %	Reference
Acetone	8.6 7	(Albemarle Corporation, 2000a) (Dead Sea Bromine Group, 2000)
Methanol	0.15	(Albemarle Corporation, 2000a)
Toluene	6.40 6.5	(Albemarle Corporation, 2000a) (Dead Sea Bromine Group, 2000)
n-Pentane	0.01	(Albemarle Corporation, 2000a)
Isopentane	0.01	(Albemarle Corporation, 2000a)
Cyclopentane	0.05	(Albemarle Corporation, 2000a)
Styrene	8.00 10	(Albemarle Corporation, 2000a) (Dead Sea Bromine Group, 2000)
Chlorobenzene	2.80	(Albemarle Corporation, 2000a)
Methylene dibromide	3.60	(Albemarle Corporation, 2000a)
Dimethyl formamide	33.90	(Albemarle Corporation, 2000a)

1.3.7 Partition coefficient n-octanol/water

The log n-octanol/water partition coefficient (log K_{ow}) of HBCDD was determined to 5.625 at 25±0.05 °C using the generator column method (USEPA Product Properties Test Guidelines, OPPTS 830.7560, 1996) and in accordance with GLP practices (MacGregor and Nixon, 1997). The test sample consisted of a composite of HBCDD samples from three manufacturers. The composite contained 8.5 % β-, 6.0 % α- and 79.1 % γ-HBCDD (total HBCDD 93.6 %). There was no information on the identity and properties of the remaining 6.4 %. All solvents used were suitable for HPLC and residue analysis or certified reagents. A 0.2 % stock solution (w/w) was used. The concentration of HBCDD was determined in the stock solution and in the individual aqueous samples collected from the generator column, using a HPLC system. The recovery was 104 and 85 % in the two matrix fortifications at 1.0 and 10.0 µg HBCDD/l, respectively. The study was in general carried out according to the given guideline. The concentration of the stock solution was approximately 0.2 % instead of 1 %, which was recommended in the guideline.

Individual Log K_{ow} values have been estimated for α-, β- and γ-HBCDD using reversed-phase (RP) high-performance liquid chromatography (HPLC) methods (Hayward et al, 2006). Of the three RP-HPLC methods tested in the study, the method based on a gradient elution combined with an exponential calibration is recommended for estimating the log K_{ow} of highly hydrophobic compounds. The estimated log K_{ow} values for α-, β- and γ-HBCDD using the gradient elution method were 5.07 ± 0.09, 5.12 ± 0.09 and 5.47 ± 0.10, respectively. The test was performed at 25ºC on a short Supleco ODP-50 cartridge column, with a flow of 1.8 ml/min with MeOH (10 -100%, increasing linearly over 40 min.) as mobile phase. There is no information on the test substances. The calibration compounds used was chlorobenzenes and PCBs, which is not optimal for the estimation of Log K_{ow} values

for α-, β- and γ-HBCDD because of the structural dissimilarities (Hayward *et al.*, 2006)

.

log K_{ow} given in the HEDSET was 5.81 (determined from BCF).

Two studies are available where the methodologies were shortly described and therefore it was not possible to establish the reliability of these studies. The log K_{ow} of HBCDD was determined to be 3.265 using a method described by Leo, Hansch and Elkins in 1971 (Yu, 1979a). The concentration of HBCDD in the aqueous and octanol phases was determined by radioassay. In the second study the log K_{ow} of HBCDD (CAS No.: 25637-99-4) was calculated using the pro-logP computer programme to 7.59 (Krämer and Wittlinger, 1989)

The log K_{ow} value of 5.625 that is in the same order of magnitude as the log K_{ow} determined from BCF will be used in the risk characterisation of HBCDD.

1.3.8 Granulometry

Particle size of the commercial HBCDD is largely governed by application. Thus, suppliers offer a wide range of particle sizes.

A particle-size description of one technical HBCDD has been submitted. The method used is called the polydisperse model and the mean diameter in three experiments is given as 62.69, 64.80 and 66.61 μm (Anonymous, 1996).

Another commercial HBCDD is sold in powder or granule form. The powder form of HBCDD has been said to have an average particle size of 110 μm. The granular form is prepared from powder, which has been compacted into bars and broken down into pieces. The resulting pieces are irregular in shape with great variation.

Albemarle has two technical products. One product, used primarily in extruded polystyrene (XPS) or expandable polystyrene (EPS), is available in powder form with particle size of 53-279 μm or granular form with the average particle size of 2410 μm (Albemarle Corporation, 2000a). The other product, designed for use in coating applications but can also be used in EPS, is grounded to a fine particle size with the average size of 3.2-4 μm (Albemarle Corporation, 2000b).

Information from Industry 2003 on particle sizes is shown in Table 1-5.

Table 1-5 Particle sizes and distribution of commercial HBCDD.

Albemarle	**Finely ground form**		**Powder**		**Granules**	
(Letter February 2003)	Size (µm)	Use	Size (µm)	Use	Size (µm)	Use
	In a 500 kg sample: 50% 3.2-4; <10% 0.79 - 0.85; 40% 4-9.8 to 13.2	In textile applications to ease its dispersion in the aqueous suspensions.	Sample of known quantity: 87% >53; 98% < 279	In EPS applications in Europe and to formulate a master batch for certain XPS applications. The master batch is used by ~10% of the European XPS applications.	Sample of known quantity: 3.1% < 500; less than 1% >5600	In ~90% of the XPS applications in Europe.
Dead Sea Bromine Group (Letter February 2003)	**Ground FR 1206 I-LM**		**Standard FR 1206 I-LM**		**Compacted FR 1206 I-CM**	
	Size (µm)	Use	Size (µm)	Use	Size (µm)	Use
Particle size distribution performed with laser diffraction analyser	D10: 0.5-0.7 D50: 1.7-4 D90: 12-20	textile application	D10: 6-8 D50: 20-26 D90: 40-50		D10: 910 D50: 1998 D90: 3297	
Great Lakes Chemical Corporation (Letter February 2003)	**CD75XF**		**CD75P**		**CD75**	
	Size (µm)	Use	Size (µm)	Use	Size (µm)	Use
	D10: 5-6 D50: 15-17 D75: 21-25 D90: 29-34 D99: 58-62 Mean: 16-19	textile application	D10: 2.7-3.5 D50: 22-26 D75: 72-92 D90: 145-175 D99: 240-280 Mean: 50-60	Standard grade	D10: ~80 D50: ~110 D75: ~130 D90: ~160 D99: ~250 Mean: ~115	
Great Lakes Chemical Corporation (Letter February 2003)	**CD75PC (compacted)**				**Industry composite used for the toxicity studies**	
	Size (µm)	Use			Size (µm)	Use
	D10: 14-150 D50: 570-750 D75: 820-950 D90: 1040-1140 D99: 1370-1440 Mean: 560-710				D10: 3.6 D50: 65 D75: 195 D90: 280 D99: 1010 Mean: 142	toxicity studies

According to the most recent information, the mean particle sizes for the different applications vary considerably between the producers. In general, the size of the fine

powder used by the textile industry is in the range of 2-19 μm, the powder 20-150 μm, and the granules 560-2400 μm (personal communication D. Sanders, GLCC).

In addition, the mean particle size of the composite (of three producers) used for the toxicity studies has been determined to 142 μm.

1.3.9 Derogation from testing of physico-chemical properties

Derogations submitted by the rapporteur for exemption from testing requirements for melting point, boiling point, flash point, auto flammability, flammability, explosive properties and oxidising properties was presented in **Document ECB 4/DERO/03/97** at the Technical meeting TMIII/97 in July 1997. After having heard the arguments put forward by industry and accepted by the rapporteur in the document, the TM agreed to the derogation statements (TM397 minutes), July 1997.

1.4 CLASSIFICATION

1.4.1 Current classification

Hexabromocyclododecane is currently not included in Annex I to Directive 67/548/EEC.

1.4.2 Proposed classification

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 study 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.

2 GENERAL INFORMATION ON EXPOSURE

General information on exposure is important both for estimations of environmental and human exposure as well as for risk characterisation and risk management of the substance.

The German company BASF used HBCDD for the first time in their production of flame-retarded polystyrene foams in the late 1980s. However, the substance has been on the world market since the 1960s. HBCDD was named Hexabromid with the CAS No 3194-55-6 when it was synthesised at BASF.

HBCDD is used industrially in the life cycle steps; -production (I), formulation (II) and industrial use (III) with the aim to increase the flame resistance of different end-products. The end-products are used (IV) both professionally and by consumers, have a relatively long service life (V) and are disposed (VI) of by different means; incinerated, recycled, put on landfill or left in the environment. The life cycle of HBCDD is shown in Figure 2-1. A summary of the production, formulation and industrial use of HBCDD is given in Table 2-3.

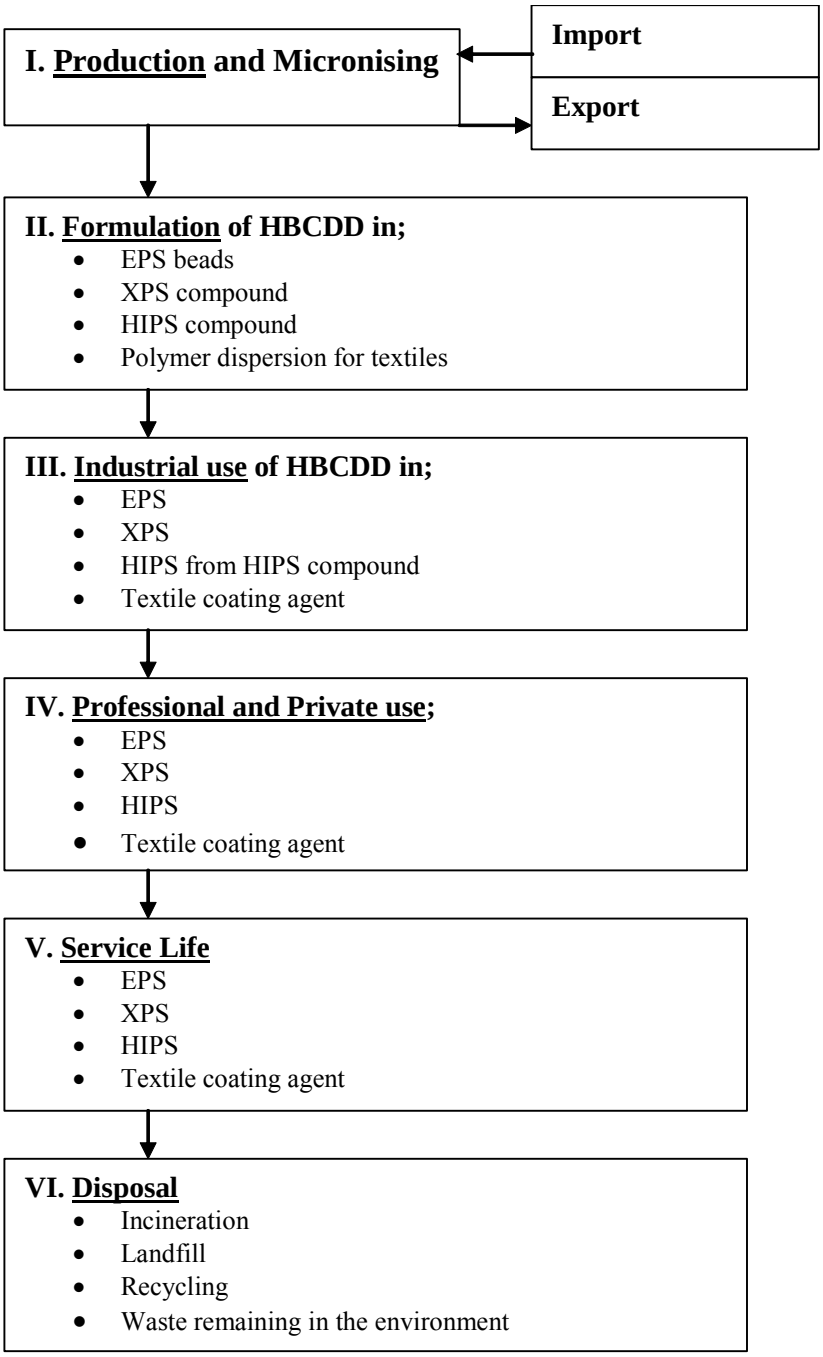


Figure 2-1 The life cycle stages of HBCDD. Roman numerals are used for different life cycle stages.

2.1 PRODUCTION

HBCDD is presently only produced at one site in EU15, located in the Netherlands. The total annual (2005) production of HBCDD is assumed to be 6000 tons. 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.

Data presented in this chapter include both closed and active production sites. A summary of the production, formulation and industrial use of HBCDD is given in Table 2-3.

2.1.1 Production method

The production of HBCDD is a batch-process. Elementary bromine is added to cyclododecatriene in the presence of a solvent. The process temperature is 20 to 70 °C, and the reaction takes place in closed systems. The suspension obtained is filtered, the solvent is removed with water, and the product is dried, stored in a silo and packed. According to one producer, production and transportation of the material to silo and the packaging are done in a closed system. The product is delivered as powder or pellets.

The production method as described in general terms in the IUCLID Data Sheet is as follows:

- Loading of raw materials
- Bromination
- Filtering
- Drying
- Storage in silo
- Packaging

2.1.2 Micronising (grinding to smaller particles)

The HBCDD particles in some applications (e.g. for use in textile back-coating) need to be very small. Therefore some quantities of HBCDD are micronised in a grinding process. No information on where micronising takes place is available, but it is assumed to occur at a very limited number of sites.

2.1.3 Tonnage – Production, Import and Export

It is not possible to give an exact tonnage for HBCDD since information on production and import was given by industry in ranges and for different years. Due to this fact it is also not possible to determine the consumption and production trends of HBCDD in the EU. In Table 2-1 the ranges given industry are presented.

Table 2-1 EU production and import of HBCDD

Production and import country	Quantity produced (t/a)	Quantity imported (t/a)	Year
The Netherlands	500-1,000 >1000- <5000 5000-7000		1996 1999-2002 2002
United Kingdom + import	1000-5000	100-500	1996

Production and import country	Quantity produced (t/a)	Quantity imported (t/a)	Year
from the USA	0		2003-
Import from the USA	0	1000 - 5000	1995
Germany	0	0	1997#

#: Second half of 1997. Information on import of HBCDD to the EU from other countries than the USA has not been reported. Import of amounts less than 1,000 t/a by one or several companies cannot be excluded.

Using the information in Table 2-1 the maximum total tonnage of HBCDD produced and imported for one year, between 1995 and 1997, is summed up to 11,500 tonnes based on 6,000 tonnes produced in the EU and 5,500 tonnes imported from the US. However, industry has informed that the 1999 market in Europe for HBCDD was 8,900 tonnes (M. Hardy, Albemarle personal communication). When adding the amounts used for the single uses of HBCDD, (see chapter 3.1.2) the total consumption is 9,618 tpa. In the RAR the figure 9,618 tonnes per year will be used for consumption of HBCDD in the European Union.

No information on export of HBCDD from the EU is provided. The export of HBCDD from the EU can be calculated from the production, the import and the consumption of HBCDD in the EU (6000+5500-9,618 = 1,882 tpa). The import and export of articles containing HBCDD is not included in this estimation.

According to industry (D. Lausberg, BASF, personal communication), the consumption of HBCDD in Eastern Europe, for instance in Poland, is considerable. The estimated market for flame retarded EPS for construction is around 30,000 tonnes/year in Western Europe and around 17000 tonnes/year in Eastern Europe. In Eastern Europe there is no market for non-flame retarded EPS. The estimated market for flame retarded XPS is 5000000 m^3/year in Western Europe and around 500000 m^3/year in Eastern Europe. There is however no information provided on from where and in what form HBCDD is delivered to Eastern Europe

The import of HBCDD to Sweden, as raw material and in chemical products, increased from around 50 tonnes per year 1993 to 120 tonnes per year 1997. Thereafter the import has almost ceased and was year 2000 less than 10 tonnes per year. Approximately 60 tonnes were imported in 2001, 30 tonnes in 2002, and 2003 and 2004 the import had decreased further to approximately 2 tonnes. (The Swedish Product Register, personal communication).

Countries outside the EU known to produce HBCDD are the USA and Japan. The annual consumption of HBCDD in Japan has increased from 600 tonnes in 1986 to 1600 tonnes in 1994. Data from Japan indicate that the consumption of HBCDD is about 2000 t/a (Toyozo Kaneko, letter of 4 March 1998). Some information on the worldwide consumption is available on the BSEF website that indicates a worldwide consumption of 16,700 tonnes/year in 2001 (available from http://www.bsef.com/publications/BSEf_factsheet_HBCD.pdf).

2.1.3.1 Import of HBCDD in end-products

HBCDD contained in expanded polystyrene (EPS) and extruded polystyrene (XPS) for the construction industry is not likely to be transported long distances due to the bulkiness of the material. Furthermore, the extrusion and expansion processes are relatively straightforward industrial processes, which allow them to be carried out in the region where the products are needed. However, transport of compounded polystyrene (PS) with HBCDD (granules, masterbatch or beads) over long distances cannot be excluded, but information on this is lacking.

Packaging material containing HBCDD (e.g. EPS), for protection of sensitive equipment, is likely to be transported worldwide. High impact polystyrene (HIPS) containing HBCDD is likely to be imported to the EU in electrical and electronic equipment, but no data on this has been submitted. Textile that is back-coated with a HBCDD-containing layer is imported from the US and other countries in unknown quantities. Import of polymer dispersions for textiles containing HBCDD cannot be excluded. In summary, import (and export) of HBCDD in articles is likely to occur but has not been possible to quantify.

2.2 USE

2.2.1 Introduction

The main downstream uses of HBCDD are in the polymer and textile industries. HBCDD can be used on its own or in combination with other flame retardants e.g. antimony trioxide and decabromodiphenyl ether.

HBCDD is used in four principal product types, which are:

- Expandable Polystyrene (EPS)
- Extruded Polystyrene (XPS)
- High Impact Polystyrene (HIPS)
- Polymer dispersion for textiles

These uses are described in more detail in chapters 2.2.3 through 2.2.6

According to industry information, the main use (90 %) of HBCDD is in polystyrene (PS) ((Frölich, 2002)) The predominant use of PS is in rigid insulation panels/boards for building construction (EPS and XPS). About 2 % of the total use of HBCDD is in “high impact polystyrene” (HIPS). Examples of end-products containing HBCDD are given in Table 2-2.

Table 2-2 Use pattern of HBCDD.

Material	Use/Function	End-products (Examples)
EPS	Insulation	• Construction, insulation boards, (packaging material)

Material	Use/Function	End-products (Examples)
		• 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
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
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

Some other minor uses have been reported, but it is not clear whether they are relevant for EU, and they are not included in the current work. For example, For example, the use of HBCDD in polypropylene (PP), adhesives, latex binders and unsaturated polyester has been reported in the USA (Anonymous, 1989). According to Kirk-Othmer (Kroschwith, 1993), the use of HBCDD in polypropylene (PP) is confirmed. HBCDD can be used in adhesives and coatings (Dead Sea Bromine Group, 1995), (Albemarle, 1998)and in SAN resins (styrene-acrylonitrile copolymer) (Albemarle, 1998). It may also be used in PVC (wires, cables and textile coatings) (National Research Council, 2000)however; industry has not confirmed that these uses are relevant in the EU. Thus, it cannot be excluded that HBCDD has been, is, or will be used in these materials.

2.2.2 Use - Tonnage and Categories

Information on *Industry categories, Use categories and Main categories* is needed when using tables for release estimations in the TGD (or EUSES) for the quantitative estimation of releases to the environment in the case of limited of site-specific information. Information on Main category may be needed for the estimation of release to air according to the release Tables of the TGD, but not for these combinations of IC/UC. In the life cycle stages Use and Disposal, qualitative approaches for estimation of releases have been used and therefore Use and Industry categories are not relevant. Industry categories relevant for estimations of release to the environment are given in the Table 2-3 below. Table 2-3 also shows a summary of number of sites and tonnage for different production and use scenarios. For details see chapter 3.1.2. The reason why the sum of the tonnages in the formulation step (II) and the step of Industrial Use (III) is that at some of the Industrial Use, XPS, HBCDD powder is used and therefore is not showed in the formulation step.

Table 2-3 Production and use of HBCDD.

Life cycle stage	Source/scenario	Use category	Industry category	Main category	Number of sites in EU	Tonnage
I. Production	Production	UC 22	IC 2	MC 1b	1	6000
	Micronising	UC 22	IC 2	MC 1b	few	1000 (assumption)
II. Formulation	Formulation, EPS	UC 22	IC 11	MC 1c	>18	3392
	Formulation, XPS	UC 22	IC 11	MC 1c	>14	1730
	Formulation, HIPS	UC 22	IC 11	MC 1c	4	>210
	Formulation, polymer dispersion	UC 22	IC 13	MC 1c	16	1054 (assumption)
III. Industrial use	Industrial use, EPS	UC 22	IC 11	MC 2	Hundreds	3400 (approx.)
	Industrial use XPS, compound	UC 22	IC 11	MC 2	17 (assumption)	1730
	Industrial use XPS, powder	UC 22	IC 11	MC 2	18	3232
	Industrial use of HIPS	UC 22	IC 11	MC 2	Not available	>210
	Industrial use, textile ind.	UC 22	IC 13	MC 2	24	1054

IC 2; Chemical industry, basic chemicals

IC 11; polymers industry

IC 13; Textile processing industry.

UC 22; flame retardant

MC1b. dedicated equipment, (very) little cleaning

MC 1c. Intermed stored off-site/dedicated equip.

MC 2; Inclusion onto or into a matrix

n.r.; not relevant

2.2.3 Use of HBCDD in Expandable Polystyrene (EPS)

2.2.3.1 Formulation of EPS compound

Expandable polystyrene is produced in a batch process, i.e. discontinuously, by suspension polymerisation of styrene in water. Styrene is dispersed in water in the form of small droplets. Prior to combining the water with the organic phase, additives are introduced. Typically these include suspension agents, free-radical-forming initiators and HBCDD flame-retardant. HBCDD-powder, most often delivered in paper bags with a plastic liner, with typically a content of 25 kg, is suspended at low temperatures in styrene prior to the addition of the water phase. Normally the bags are emptied into an intermediate storage container from where the HBCDD is transported via pipes and a weighing station prior the addition to the styrene. In the reactor, styrene forms the disperse phase as small monomer droplets in the continuous water phase. Final droplet size (0.01 to 0.5 mm) is determined by the ratio of disperse to continuous phase (typically 50:50) and by stirrer speed. The suspension agents prevent coalescence.

Within the monomer droplets (bulk), polymerisation occurs while the reactor content is heated up and held at its reaction temperature. During this free-radical polymerisation an expansion agent (e.g. pentane) is added to the reactor under pressure, where it is absorbed in the polymer droplets. In the final EPS beads, HBCDD is incorporated as an integral and encapsulated component within the polymer matrix with uniform concentration throughout the bead.

After complete conversion of the styrene monomer to EPS-beads, the reactor is cooled down and the beads are separated from the water by centrifugation. The decanted water, which could contain dissolved and dispersed HBCDD, is reused and exchanged on an annual basis or less frequently. The EPS beads are dried, and thereafter classified into various size fractions and surface coated. These different grades are packed in bins, bags, or transported in bulk trucks to the EPS-converters. The maximum concentration of HBCDD in EPS beads is assumed to be 0.7 %.

The numbers of sites in the EU area, which are manufacturing flame retarded EPS (formulation) and are members in APME, are 12. They represent about 2/3 of the number of sites producing EPS, but more than 80 % of the amount of HBCDD used for EPS. No data on where the sites are located have been submitted.

2.2.3.2 Industrial use of EPS compound

EPS foam is produced from EPS beads via pre-expansion of the beads with dry saturated steam, drying with warm air and shaping in shape moulds or in a continuous moulding machine. First, the raw material beads are pre-expanded in loose form with the help of dry saturated steam in pre-expanders. The raw materials are transported via pipes or tubes from the packaging containers to these stirred vessels. After expansion the beads are partly dried in fluid bed driers with warm air. The beads are subsequently stored in large permeable silos to “mature” for several hours up to 24 hours. During this stage the beads dry further and reach equilibrium with the ambient atmosphere around them. In the third phase the beads are

transported/blown, via pipes/tubes into block or shape moulds or in a continuous moulding machine in which the product gets its shape. The foam can then be further formed by cutting, sawing or other machine operations.

The number of sites with industrial use of PS beads containing HBCDD is likely to be very large, but no detailed information has been submitted by industry.

2.2.3.3 Professional and private use of EPS containing HBCDD

EPS containing HBCDD is used in end-products such as:

- insulation panels/boards in the construction sector
- automobile cushions for children (KemI, 1994) to meet the needs of the FMVSS 302 standard
- rigid packaging material for fragile equipment (minor use)
- packaging material such as ”chips” and shaped EPS-boards (minor use)

HBCDD-containing EPS can also be used in props for theatre, film and also for exhibitions. There is no information on that use other than that Industry claims that the main flame retardant for EPS is HBCDD and that properties for theatre, film and exhibitions probably is flame-retarded.

Secondary process activities of the EPS foam products, especially block foam, can be cutting, sawing and machining to manufacture shaped products such as interlocking boards. The cuttings and sawdust can be recycled in the moulding process within the plant. The EPS foam products, e.g. insulation boards, are normally transported shrink-wrapped, or packed in cartons.

Nearly all EPS foam containing HBCDD is used in the Building and Construction Industry, 1997: 377,500 tonnes, in Western Europe. The use of these flame-retarded products is often required in the EU countries to comply with the existing standards and/or building regulations and codes.

The market for EPS in Europe in 2001 is according to industry wide collected data through an external organisation (auditable data collection) approx. 420,000 tonnes for construction applications. In Western Europe approx. 70 % of the EPS are fire retardant grades. The Eastern European volume is approximately 170,000 tonnes for construction out of which more than 99 % is flame-retarded grades. The use of HBCDD in EPS building materials in Denmark was in 1997, 0.5-27 tonnes (Danish Environmental Protection Agency, 1999).

EPS used in packaging does generally not contain any flame retardant additive. Only in some cases, e.g. when requested by customers to minimise for instance the effect of fires, flame retarded EPS-material is used for non-food packaging applications. Packaging is approx. 250,000 tonnes in Western Europe with approx. 10 % flame-retarded grades. The consumption in Eastern Europe is approx. 20,000 tonnes in packaging but more flame-retarded grades. These numbers should represent >90 % of the volumes sold, as not all companies are reporting and some market estimates are made by the contributing producers.

2.2.4 Use of HBCDD in Extruded Polystyrene (XPS)

At the production of XPS-material the formulation stage can take place either at a separate site or at the same site as the following stage of industrial use.

2.2.4.1 Formulation of XPS compound

The HBCDD is supplied either in powder or in low-dust granulated form in either 25 kg bags or in 1 ton supersacks or "big bags". The supersacks are emptied into hoppers designed to minimise dust emissions. The HBCDD is then carried to the point of mixing with screw or air driven metering equipment. The compounded polystyrene is extruded and cut into granules, and packaged. The extrudate is either air-cooled or cooled by running in a water bath. According to industry information (personal communication) the masterbatch can contain approx. 40 % (w/w) of HBCDD.

Information that the number of this kind of sites is at least 14 in the EU countries has been submitted from Industry. Table 2-4 classifies the 14 companies in decreasing order of HBCDD consumption.

Table 2-4 EU XPS Compounders and Masterbatchers

Code	Decreasing HBCDD Consumption Size Order	Country
C1	1	Germany
C2	2	Austria
C3	3	Italy
C4	4	Italy
C5	5	Italy
C6	6	Italy
C7	7	Italy
C8	8	Italy
C9	9	UK
C10	10	Belgium
C11	11	?
C12	12	Italy
C13	13	Italy
C14	14	Italy

2.2.4.2 Industrial use of XPS- compound/HBCDD powder at the manufacture of XPS

The manufacture of XPS materials is carried out in the following way:

- The polystyrene, the additives such as processing aids, flame retardant, dye and blowing agent are fed continuously to an extruder.
- The polymer is melted; the blowing agent is mixed with the melted polymer and a "foamable gel" is formed.
- The gel is then cooled before it exits through an orifice called a die, where the blowing agent volatilises, causing the plastic to assume a foam structure. The blowing agent is usually a volatile, chemically stable compound, and by its introduction into the molten polymer, it reduces the density of the product by the formation of a myriad of closed cells within its structure.
- The foam is then trimmed to desired shape. The boards are packed into shrink-wrapped bundles and pallated. The pallets are stored for curing (usually 1 week) and are then ready for shipment.
- A remainder of about 25 % of the material is recycled to the extruder. This material is mainly "skin" from the surface, with higher density. The recycled material comes in contact with water.

One technology usually known as the UCI technology uses a vacuum in addition to blowing agents to produce the lighter (i.e., lower density) foams. In this technology, the product comes into contact with water in a water pond directly after the extrusion. This technology is used at two plants in the EU.

There are approximately 35 sites in the EU15 area manufacturing flame retarded XPS containing HBCDD. Presently there are also 3 sites in Eastern Europe and 4 sites in Turkey.

The use of XPS is as follows:

Cold bridge insulation	10 %
Sandwich Panels and Laminates	4 %
Cavity Insulation	17 %
Floors	10 %
Basement Walls and Foundations	30 %
Inverted Roofs	18 %
Ceilings	5 %
Miscellaneous	6 %

Insulation panels made from XPS contain 1 to 3 % (w/w) HBCDD.

2.2.4.3 Professional and private use of XPS

The XPS product is transported usually to a main distributor's warehouse, perhaps from there to a local distributor/dealer and hence to a building site on the orders of

the building contractor. There is a small amount (5 %) of "do it yourself"-business" via "do it yourself"-stores/building material suppliers. Clearly for major building sites the building contractor can have material delivered directly from the plant to the building site.

For prevention of frost damages on roads and in railway embankments it is not necessary to use flame retarded polystyrene, however that occurred earlier mainly to prevent fire during transport and storage (Bernes, 1998; Jansson, 2004). HBCDD is no longer used in these applications in Sweden.

It will be assumed for the calculations of exposure, as a realistic worst case, that the concentration of HBCDD in XPS is 3 %.

2.2.5 Use of HBCDD in High Impact Polystyrene (HIPS)

2.2.5.1 Formulation of polystyrene compound for the production of HIPS

High Impact Polystyrene is produced either in a batch or continuous polymerisation process. The final raw material is homogenised and extruded into HIPS pellets either strand- or face-out. These pellets are the starting material for the production of flame-retarded HIPS. Different flame retardant additives are used of which HBCDD constitutes only a small part.

The HBCDD powder, delivered in plastic bags, is filled in intermediate storage containers from where the HBCDD is transported to a weighing station. HBCDD and other ingredients required for the particular HIPS formulation are weighed and transported further to the feeding hopper of the extrusion equipment. In the feeding hopper all ingredients together with the HIPS pellets are metered in the extruder for further mixing, homogenisation and granulation into pellets.

An alternative route for HIPS production is via an intermediate-compounding route. First a masterbatch of general-purpose polystyrene pellets and HBCDD at a high concentration is prepared, followed by compounding this masterbatch with virgin HIPS material in a conversion step. The process of preparing the HBCDD masterbatch is similar to that of the HIPS production but at a higher HBCDD concentration.

After the molten mass at the end of the extruder is pressed through a plate with holes (die/plate), different granulation processes take place, for example:

- face cutting in air; a rotating knife directly after the plate cut the extruded "strands" into pellets cooled by air.
- under water face cutting; a rotating knife directly after the plate in a water bath cuts the extruded strands in pellets cooled by water.
- strand cutting; the molten strands are passed through a water bath to solidify and cool and are cut in a granulator

After the granulation process the HIPS pellets are dried and packed, either in bulk silos/containers or 25 kg bags, ready for conversion into HIPS products. The HBCDD masterbatch process normally uses the strand-cutting route.

2.2.5.2 Industrial use of HIPS-compound

HIPS materials can be converted into HIPS products using various extrusion techniques and injection moulding. HIPS products can also be manufactured via a compounding route, i.e. mixing virgin HIPS raw material with a HBCDD masterbatch during the extrusion or injection moulding process.

2.2.5.3 Professional and private use of HIPS

Most of the flame retarded HIPS products are used in electrical and electronic appliances (Ransbotyn, 2000).

HBCDD in HIPS is used in e.g.

- audio visual equipment cabinets (video and stereo equipment)
- distribution boxes for electrical lines in the construction sector
- refrigerator lining

In Germany, about 8,000 - 16,000 tonnes of HIPS with HBCDD were produced in 1990. The HBCDD content is 1 - 3 % (w/w) (BASF, 1996)or in other cases 5 or 7 % (Albemarle, March 1996). It will be assumed for the calculations of exposure, as a realistic worst case, that HIPS contains 7 % HBCDD.

2.2.6 Use of HBCDD in textile coatings

Flame retardant systems are used in textile application to comply with British Standard and German DIN flame retardant standards.

BSEF estimates the annual HBCDD usage for textile application in the EU to be in the range of 800 tonnes.

HBCDD is formulated to polymer-based dispersions (e.g. acrylic or latex) of variable viscosity in the polymer industry. The dispersions are then processed in the textile finishing industry.

The HBCDD particles used for textile back-coating need to be very small. Therefore micronising (see chapter 2.1.2) is performed before the formulation step.

2.2.6.1 Formulation of polymer dispersion for textiles

Textile formulators prepare flame retarded formulations, which are water-based dispersions and can contain a binder system and HBCDD as well as up to 20 other ingredients. These flame retarded formulations, mostly custom tailored, are supplied

as dispersion to back-coaters. In this scenario, formulation is carried out in an open batch system. HBCDD is added to a dispersion containing water, a polymer e.g. synthetic latex, acrylates or PVC, thickener and dispersion agent. The chemical preparation can also contain other brominated flame-retardants such as decabromodiphenyl ether. In addition, synergists such as antimony trioxide and antimony pentoxide could also be included in the end-product. According to industry information, the concentration of HBCDD in the dispersion may range from 5 to 48 %. However, additional product information indicates that a likely concentration of HBCDD in the coated layer may be about 25 % corresponding to 10 - 15 % in the final dilution of the dispersion. Water and solvents will leave the preparation when dried and concentrations of flame-retardants in the coating layer will be higher than in the preparation. Preparations with the highest concentration of HBCDD are assumed to be diluted before use.

The identified number of sites in the EU area, which are manufacturing polymer dispersion containing HBCDD, is 16.

The water based dispersion used by the backcoaters; both paste as well as foam, need to be stable (no precipitation and no viscosity change) and should not contain particles clogging the system. This is why the particle size of the solids is so important. Too fine particles act as thickener, where too course material will lead to a non-stable dispersion (precipitation) and an applied coated film with a non acceptable rough surface.

2.2.6.2 Industrial use of textile back-coating agent

Applying a back-coating to textile can be carried out in the following ways:

1- as paste where a layer is “glued” to the textile and a scratch knife defines the final thickness depending on the flame retardant standard, the textile used and the flame retardant concentration in the dispersion or
2- as foam, where a foam layer is pressed on the textile through a rotating screen. Once applied the foam cells will break resulting in a thin coating film.

The coating is dried and fixated in an oven at temperatures between 140 till 180 °C, (Delgado and (EIPPCB, 2003)

The formulated product is used on technical textile and furniture fabric, on cotton fabrics and cotton polyester blends. For the calculations of exposure, it will be assumed that the backcoating layer of the finished textile contain 25 % HBCDD.

HBCDD is usually applied with antimony trioxide as a backcoating in a mass ratio of 2,1 (i.e. about 6-15 % HBCDD and 4-10 % antimony oxide by weight) (National Research Council, 2000).

2.2.6.3 Professional and private use of textiles with back-coating containing HBCDD

The textiles with the back-coating containing HBCDD can be used for e.g. flat and pile upholstered furniture (residential and commercial furniture), upholstery seatings

in transportation, draperies, and wall coverings, bed mattress ticking, interior textiles e.g. roller blinds, automobile interior textiles and car cushions.

2.3 SERVICE LIFE

2.3.1 Accumulation of HBCDD - stock piling

HBCDD has been used for several decades. Assuming a constant annual consumption, of more than 9,000 tonnes of HBCDD is incorporated in articles made of polystyrene and in coating layers for textile backcoating for the EU market. The amount of HBCDD in the society is accumulating, forming a stockpile, since the service life of these end-products are estimated to be generally longer than 1 year. They vary between 1 year and more than 20 years and in some cases more than 100 years..

Data on the life expectancy of plastics is given in the Emission Scenario Document on Plastic Additives ((OECD, 2004)). Applications relevant for HBCDD are given in Table 2-5.

Table 2-5 Life expectancy of plastics

Applications	Service Life (years)
Building and construction	>10
Electronic	0 to 5
Electrical	10 to 20
Furniture	5 to 10
House wares	0 to 5
Packaging	2

Data from (OECD, 2004)

2.3.2 Losses of HBCDD from end-products

HBCDD is added to the matrix, not chemically bound, and does not seem to be degraded in the matrix.

The release of HBCDD from end-products depends on chemical and physical processes. Physical processes determining loss of HBCDD from the polymer matrix are (1) migration of HBCDD in the polymer, (2) the loss of HBCDD from the surface and (3) emission of particles lost at processing of the material or because of weathering and abrasion. The migration rate depends on the diffusion rate (concentration and mobility) and the solubility of the substance in the polymer. The loss from the surface depends on the volatility and/or physical conditions e.g. temperature and the solubility of HBCDD in a contacting media.

For quantitative estimation of the release from an end-product, the relative surface area of the product needs to be taken into account.

The rate-limiting step of release of HBCDD from end-products could be the migration rate in the polymer or the rate of loss from the surface depending on parameters such as:

- concentration of HBCDD in the polymer
- nature of the polymer
- nature of the surrounding media

The concentration in the polymer varies between 0.7 (EPS) and 25 % (textile backcoating). The polymers are various kinds of polystyrene (EPS, XPS, HIPS), latex, acrylics etc. The surrounding media are air (most uses), water (outdoor uses e.g. inverted roofs flushed by precipitation) and soil (buried construction material).

2.4 DISPOSAL AND RECOVERY

Waste containing HBCDD is generated at each life cycle step. In some cases the waste material, so called pre-consumer waste, could be recycled into the process. Wasted end-products (post-consumer waste) are incinerated, put on landfill, left in the environment or recycled.

2.4.1 Waste formation rate

According to the manufacturing industry, the main use of HBCDD is insulation in the construction sector. The consumption of plastics in this sector will increase (A P M E, Sofres Conseil for the Association of Platics Manufacturers Europe., 1995). However the quantities of plastic wastes will increase more quickly since only the products, which have a service lifetime shorter than 20 years, have been wasted so far. Therefore, in the future, we can expect an increasing amount of HBCDD to leave the use and enter the waste-handling sector of society. This, in turn, means that the amounts of HBCDD incinerated, accumulating on landfills and maybe leaching, as well as the amounts left in the environment will increase. Information from industry (A P M E, Sofres Conseil for the Association of Platics Manufacturers Europe., 1995)also shows that, in 1992, out of the total construction- and demolition (C&D) waste, plastic waste was 0.4 % of the weight. EPS and XPS were representing about 3 % of the plastic waste and therefore 0.01 % of the total building waste. The extrapolation to 2010 shows that the total plastic wastes in C&D waste if multiplied by a factor of 2.5, EPS and XPS will represent a fraction of 8 %. Therefore a total of 0.08 % of the total C&D waste if this total will remain the same. This indicated the small proportion of total waste related to EPS and XPS.

2.4.2 Waste handling

It is not known to what extent end-products containing HBCDD is landfilled, incinerated, left in the environment or recycled. Waste ending up in the municipal waste streams is likely to be put on landfill or incinerated. Construction material used on or under the soil could be left in the environment after use or be part of wasted construction material that is used as filling material e.g. for road construction.

Larger pieces of polystyrene board may be recycled, and recycling of EPS does occur in an organised way in several European countries (personal communication, Industry). Wasted EPS-boards are grinded and put back into the moulding process together with virgin EPS to form new boards. Increasing quantities of EPS waste including post-consumer waste are recovered and collected via national waste management schemes, e.g. in Germany, and primarily mechanically recycled into EPS or PS products for construction applications. However, it is not clear to what extent, or if at all, a distinction is made between streams of flame retarded and non-flame retarded material. There is no labelling/marking system so that the waste streams could be kept apart. Theoretically, recycling can lead to an uncontrolled contamination of polystyrene products, which do not need to be flame retarded, with HBCDD. The overall recycling of demolition material is estimated to be 30 % (RTDinfo,no26**,** http://europa.eu.int/comm/research/rtdinfo/en/26/constr3), although it is likely to increase with time.

2.5 CONTROLS ON HBCDD

2.5.1 Legislative controls

We are not aware of any Occupational Exposure Limit Values (OELV) for HBCDD. OELVs for organic dust and mist may be applied for HBCDD. For e.g. Sweden this level is 5 mg/m^3 and in many other countries 10 mg/m^3 (Table 2-6).

Table 2-6 Occupational Exposure Limit Values for organic dust and mist.

Country	Limit value
Sweden	5 mg/m^3
USA	TLV = 10 mg/m^3

2.5.2 Voluntary measures

Industry has started a voluntary emission control programme, VECAP (http://www.bsef.com/newsmanager/uploads/vecap_brochure_pstextiles.pdf), in the UK, to decrease the environmental releases of HBCDD from the textile industry. The participating companies in this programme work with a Code of Good Practice

Further voluntary emission control and reduction programme on HBCDD is known as SECURE (Self Enforced Control of Use to Reduce Emissions) and is subscribed to by the HBCDD producers and the EPS bead/XPS foam manufacturers.

2.5.3 Requirements for using flame retardant insulation materials

Industry has delivered information on general requirements for using flame retarded insulation material (D. Lausberg, BASF personal communication), Table 2-7.

Table 2-7 Requirements for using flame retarded insulation materials.

Country	Remarks on requirements
Austria, Germany, Switzerland	Strict legal requirements on all building materials concerning the reaction to fire.
Belgium, France, Luxemburg, Spain, The Netherlands	Limited requirements on insulation materials depending on the size and use of buildings.
United Kingdom	No legal requirements. There is a tendency to increasing demand.
Denmark, Finland, Norway, Sweden	Is practically not used - for most applications above ground level (except roof insulation) non-combustible insulation are used.
Eastern Europe	Authorities and fire brigades have very strict requirements on fire safety. Almost 100 % flame retarded material is used.

(D. Lausberg, BASF personal communication).

3 ENVIRONMENT RISK ASSESSMENT

3.1 ENVIRONMENTAL EXPOSURE

3.1.1 General discussion

The life cycle stages and scenarios for HBCDD are described in Chapter 20. During the handling of HBCDD in all life cycle stages, releases of HBCDD to the environment can be expected. Site-specific information is available for many of the HBCDD uses with the exception of industrial use of EPS and HIPS. The extent and quality of the information varies. For some sites the information is limited to the consumed amount of HBCDD, whereas the information from other sites is more detailed. An overview of the available site-specific information is shown in Table 3-1. In some cases Industry has asked to keep sensitive data confidential. Such confidential data are compiled in Annex C.

Releases are determined for all sites where site-specific data have been provided from Industry. In addition, generic local release estimations are performed in order to represent sites for which no information has been submitted. There is no site-specific information or any other information available on direct release to soil. Direct release to soil is not considered to be a relevant route for releases of HBCDD.

Table 3-1 Overview of the available site-specific information.

Life-cycle stage and scenario		Total number of sites identified	Number of sites with data provided[1]		
			Type of data provided		
			HBCDD volume per site	Emission to water	Emission to air
I.	Production	2	2	2	2
	Micronising	<5	1	1	1
II.	Formulation, EPS and production of HIPS	13 (19)*	13	5	2
	Formulation, XPS	3(>14)*	2	2	2
	Formulation, polymer dispersion	7 (16)*	6	4	2
III.	Industrial use, EPS.	(>100)*	-	-	-
	Industrial use, HIPS.	-	-	-	-
	Industrial use XPS, (masterbatch)	5(17)	5	5	5
	Industrial use XPS (powder HBCDD)	18 (18)*	18	18	1
	Industrial use, textile ind.	24	5	4	4

*Total number of sites given by industry within brackets

3.1.2 Environmental releases

3.1.2.1 Release during production and micronising of HBCDD (Life Cycle Stage I)

Site-specific information on the annual release of HBCDD to air and wastewater is available for two sites (site A located in the UK and site B located in the Netherlands). The site located in the UK closed in 2003 and a third site in Germany closed in 1997. No emission data from this third site has been provided.

SITE A

This site used to be the largest single source of emissions of HBCDD to the environment. It closed down in December 2003. It is kept in the risk assessment for illustrative purposes only and the emissions are not added to the total releases of HBCDD.

The production at this site is assumed to have been 5000 tonnes/year with production during 358 days/year. This production volume is based on IUCLID data. An environmental assessment of the site was carried out in 2000. This assessment covers sampling of water and analysis of HBCDD in wastewater streams, air sampling for estimation of air releases, soil sampling to provide indications on air deposition of HBCDD on unprotected ground around

the site and generation of waste. Data from this assessment showed releases, to air of 1.68 kg/day (602 kg/year) and to surface water of 3.73 kg/day (1336 kg/year) during the year 2000 (Anonymous, 2000f).

A similar survey was performed 2001 (Lowe and Huxham, 2002) showing releases to air of 9.5 kg/day (3400 kg/year) and to wastewater from the onsite STP of 5.59 kg/day (2000 kg/year). The effluent from the onsite STP is further treated in a municipal STP. The annual release of HBCDD in solid waste including sludge from the onsite STP was estimated to 1136 tonnes/year. This waste is collected and put on landfill. The data from this latter survey are used in the risk assessment, as a reasonable worst case. However, this site is included as an example for information only.

SITE B

The reported amount HBCDD produced at site B varies from one year to another. However, for the purpose of this risk assessment the production is assumed to be 6000 tonnes/year with 358 production days/year (Tange, 2003). This value is not used in any further calculations in the risk assessment due to the fact that available emission data are used in the calculations of PECs.

The annual release 2001 was 6.2 kg mainly via wastewater to a STP (Sanders, 2002). The annual release to wastewater has been reported to be 0.73 kg (2002), 0.13 kg (2003) and 0.20 kg (2004) (Tange, 2005). The highest release figure (0.73 kg/year) from year 2002- 2004 is taken as a reasonable measure of the emissions to wastewater from this site.

The emission to air is 21.7 kg/year, according to site-specific data (Tange, 2003). The site has installed an "absolute filter" for outgoing air. First measurements indicate that particulate emissions to air now are lower than 0.03 mg/m^3. Using a worst-case assumption (assuming all dust would be HBCDD) this would correspond to less than 2 kg/year emissions to air.

No information has been submitted on the formation of solid waste containing HBCDD, but information saying waste from the production, including sludge from the STP, is taken care of by incineration and reuse in the process is given.

TOTAL, REGIONAL AND CONTINENTAL EMISSIONS FROM PRODUCTION

Since there is only one active producer in the EU (site B), the emissions from this site are used for the estimation of regional background concentrations. The resulting total, regional and continental emissions from production of HBCDD are given in Table 3-2.

Table 3-2 Total, regional and continental emissions from production of HBCDD .

	Total (kg/year)	Regional (Site B) (kg/year)	Continental (kg/year)
Air	2	2	0
Wastewater	0.73	0.73	0
Surface water	0	0	0

RELEASE DURING MICRONISING OF HBCDD

Micronising is a process of grinding HBCDD powder or granules to a more fine powder, primarily used in the formulation of textile backcoating. The total amount of HBCDD used for micronising is about 1,000 tonnes per year, based on information from the textile Industry

There is no information on the number of sites involved in micronising, however the number of sites is assumed to be low. Data from one single site with micronising have been submitted from Industry (Esser *et al.*, 2003). According to industry this site uses the major part of the amount of HBCDD processed by micronising.

Local emissions

No water is used in the process; except for the cleaning-water used, which is collected in a tank and disposed off to a "waste management treatment company". It is therefore assumed that there are no emissions to wastewater or surface-water.

The emission to air from the site that submitted data was determined to 0.3 kg/year based on site-specific data (Esser *et al.*, 2003). No data on the number of emission days per year are given why the TGD default value from the B-tables is used (300 days/year).

Regional and continental emissions

Based on the information given by industry that the site for which data are given is the by far largest microniser it is assumed that the emissions from possible other sites involved in micronising are negligible. Thus, the continental release is assumed to be zero.

The resulting total, regional and continental emissions of HBCDD for micronising are given in Table 3-3.

Table 3-3 Total, regional and continental emissions of HBCDD from micronising

	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	0.28	0.28	0
Wastewater	0	0	0
Surface water	0	0	0

3.1.2.2 Release during formulation (Life Cycle Stage II)

HBCDD is used in four different types of formulation:

- Formulation of EPS compound, for the manufacture of flame retarded EPS
- Formulation of polystyrene compound for the production of HighImpact PolyStyrene (HIPS)
- Formulation of XPS-compound for the manufacture of flame retarded XPS

- Formulation of polymer dispersions, for textile backcoating

FORMULATION OF POLYSTYRENE COMPOUND FOR THE MANUFACTURE OF EPS AND/OR HIPS

Industry has requested that the site-specific information given for this use area should be kept confidential. Therefore, only release figures are presented here. Detailed data and calculations are given in the confidential Annex C.

The emissions from both the formulation of EPS-beads and HIPS are dealt with in this scenario. The reason for that is that the information on emissions received from most of these sites cannot be separated between these two activities.

There are 19 sites within the EU with either formulation of EPS beads or HIPS or both (Ransbotyn, 1999, 2000). Fifteen are only producing EPS, three are producing EPS and HIPS and one only HIPS. Site-specific information for year 2000 has been provided from 13 sites however, no information on locations has been submitted.

EPS - The 12 sites providing site-specific data used 2714 tonnes (year 2000). This represents 80 % of the amount of HBCDD used for the formulation of flame retarded EPS compound within the EU (Ransbotyn, 2000). The total annual use of HBCDD is calculated to be 2714/0.8 = 3392 tonnes/year. Furthermore it is known that six additional sites of this kind use HBCDD. These sites are thus assumed to represent the remaining 20 %, i.e. 0.20×3392 = 678 tonnes/year.

HIPS - Only three of the HIPS producing sites used HBCDD in year 2000. The annual amount used this year was 210 tonnes (Ransbotyn, 2000).

Taken together the calculated total annual use of HBCDD, for EPS and HIPS, becomes 3602 tonnes/year (3392+210).

More recent information on the use of flame retarded EPS in Western Europe (Anonymous, 2002b) gives a used amount of 2233 tonnes/year HBCDD. This is based on the amount of EPS used in material for insulation and packaging. In EPS there is 0.7 % (w/w) HBCDD. A fraction of 70 % of the insulation material and 10 % of such packaging material are flame retarded and principally with HBCDD. These data, based on rather uncertain estimations, indicate lower amounts used than the amount estimated from the site-specific data above. Therefore, this estimation is not used further in the RAR. Data on amounts of HBCDD (year 2000) used for formulation of EPS compound and for production of HIPS are shown in Table 3-4**.**

Table 3-4 Data on amounts of HBCDD (year 2000) used for formulation of EPS compound and for production of HIPS.

	HBCDD use (tonnes/year)	
	EPS	**HIPS**
Total amount for the 13 sites with site-specific data	2714	210
Total amount for the remaining six sites with no site-specific data	678	0
Total use (19 sites)	**3392**	**210**
Total use, sum for EPS and HIPS	**3602**	

Local emissions

Site-specific release estimation

The site-specific emissions of HBCDD to water and air are presented in Table 3-5 below. Data and calculations are presented in Annex C.

Data on the emissions to wastewater were given by 10 out of the 13 sites that provided data (Anonymous, 2000a). For 9 of the sites measured concentrations in the wastewater from the on site STP were used as the basis for calculation of emission factors for these sites. The 90th percentile of the calculated emission factors for these 9 sites (76×10^{-6}) was used for estimating emissions from the remaining three sites.) Emissions to air have been measured at three of the sites (Anonymous, 2000a). The emission reported from one of the sites most likely represents HBCDD captured in the filters of the ventilation system and will therefore not be used in the assessment. For the estimation of emissions to air from the sites lacking information an emission factor of $7.31\text{x}10^{-6}$ has been used. This factor is the highest known emission factor from the three use areas EPS formulation, XPS formulation and Textile formulation. It is assumed that the emissions to air are in the form of dust emanating from the handling of finely ground powder and that the handling situation is similar in all these three use areas.

Table 3-5 Local emissions of HBCDD to water and air from sites with formulation of compound for EPS and HIPS.

Site*	Connected to municipal STP (Yes/No)	Emission to surface water[1] (kg/year)	Emission to waste water [1] (kg/year)	Recipient	Emission to air[1] (kg/year)	Number of emission days per year

Site A	No	113.4	-	River[2]	0.40	350[2]
Site F	No	5.3	-	River[2]	2.0	174[2]
Site I	No	7.8	-	River[2]	2.2	183[2]
Site B	Yes	-	0.14	River[2]	1.1	340[2]
Site C	No	0.85		Nd	1.9	300[3]
Site D	No	13.6	-	River[2]	1.5	340[2]
Site E	No	2.1	-	River[2]	0.26	61[2]
Site G	Yes	-	2.6	Nd	1.8	300[3]
Site H	No	0.63	-	River[2]	0.41	94[2]
Site J	No	9.1	-	Nd	0.88	300[3]
Site K	Yes	-	20	Nd	1.1	300[3]
Site L	Yes	-	10.3	Nd	0.99	300[3]
Site P	No	0.30	-	Nd	0.029	1[2]

* The designation of the sites is the same as used by industry.

1. Details are given in annex C.

2. Data provided from industry.

3. The value is determined in EUSES from the B-Tables.

- No emissions.

Nd = No data.

Generic release estimation

The total emissions from the 6 sites not providing any information have been calculated using the 90th percentile of the 9 calculated emission factors for water emissions (76×10^{-6}) and the emission factor from site-specific data from a similar handling of HBCDD powder, to air (7.31×10^{-6}). For the emissions to water, 30 % of the water is lead to a municipal STP and 70 % to surface water, based on the data saying that 4 of the 13 sites with site-specific data are connected to a municipal STP.

The emissions from a generic local site have also been determined, using the same emission factors. The volume of HBCDD used at the generic local site has been determined as being 300 tpa which is equal to the 90th percentile of the annual use of the sites for which the HBCDD consumption is known. The number of emission days is 300. As the emission factor to water used in the calculation is relevant for emissions after treatment in an on site STP the EUSES model is run with the setting bypass STP.

The emission of HBCDD to water and air from the generic local site and the total emission for the sites with no site-specific data are given in Table 3-6.

Table 3-6 Emissions of HBCDD to water and air from a generic local site and the total emissions for the sites with no site-specific data with formulation of compound for EPS and HIPS

Site	Emission to water (kg/year)	Emission to air (kg/year)
Generic Local site	22.8(surf. water)	2.2
Total emissions for the sites with no site-specific data Waste water Surface water	 15 36	4.9

Regional and continental emissions to water and air from EPS formulation and HIPS production

The sites, with formulation of EPS and/or HIPS, are because of the relatively expensive transport of products of low density assumed to be evenly distributed within the EU. Therefore the 10 % rule for assigning emissions to the region could be applicable (from TGD). However, one of the sites uses a large proportion of the total volume of HBCDD in this use area. Therefore, the emissions from this site are assigned to the region. The total emissions are the sum of the site-specific emissions in Table 3-5 and the generic emissions in Table 3-6. The resulting total, regional and continental emissions of HBCDD at sites involved in formulation of EPS and HIPS are given in Table 3-7.

Table 3-7 Total, regional and continental emissions of HBCDD from formulation of EPS and production of HIPS.

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	19.5	0.4	19.1
Surface water	212	113.4	98.6
Wastewater	48	0	48

FORMULATION OF XPS COMPOUND FOR THE MANUFACTURE OF FLAME RETARDED XPS

Industry has requested that the site-specific information given for this use area should be kept confidential. Therefore, only release figures are presented here. Detailed data and calculations are given in the confidential Annex C.

According to information from industry there are at least 14 sites using HBCDD at the production of XPS-compound (Frölich, 2002). Nine out of these sites are located in Italy and the remaining five are located in five different countries in Europe (Germany, Austria, UK, Belgium and one unknown).

Information on the annual use at three single sites has been submitted and the total annual use of HBCDD is reported to be 1730 tonnes, see Table 3-8 (Frölich *et al.*, 2003).

Table 3-8 Data on amounts of HBCDD (year 2003) used for formulation of XPS compound .

	HBCDD use (tonnes/year)
Total amount for the sites with site-specific data	1160
Total amount for the remaining 11 sites with no site-specific data	570
Total use	**1730**

Local emissions

Site-specific release estimation

Three sites (Materbatch G, Masterbatch H and Masterbatch I) have provided data on emissions to water and air (Frölich *et al.*, 2003).

The site-specific emissions of HBCDD to water and air are presented in Table 3-9 below.

Table 3-9 Local emissions of HBCDD to water and air from sites with formulation XPS compound.

Site*	Connected to municipal STP (Yes/No)	Emission to waste water[1] (kg/year)	Recipient	Emission to air[1] (kg/year)	Number of emission days per year
MasterbG	Yes	0.12	Nd	2.6	300[3]
MasterbH	Yes	0.27	River[2]	1.2	300[3]
MasterbI	Yes	37	Nd	3.3	300[3]

* The designation of the sites is the same as used by industry.

1. Details are given in annex C.

2. Data provided from industry.

3. The value is determined in EUSES from the B-Tables.

Nd = No data.

Generic release estimation

The total emissions from the 11 sites not providing information have been calculated by using the worst case emission factors from the site-specific data (74.2×0^{-6} for water and 7.31×10^{-6} for air). According to the TGD default assumption of STP-connection 80 % of the emission is lead to a municipal STP and 20 % to surface water. The emissions from a generic local site have also been determined, using the same emission factors. The volume of HBCDD used at the generic local site is assumed to be 350 tpa which is approximately the same as the mean of the annual use of the three sites that provided data. The number of emission days is 300.

The emissions of HBCDD to water and air from the generic local site and the total emission for the 11 sites with no site-specific data are given in Table 3-10.

Table 3-10 Emissions of HBCDD to water and air from a generic local site and the total emission for the 11 sites with no site-specific data with formulation of XPS compound.

Site	Emission to water (kg/year)	Emission to air (kg/year)
Generic Local Site	26 (waste water)	2.6
Total emission for the 11 sites with no site-specific data		4.2
Waste water	33.8	
Surface water	8.5	

Regional and continental emissions to water and air from formulation XPS compound

Based on the information that around 65 % of the sites are situated in the same country, we assume 50 % of the annual use of HBCDD to take place in the region as a reasonable worst case. The total emissions are the sum of the site-specific emissions in Table 3-9 and the generic missions in Table 3-10. The resulting total, regional and continental emissions of HBCDD at sites with formulation of polystyrene compound containing HBCDD for manufacture of XPS production are given Table 3-11.

Table 3-11 Total, regional and continental emissions of HBCDD from formulation of XPS compound.

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	11.3	5.7	5.7
Wastewater	71.2	35.6	35.6
Surface water	8.5	4.3	4.3

FORMULATION OF POLYMER DISPERSIONS FOR TEXTILE BACKCOATING

A total of 16 sites within the EU have been identified using HBCDD in the formulation of polymer dispersions for textile back coating according to industry information (De Poortere, 2001). Seven sites of these sites are located in the UK, four in Germany and five in Belgium. Some site-specific information on emissions to wastewater has been submitted for five sites (TexForm1-5), together using approximately 500 tonnes HBCDD/year. Furthermore, the total use of HBCDD for polymer dispersions has been estimated by Industry to 1054 tonnes/year (De Poortere, 2001).

Further data from two sites (TexFormA and TexFormB) together using 125 tonnes/year HBCDD has been submitted (Frölich *et al.*, 2003). Whether any of these two sites are the same as any of the previous five sites is not known, but we assume it is seven individual sites.

Data on used amounts of HBCDD (2003) at six of the seven sites are given in Table 3-12 (TexForm2 did not report their HBCDD consumption).

Table 3-12 Data on amounts of HBCDD (year 2003) used for formulation of polymer dispersions for textiles.

Site*	HBCDD use (tonnes/year)
TexForm1	197.5
TexForm2	Nd**
TexForm3	5
TexForm4	90
TexForm5	150
TexFormA	50
TexFormB	75
Total amount for the sites above with site-specific data	>568
Total amount for the remaining 10 sites with no site-specific data	<486
Total use	1054

*The designation of the sites is the same as used by industry
**No data

Local emissions

Site-specific release estimation

Some site-specific information on emissions to wastewater was originally submitted for five sites (TexForm1-5), together using approximately 500 tonnes HBCDD/year. Later further data from two sites (TexFormA and TexFormB) together using 125 tonnes/year HBCDD was submitted (Esser *et al.*, 2003).

For Tex_Form1-5 there is information available on the type of on-site wastewater treatment used. For three of these sites, zero emission is indicated due to recycling of wastewater. The remaining two sites (TexForm1 and TexForm4) treat their wastewater by flocculation, settling and decantation. From one of them, measurements of the concentration of HBCDD in the outflow from the STP gave an emission factor of 5×10^{-7}. Industry claims that this emission factor is applicable to sites having similar type of wastewater treatment. Using this emission factor, the resulting emissions to surface water from the two sites are 0.1 and 0.045 kg/year, respectively.

All wastewater, originating from cleaning processes at TexFormA, is treated and reused within the production. There are no emissions from this site. At TexFormB the wastewater from cleaning processes is diluted with wastewater from other sources and then treated by hydroxide precipitation. This treated wastewater contains HBCDD and is released to the public sewage system. The emission factor for this site is 113×10^{-6}.

Solid waste, including sludge from on-site STP from the sites with formulation of polymer dispersions for textiles, is either incinerated or put on controlled landfills

The emission factors for emissions to air calculated from the two sites (TexFormA and TexFormB) were 0.27×10^{-6} and 0.094×10^{-6}, respectively. For the estimation of emissions to air from the sites lacking information, an emission factor of 7.31×10^{-6} has been used. This factor is the highest known emission factor from the three use areas EPS formulation, XPS formulation and Textile formulation. The site-specific emissions of HBCDD to water and air from the sites with formulation of polymer dispersions for textiles are given in Table 3-13.

Table 3-13 Local emissions of HBCDD to water and air from sites with formulation of polymer dispersions for textiles.

Site*	Emission factor (10^{-6})	Connected to municipal STP (Yes/No)	Emission to waste water (kg/year)	Recipient	Emission factor (10^{-6})	Emission to air (kg/year)	Number of emission days per year
TexForm1	0.5[1]	Nd	0.099	Nd	7.31[2]	1.4[2]	300[3]
TexForm2	0[1]	Nd	0	Nd	?	?	?
TexForm3	0[1]	Nd	0	Nd	7.31[2]	0.037	300[3]
TexForm4	0.5[1]	Nd	0.045	Nd	7.31[2]	0.66	300[3]
TexForm5	0[1]	Nd	0	Nd	7.31[2]	1.1	300[3]
TexFormA	0[1]	Nd	0	Nd	0.27[1]	0.014[2]	300[3]
TexFormB	113[1]	Nd	8.5	Nd	0.094[1]	0.0071[2]	300[3]

*The designation of the sites is the same as used by industry.

1. Emission factors provided from industry.

2. Largest site specific emission factor from the three use areas EPS formulation, XPS formulation and Textile formulation used as realistic worst case.

3. The value is determined in EUSES from the B-Tables.

Nd = No data.

Generic release estimation

The **total** emissions from the 10 sites not providing information have been calculated by using the worst case emission factors from site-specific data. The emission to water is calculated from the emission factor for site TexFormB assuming no waste water treatment. If the wastewater treatment at this site has the same efficiency as that estimated by EUSES i.e. 79.4 % removal, the emission factor becomes 549×10^{-6} (= $113\times10^{-6}/0.206$).

The estimation is slightly biased due to the fact that total HBCDD consumption at the unknown sites also includes the unknown consumption volume at TexForm2 which claims they have no emissions to water. The emissions to water are split between waste-water and surface water according to the TGD default assumption of STP-connection of 80 %.

The emission factor (7.31×10^{-6}) is used for estimating the emissions to air.

The emissions from a generic worst case **local** site have also been estimated using the same emission factors. The generic local site is assumed to use 174 tonnes/year of HBCDD. This corresponds to the 90th percentile use of the six sites, which provided data on their HBCDD consumption. The number of emission days is 300.

The site-specific emissions of HBCDD to water and air from this generic local site and the total emission for the 10 sites with no site-specific data are given in Table 3-14.

Table 3-14 Emissions of HBCDD to water and air from a generic local site and the total emission for the 10 sites with no site-specific data with formulation of polymer dispersion for textiles.

Site	Emission factor (10^{-6})	Emission to water (kg /year)	Emission factor (10^{-6})	Emission to air (kg/year)
Generic Local	549	95 (waste water)*	7.31	1.3
Total emission for the 10 sites with no site-specific data				3.55
Waste water		213		
Surface water		53		

Regional and continental emissions to water and air from formulation of polymer dispersions for textiles

Based on the information that the formulation of polymer dispersions takes place in three countries, 1/3 of the used amount and the total emissions are assigned to the region. The total emissions are calculated as the sum of the site-specific emissions in Table 3-13 and the emissions from the generic sites in Table 3-14. The resulting total, regional and continental emissions of HBCDD at sites involved in formulation of polymer dispersions for textiles are given in Table 3-15

Table 3-15 Total, regional and continental emissions of HBCDD from formulation of polymer dispersions for textiles.

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	6.8	2.3	4.5
Wastewater	221	74	146
Surface water	55	18	37

3.1.2.3 Release during industrial use (Life Cycle Stage III)

There are five types of industrial use involving HBCDD:

- Industrial use of EPS compound at the manufacture of flame retarded EPS
- Industrial use of HIPS compound at the manufacture of flame retarded HIPS
- Industrial use of XPS compound at the manufacture of flame retarded XPS
- Industrial use of HBCDD-powder at the manufacture of flame retarded XPS
- Industrial use of polymer dispersion at the backcoating of textile

INDUSTRIAL USE OF EPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED EPS

The number of sites with Industrial use of EPS compound at the manufacture of flame retarded EPS is assumed to be hundreds, because of the nature of this process. It is production of a lot of EPS-products for different uses and of many different shapes. The amount of HBCDD for this life cycle stage is relatively large

It is assumed that the HBCDD volume used in the manufacture of EPS within the EU equals the volume HBCDD used in the formulation of EPS compound (i.e. approx. 3400 tpa) because of relatively expensive transports due to the low density of the material. The regional tonnage is estimated to 340 tonnes/year using the 10 % rule. This is supported by the large number of sites and the fact that the compound and the product have low density and therefore not can be transported economically. The HBCDD consumption at the generic local site has been estimated to 17 tpa based on table B3.9 (IC=11 "Polymers Industry") in TGD which gives a **"fraction of the main local source"** of 0.05. The number of emission days is 300.

The emission factor, L3 (conversion, partially open processes) from the Emission Scenario Document on Plastic Additives, ((OECD, 2004)) is used. For organic flame-retardants this factor is 0.006 %. In this scenario, half of the emission is supposed to go to water as a result of wet cleaning of surfaces contaminated with HBCDD and EPS dust spread from the process (transport, sawing, and cutting). The other half is assumed to be directed to air as a result of HBCDD and EPS dust spread to the air from the process and released to the atmosphere via the ventilation. The resulting emission factors to both water and air are thus $30 \cdot 10^{-6}$. An STP connection rate of 80 % is assumed for the total, regional and continental release estimation according to the TGD default.

The resulting total, regional, continental and generic local emissions of HBCDD to water and air are given in Table 3-16.

Table 3-16 Total, regional, continental and local emissions of HBCDD from industrial use of EPS compound

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)	Local (kg/year)
Air	102	10.2	92	0.51
Wastewater	82	8.2	74	0.51
Surface water	20.4	2	18	-

INDUSTRIAL USE OF HIPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED HIPS

No site-specific information has been submitted and the number of sites involved is not known. It is assumed that the HBCDD volume used in the manufacture of HIPS within the EU equals the volume HBCDD used in the formulation of HIPS compound (i.e. approx. 210 tpa). The regional tonnage is estimated to 21 tonnes/year using the 10 % rule (TGD). The HBCDD consumption at the generic local site has been estimated to 5.25 tpa based on table B3.9 (IC=11 "Polymers Industry") in TGD which gives a **"fraction of the main local source"** of 0.25. The number of emission days is 30.

The emission factor, L3 (conversion, partially-open processes) from the Emission Scenario Document on Plastic Additives, ((OECD, 2004)) is used. For organic flame-retardants this factor is 0.006 %. In this scenario, half of the emission is supposed to go to water as a result of wet cleaning of surfaces contaminated with HBCDD and HIPS dust spread from the process (transport, sawing, and cutting). The other half is assumed to be directed to air as a result of HBCDD and HIPS dust spread to the air from the process and released to the atmosphere via the ventilation. The resulting emission factors to both water and air are thus $30 \cdot 10^{-6}$. A STP connection rate of 80 % is assumed for the total, regional and continental release estimation according to the TGD default. The resulting total, regional, continental and generic local emissions of HBCDD to water and air are given in Table 3-17.

Table 3-17 Total, regional, continental and local emissions of HBCDD from industrial use of HIPS compound

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)	Local (kg/year)
Air	6.3	0.63	5.7	0.16
Wastewater	5.0	0.5	4.5	0.16
Surface water	1.3	0.13	1.2	-

INDUSTRIAL USE OF HBCDD AT THE MANUFACTURE OF FLAME RETARDED XPS

Industry has requested that the site-specific information given for this use area should be kept confidential. Therefore, only release figures are presented here. Detailed data and calculations are given in the confidential Annex C.

There are, according to industry, 35 sites in Europe (EU 15) producing flame retarded XPS (Mills, 2003a). The sites use HBCDD either as powder or as compound. According to Industry information 70 % of the HBCDD used for XPS is used as compound (Klatt, 2003b). This is however contradicted by the site-specific data given, which indicate a much lower percentage. Site-specific data on used amounts of HBCDD is provided for 22 sites (Banner, 2003)). The use of HBCDD at these 22 sites is 3950 tonnes/year. Of these 22 sites 18 sites use the HBCDD as powder, 3232 tonnes/year. Four sites use HBCDD as compound, 719 tonnes/year (Abbott, 2001; Banner, 2003). It is assumed that the remaining 13 sites use HBCDD in the form of compound.

The total amount of HBCDD used for XPS can be calculated using the information that the total amount of HBCDD being used at the 22 sites with site-specific data represents 95 % of the total use for this application (Abbott, 2001). This gives a total amount of 4158 tonnes/year (3950/0.95 = 4158 tonnes/year).

However, it can be assumed that all HBCDD produced as polystyrene compound in the EU, 1730 tonnes/year, is used for the manufacturing of XPS within the EU, because of expensive transports. Adding this amount of HBCDD to the amount used as powder (3232 tonnes/year) gives a total amount of 4962 tpa. This latter value will be used in this risk assessment.

Industrial use of XPS compound for flame retarded XPS

The total amount of HBCDD used as compound is assumed to be 1730 tonnes/year (4962tpa-3232 tpa). The amount of HBCDD used at the four sites that have provided site-specific data is 719 tonnes/year. Thus, the amount used at sites with no site-specific data submitted is 1011 tonnes/year.

Data on used amounts of HBCDD in compound for manufacturing of flame retarded XPS are given in Table 3-18.

Table 3-18 Data on used amounts of HBCDD in compound (year 2002) for manufacturing of flame retarded XPS.

Site	HBCDD use (tonnes/year)
Total amount for the 4 sites with site-specific data	719
Total amount for the remaining 13 sites with no site-specific data	1011
Total use	1730

Local emissions

Site-specific release estimation

Site-specific data on the emission to water have been submitted from four sites. These sites did not deliver data on emissions to air. The emissions to air have been calculated using emission factors from a study on emissions to air from three XPS plants chosen to be representative in Europe (Mills, 2003b).

The site-specific emissions of HBCDD to water and air from the sites with industrial use of compound for flame retarded XPS are given in Table 3-19.

Table 3-19 Local emissions of HBCDD water and air from sites with industrial use of XPS compound for flame retarded XPS.

Site*	Emission to waste water [1] (kg/year)	Connected to municipal STP (Yes/No)	Recipient	Number of emission days per year	Emission to air (kg/year)	Number of emission days per year
XPS 1	2.2[1]	Nd	Nd	15[2]	0.31[1]	15[2]
XPS 2	0[1]	Yes[2]	Nd	1[2]	18[1]	300[4]
XPS 3	1.3[1]	Nd	Nd	1[3]	14[1]	300[4]
XPS 11	4.2[1]	Nd	Nd	2[2]	9.3[1]	300[4]

*The designation of the sites is the same as used by industry.

1. Details given in Annex C.

2. Data provided from industry.

3. No information on the number of emission days has been submitted. One day per year is used as a reasonable worst case.

4. The value is determined from the B-Tables in TGD.

Nd = No data.

Generic release estimation

The emissions from the 13 sites for which no site-specific data are available have been estimated using the worst case emission factors derived from the site-specific data (see Annex C for details). For the emissions to water, 80 % of the water is lead to a municipal STP and 20 % to surface water according to the TGD default. The generic local site is assumed to have a HBCDD consumption that equals the 90th percentile of the 22 sites with industrial use of HBCDD powder and compound for the production of flameretarded XPS, for which the consumption is known. The number of emission days is 1 for waste water emissions, as a reasonable worst case.

The emissions of HBCDD to water and air from the generic local site and the total emission for the 13 sites with no site-specific data are given in Table 3-20.

Table 3-20 Emissions of HBCDD to water and air from a generic local site and the total emission for the 13 sites with no site-specific data involved in industrial use of XPS compound

Site	Emission to water (kg/year)	Emission to air (kg/year)
Generic Local	7.9 (waste water)	17.4
Total emission for the 13 sites with no site-specific data Waste water Surface water	21.3 5.3	58.6

Regional and continental emissions

The amount used in the region is assumed to be 350 tonnes/year, as a reasonable worst case. This is about 20 % of the total amount. This assumption is based on information saying that around 65 % of the sites producing the compound are located in one country (Frölich, 2002). The industrial use of the compound is assumed to be located relatively close to the formulation sites.

The resulting total, regional and continental emissions of HBCDD at sites involved in XPS industrial use from compound are given in Table 3-21.

Table 3-21 Total, regional and continental emissions of HBCDD from industrial use of XPS compound.

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	100	20	80
Wastewater	27	5.4	21.6
Surface water	7	1.4	5.6

Industrial use of HBCDD powder for XPS

There are 18 sites producing flame retarded XPS using HBCDD powder according to Industry information. Site-specific data have been provided for all these sites. The total use of HBCDD for these 18 sites sums up to a total of 3232 tonnes/year.

Local emissions

Site-specific

Data on the emission to water are reported from 17 of the 18 sites. The figures represent either the concentration in the process effluent or the concentration in the outflow from an on-site STP. The site with the largest emission, reports an emission to waste water of 15.1 kg/year. The total emission of HBCDD to waste water from all of these 17 sites is approximately 30.5 kg/year. The emission from the 18th site (XPS 27) is calculated using the largest of the known emission factors giving a total emission for all 18 sites of approx. 33 kg/year.

Emissions to air have been calculated using data from a study were emissions to air from three XPS plants was studied (Schröder, 2002)

The site-specific emissions of HBCDD to water and air from the sites with industrial use of HBCDD powder for flame retarded XPS are given in Table 3-22.

Table 3-22 Local emissions of HBCDD to water and air from sites with industrial use of HBCDD powder for flame retarded XPS.

Site*	Emission to waste water[1] (kg/year)	Connected to municipal STP (Yes/No)	Recipient	Number of emission days per year to water	Emission to air[1] (kg/year)	Number of emission days per year to air
XPS 4	4.4	Nd	Estuary/sea	2[2]	1.5	300[4]
XPS 5	1.2	Nd	River	2[2]	1.4	300[4]
XPS 6	0.055	Nd	River	1[3]	3.7	300[4]
XPS 7	3.7	Nd	River	1[3]	1.5	300[4]
XPS 8	0.0024	Nd	Nd	2[2]	1.1	300[4]
XPS 9	0	Nd	River	1[2]	0.73	200[4]
XPS 10	6.0	Nd	River	1[3]	0.54	148[4]
XPS 13	0.0029	Nd	River	1[2]	0.70	192[4]
XPS 14	0.0019	Yes	Sea	2[2]	0.15	42[4]
XPS 16	0	Nd	Nd	1[3]	0.40	108[4]
XPS 17	0	no	Nd	1[3]	1.8	300[4]
XPS 18	0	no	Nd	1[3]	1.8	300[4]
XPS 20	0.11	Yes	River	2[2]	1.2	300[4]
XPS 21	15	Nd	River	2[2]	1.5	300[4]
XPS 23	0.00004	Yes	Nd	1[2]	0.59	162[4]
XPS 24	0.0004	Yes	Nd	12[2]	0.91	248[4]
XPS 26	0.021	Yes	River	1[3]	3.8	300[4]
XPS 27	2.5	Nd	Nd	1[3]	0.23	62[4]

1. Emission data provided from industry. Details are given in Annex C.

2. Data from industry.

3. No information on the number of emission days has been submitted. One day per year is used as a reasonable worst case.

4. The value is determined from the B-Tables in TGD.

*The designation of the sites is the same as used by industry.

Nd = No data

Regional and continental emissions

The two sites having the largest emissions to water (XPS10 and XPS21) are chosen for the region, as a reasonable worst case. If the emission were proportional to the use of HBCDD the tonnage at these sites would be approximately 47 % of the total use. However, the annual use at these sites is only about 9 % of the total annual use for this life cycle stage and it is this latter figure that is used when the total emission to air is assigned to the region. The resulting total, regional and continental emissions of HBCDD at sites involved in XPS industrial use using HBCDD powder are given in Table 3-23.

Table 3-23 Total, regional and continental emissions of HBCDD at sites involved in industrial use of HBCDD powder for flame retarded XPS.

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	23.6	2.1	21.5
Wastewater	26.4	16.9	9.5
Surface water	6.6	4.2	2.4

INDUSTRIAL USE OF TEXTILE BACK-COATING AGENT

The annual amount of HBCDD used for textile backcoating is assumed to be 1054 tonnes. According to information from industry, 24 sites have been identified in the EU (De Poortere, 2001). The sites are located in Belgium (15 sites) and the UK (9 sites). For 5 of the sites information on HBCDD use has been submitted (total annual use 276.6 tonnes/year) (Frölich *et al.*, 2003). Solid waste including sludge from on-site STPs at textile backcoaters is either incinerated or put on controlled landfills, due to the potential of heavy metal contamination.

The regional volume is assumed to represent 50% of the total use because of the geographic concentration according to this information.

Data on used amounts of HBCDD for Industrial use of textile back-coating agent are given in Table 3-24.

Table 3-24 Data on used amounts of HBCDD (2000) for Industrial use of textile back-coating agent.

Site*	HBCDD use (tonnes/year)
Backcoat.1	140.7
Backcoat.2	33.6
Backcoat.3	52.8
Backcoat.4	22.5
BackcoatC	27
Total amount for the sites above with site-specific data	276.6
Total amount for the remaining 19 sites with no site-specific data	777.4
Total use	1054

*The designation of the sites is the same as used by industry

Local emissions

Site-specific release estimation

Three different emission factors are used depending on type of wastewater treatment.

A: $8.5 \cdot 10^{-3}$ is representative for sites with no STP.

B: $5 \cdot 10^{-5}$ is representative for sites using settling and, decantation.

C: $5 \cdot 10^{-7}$ is representative for sites using flocculation, settling and, decantation.

The above emission factors are derived from measurements at two sites, one being a backcoater (emission factor A and B) and the other a polymer dispersion formulator (emission factor C). At the backcoating site the concentration of HBCDD was measured before and after wastewater treatment. The concentration of HBCDD in the process water before treatment was equivalent to an emission factor of $8.5 \cdot 10^{-3}$. The concentration in process water after treatment (settling and, decantation in three consecutive sumps) was equivalent to an emission factor of $5 \cdot 10^{-5}$. The third emission factor of $5 \cdot 10^{-7}$ was derived from measurements at a site formulating polymer dispersions treating the wastewater by flocculation, settling and decantation.

Only four of the backcoating sites have submitted information on what type of wastewater treatment they use. One site has no on-site STP, two of them have type B wastewater treatment and the fourth has type C treatment.

Information on HBCDD emissions to air has been submitted from the five sites. The emission factors range from 0 to 0.7×10^{-6}. The emissions are attributed to volatile losses through the stack from the drying oven.

The site-specific emissions of HBCDD to water and air from the sites with industrial use of textile back-coating agent are given in Table 3-25.

Table 3-25 Local emissions of HBCDD to water and air from sites with industrial use of textile back-coating agent.

Site*	Emission factor (10-6)**	Emission to waste water (kg/year)	Connected to municipal STP (Yes/No)	Recipient	Emission factor (10^{-6})	Emission to air (kg/year)	Number of emission days per year
Backcoat.1	50[1]	7.0[2]	Nd	Nd	0.5[1]	0.070[2]	225[3]
Backcoat.2	50[1]	1.7[2]	Nd	Nd	0.5[1]	0.017[2]	54[3]
Backcoat.3	8500[1]	450[2]	Nd	Nd	0[1]	0[2]	84[3]
Backcoat.4	0.5[1]	0.011[2]	Nd	Nd	0.7[1]	0.016[2]	180[3]
BackcoatC	16[1]	0.43[2]	Nd	Nd	0.009[1]	0.00024[2]	43[3]

*The designation of the sites is the same as used by industry.

1. Emission factor provided from industry.

2. The emission is calculated from the data on amount HBCDD used at the site and the emission factor.

3. The value is determined from B-Table 3.12 in the TGD.

Nd = No data.

Generic data release estimation

The emission to water from the 19 sites for which no site-specific data are available is estimated to 777400 kg/year$\times 8500 \times 10^{-6}$ = 6608 kg/year using the emission factor from Backcoat3 (8500×10^{-6}) as a worst case. Assuming 80 % STP connection according to the TGD default gives 5286 kg/year to wastewater and 1321 kg/year direct to surface water.

The total emission to air from the 19 sites for which no site-specific data are available is estimated to 777400 kg/year×0.7×10^{-6} = 0.54 kg/year using the emission factor determined from Backcoat4 (0.7×10^{-6}).

A generic local site has been constructed assuming that 158 tonnes/year of HBCDD is used at the site. This is about 30 % of the regional volume and somewhat higher than the 90th percentile (106 tonnes/year) for the used amounts at the five sites which have delivered data. The number of five site-specific data on used amount of HBCDD is too few for using the 90th percentile as the amount used at the generic site.

The emissions of HBCDD to water from the generic local site and the total emission for the 19 sites with no site-specific data are given in Table3-26.

Table3-26 Emissions of HBCDD to water and air from a generic local site and the total emission for the 19 sites with no site-specific data with industrial use of textile back-coating.

Site	Emission factor (10^{-6})	Emission to wastewater/ surface water (kg/year)	Emission factor (10^{-6})	Emission to air (kg/year)
Generic Local site	8500	1343	0.7	0.11
Total emissions for the 19 sites with no site-specific data Waste water Surface water		 5286 1321		0.54

Regional and continental emissions to water and air from industrial use of textile back-coating

Since backcoating is allocated to two countries in the EU, 50 % of the total HBCDD use is assigned to the region as a reasonable worst case. The resulting total, regional and continental emissions of HBCDD at sites involved in industrial use of textile back-coating are given in Table 3-27.

Table 3-27 Total, regional and continental emissions of HBCDD at sites involved in industrial use of textile back-coating

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	0.64	0.32	0.32
Wastewater	5653	2826	2826
Surface water	1413	706.5	706.5

3.1.2.4 Release during professional and private use (Life Cycle Stage IV)

No information on private use of sole HBCDD has been provided and is not considered probable. Release of HBCDD to the environment, due to the professional and private use of end-products can occur via

i) generation of waste and/or

ii) losses during service life due to migration and wear

These losses can occur from end-products during professional uses, such as for instance through:

- professional use of PS blocks, e.g. cutting, gluing and heating

- manufacturing of upholstered furniture and sewing of interior textiles and mattress ticking etc.

- insulation work in buildings, parking decks, roads, inverted roofs

Indoor activities may give rise to direct releases of HBCDD to the work environment. Outdoor activities could give rise to uncontrolled spreading of surplus material, which will end up in the environment.

Work with insulation boards in buildings result in emissions of HBCDD through minor polystyrene particles (dust) during e.g. sawing.

Emissions due to private use of articles containing HBCDD are assumed to be much less than the emissions from professional use. In this risk assessment the emissions from private use is included in the emissions from professional use.

Release of HBCDD containing particles may also take place during demolition of buildings. This is handled in Chapter 3.1.2.6.

PROFESSIONAL USE

Professional use of articles containing HBCDD, which is assumed to result in releases to the environment and for which the releases are estimated is the installation of building insulation at construction and renovation of buildings.

Building insulation

General

Release occurring during construction and renovation of buildings can be estimated (e.g., caused by sawing the polystyrene boards), the potential release of polystyrene (PS)-particles from PS-boards during construction has been provided (Klatt, 2003a).

The weight of the particles being formed at sawing of boards (XPS and EPS) has been measured. It is assumed that every tenth board is sawed, and always along the short side. The boards are 6 cm thick, the short side 60 cm, and the long side either 125 or 104 cm.

The release of particles from sawing of *XPS-boards* is estimated to be 5.0 g XPS-particles per tonne XPS. With a yearly use of 4.962 tonnes HBCDD in XPS, a worst-case estimate would result in a yearly release of 25 kg HBCDD (via particles) from the use of XPS in constructions.

The corresponding figures for sawing *EPS-boards* are a release of 445 g particles per tonne EPS. However, it is more common to make a cut in the board with a sharp knife and then to break the board, or -at large sites, to cut the boards with a “hot wire”. In both methods of cutting EPS-boards result in much less particles being formed, as compared to sawing. Considering the use of these different methods, a worst-case estimate is a release of 100 g

particles per tonne EPS-board, resulting in a yearly release of 339 kg HBCDD from the 3392 tonnes used in EPS. The release of HBCDD from EPS used for packaging materials is assumed to be the same as for EPS used in insulation material.

The total figure would be a yearly release of 364 kg HBCDD from PS-boards during construction.

The distribution of this release is assumed to be 50 % to surface water and 50 % to air. These activities are assumed to take place outdoors or at open building sites why the particles are small and relatively light will be released to the surrounding environment. The relatively large area per mass unit of polystyrene of the particles may facilitate the release of HBCDD to the air in a relative short time.

Emissions to water

Fifty percent of the total emissions from insulation boards at the installation in buildings are assumed to be directed to surface water. Thus, it is assumed that 182 kg/year is emitted to surface water.

Emissions to air

Fifty percent of the total emissions from insulation boards at the installation in buildings are assumed to be directed to air. Thus, it is assumed that 182 kg/year are diffusively spread to air.

Regional and continental emissions

For professional use of HBCDD, the regional release is assumed to be 10 % of the EU release.

Total, regional and continental emissions of HBCDD from installation of professional insulation boards are given in Table 3-28.

Table 3-28 Total, regional and continental emissions of HBCDD from installation of professional insulation boards

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	182	18	164
Wastewater	0	0	0
Surface water	182	18	164

PRIVATE USE

The private use of HBCDD containing articles such as e.g. insulation boards is small compared to the professional use. Emissions from private use are considered to be covered by the calculation of emissions during professional use.

3.1.2.5 Releases during service life (Life Cycle Stage V)

GENERAL

A diversity of flame retarded products containing HBCDD is put on the market. Examples are given in Chapter 2.

Releases during service life include emissions caused by migration of HBCDD from the materials and wear leading to release of particles containing HBCDD. The emissions from sawing of polystyrene insulation-boards are dealt with in section 3.1.2.4. Professional use. The emissions from demolition of buildings are dealt with in section 3.1.2.6. Releases from waste and waste management.

Assuming a constant annual use of HBCDD of 9,000 tonnes, and an average product service lifetime of 50 years, this will lead to an amount of about 450000 tonnes constantly in use in the society, when a steady state is reached. However, the lifetime for the various products containing HBCDD varies considerably, with a lifetime in the order of 10-30 years for textiles and 30-100 years for building insulation. Therefore, separate calculations are performed below for textiles and for building insulation.

Some estimations of the release of HBCDD from different materials have been performed. The estimates of release factors are presented in Table 3-29. The German UBA has used a release factor of 1 % from polystyrene (Ahlers *et al.*, 1996).

In the BUA report (BASF, 1996) on HBCDD it was concluded that a migration study of HBCDD from polystyrene should be performed to clarify the environmental releases. This was done by BASF in 1996 using a study design where a board of XPS containing 0.54 % of HBCDD was put into contact with (1) still distilled water and (2) flowing distilled water.

(1) It was reported (BASF, 1996) that the loss of HBCDD from XPS to 2.5 litre of distilled still water was 0.5 µg/l after one week. The concentration in the water was constant between one and six weeks. It was stated that the loss was independent of the piece of Styrodur (XPS) was fresh or used. However, the conditions of use were not stated. The release was calculated to be 7.5 µg/m^2.
(2) In the study with flowing water the release was 0.083 % (BASF, 1996). It was also found that the loss of HBCDD would level off with time. It was stated that the results of the study indicate that only substance situated at the surface of the material will be lost.

However, considering the very low solubility of HBCDD in distilled water, these studies do not seem very relevant. The results from the experiment are not considered to be useful for any of the scenarios in Chapter 3.1, because the surrounding for XPS in most situations is not water.

A release study was carried out on fabric treated by high temperature exhaustion dyeing with 2.5 % HBCDD. The flame retarded PET fabric was washed about 40 times. Measurements of bromine left in the fabric showed that after ten washes no more loss occurred. It was also shown that the magnitude of the loss of HBCDD was concentration-dependant. The loss of HBCDD was 17 % from a PET fabric with an initial concentration of 2.5 % (McIntyre *et al.*, 1995). The magnitude of the release in this study is not believed to be representative for the washing of fabric today. And, based on the knowledge on how HBCDD-treated fabric is being used, it is not likely that these fabrics will be washed very often, if at all.

In the Emission Scenario Document- 'Additives Used in Plastics Industry', loss factors over service life for products used indoors are set for organic flame-retardants to 0.05 % volatile loss to atmosphere over lifetime and 0.05 % leaching loss to liquid waste over lifetime (totally 0.1 %).

Table 3-29 Estimations made of losses from polymers could be summarised as follows

Material	Surrounding medium	Losses	Reference
Polystyrene	Environment not specified	1 %	**(Ahlers *et al.*, 1996)**
Exhaustion dyed PET fabric (2.5 % HBCDD)	Washing water	17 % total	**(McIntyre *et al.*, 1995)**
Organic flame retardant in polymer matrix (unspecified), Indoor service	Atmosphere Leaching loss to liquid waste	0.05 %/service life 0.05 %/service life	**(Anonymous, 1998)**
XPS (0.54 % HBCDD)	Distilled still water	7.5 μg/m²/7 days	(BASF, 1996)
XPS (0.54 % HBCDD)	Distilled flowing water	0.083 %/3 months	(BASF, 1996)
XPS (1.1-2.0 % HBCDD)	Air	5 μg/m²/year	**(Klatt, 2003a) ***

* For more information about on this study, see below under "Releases from polystyrene boards during service life"

Swedish IVL (Palm *et al.*, 2002) has made a model for the flow and emission of HBCDD in products in the Stockholm area, Sweden, based on analysis of the material flow and estimations of emissions. The main emission route is then to the air from EPS and textiles. Since almost no XPS containing HBCDD nowadays is used in Sweden, the emissions originate from former use of XPS. Estimation of HBCDD in products in use in the Stockholm society, inflow of HBCDD and emissions to air and ground are given in Table 3-30

Table 3-30. Estimation of HBCDD in products in use in the Stockholm technosphere, inflow of HBCDD and emissions to air and ground.

HBCDD in product	Inflow of HBCDD with products (kg/year)	Amount of HBCDD in use in the Stockholm area (tonnes)	Emission of HBCDD to the ground/soil (kg/year)	Emission of HBCDD to the air (kg/year)
EPS	50-200	1.4-8.4		0.7-4.2
Textiles	200-2100	1.9-22		0.94-10.9
Car equipment/textiles	?	13.6-48.6		6.8-24.4
XPS	0	12-19	82-118	5.8-8.4

Data from **(Palm *et al.*, 2002)**

The approach used in the calculations above assumes a constant release of HBCDD. This approach also assumes a constant continuous use of the substance, which is difficult to predict. It is likely that the total amount of construction material and consumer goods containing HBCDD will increase with increasing population and increasing welfare, unless HBCDD is substituted in the current applications. This would indicate that the amount of HBCDD in the society would increase with time and thereby resulting in increasingly releases

from end-products and waste. However, this may not be the case in Sweden because of the minor use of HBCDD

The results from this study are not further used in this risk assessment, because it is limited to Stockholm and therefore not considered representative for the scenario.

RELEASES FROM TEXTILES DURING SERVICE LIFE

The use of HBCDD as flame-retardant additive in textiles could possibly lead to contamination of surface-water during washing of the fabric. Furthermore, emissions due to the wear of the fabric during its service life can also be expected. The annual use of HBCDD in textiles is assumed to be 1050 tonnes. The lifetime of this kind of textiles may be 10-30 years.

The Polymer Research Centre at the University of Surrey and the Bolton Institute undertook studies to determine the release of flame-retardants from backcoated textiles (Thomas and Stevens, 2006). The effects of ageing and wear on the potential release were investigated. More information from this study is presented in Chapter 4.1.1.3. Consumer exposure. Where possible, accepted standard methodologies were used for testing. Ageing was simulated by exposure to higher temperatures, alone or in combination with increased humidity, exposure to UV-A light and by a physical process, the latter being a modified standard method for testing abrasion resistance of fabrics. The wear was simulated by a "Martindale abrasion test" according to ISO 12947-1:1999; this test is commonly used in the textile industry to simulate abrasion of textiles over their product life. The release was measured both as formation of debris (and size of the particles/fibres in the debris) and as volatile release. The ageing and wear simulations were designed to simulate the entire lifetime of the textile.

The tests were performed on one single cotton fabric with 7.7 w.t.-% HBCDD, but the textile is not further described in the report. The coating was applied at a rate of 271 g/m^2, giving an area density of HBCDD in the final coating of around 1.98 mg/cm^2.
It is assumed to be a representative cotton fabric, but the lack of information on the textile makes it difficult to evaluate the representatively of both the textile and the results, as abrasion depends a lot on the structure/construction of the textile. Fabric only based on cotton is not commonly used in furniture fabric, but blends of cotton and e.g. wool and synthetic polymers are more common. According to the study director, the choice of cotton would possibly be a worst-case choice, as other materials wear at slower rates.

It is an extensive study, which has generated a huge amount of data. However, the methods used in the study are poorly described and the calculations are often difficult to follow. Therefore, it is difficult to evaluate the results and how representative they are for real world conditions.

In the study, the samples were subject to various standard accelerated ageing protocols as follows:
a) control conditions (20°C and 65% relative humidity for either 18 or 36 weeks),
b) environmental ageing (30°C at 65% relative humidity for 20 weeks or 60°C at 75% relative humidity for 2, 8, 14 or 20 weeks),
c) thermal ageing (60°C, 70°C[4] or 90°C all at ambient relative humidity for up to 10 weeks (60 and 70°C) or 4 weeks (90°C) and

[4] This temperature appears as 75°C in places in the report.

d) UV-ageing (exposed to 340 nm at room temperature for 5, 10, 20 or 30 days).

Following ageing under the various conditions, the samples were subject to various wear and leaching tests. Volatile emissions to the head space were also determined during the thermal ageing tests. No HBCDD could be detected in these samples indicating minimal loss by volatilisation during the ageing process.
To determine particulate and volatile release during wear of the textile, an adaptation of the Martindale abrasion test using an enclosed system was used. The test equipment was modified to allow settled debris and particulates to be collected and the concentrations of volatiles and airborne particulates to be determined. Using this equipment, the mass balance was found to be over 99.5% over a period of up 30,000 wear cycles (this was taken to be representative of a full service-life wear history).

The tested fabric exhibit a low wear rate for all ageing conditions with the exception of UV aged (wavelength 340 nm) materials that wear with increased ageing. The UV exposure is thought to be representative for the indoor environment as UV-A goes through glass windows, and it is well known that cotton and wool fibres are weakened by sunlight.

The debris produced in the fabric wear test contains a large number of short and long cotton fibres from the fabric. In addition, particulates are present in the debris with the largest quantity (both number and weight of particles) in the 10-90 μm size ranges with a low quantity by weight of smaller particles. Some are aggregates from the backcoating material and these consist of submicron particles in many cases.

Flame-retardant compounds were found to be heterogeneously distributed throughout the debris which is consistent with the fact that the deposition of the backcoating on the fabric is non-continuous and the distribution of flame-retardant compounds within the fabric is non-uniform.

UV-aged fabric produced greater amounts of debris during wear testing than the non-aged fabric, which seems reasonable. No HBCDD were detected as volatiles. After the maximum UV-aging and number of wear cycles, the report says that debris contained 0.47 % HBCDD by debris weight, which would represent a maximum of 44 or 84 mg of HBCDD per m^2 of fabric surface area released through wear of the unaged or UV-aged samples.

Extraction of the unaged fabrics, to determine the total amount of HBCDD potentially available for recovery, showed a recovery of 93 ± 5 % of the load level. The corresponding values after thermal or UV-aging was 72.6 % or 74.6 %, respectively, indicating that 20 % of the HBCDD could not be recovered after aging. Thus, thermal or UV-aging resulted in similar reductions in total extractability. These reductions could be due to an actual reduction in the fabric content of HBCDD or a reduction in the extraction efficiency due to an increase in the binding of the flame-retardant to the fabric or other processes. The reductions could also be related to, e.g., debromination of the flame retardant in the textile, and that the modified (debrominated) substance is not identified in the chemical analyses as mentioned. A very small follow-up test by Industry indicates that when measuring the bromine content of the textile before and after aging, the reduction was less (approx. 7 %) than reported in this study (personal comm. BSEF), supporting that release of HBCDD is not the only reason to the observed reduced amount of HBCDD in the textile after aging.

Overall, the most important information from this study is that debris that contains HBCDD is being formed, and that the concentration of HBCDD in the debris is at least 0.47 %. The

amount of dust generated under conditions said to mimic life-time wear (UV-light and mechanical wearing) was 0.86 % of the weight of the textile (2.3 g dust/m^2).

The analyses of the bromine content in the debris can be used to estimate the release resulting from wear of textiles. This analysis is made directly on the debris (SEM and EDX analysis) without extraction. The result is the amount of bromine as an element. The mean concentration of HBCDD in the analysis of three samples of debris was 0.47 %. If this release is expressed in an average area release with respect to fabric surface area it will be 84 mg/m^2. The HBCDD area density in the samples was 1.98 mg/cm^2 (1.98×10^4 mg/m^2). The textile area of the textiles containing the annual use of 1050 tonnes (1.05×10^{12} mg) will be 1.05×10^{12} mg/1.98×10^4 mg/m^2 = 5.3×10^7 m^2. The release from this area will be 84 x 5.3×10^7 mg = 4.4545×10^9 mg = 4450 kg.

When considering these emissions, it should be noted that they are based on an assumption that all of the textiles are subject to UVA-ageing (exposure to wavelengths of 340 nm). In practice, not all textiles will be exposed to such radiation, and so the actual emissions from this source are likely to be lower than estimated here. In addition, the estimate assumes that all textiles treated with HBCDD will be subject to wear during their lifetime. This is not likely to be the case in practice as some items, for example window blind fabrics, are not likely to be subject to such wear and even in the case of furniture, only a fraction of the total fabric area on the furniture will be subject to wear.

To illustrate this, further information on these aspects has been provided by Stevens (2007). Based on examination of a typical large fabric-covered three-piece suite that was 17 years old and was in regular use, it was estimated that the area of the fabric subject to high wear was around 10-11% of the total surface area. The main areas of high wear were found to be the top face of the seat cushion and a portion of the arms. The back cushion received far less wear, as did the other areas where the fabric is generally compressed or flexed rather than abraded. These areas defined subject to medium wear was estimated to be 19-25% of the total fabric area. Assuming that the 11% figure can be generally applied, the estimated emission figure of 4450 kg/year would be reduced to around 490 kg/year (= 84 x 5.3×10^7 x 0.11 mg/year) when the area of the fabric most subject to wear is taken into account.

Stevens (2007) also considered that not all of the fabric will be exposed to direct sunlight, and estimated that a figure of around 5% of the total area that would be exposed would be reasonable. Taking this into account, and assuming that the lifetime emission figure for the non-UVA-exposed area would be around 44 mg/m^2 (as found in the experiments with the non-aged fabric), and again assuming that only 11% of the surface area of the UVA-exposed and non-UVA-exposed parts of the fabric were subject to the highest levels of wear, the estimated emissions are further reduced to around 268 kg/year (= 84 x 5.3×10^7 x 0.11 x 0.05 + 44 x 5.3×10^7 x 0.11 x 0.95 mg/year).

It is decided to use the emission figure 268 kg/year in the risk assessment.

The use of flame retarded textiles in the EU is not evenly distributed throughout the EU owing to the differing fire safety regulations - particularly for domestic upholstery - in various countries. In order to take this distribution into account, it will be assumed that around 25% of these releases occur in a region (in line with the approach taken in the risk assessment of decabromodiphenyl ether (UK Environment Agency, 2007).

This release is assumed to be to waste-water, via wet cleaning of indoor floors and via solid waste from vacuum cleaning and sweeping. A minor release will be via the ventilation. This is assumed to be negligible. Information for assuming the distribution of this release is very limited. A distribution of 50 % to waste water and 50 % to solid waste is assumed. The STP connection rate is assumed to be 80 % according to the TGD default.

The emissions of HBCDD from textiles during service life are given in Table 3-31.

Table 3-31 Total, regional and continental emissions of HBCDD from textiles during service life.

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	0	0	0
Wastewater	107	27	80
Surface water	27	7	20

Textile washing,

General

The flame retardant is physically incorporated into a polymer on the back of the fabric and so this would be expected to minimise the loss of HBCDD during washing. The types of fabrics that HBCDD is used in, upholstery fabrics, are unlikely to be washed frequently. Indeed, for many items of furniture, no washing is envisaged, as the covers are not removable. Even for furniture with removable covers, it would be expected that washing would be infrequent, perhaps of the order of once per year. A figure of around 2 % has been suggested as a reasonable estimate of the percentage of the current textiles that contain HBCDD that may be subject to washing during use.

For HBCDD-treated textiles a release factor for release via washing water of 17 % has been mentioned, see Table 3-29 (McIntyre *et al.*, 1995). However, the release factor of 0.05 % (5×10^{-4}) to wastewater during service-life is used as a realistic worst case (Anonymous, 1998). This would be the emission of substance as long as the matrix is intact and the surface area remains unchanged. No significant emissions to air are assumed to take place during washing of textiles.

If the release factor of 5×10^{-4} to waste water during service-life is used, the resulting releases to wastewater would become 10.5 kg HBCDD per year ($0.0005\times1050000\times0.02 = 10.5$ kg). There is considerable uncertainty in this estimated figure.

Regional and continental emissions

The use of flame-retarded textiles is not evenly distributed throughout the EU owing to the differing fire safety regulations in various countries. In order to take this distribution into account, the regional release is assumed to be 25 % of the EU release.

The resulting total, regional and continental emissions of HBCDD from professional use of textiles are given in Table 3-32.

Table 3-32 Total, regional and continental emissions of HBCDD from washing of textiles.

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	0	0	0
Surface water	0	0	0
Wastewater	10.5	2.6	7.9

RELEASES FROM POLYSTYRENE BOARDS DURING SERVICE LIFE

Emissions to water

No emissions to water from polystyrene boards are assumed to take place during service life.

Emissions to air

Emission experiments have been conducted to measure the loss of HBCDD from foamed polystyrene. Air was blown through a tube with a surface of 0.15 m^2 (40 mm diameter and length of 1200 mm, 15 l/h flow) of foamed polystyrene (extruded polystyrene -XPS, containing respectively 1.2 and 2.0 % HBCDD with a density of 35 g/l). The emitted HBCDD was adsorbed on PUR-foams thus minimising any surface effects. After the sampling period the PUR-foams were extracted and HBCDD was determined by LC/MS with a detection limit of about 4 ng HBCDD per sample.

Given a total annual use of 350 million m^2 flame retarded foamed polystyrene in Western Europe and an emission of 70 ng/m^2 HBCDD in five days (= 5 μg/m^2 per year), calculated from a released amount of during hours. From this one can calculate the emission of HBCDD per year to air to about 1.8 kg HBCDD per year in Western Europe (Mills, 2002; Klatt, 2004). The consumed annual amount of HBCDD in these applications is 7364 tonnes giving a loss factor to air of about 2.4×10^{-5} %. This factor is used in the consumer scenario 'indoor air' (Chapter 4.1.1.3).

The steady state emissions from the polystyrene boards are the emissions from the annual consumed amount multiplied with the service life of the boards (30-100 years). To reflect the emissions today we assume a service life of 30 years, because the boards are not believed to have been used much more than 30 years back yet.

If the calculations are modified to reflect the market proportions of EPS and XPS and the release of HBCDD per area unit is proportional to the concentration in the polystyrene matrix the annual release will be 0.7 kg. With a service life of 30 years the release will be 21 kg. There are no data available on the release of HBCDD from EPS foam. The assumption that the release should be proportional to the concentration in the polystyrene matrix is uncertain, because of differences of the structure of the matrixes of foams made of XPS and EPS.

The material used in the experiment is new and the release may differ over time because of migration. The environment in which the material is located during use may be different to the experiment. The experiment does not consider air saturation.

The value used in the calculations is the highest measured in the experiment.

Due to the uncertainties, the emission 1.8 kg/year×30 years = 54 kg/year is used as a reasonable worst case.

REGIONAL AND CONTINENTAL EMISSIONS

For private use of HBCDD, the regional release is assumed to be 10 % of the EU release.

The resulting regional and continental emissions used as input to EUSES are given in Table 3-33.

Table 3-33 Total, regional and continental emissions of HBCDD from building insulation

HBCDD emissions	Total (kg/year)	Regional (kg/year)	Continental (kg/year)
Air	54	5.4	48.6
Surface water	0	0	0
Wastewater	0	0	0

3.1.2.6 Releases from waste and waste management (Life Cycle Stage VI)

Waste is generated mainly in three situations: HBCDD/material losses at all industrial scenarios (point sources). This waste is generally; recycled into the process, put on landfill or incinerated. Emissions from this kind of waste generation are assumed to be included in the release estimates of the TGD for the corresponding life-cycle step.

1. Generation of waste during the professional and private use as described in section 3.1.2.4
2. Disposal of worn-out articles. This waste is thought to be treated as follows;- recycled - remained in the environment, put on landfill, incinerated
3. Releases from demolitions of buildings are also relevant to take into account in the waste life-cycle stage.

There is a big uncertainty in future releases of HBCDD from demolition of buildings insulated with flame retarded polystyrene (EPS and XPS). The uncertainties depend, among other things, on how and by whom the demolition will be performed. Current methods of demolition include; implode a structure with explosives, use a crane and wrecking ball technique, or deconstruct the structure (US EPA; www.epa.gov/epaoswer/non-hw/debris/). This handling may contribute to release of minor particles and fragments of the material spread in the environment. HBCDD may then be released from these particles to the environmental compartments. Larger pieces of polystyrene board may be recycled, and recycling of EPS does occur in an organised way in several European countries (personal communication, Industry). However, the overall recycling of demolition material is still only estimated to be 30 % (RTD info, no 26, **http://europa.eu.int/comm/research/rtdinfo/en/26/constr3**), although it is likely to increase with time.

Manual deconstruction was mimicked by manual breaking of boards. The release was measured by weighing of particles being formed. No particles were formed by two breaks of a 456 g XPS-board, whereas two breaks of a 287 g EPS-board was estimated to generate 0.026 g particles, giving an emission factor of 90 g EPS-particles/tonne EPS. If assuming that the

overall recycling of 30 % is also valid for PS-boards (manually removed from building), a yearly release of 108 kg HBCDD can be calculated based on an annual use of 8,000 tonnes HBCDD in EPS and XPS of which approximately 50% is EPS (90 g/tonne×4,000 tonnes×30 % recycling).

For the remaining fraction of demolition material not being recycled (70 %), the lack of information on how much the PS-boards are being broken during demolition makes it difficult to calculate the potential emissions. Therefore, the release factor of 0.1 % (Anonymous, 1998) is used for the remaining fraction of 70 % (i.e., 70 % of 8,000 tonnes used in XPS and EPS), resulting in a potential worst-case estimate of future emissions of 5,600 kg HBCDD per year, when buildings insulated with HBCDD-containing PS-boards starts to get demolished. As comparison, most buildings being demolished today were built in the 1920-ies (RTD info, no 26). However, it is a lot of uncertainty in this estimate, and with increasing recycling of demolition material, the emissions will decrease. Still, it is likely that the future releases will be bigger than those occurring during construction.

Recycling of EPS does occur in several European countries. The recycling will increase the control of the material flow, and lead to decreased emissions. However, theoretically, recycling can also lead to an uncontrolled contamination of polystyrene products, which do not need to be flame retarded, with HBCDD. It is not clear whether flame retarded expanded and extruded polystyrene end-products are treated separately in the recycling process. There is no labelling/marking system so that the waste streams could be kept apart. Recycling of EPS could lead to that concrete mixed with EPS in the future contains HBCDD (Freilich, 2004). Recycling XPS from demolition is not an obvious option as the material is not clean. Building job site waste on the other hand, if properly collected, could be recycled.

Construction material can be categorised into buried and not buried material. HBCDD is present in both categories. Waste remaining in the environment applies to use of construction material on or in the ground, such as insulation under parking decks, rails, roads, exterior insulation of cellars etc. These kinds of end-products are expected to remain in the environment also after use. Service lifetime expectancy is claimed to be 50 years or more for extruded insulation.

One important difference between landfilling and most other life-cycle steps is the time frame. Emissions from landfills may prevail for a very long time, often thousands of years or longer. Polymer end-products containing HBCDD will accumulate on landfill sites. Degradation of the matrix will sooner or later cause release of the substance from the matrix. Besides this, biodegradation of HBCDD that occur in the landfill will limit potential future emissions.

Polystyrene is degraded in the landfill due to UV-light, micro-organisms and physical impact. According to Sundqvist and co-authors, PS is degraded through an ”unzipping” of the polymeric chain giving mostly the styrene monomer as degradation product **(Sundqvist *et al.*, 1994)**. During approximately 100 years. Some 1-10 % of PS will be degraded in the landfill.

Incineration

An unknown amount of HBCDD-containing end-products are incinerated every year in incineration plants.

Co-combustion of building insulation foams with municipal solid waste has been investigated 1993-1994 in a well-functioning municipal solid waste incinerator (MSWI) (Vehlow and Mark, 1995, 1996). The combustion temperature was 850-950 °C. The MSWI was equipped

with an fabric filter and two Venturi scrubbers. Polybrominated dibenzodioxins (PBDDs) were generated at the levels of pg/m^3 and typically for polybrominated dibenzofurans (PBDFs)<1 ng/m^3. Even if the amount of bromine increases at co-combustion, the amount is still much lower than the amount of chlorine. At the incineration there is a total combustion of organic compounds in the waste. When cooling of the exhaust emissions a certain *de-novo* formation of halogenated compounds occurs. However, this formation results in very low (close to or below the detection limit) amounts of brominated and mixed halogenated dioxins and furans, which has also been shown in another investigation (Öberg and Bergström, 1990). Thus, in well-functioning incinerators this seems to be an acceptable way of taking care of waste.

In case of uncontrolled fires (accidental fire) and at co-combustion at lower temperatures or not well functioning incinerators there is a risk of formation of PBDDs and PBDFs. The same has been concluded in chapters 3.7-3.9 of the Environmental Health Criteria Document on dioxins and furans (International Programme on Chemical Safety, 1998).

3.1.2.7 Natural sources

There is no information on natural occurrence of HBCDD.

3.1.2.8 Summary of release estimations

The estimations of the total regional and continental emissions used for further calculations with EUSES are summarised in Table 3-34 below.

Table 3-34 Summary of releases

	Total (kg/year)			Continental (kg/year)			Regional (kg/year)		
Life-cycle stage	**Air**	**Waste-water**	**Surface-water**	**Air**	**Waste-water**	**Surface-water**	**Air**	**Waste-water**	**Surface-water**
Production	2.0	0.73	0	0	0	0	2.0	0.73	0
Micronising	0.3	0	0	0	0	0	0.3	0	0
Formulation EPS and HIPS	19.5	48	212	19.1	48	99	0.4	0	113.4
Formulation XPS	11.3	71.2	8.5	5.7	35.6	4.3	5.7	35.6	4.3
Formulation polymer dispersion (for textiles)	6.8	220	55	4.5	146	37	2.3	74	18
Industrial use EPS	102	82	20.4	92	74	18	10.2	8.2	2
Industrial use HIPS	6.3	5.0	1.3	5.7	4.5	1.2	0.63	0.5	0.13
Industrial use XPS (compound)	100	27	7	80	21.6	5.6	20	5.4	1.4
Industrial use XPS (powder)	23.6	26.4	6.6	21.5	9.5	2.4	2.1	16.9	4.2
Industrial use textile (backcoating)	0.64	5653	1413	0.32	2826	706	0.32	2826	706
Professional use insulation boards (at building sites)	182	0	182	164	0	164	18	0	18
Service Life Textiles (washing)	0	10.5	0	0	7.9	0	0	2.6	0
Service Life Textiles (wear)	0	107	27	0	80	20	0	27	7
Service Life EPS&XPS	54	0	0	48.6	0	0	5.4	0	0
Total emissions	508	6251	1933	441	3253	1058	67.4	2997	874
kg/day*	1.39	17.1	5.29	1.21	8.9	2.89	0.18	8.21	2.39

*These emissions are used in the EUSES model for the estimation of the regional and continental background

3.1.3 Environmental fate

3.1.3.1 Degradation and transformation in the environment

Degradation in different media has been estimated using EPIWIN (Wania, 2003) see Table 3-35 below.

Table 3-35 First-order degradation half-lives in various environmental media, estimated by EPIWIN.

	Air	**Water**	**Soil**	**Sediment**
	Hours			

	Air	Water	Soil	Sediment
HBCDD	51.2	1440	1440	5760

*Data from Wania (**Wania, 2003**)

3.1.3.1.1 Abiotic degradation

Hydrolysis experiments using Firemaster 100 showed that after 39 days no bromide ion formation was detected. The detection limit of this method was 200 ppm (Wiegand, 1979). This study is not considered reliable due to its high detection limit.

Industry has requested a derogation from performing a hydrolysis study as they are at the limit of their analytical method and do not believe that they can monitor for the disappearance of parent substance or for appearance of metabolites. Hydrolysis should not be a significant route of environmental degradation for this product due to HBCDD's low water solubility. HBCDD can be predicted to adsorb to soil on the basis of its water solubility and vapour pressure. The rapporteur considers hydrolysis to be a route of degradation of minor importance. The rapporteur has the opinion that this request for derogation from testing is acceptable (Letter to ECB from KemI 1999-03-10).

Theoretically abiotic degradation of hexabromocyclododecane is possible. According to Kirk-Othmer ((1993)) HBCDD is rather easily dehydrobrominated and has a lower thermo-stability than aromatic brominated flame retardants. In practice abiotic degradation is probably of low significance because of the rather rigid ring-structure of HBCDD and its low water solubility. Anyhow, the following degradation pathways are theoretically possible for aliphatic brominated substances:

1. when exposed to sunlight and air

$$\text{H-R-Br} + h\nu \rightarrow \text{H-R}\cdot + \text{Br}\cdot \rightarrow \text{R}' + \text{HBr}$$

where R´ is an unsaturated substance

When the substance is encased in a polymeric structure this reaction is of little significance.

2. an elimination process catalysed by Lewis base in an E2-reaction or spontaneously in an E1-reaction

$$\text{H-R-Br} + \text{L} \rightarrow \text{L-H}^+ + \text{R}' + \text{Br}^- \qquad \text{(E2)}$$

$$\text{H-R-Br} \rightarrow \text{H-R}^+ + \text{Br}^- \rightarrow \text{H}^+ + \text{R}' + \text{Br}^- \qquad \text{(E1)}$$

These reactions are also considered to be of low significance considering the low water solubility of HBCDD and the low rate of migration of HBCDD from the polymer.

3. a nucleofilic substitution reaction of S_N1 or S_N2 type

$$\text{H-R-Br} \rightarrow \text{H-R}^+ + \text{Br}^- \xrightarrow{\text{Nu}^-} \text{H-R-Nu} + \text{Br}^- \qquad (S_N1)$$

$Nu^- + H\text{-}R\text{-}Br \rightarrow Nu\text{---}HR\text{---}Br^- \rightarrow Nu\text{-}R\text{-}H + Br^-$ (S_N2)

These reactions need a nucleofile and a solvent to solvatize the ion to be present.

One further factor making the above reactions of low significance is the rather rigid ring-structure of HBCDD. None of the reactions will have a transition state favoured by the ring-system.

In conclusion, a chemical degradation of HBCDD is theoretically possible. It can probably not be of quantitative importance in the environment other than in anaerobic, reducing sediments.

3.1.3.1.2 Biodegradation

STANDARD READY AND INHERENT BIODEGRADATION TESTS

The degradation of HBCDD was studied in a Closed Bottle Test carried out in accordance with OECD Guideline 301D and GLP (Schaefer and Haberlein, 1996). The test sample was a compilation of equal parts of HBCDD in current production and use from three manufacturers. There is no information on composition of the sample in the report even though it was required according to the protocol. Sodium benzoate was used as reference substance.

No biodegradation of HBCDD at 7.7 mg/l was observed over the 28-day test period. An average percent of biodegradation of the reference substance greater than 60 % (82 %) was achieved by day 7. The study is considered reliable. The results indicate that HBCDD will not be readily biodegradable under aerobic conditions.

The results from one further study on the biodegradation of HBCDD (CAS 3194-556) with complementary measurements (Anonymous, 1973) indicate that HBCDD could biodegrade to a certain extent. However, this study is very shortly described and could therefore not be satisfactorily evaluated.

AEROBIC AND ANAEROBIC TRANSFORMATION OF HEXABROMOCYCLODODECANE IN AQUATIC SEDIMENT SYSTEMS AND SOIL - SIMULATION STUDY 1

Sediment systems

A water/sediment simulation test according to OECD guideline 308 and GLP was performed to assess the degradability of technical HBCDD under aerobic and anaerobic conditions (Davis *et al.*, 2003a). The test material had the following composition: 5.8 % α-HBCDD, 19.3 % β-HBCDD and, 74.9 % γ-HBCDD. Sediment and water samples were collected from two freshwater systems in Pennsylvania, USA (Schuylkill River and Neshaminy Creek). Microcosms were prepared in 250 ml serum bottles. The amount of sediment added to the bottles ranged from 14 to 37 g dwt. Water was added so that the resulting water to sediment

ratio ranged between 1.6 and 2.9. The sealed anaerobic microcosms were pre-incubated at 23±1 °C for 43 to 44 days under anaerobic conditions. Aerobic microcosms were pre-incubated at 20±1 °C for 49 days.

After the acclimation period 5 µl of a stock solution of HBCDD dissolved in acetone (250 ng/µl) was added near the centre of the sediment layer of the microcosms leading to nominal concentrations ranging from 34 to 89 µg/kg (sediment dwt). Background microcosms, included to identify possible analytical interferences, and received 5 µl acetone only. In order to study the importance of abiotic processes biologically inhibited microcosms were included in the study. The abiotic microcosms were prepared by steam sterilisation (121 °C; 15 psi; 60 minutes).

The sealed microcosms were incubated in the dark at 20±1 °C for up to 119 days. Oxygen concentrations in the headspace of the aerobic microcosms were monitored regularly. When the oxygen concentration in the headspace of the microcosms decreased to the range of 10-15 % the microcosms were opened and the headspace exchanged with ambient air. This was done at day 79 for the Schuylkill river microcosms and at days 21, 51 and 92 for the Neshaminy creek microcosms. Conductivity and pH was also monitored during the incubation period.

Duplicate viable microcosms were sacrificed for analysis on at least seven separate sampling days including day 0. Duplicate abiotic controls were analysed on four separate sampling days including day 0. Water and sediment were separated by centrifugation, extracted with hexane, further processed and analysed for HBCDD with LC/MS. At each sampling day background microcosms were spiked with HBCDD and analysed in order to determine the recovery. The recovery was in most cases below 70 % and ranged from 33 to 125 %. The detection limit was 0.5 ng HBCDD/g. It was not possible to quantify α- and β-HBCDD. The quantification of HBCDD in the study is therefore based only on γ-HBCDD. Measured γ-HBCDD-concentrations were corrected for recovery. It is, however, unclear from the report if the HBCDD-concentrations reported are γ-HBCDD concentrations or total HBCDD concentrations recalculated from the quantification of γ-HBCDD. In Neshaminy creek control samples (not amended with HBCDD) an interfering peak was observed in LC/MS-chromatograms. The identity of this compound was not determined. γ-HBCDD concentrations in the Neshaminy creek microcosms were corrected for this background interference. In order to identify possible volatile transformation products the headspace of selected microcosms was analyzed at the termination of the study.

The results from the study are given in Table 3-36 and Table 3-37 below. In the viable **aerobic** sediments the γ-HBCDD-concentrations decreased fairly rapidly to around 7-10 % of the measured day 0 concentrations within 21 days. In the abiotic aerobic microcosms the disappearance was slower with 66 % of the measured day 0 concentration remaining after 119 days in the Schuylkill river sediment. In the abiotic Neshaminy creek sediment 54 % of the measured day 0 γ-HBCDD concentration remained after 64 days whereas after 119 days only 5 % remained. The reported disappearance half-lives for γ-HBCDD in the aerobic sediments at 20 °C were:

	Viable	Abiotic
Schuylkill River:	11 days	190 days
Neshaminy creek:	32 days	30 days
With temperature correction to 12 °C (eq. 25 TGD) this means:		
Schuylkill River:	21 days	360 days

Neshaminy creek: 61 days 57 days

No degradation products were detected neither in the headspace of the microcosms nor in the water or sediment phases.

Table 3-36 HBCDD concentration in aerobic Schuylkill river and Neshaminy creek sediments at different sampling times.

Schuylkill river *Nominal HBCDD concentration: 34 ng/g dwt*			**Neshaminy creek** *Nominal HBCDD concentration: 60 ng/g dwt*		
Day	**viable**	**abiotic**	**Day**	**viable**	**abiotic**
	HBCDD* (ng/g dwt)	**HBCDD* (ng/g dwt)**		**HBCDD* (ng/g dwt)**	**HBCDD* (ng/g dwt)**
0	31.9±2.5	31.3±0.5	0	80.7±5.8	58.3±10.0
1	11.2±3.4	-	1	42.4±9.5	-
7	31.2±0.3	-	7	19.1±9.3	-
21	3.2±1.2	29.0±2.7	21	5.7±2.9	22.0±10.7
47	1.0±0.8	-	47	-	-
64	nd	21.6±3.3	64	3.0±3.4	31.1±4.7
91	nd	-	91	0.6±0.9	-
119	3.3±4.6	20.7±0.7	119	9.2±4.1	2.7±3.8

*Not clear from the report if the concentrations given refer to total HBCDD or only γ-HBCDD.

nd: not detected. Detection limit 0.5 ng/g.

- not measured.

In the viable **anaerobic** sediments the γ-HBCDD-concentrations decreased to non-detectable levels within a week. Already at the day 0 sampling only 44 % of the nominal concentration was detected indicating a very rapid disappearance mechanism. The disappearance of γ-HBCDD was rapid also in the abiotic sediments. The measured day 0 concentration was around 40 % of the nominal concentration and at day 14 only 16 and 22 % of the nominal concentration was detected in the two sediments, respectively.

The reported disappearance half-lives for γ-HBCDD in the anaerobic sediments at 20 °C were:

	Viable	Abiotic
Schuylkill River:	1.5 days	10 days
Neshaminy creek:	1.1 days	9.9 days
With temperature correction to 12 °C (eq. 25 TGD) this means:		
Schuylkill River:	2.8 days	19 days
Neshaminy creek:	2.1 days	18.8 days

It can be concluded that the disappearance is very rapid with half-lives around 1 day or less for viable anaerobic sediments and less than 14 days and maybe even <1 day (based on the low percentage recovered from the day 0 samples) for abiotic anaerobic sediments. No

degradation products were detected neither in the headspace of the microcosms nor in the water or sediment phases.

Table 3-37 HBCDD concentration in anaerobic Schuylkill river and Neshaminy creek sediments at different sampling times.

Schuylkill river *Nominal HBCDD concentration 63 ng/g dwt*			**Neshaminy creek** *Nominal HBCDD concentration 89 ng/g dwt*		
Day	**viable**	**abiotic**	**Day**	**viable**	**abiotic**
	HBCDD* (ng/g dwt)	**HBCDD* (ng/g dwt)**		**HBCDD* (ng/g dwt)**	**HBCDD* (ng/g dwt)**
0	27.7±6.7	27.2±1.6	0	39.1±5.4	37.5±6.6
1	6.2±1.4	-	1	55.9±18.8	-
7	nd	-	7	nd	-
14	nd	14.2±0.3	14	nd	14.2±6.3
61	nd	nd	62	nd	nd
91	nd	-	91	nd	-
119	nd	nd	119	nd	1.4±2.0

*Not clear from the report if the concentrations given refer to total HBCDD or only γ-HBCDD.

nd: not detected. Detection limit 0.5 ng/g.

- not measured.

The study seems to have been performed in agreement to the referred guideline although several deviations can be noted, e.g.:

- the sediment sample sizes were smaller than recommended (14-37 g vs. the recommended minimum of 50 g),
- the volume ratios 'water/sediment' were lower than recommended (≥1.6, but guideline recommends a ratio of 3-4).
- the acclimation period were 43-49 days vs. the recommended maximum of 4 weeks, the test substance was applied into the centre of the sediment instead of to the water phase.

However, these deviations probably have little impact on the outcome of the study.

More important, the interfering peak (with characteristics identical to γ-HBCDD, at concentration of 1/3 of added HBCDD) in the unamended Neshaminy creek sediment indicates a possibility that this sediment could be contaminated with HBCDD. The authors do not discuss this and no attempt has been made to explain the interfering peak. Furthermore, the concentration of HBCDD chosen was so low that α- and β-HBCDD were barely detectable even at start of incubation, and these diastereomers could consequently not be quantified in the study. Despite its lower content in the technical product, α-HBCDD is found in higher concentrations than γ-HBCDD in, e.g., eels, seals & porpoise (c.f. section 3.2.4.2.2). This indicates that the α- diastereomer is more stable in the environment than the γ-diastereomer. A recent rat study supports this conclusion by showing a 100-fold higher

accumulation (which is dependent on the degradability) of α-HBCDD than of γ-HBCDD in rat adipose tissue (with β-HBCDD being intermediate) after 90 days exposure to technical HBCDD (c.f. section **4.1.2.6**). Also in earthworms the α- diastereomer is bioaccumulated to a higher extent than the γ- diastereomer (c.f. chapter 3.1.3.2.4). Both these diastereomer s (α and β) are also more thermally stable than the γ- diastereomer (c.f. section **1.3.3**).

In this perspective it is a serious shortcoming of the study that the fate of the α- and β- diastereomer s has not been studied.

In conclusion, the results show that γ-HBCDD very rapidly disappears from viable anaerobic sediments with half-lives <1 day. The rapid dissipation indicates that abiotic processes (chemical reaction or adsorption) are involved. This is further supported by the rapid dissipation also from the abiotic anaerobic microcosms.

Under aerobic conditions the dissipation seems to be slower and the difference in disappearance time between viable and abiotic sediments greater. This may be an indication that biotic processes are involved. However, as no degradation products (including CO_2) were identified it is not possible to firmly draw such a conclusion. It is also not possible to draw any conclusion with respect to the fate of the α-and β- diastereomers.

Soil systems

A soil simulation test according to OECD guideline 307 and GLP was performed to assess the degradability of technical -HBCDD under aerobic and anaerobic conditions (Davis *et al.*, 2003b). The test material came from the same batch as used in the sediment study (c.f. above). The soil was collected in North Dakota, USA and was characterised as a sandy loam soil with 64 % sand; 20 % silt and 16 % clay and an organic carbon content of 1.8 %.

Microcosms were prepared by adding 50 g dwt of soil to 250 ml serum bottles. The moisture content of the soil was adjusted to 20 % by weight (corresponding to a water holding capacity between 40 and 60 %). Aerobic microcosms were pre-incubated in the dark for 35 days at 20±1 °C. Anaerobic microcosms were prepared in an anaerobic atmosphere and pre-incubated in the dark at 23±1 °C for 43 days. Following the pre-incubation period, activated sludge (from the Midland Municipal Wastewater Treatment Plant) was added to the microcosms to a concentration of 5 mg/g dry soil. HBCDD was added to the soil in 5 µl of acetone leading to nominal concentration of 25 ng/g dwt

The test microcosms were then incubated for 120 days aerobically or anaerobically in the dark at 20±1 °C. Abiotic controls, achieved by steam sterilisation of soil and sludge, were run in parallel. The headspace of the viable aerobic microcosms was exchanged with laboratory air after 14, 80 and 101 days of incubation.

Duplicate viable microcosms were sacrificed for analysis on seven separate sampling days including day 0. Duplicate abiotic controls were analysed on four separate sampling days including day 0. The soil samples were treated and analysed using the same method as for the sediment test (c.f. above). The headspace of selected microcosms was analyzed for possible volatile metabolites at the termination of the study. Also in this study, it was not possible to quantify the α- and β- diastereomers. The quantification of HBCDD in the study is therefore based only on the γ- diastereomer. It is, however, unclear from the report if the HBCDD-concentrations reported are γ-HBCDD concentrations or total HBCDD concentrations recalculated from the quantification of γ-HBCDD.

The results from the study are given in Table 3-38 below. At day 0 the measured concentrations were far below the nominal test concentrations in both aerobic and anaerobic samples as well as in the abiotic controls. This may be an indication of rapid adsorption to the soil. In the viable aerobic soil γ-HBCDD slowly decreased to approximately 25 % of the day 0 concentration after 119 days of incubation. In the abiotic aerobic soil almost no decrease was noted. In the anaerobic soil the disappearance rate was similar to that of the aerobic soil during the first week with 77 % of the measured day 0 concentration remaining day 7. After that γ-HBCDD rapidly decreased to 8 % of the day 0 concentration at day 21. During abiotic conditions disappearance was significantly slower.

Although the kinetic analyses were not optimal, the reported disappearance half-lives for γ-HBCDD at 20 °C were:

	Viable	Abiotic
Aerobic soil	63 days	>>120 days
Anaerobic soil	7 days	82 days
With temperature correction to 12 °C (eq. 25 TGD) this means:		
Aerobic soil	119 days	>>227 days
Anaerobic soil	13 days	155 days

There were no degradation products detected, neither in the soil nor in the headspace of the microcosms.

Table 3-38 HBCDD concentration in aerobic and anaerobic sandy loam soil at different sampling times.

Aerobic *Nominal HBCDD concentration 25 ng/g dwt*			**Anaerobic** *Nominal HBCDD concentration 25 ng/g dwt*		
Day	**viable**	**abiotic**	**Day**	**viable**	**abiotic**
	HBCDD* (ng/g dwt)	**HBCDD* (ng/g dwt)**		**HBCDD* (ng/g dwt)**	**HBCDD* (ng/g dwt)**
0	15.9±1.3	18.0	0	11.0±0.1	17.2±2.5
1	13.0±2.1	-	1	8.0±5.1	-
7	11.5±0.3	-	7	8.5±0.1	-
21	9.5±0.3	16.3±2.5	21	0.9±1.3	19.1±3.1
48	6.6±0.1	-	56	0.5±0.6	11.8±1.3
65	5.1±1.8	16.0±0.3	91	nd	-
119	4.0±1.1	17.4±7.5	119	nd	6.9±1.9

*Not clear from the report if the concentrations given refer to total HBCDD or only γ-HBCDD.

nd: not detected. Detection limit 0.5 ng/g.

- not measured.

The study seems to have been performed in agreement to the referred guideline. However, only one soil was used, whereas the guideline recommends use of four types of soil for studies of transformation rates.

The results indicate that γ-HBCDD disappears from the studied aerobic soil with an approximate half-life of 63 days. However, as no transformation products were detected, the

mechanism of the disappearance remains unexplained. The disappearance of HBCDD may to some extent be due to adsorption to the soil as indicated by the discrepancy between nominal and measured concentrations day 0. The half-life is therefore considered less reliable. Furthermore, it is not possible to assess the representatively of this half-life since only one soil was studied. Finally, the study does not give any information about the fate the α- and β-diastereomers which together constitutes 25 % of technical HBCDD and are assumed to be more stable than γ-HBCDD.

TRANSFORMATION OF ^{14}C-HEXABROMOCYCLODODECANE IN SLUDGE, SEDIMENT AND SOIL – SIMULATION STUDY 2

Studies on the degradation of 14C-hexabromocyclododecane were performed in activated and digester sludge, freshwater sediments and soil (Davis *et al.*, 2004, 2006). Tests were performed according to GLP and in general according to OECD guidelines 302B for sludge and OECD guideline 308 for sediment and 307 for soil using a tiered approach. Initial studies with municipal waste-water activated sludge for aerobic conditions and municipal waste-water digester sludge for anaerobic conditions were performed to help in the elucidation of degradation pathways. Subsequent studies were performed in sediment and soil under both aerobic and anaerobic conditions. Products produced in the transformation were identified and a pathway has been proposed for the transformation involving solely debromination. Relative concentrations of α-, β- and γ-HBCDD were monitored during the transformation studies. The overall quality of the study is very good.

The test material was a composite sample of HBCDD from three manufacturers with identification nr WIL5850. The composition of the sample was 8.68, 6.12 and 85.19 % of α-, β- and γ HBCDD, respectively. ^{14}C-HBCDD was labelled at the minimum at the 1, 5 and 9 positions. The composition of the ^{14}C-HBCDD was 7.74, 7.84 and 81.5 % of α-, β- and γ HBCDD, respectively. Both HBCDD and ^{14}C-HBCDD were dissolved in acetone for stock solutions.

Test systems

Sludge Activated sludge and digester sludge were collected at the Midland, Michigan municipal wastewater treatment plant, USA, and were used within 48 h of collection. There is no information on the content of HBCDD in neither of the sludges, other than that this facility treats predominantly domestic sewage.

Sediment: Sediment and water samples were collected from the Schuylkill River (Valley Forge, Pennsylvania, USA) close to where the same types of sampling were performed for the former tests (Study 1).

Soil: Soil was collected from the same site in Northwood, North Dakota, USA, that was used in the former test (Study 1).

Experiment

The experimental set-ups and results are summarised in Table 3-40.

Aerobic activated sludge ^{14}C-HBCDD was added to activated sludge and mineral medium in sealed 1-liter glass duplicate vessels equipped with CO_2-traps. The nominal concentration of ^{14}C-HBCDD added was 3.6 mg/l. Adding mercuric chloride prior to the addition of HBCDD made biologically inhibited controls. Biological activity in the sludge was verified by following the mineralization of ^{14}C-benzoate in parallel vessels. The toxicity of HBCDD and or acetone to the microorganisms in the sludge was evaluated by the addition of both ^{14}C-benzoate and HBCDD. The oxygen content was maintained at 12 % by introducing more oxygen at day 2, 4 and 8. Thereafter the headspace gases of the reaction mixtures were allowed to exchange approximately weekly. The vessels were incubated with continuous mixing at 22±3 °C for 56 days. $^{14}CO_2$ was measured weekly and ^{14}C-HBCDD and other ^{14}C-products were determined on days 0, 17, 14, 28, and 56. The sludge samples were extracted overnight with acetonitrile, and then filtered through a nylon membrane and the filtrate was measured by LSC and HPLC-RAM. At the end of the study phosphoric acid was added to the reaction mixtures and allowed to mix for three days to convert carbonate to carbon dioxide that was collected in traps.

Anaerobic digester sludge The sludge was filtered to remove large particles. Care was taken to handle the sludge with anaerobe-techniques. The sludge was diluted with mineral medium to a sludge concentration of 2130 mg suspended solids/l. This sludge was distributed to 60-ml serum bottles. The nominal concentration of ^{14}C-HBCDD added was 4.2 mg/l. Test mixtures, benzoate control mixtures and toxicity control mixtures were set up as outlined for the activated sludge. Biologically inhibited controls were prepared by autoclaving (30 min., 121 °C and 15 psi). In addition inoculums controls and carrier solvent (acetone) controls were set up to measure the endogenous biological activity ***i.e.*** gas production and the impact of solvent on the biological activity. The serum bottles were sealed with rubber stoppers and aluminium caps. The bottles were incubated at 35±2 °C in darkness. In all bottles the resazurin redox indicator remained colourless confirming the absence of oxygen, in addition methane concentrations were measured in selected bottles on days 34 and 60 to confirm anaerobic conditions. Bottles were sampled on days 0, 5, 7, 14, 21, 28 and 60. The headspace gases were routinely monitored for ^{14}C-products. The digester sludge samples were extracted and analysed in the same way as the activated sludge samples. The Most Probable Number Technique in fluid thioglycollate medium was used to determine total culturable microorganisms in the test and biologically inhibited sludge after one month incubation anaerobically at room temperature. No results were however presented.

Anaerobic digester sludge, supplemental study to facilitate isolation and identification of transformation products Reaction mixtures were prepared in 160 ml serum bottles. ^{14}C-HBCDD in acetone was mixed with HBCDD in the bottles and the acetone was allowed to evaporate. Approximately 100 ml anaerobic digester sludge diluted with inorganic medium, ***i.e.*** 3400 mg solid per l, were added to each of the serum bottles, which then were sealed with Teflon-coated septa. The bottles were incubated in the dark at 35 °C. The HBCDD concentrations tested were 0, 1, 50, 100 and 500 mg/l. 2.5-ml portions of the reaction mixture were removed on days 8, 14, 20, 35, 44, 69 and 93 days and analysed for ^{14}C-HBCDD and ^{14}C-products. After 106 days the remaining 0 and 500 mg HBCDD/l -mixtures were extracted overnight with acetonitrile and the degradation products formed were concentrated by solid phase extraction for product identification.

Freshwater sediment, aerobic and anaerobic Sediment samples used for aerobic microcosms were collected at the surface layer, approximately 0-5 cm depth, while samples for the anaerobic microcosms were collected at a separate site in the anoxic zone of 3 to 10 cm below the sediment-water interface. The anaerobic sediments were processed anaerobically.

Sediments were passed through a 2 mm sieve to remove stones and improve homogeneity. 28-30 g dwt sediment portions were combined with 100 ml corresponding surface water in 250 ml serum bottles and vigorously mixed. The serum bottles were sealed with Teflon-coated rubber septa. Aerobic and anaerobic serum bottles were stored for equilibration at approximately 20 °C for an equilibration period of 28 and 33 days. Headspace gases of the aerobic bottles were equilibrated with air. After the equilibration period the final reaction mixtures were prepared. Viable test mixtures, biologically inhibited control mixtures, benzoate control mixtures and toxicity control mixtures were prepared in principal as for the activated sludge. ^{14}C-HBCDD (4.3 to 4.7 mg/kg dwt) was added below the surface at multiple locations in the sediment layer. Biologically inhibited controls were prepared by autoclaving (60 min., 121 °C and 15 psi) on three separate days prior to the addition of test substance. Benzoate and toxicity controls were prepared with ^{14}C-benzoate. The microcosms were incubated at 20±2 °C. Oxygen in the headspace of the aerobic microcosms was routinely monitored at each sampling point. The oxygen concentration was maintained at approximately 12 % by injection of oxygen when necessary. Sediment microcosms were analysed on days 0, 5, 12, 21, 28, 56, 84 and 112 for the aerobic microcosms and on days 0, 5, 7, 14, 21, 28, 56, 84 and 113 for the anaerobic microcosms. Biologically inhibited controls were analysed on days 0, 5, 28, 56, 84, 112 for the aerobic and for the anaerobic microcosms on days 0, 7, 28, 56, 84, 113. The viability and toxicity controls were analysed for $^{14}CO_2$ on days 8, 13 and 28 and on days 7 and 15 for the aerobic and anaerobic microcosms, respectively. The headspace gases of selected reaction mixtures were analysed for volatile ^{14}C-products. For analysis, the water and sediment layers of the microcosms were separated by centrifugation. Each phase was extracted overnight with acetonitrile and thereafter centrifuged to separate solids from the solution, which then was filtered through nylon membrane filters and the filtrate was measured by LSC and HPLC-RAM.

Soil, aerobic The soil was sieved through a 2 mm sieve to improve homogeneity. Soil microcosms were prepared by adding 58 g portions (approximately 50 g dwt) to 250 ml serum bottles. The moisture content was adjusted to approximately 20 % by weight. The serum bottles were sealed with Teflon-coated rubber septa contained in screw-on caps. The serum bottles with soil were pre-incubated for 15 days at approximately 20 °C. Following the pre-incubation period, the headspace gases of the microcosms were exchanged with fresh air. ^{14}C-HBCDD at nominal concentration of 3.0 mg/kg dwt and ^{14}C-benzoate was added to the soil followed by mixing of soil and test substance by hand to evenly distribute the test substance. Viable test mixtures, biologically inhibited control mixtures, benzoate control mixtures and toxicity control mixtures were prepared in principal as for the activated sludge. Biologically inhibited controls were prepared by autoclaving (60 min. 121 °C and 15 psi) on three separate days prior to the addition of test substance. The microcosms were incubated in the dark at 20±2 °C. The oxygen content was maintained at 12 %. Oxygen was added to viable microcosms on days 76 and 90. Viable microcosms were analysed on days 0, 7, 14, 28, 56, 86 and 112 and the biologically inhibited controls on the same days except on day 7. The soil was extracted with acetonitrile overnight, followed by centrifugation and filtration and analysis as for sediment. The benzoate and toxicity controls were analysed for $^{14}CO_2$ on days 8, 14, 22 and 30.

Analytical methods Total radioactivity in aqueous samples was determined with a liquid scintillation counter, LSC. The distribution of radioactivity between ^{14}C-HBCDD and ^{14}C-products was determined using high performance liquid chromatography coupled to a radioactivity monitor, HPLC-RAM, where the diastereomers also were possible to separate. Methane and volatile ^{14}C-products were analysed with gas chromatography coupled to a radioactivity monitor, GC-RAM.

Product identification With a combination of HPLC-MS and GC-MS-analyses the three major ^{14}C-products were identified.

Data analysis Concentrations of ^{14}C-HBCDD and ^{14}C-products in the different reaction mixtures were reported at each sampling time as a percentage of initial radioactivities added to the test system, *i.e.* nominal concentration. Primary degradation for the α-, β- and γ-diastereomer (i) of ^{14}C-HBCDD were reported as percent based on a comparison of measured amounts of diastereomer concentrations at day t compared to day 0:

$$\text{Primary degradation}_i\,(t) = ((m_i(t_0) - m_i(t))/m_i(t_0)) \times 100$$

where $m_i(t)$ and $m_i(t_0)$ represents amount of diastereomer as determined by LSC and identified by HPLC at day 0 and day t in percent of initial radioactivity.

Results

The results together with the experimental set-ups are summarised and commented in a table at the end of Results.

The levels of HBCDD in all systems are reported as % of initial radioactivity.

In the digester sludge experiment with a series of HBCDD concentrations transformation products were identified as tetrabromocyclododecene (product I), dibromocyclododecadiene (product II) and 1,5,9-cyclododecatriene (product III). 1,5,9-cyclododecatriene has CAS-nr 4904-61-4 and 4904-62-2. 1,5,9-cyclododecatriene is the raw material for production of HBCDD. The proposed pathway for debromination is shown below.

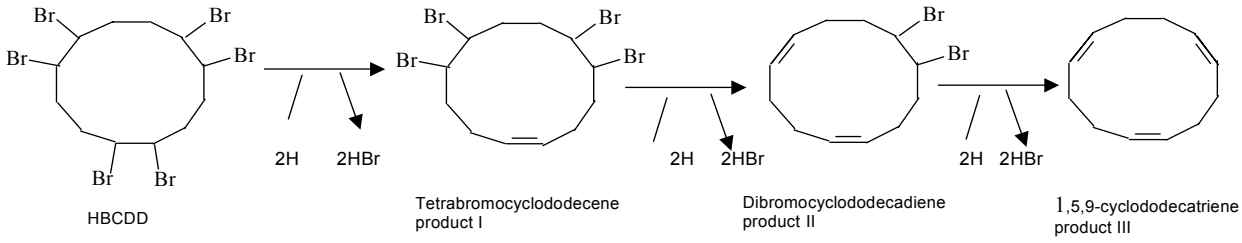


Figure 3-1 Proposed pathway for debromination of HBCDD

Degradation of ^{14}C-HBCDD was observed in the digester sludge as a decrease from 114 to 15 and 97 to 6 % initial radioactivities after 60 days in the viable and biologically inhibited systems, respectively. An explanation to the decrease in the biologically inhibited systems can be that the debromination is an abiotic process facilitated by a reduced environment. At the end of the experiment around 30 % of remaining radioactivity was recovered in the rubber stoppers. This radioactivity was mainly connected with dibromocyclododecadiene (product II) and 1,5,9-cyclododecatriene (product III) (Personal communication with Industry).

In the aerobic activated sludge there was only slight degradation in the viable systems, a decrease from 99 to 78 % of initial radioactivity after 56 days. In the biologically inhibited system the decrease was only 100 to 85 % of initial radioactivity after 56 days. In this system mercuric chloride was used for the inhibition of biological activity. The carbon-chloride bond

is stronger compared to the carbon-bromine bond and therefore chloride could have been substituted for bromine in the biologically inhibited system.

In the anaerobic freshwater sediment a decrease was observed from 96 to 37 and 112 to 75 % of initial radioactivity after 113 days in the viable and biologically inhibited systems, respectively. The recovery was 95 % or above. The results are shown in Figure 3-2. Industry chose to us the equation for primary degradation to show the relative disappearance for the diastereomers. The rapporteur preferred to use ModelMaker 4, a single first order kinetic model without log-transformation of the data.

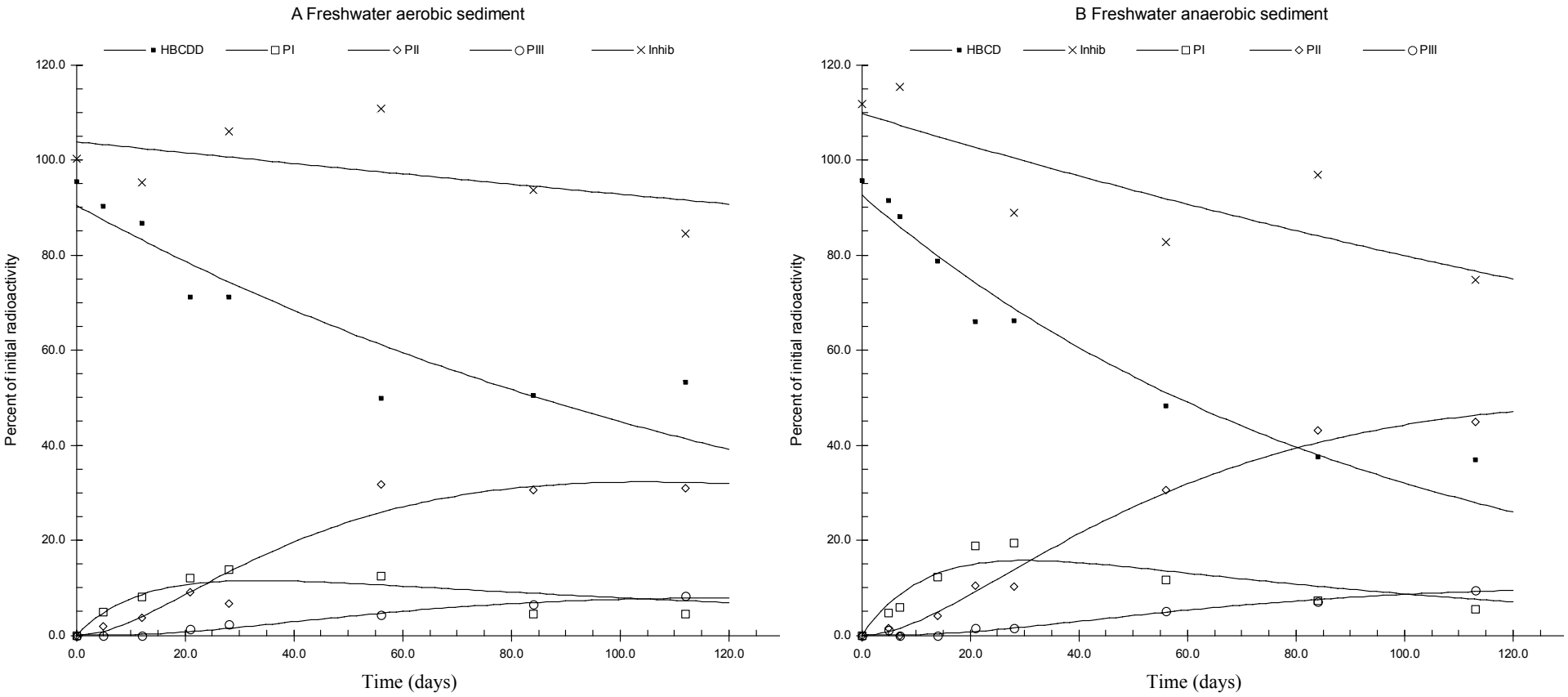


Figure 3-2 A and B. The disappearance of total HBCDD in freshwater sediment microcosms shown as percent of initial radioactivity at different times. The disappearance is shown in viable and biologically inhibited sediment as well as the appearance of products in viable sediments. A: HBCDD and products of HBCDD in aerobic microcosms; B: HBCDD and products in aerobic microcosms.

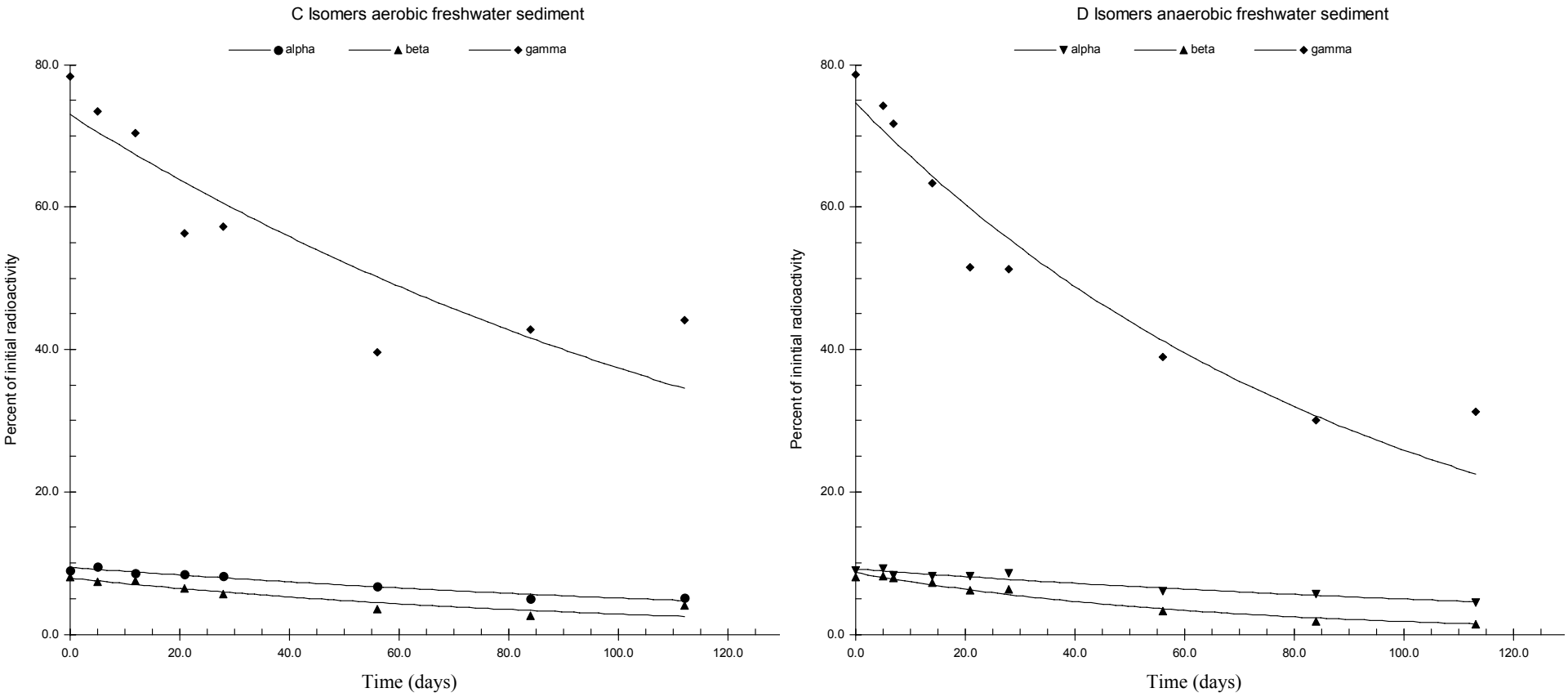


Figure 3-3 The disappearance of α-, β- and γ-HBCDD in freshwater sediment microcosms shown as percent of initial radioactivity at different times. C: α-, β- and γ -HBCDD in aerobic microcosms; D: α-, β- and γ -HBCDD in aerobic microcosms.

In the aerobic freshwater sediment a decrease was observed from 95 to 53 and 100 to 85 % of initial radioactivity after 112 days in the viable and biologically inhibited systems, respectively. The recovery was 92 % or above. The results are shown in Figure 3-2.

In soil a decrease was observed from 98 to 88 and 110 to 103 % of initial radioactivity after 112 days in the viable and biologically inhibited systems, respectively. The recovery was 89 % or above.

The rapporteur has chosen to calculate half-lives for the aerobic and anaerobic freshwater sediments for total HBCDD and in addition for the respective diastereomers. There is no indication of toxicity, why it should be acceptable to approximate DT50 from the data submitted. For the kinetic modelling the rapporteur used ModelMaker 4, a single first order kinetic model without log-transformation of the data. With this modelling also other kinetics than first-order can be calculated, however for these data the best fit was with the single first order kinetic model. The exact kinetics is difficult to determine due to factors like: the type of microorganisms in the current experimental set-ups, due to extracellular enzymes, due to interaction of HBCDD with the environment and others. The results are shown in Table 3-39. DT50s at 12 °C for aerobic and anaerobic freshwater sediment are used in the EUSES modelling. According to TGD data on half-lives for substances in anaerobic sediments are rarely available and in EUSES this rate constant is by default set to 0. In the current case the anaerobic degradation has specifically been investigated why the default value in EUSES has been changed.

Table 3-39 Calculated DT50s for HBCDD and diastereomers in aerobic and anaerobic freshwater sediments (performed by the rapporteur) to be used in the EUSES modelling.

Sediment	Test substance	DT50 (days)		Comments
		20°C*	12°C**	
Aerobic freshwater sediment	HBCDD	101	191***	Around half (44 %) of the initial amount of the substance was transformed at the end of the study. Only duplicate samples were measured and the variation was sometimes large. Taken together the exact values are uncertain. However, DT50 12 °C is considered enough certain to be used in the EUSES-modelling.
	α-HBCDD	113		Only around half of the initial amount of the diastereomers was transformed at the end of the study. Only duplicate samples were measured and the variation was sometimes large. Taken together the exact values are uncertain. DT50s can only be taken as an indication of that there are differences between the diastereomers.
	β- HBCDD	68		
	γ -HBCDD	104		
Anaerobic freshwater sediment	HBCDD	66	125***	Only slightly more than half of the initial amount of substance (59%) was transformed at the end of the study. Only duplicate samples were measured and the variation was sometimes large. Taken together the exact values are uncertain. However, DT50 12 °C is considered enough certain to be used in the EUSES-modelling.
	α- HBCDD	113		For α- and γ-HBCDD only slightly more than half of the initial amount of substance was transformed at the end of the study. Only duplicate samples were measured and the variation was sometimes large. Taken together the exact values are uncertain. DT50s can only be taken as an indication of that there are differences between the diastereomers.
	β -HBCDD	44		
	γ -HBCDD	65		

*The kinetic modelling is performed with ModelMaker 4, a single first order kinetic model without log-transformation.

**Transformed according to TGD.

***Used in the EUSES modelling.

Even though the data for the diastereomers only can be taken as an indication 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.

Table 3-40 Summary of test set-ups and results with comments.

Test set-up	Source	Test conditions		Test substance	Analysis	Results and comments		
		Specific	General			Results given in % of initial nominal radioactivity		
Aerobic activated sludge	Midland, Michigan municipal wastewater treatment plant, USA HBCDD content of sludge not measured.	Viable test	300 ml corresponding to 1060 mg mixed liquor suspended solids/l Duplicates, sub-samples taken out. 12 % oxygen maintained 22±3 °C in the dark Continuous stirring Sampling at 0, 7, 14, 28 and 56 days. End of study reaction mixtures acidified with phosphoric acid, mixing for three days to convert carbonate to CO_2 collected in caustic traps.	^{14}C-HBCDD nominal concentration 3.6 mg/l corresponding to 3430 mg/kg sludge dwt	Extraction overnight with acetonitrile, filtered through 0.45 µm filter. Filtrate assayed: LSC for radioactivity; and HPLC-RAM to measure concentrations of ^{14}C-HBCDD and ^{14}C-products GC-RAM to measure headspace gases	*^{14}C-HBCDD*	*Day 0 (%)*	*Day 56 (%)*
						Total	99.1±0.57	77.7±2.2
						α	8.8±0.3	8.2±0.5
						β	8.8±0.5	6.4±0.4
						γ	81.6±1.3	63.1±1.3
						±Product I, II and III barely detectable. Slightly decreasing recovery with time, indicating stronger binding to particles with time. The extraction efficiency increased at day 56 with two extractions.		
		Abiotic control, biologically inhibited		^{14}C-HBCDD nominal concentration 3.6 mg/l corresponding to 3430 mg/kg sludge dwt + $HgCl_2$ 250 mg/l		*^{14}C-HBCDD*	*Day 0 (%)*	*Day 56 (%)*
						Total	99.8±0.3	40.37±7.7
						α	9.5±0.9	4.5±0.9
						β	9.6±0.8	Not detected
						γ	80.7±1.5	35.8±6.8
						Larger decrease of HBCDD than in the viable test indicating transformation. However, it is possible that the $HgCl_2$ is by some way involved in this transformation. Products barely detected. Slightly decreasing recovery with time, indicating stronger binding to particles with time. The extraction efficiency increased at day 56 with two extractions.		
		Toxicity control for impact on biological activity		HBCDD 3.6 mg/l + ^{14}C-benzoate 50 mg/l	GC-RAM to measure headspace gases		*Day 4 (%)*	*Day 56 (%)*
						$^{14}CO_2$	37.9±0.8	69.8±6.1
						No indications of toxicity.		

Test set-up	Source	Test conditions		Test substance	Analysis	Results and comments		
		Specific	**General**			Results given in % of initial nominal radioactivity		
		Reference control to verify biological activity		^{14}C-benzoate 50 mg/l	GC-RAM to measure headspace gases		*Day 4 (%)*	*Day 56 (%)*
						$^{14}CO_2$	39.4±1.4	77.0±8.5
Anaerobic digester sludge	Midland, Michigan municipal wastewater treatment plant, USA HBCDD content of sludge not measured.	Viable test	30 ml corresponding to 2130 mg solids/l Duplicates were analysed 35±2 °C in the dark, under anaerobic conditions. pH 7.4 nominal Gentle mixing by hand weekly. Serum bottles sealed with rubber stoppers. Sampling at 0, 5, 7, 14, 21, 28 and 60 days.	^{14}C-HBCDD nominal concentration 4.22 mg/l corresponding to 1980 HBCDD/kg sludge dwt	Extraction overnight with acetonotrile, filtered through 0.45 µm filter. Filtrate assayed: LSC for radioactivity; and HPLC-RAM to measure concentrations of ^{14}C-HBCDD and ^{14}C-products GC-RAM to measure headspace gases Total culturable organisms determined by growth on thioglycollate medium. Serial dilutions in duplicates and incubation one month at ~22 °C. Turbidity determined.	*^{14}C-HBCDD*	*Day 0 (%)*	*Day 60 (%)*
						Total	114.2±3.4	14.5±9.1
						α	10.1±0.3	1.2±1.7
						β	9.7±0.1	<1
						γ	94.4±3.0	12.5±6.3
						Product I, II and III and trace products in measurable amounts. Decreasing recovery with time, indications of accumulation of ^{14}C in the rubber stoppers.		
							Day 34 (%)	*Day 60 (%)*
						^{14}C as methane	4.0±0.1	30.7±7.7
		Abiotic control, biologically inhibited		^{14}C-HBCDD nominal concentration 4.22 mg/l corresponding to 1980 HBCDD/kg sludge dwt + Heat sterilisation 30 min, 121 °C, 15 psi		*^{14}C-HBCDD*	*Day 0 (%)*	*Day 60 (%)*
						Total	96.5±3.1	6.3
						α	8.2±0.1	<0.6
						β	8.1±0.2	<0.6
						γ	80.2±3.2	5.4
						Product I, II and III and trace products in measurable amounts. Decreasing recovery with time, indications of accumulation of ^{14}C in the rubber stoppers.		
							Day 34 (%)	*Day 60 (%)*
						^{14}C as methane	0.4±0.0	0.3±0.0
		Toxicity control		HBCDD 3.6 mg/l	GC-RAM to measure		*Day 8 (%)*	*Day 21 (%)*

Test set-up	Source	Test conditions		Test substance	Analysis	Results and comments		
		Specific	**General**			Results given in % of initial nominal radioactivity		
		for impact on biological activity		+ ^{14}C-benzoate 50 mg/l	headspace gases	$^{14}CO_2$	38.29±0.0	47.0±0.5
						No indications of toxicity.		
		Reference control to verify biological activity		^{14}C-benzoate 50 mg/l	GC-RAM to measure headspace gases		*Day 8 (%)*	*Day 21 (%)*
						$^{14}CO_2$	21.4±2.6	48.0±0.3
		Inoculum control to determine endogenous gas production		Dilution medium	GC-RAM to measure headspace gases Pressure transducer to measure gas volume		*Day 60*	
						^{14}C as methane (%)	14.3±4.8	
						Cumulative gas volume (ml)	28.8	
		Carrier solvent control to determine effect of acetone on gas production		Dilution medium + 5 µl acetone	GC-RAM to measure headspace gases Pressure transducer to measure gas volume		*Day 60*	
						^{14}C as methane (%)	30.7±6.9	
						Cumulative gas volume (ml)	34.9	
Supplemental anaerobic digester sludge	Midland, Michigan municipal wastewater treatment plant, USA HBCDD content of sludge not measured.	Viable test with 100 ml corresponding to 3400 mg solids/l Sealing with Teflon-coated septa. Mixing by hand, several times per week. 35 °C in the dark Subsamples at 8, 14, 20, 35, 44, 69 and 93 days. After 106 days the remaining 0 and 500 mg/l mixtures extracted for product identification.		^{14}C-HBCDD 0, 1, 50, 100 and 500 mg/l	LSC for radioactivity HPLC-RAM to measure concentrations of ^{14}C-HBCDD and ^{14}C-products GC-RAM to measure headspace gases	*Product identification:* Product I = tetrabromocyclododecene Product II = dibromocyclododecadiene Product III = t,t,t-1, 5,9-cyclododecatriene with CAS-no. 676-22-2		
Aerobic freshwater sediment	Schuylkill River, Valley Forge, Pennsylvania,	Viable test Sampling at 0, 5, 12, 21, 28, 56, 84	0-5 cm depth (3.7 % organic carbon, 59 % sand, 28 % silt, 13 % clay; cation exchange	^{14}C-HBCDD nominal concentration 4.67 mg/kg.	Water and sediment layers separated by centrifugation. Each phase extracted	*^{14}C-HBCDD*	*Day 0 (%)*	*Day 112 (%)*
						Total	95.4±4.6	53.3±9.7
						α	9.0±1.0	5.1±0.6

Test set-up	Source	Test conditions		Test substance	Analysis	Results and comments		
		Specific	**General**			Results given in % of initial nominal radioactivity		
	USA HBCDD content of sediment not measured.	and 112 days. Most often duplicate samples.	capacity 12.3 meq/100g; microbial biomass 175.5 µg/g), sieved through a 2 mm sieve to remove stones. Moisture content 43.9 % wwt 28 g dwt mixed with 100 ml surface water. Serum bottles sealed with Teflon-coated rubber septa. Equilibration period at 20 °C for 28 days in the dark. Test substance added below the surface at multiple locations. Aerobic conditions maintained. 20±2 °C in the dark.		overnight with acetonitrile, centrifuged and filtered through 0.45 µm filter. Filtrate assayed: LSC for radioactivity; and HPLC-RAM to measure concentrations of ^{14}C-HBCDD and ^{14}C-products GC-RAM to measure headspace gases	*β*	8.0±1.1	4.0±0.7
						γ	78.4±2.6	44.1±8.5
						Product I, II and III at measurable levels, trace products barely detectable. See Figure 3.2 A for levels.		
		Abiotic control, biologically inhibited Sampling at 0, 12, 28, 56, 84 and 112 days.		^{14}C-HBCDD nominal concentration 4.67 mg/kg + Heat sterilisation 60 min, 121 °C, 15 psi on three separate days.		*^{14}C-HBCDD*	*Day 0 (%)*	*Day 112 (%)*
						Total	100.2±16.5	8.4±5.4
						α	9.3±2.0	8.6±1.3
						β	9.0±1.9	6.8±0.8
						γ	81.9±12.6	69.1±3.4
						Product I, II and III and trace products barely detectable.		
		Toxicity control for impact on biological activity Analysed for $^{14}CO_2$ at 8, 13, and 28 days		HBCDD 4.67 mg/kg + ^{14}C-benzoate 13.6 mg/kg	GC-RAM to measure headspace gases		*Day 8 (%)*	*Day 28 (%)*
						$^{14}CO_2$	27.2±2.3	21.4±0.5
						No indications of toxicity.		
		Reference control to verify biological activity Analysed for $^{14}CO_2$ at 8, 13, and 28 days		^{14}C-benzoate 13.6 mg/kg	GC-RAM to measure headspace gases		*Day 8 (%)*	*Day 28 (%)*
						$^{14}CO_2$	24.3±2.3	11.7±15..2
Anaerobic freshwater sediment	Schuylkill River, Valley Forge, Pennsylvania,	Viable test Sampling at 0, 5, 7, 14, 21, 28, 56,	3-10 cm depth (3.4 % organic carbon, 61 % sand, 28 % silt, 11 % clay, cation exchange	^{14}C-HBCDD nominal concentration 4.31 mg/kg	Water and sediment layers separated by centrifugation. Each phase extracted	*^{14}C-HBCDD*	*Day 0 (%)*	*Day 113 (%)*
						Total	95.6±2.2	37.0±6.9
						α	9.0±0.6	4.4±1.8

Test set-up	Source	Test conditions		Test substance	Analysis	Results and comments		
		Specific	**General**			Results given in % of initial nominal radioactivity		
	USA HBCDD content of sediment not measured.	84, and 113 days. Most often duplicate samples.	capacity 10.6 meq/100 g; microbial biomass 197.6 µg/g) sieved through a 2 mm sieve to remove stones. Moisture content 40.3 % wwt 30 g dwt mixed with 100 ml surface water. Serum bottles sealed with Teflon-coated rubber septa. Equilibration period at 20 °C for 33 days in the dark. Test substance added below the surface at multiple locations. Anaerobic conditions maintained, methane gas production and resazurin colour (redox dye) controlled. 20±2 °C in the dark.		overnight with acetonitrile, centrifuged and filtered through 0.45 µm filter. Filtrate assayed: LSC for radioactivity; and HPLC-RAM to measure concentrations of ^{14}C-HBCDD and ^{14}C-products GC-RAM to measure headspace gases	*β*	8.0±0.9	1.4±0.6
						γ	78.6±3.8	31.2±5.0
						Product I, II and III at measurable levels. Trace products barely detectable. See Figure 3.2 B for levels.		
		Abiotic control, biologically inhibited Sampling at 0, 7, 14, 28, 56, 84 and 113 days.		^{14}C-HBCDD nominal concentration 4.31 mg/kg + Heat sterilisation 60 min, 121 °C, 15 psi on three separate days.		*^{14}C-HBCDD*	*Day 0 (%)*	*Day 113 (%)*
						Total	111.7±3.5	74.7±6.0
						α	10.3±1.9	8.9±1.2
						β	10.6±0.3	5.2±0.7
						γ	90.8±1.8	60.6±7.9
						Product I, II and III at measurable levels. Trace products barely detectable.		
		Toxicity control for impact on biological activity Analysed for $^{14}CO_2$ at 7 and 15 days		HBCDD 4.31 mg/kg + ^{14}C-benzoate 12.8 mg/kg	GC-RAM to measure headspace gases		*Day 7 (%)*	*Day 15 (%)*
						$^{14}CO_2$	28.8±2.5	37.0±2.1
						No indications of toxicity.		
		Reference control to verify biological activity Analysed for $^{14}CO_2$ at 7 and 15 days		^{14}C-benzoate 12.8 mg/kg	GC-RAM to measure headspace gases		*Day 7 (%)*	*Day 15 (%)*
						$^{14}CO_2$	32.2±0.1	34.2±1.41
Aerobic soil	Northwood, North Dakota, USA	Viable test Sampling at 0, 7, 14, 28, 56, 86	58 g soil i.e. ~50 g dwt, sieved through a 2 mm sieve to remove stones (2 % organic carbon, 64	^{14}C-HBCDD nominal concentration 3.04 mg/kg dwt	Extraction overnight with acetonitrile, centrifuged and filtered through 0.45 µm filter.	*^{14}C-HBCDD*	*Day 0 (%)*	*Day 112 (%)*
						Total	97.6±0.2	88.2±1.4
						α	8.6±0.5	8.2±0.2

Test set-up	Source	Test conditions		Test substance	Analysis	Results and comments		
		Specific	**General**			Results given in % of initial nominal radioactivity		
	HBCDD content of sludge not measured.	and 112 days.	% sand, 18 % silt, 18 % clay; cation exchange capacity 12.3 meq/100 g; microbial biomass 407.8 µg/g). Adjusted to 20 % moisture. Serum bottles sealed with Teflon-coated rubber septa. Equilibration period at 20 °C for 15 days. After addition of HBCDD mixed by hand. Aerobic conditions maintained. 20±2 °C in the dark. Oxygen added at days 76 and 90 to viable soils.		Filtrate assayed: LSC for radioactivity; and HPLC-RAM to measure concentrations of ^{14}C-HBCDD and ^{14}C-products GC-RAM to measure headspace gases	*β*	8.3±0.5	7.5±0.3
						γ	80.7±0.9	72.6±1.1
						Product I, II and III and trace products barely detectable.		
		Abiotic control, biologically inhibited Sampling at 0, 14, 28, 56, 86 and 112 days.		^{14}C-HBCDD nominal concentration 3.04 mg/kg dwt + Heat sterilisation 60 min, 121 °C, 15 psi on three separate days		*^{14}C-HBCDD*	*Day 0 (%)*	*Day 112 (%)*
						Total	109.7±4.1	102.8±0.2
						α	10.0±0.6	8.6±0.2
						β	9.5±0.2	8.7±0.1
						γ	90.2±3.3	85.5±0.2
						Product I, II and III and trace products barely detectable.		
		Toxicity control for impact on biological activity Sampling at 0, 7, 14, 28, 56, 86 and 112 days.		HBCDD 3.04 mg/kg dwt + ^{14}C-benzoate 10.6 mg/kg	GC-RAM to measure headspace gases		*Day 8 (%)*	*Day 30 (%)*
						$^{14}CO_2$	36.1±3.6	6.3
						No indications of toxicity.		
		Reference control to verify biological activity Sampling at 0, 7, 14, 28, 56, 86 and 112 days.		^{14}C-benzoate 10.6 mg/kg	GC-RAM to measure headspace gases		*Day 8 (%)*	*Day 30 (%)*
						$^{14}CO_2$	19.5±4.5	9.9

ANAEROBIC DEGRADATION IN SEWAGE SLUDGE

Degradation rate constants of six HBCDD stereoisomers, under anaerobic conditions in sewage sludge have been reported (Gerecke *et al.*, 2004). Sewage sludge was seen as a relevant temporary storage compartment or terminal sink for these compounds, since the retention time in a digester is around 15 days. Experiments were conducted by adding individual target compounds or mixtures to freshly collected digested sewage sludge from a mesophilic digester. The sewage sludge was amended with yeast and starch. Experiments, performed at 37 °C, with racemic mixtures of individual diastereoisomers showed that (+/-)-β-HBCDD and (+/-)-γ-HBCDD degraded faster than (+/-)-α-HBCDD by an estimated factor of 1.6 and 1.8, respectively. The technical HBCDD mixture degraded with an apparent pseudo first order rate constant of 0.46±0.13 d^{-1}, corresponding to a half-life of 1.6 d in the sewage sludge.

DEGRADATION OF METABOLITES

It was shown in simulation study 2 that HBCDD was degraded via stepwise dehalogenation to 1,5,9-cyclododecatriene (CDT) which did not seem to be further degraded.

In order to elucidate the degradability of CDT industry has performed four screening degradation studies according to OECD guidelines 301.

The first study performed according to OECD guideline 301 D (Hamwijk and Cremers, 2005a) was inconclusive as the test substance (c,t,t-CDT) was inadequately dosed to the test system. In this study CDT, dissolved in hexane, was applied to filter paper which after evaporation of the hexane was added to the test medium. It appeared that CDT had evaporated from the filter paper together with the hexane before it was added to to the test medium.

In the second study which also was performed according to OECD guideline 301 D (Hamwijk and Cremers, 2005b) t,t,t-CDT, was added directly to the filter paper to circumvent the problem of evaporation. Analytical measurements confirmed that the test substance was adequatley dosed. No mineralisation was observed and a recovery of 84-113 % of the nominal dose after 28 d of incubation indicated that no primary degradation had occurred. The test substance appeared to remain on the filter as a small clot and the authors speculate that the absence of degradation may have been caused by a small surface area of the substance on the filters, thus reducing the bioavailability.

In the third study performed according to OECD guideline 301 F (Davis *et al.*, 2006a) t,t,t-CDT was coated on silica gel by mixing on a roller bank for 72 h. Two concentrations were tested, 1 mg/l and 10 mg/l. The test substance was coated on silica gel at a loading of 1 mg CDT/g silica gel. The inoculum concentration was 30 mg/l which is the highest inoculum concentration allowed according to the guideline. The reaction mixtures were incubated in 160 ml glass serum bottles (with a nominal 85 ml volume headspace) for 60 days at 20 °C. Triplicate reaction mixtures were analysed for CDT on days 0, 7, 14, 21, 28, 42 and 60. The oxygen concentration in the headspace was also analysed. A parallell set of mixtures were used to measure CO_2 production by converting inorganic carbon to carbonate with 5N NaOH. In addition several controls were employed; inoculum blanks for determining background oxygen consumption, viability controls (70 mg aniline/l), toxicity controls (70 mg aniline/l + 1 or 10 mg CDT/l), abiotic controls (1 or 10 mg CDT/l, heat sterilised for 30 minutes), and water controls with CDT and mineral medium only to evaluate losses in absence of sludge.

CDT disappeared at both test concentrations after a lag phase of at least 14 days (see Table 3-41

Table 3-41 Percentage of applied CDT dose remaining after after 14, 21, 28, 42 and 60 days of incubation

	Day				
Test conc.	**14**	**21**	**28**	**42**	**60**
1mg/l	86 ± 3[a]	72 ± 14	55 ± 17	<7	na
10mg/l	93 ± 2	82 ± 6	79 ± 4	57 ± 19	38 ± 5

[a]mean ± standard deviation.

In the abiotic control and the water control no significant disappearance was observed indicating that biological processes are involved in the disappearance of CDT from the viable test systems. In the 10 mg/l CDT toxicity control there was a lag phase of 7 days before the degradation of aninline started, indicating some inhibition of the sludge.This may be the explanation to the slower disappearance rate of CDT at 10 mg/l compared to 1 mg/l.

There was no difference in CO_2 production between blank controls (inoculum only) and the reaction mixtures containing 1 mg/l CD. At 10 mg/l CDT the CO_2 production was slightly higher in the reaction mixtures compared to the blank control day 60. If the difference was due to mineralisation only it would represent approx. 6 % mineralisation. However, due to the high variation in respiration no firm conclusion regarding mineralisation can be drawn.

To conclude, the study confirms that CDT is not ready biodegradable. The study indicate however, that CDT is primary degradable but there are no convincing evidence that the substance is mineralised.

In a fourth study (Davis, 2006b) using essentially the same test set up as in study 3 the degradation of ^{14}C-labelled CDT was studied. CDT was ^{14}C-labelled at 6 carbon atoms and the test was run at two concentrations 1 and 0.2 mg CDT/l. The test bottles were incubated on rotary shakers at 20 °C for up to 77 days. Duplicate reaction mixtures were analysed for CDT on days 0, 7, 14, 21, 28, 35, 49, 63 and 77. Duplicate samples from a separate set of bottles were analysed for $^{14}CO_2$ at the same time intervals.

CDT decreased steadily at both test concentrations with no obvious lag phase (Table 3-42). At 0.2 mg CDT/l approximately 40 % remained after 35 days of incubation and 8 % after 63 days of incubation. At 1 mg CDT/l the disappearance was somewhat slower with over 70 % remaining after 35 days of incubation and 44 % remaining after 63 days of incubation. After 77 days of incubation the CDT concentrations were below the detection limit.

The HPLC analysis revealed a peak with shorter retention time than CDT. This peak was, likely a transient intermediate produced from the degradation of t,t,t-[^{14}C]CDT. For both sets of viable reaction mixtures the levels of this material continued to increase during the first month of the study and reached its maximum concentration by the study midpoint (i.e., day 35 values of ~30 and 17 % of initial radioactivity for the 0.2 and 1 mg/l reaction mixtures, respectively. With subsequent incubation time these levels decreased with the final values reaching ~18 % and non-detectable for the 0.2 and 1 mg/l reaction mixtures, respectively.

Carbon dioxide was formed at both test concentrations (Table 3-43). The rate was initially slow with 11 % CO_2 formed after 35 days of incubation at the lower test concentration. After that the formation rate seemed to increase and after 63 days of incubation nearly 50 % of the initial radioactivity had been converted to carbon dioxide. At the higher test concentration the CO_2 formation rate was slower. After 49 days approx. 18 % of the initial radioactivity had

been converted to CO_2. Similar to the lower test concentration the CO_2 formation rate increased towards the end of the study and at day 77 approximately 70 % of the initial radioactivity had been recovered as CO_2. In contrast, levels of $^{14}CO_2$ in the abiotic controls remained at ~1-2 % during this period.

Table 3-42 *t,t,t-[^{14}C]*CDT Concentrations at different sampling times **(Percent of Day 0)**

Test conc	Replicate	Day								
		0	**7**	**14**	**21**	**28**	**35**	**49**	**63**	**77**
0.2mg/l	1	96.9	84.6	75.0	67.3	19.4	46.7	nd[a]	nd	–[b]
	2	99.2	77.0	73.2	63.4	58.6	42.3	51.3	12.7	–
	Average	98.1±1.6[c]	80.8±5.4	74.1±1.3	65.3±2.8	39.0±27.7	44.5±3.1	27.7±33.5	8.3±6.1	–
1mg/l	1	95.8	86.4	86.4	80.3	86.2	68.1	67.9	65.7	nd
	2	96.2	86.2	75.2	73.6	79.0	80.0	nd	23.3	nd
	Average	96.0 0.3	86.3±0.1	80.8±8.0	77.0±4.7	82.6±5.1	74.0±8.4	34.3±47.4	44.5±29.9	nd

[a]not detected – Approximate detection limit (3 x signal/noise) was 4 % of initial radioactivity for 0.2 mg/l t,t,t-[^{14}C]CDT test mixtures and 0.8 % of initial radioactivity for 1 mg/l t,t,t-[^{14}C]CDT test mixtures.

[b]not analyzed.

[c](± 1 Standard Deviation).

Table 3-43 ^{14}CO2 Concentrations at different sampling times (Percent of Day 0)

Test conc	Replicate	Day[a]						
		15	**21**	**28**	**35**	**50**	**63**	**77**
0.2mg/l	1	3.6	5.6	9.4	11.9	42.6	56.5	–
	2	3.3	5.7	6.7	10.3	19.7	42.8	–
	Average	3.4±0.2[b]	5.6±0.1	8.1±1.8	11.1±1.2	31.2±16.1	49.7±9.6	–
1mg/l	1	3.3	4.6	8.9	–	23.5	24.1	70.3
	2	5.7	6.3	7.4	–	11.7	39.1	66.1
	Average	4.5±1.7	5.5±1.2	8.1±1.0	–	17.6±8.3	31.6±10.6	68.2±3.0

[a]No CO_2 was detected day 7.

[b](± 1 Standard Deviation).

To conclude, similar to the study using unlabelled CDT this study also shows that CDT is not ready biodegradable. It also indicates that CDT is biodegraded to carbon dioxide via at least one intermediate degradation product.

3.1.3.1.3 Summary of biodegradation

HBCDD is not ready biodegradable according to the results from a Closed Bottle Test where no biodegradation was observed during 28-days at a test concentration of 7.7 mg HBCDD/l.

A simulation biodegradation study (simulation study 1) was performed in 2003 (Davis *et al.*, 2003a), indicating rather fast disappearance of HBCDD from soil and sediment, especially at

anaerobic conditions. However, many questions were raised regarding the results, *e.g.*, what the disappearance represented since no degradation products were found. It was noted that the recovery varied a lot (33-125 %), even in un-aged samples, indicating problems with the extraction method In addition, only the γ diastereomer was studied, raising concerns about the biodegradability of the α- and β-diastereomers. Taken together, the half-life values that can be calculated from this study may overestimate the degradability of HBCDD, and these figures are therefore considered less reliable.

A second study (simulation study 2) has been performed with the aim to identify potential metabolites by means of using ^{14}C-labelled HBCDD. In this study the methods for extraction and analyses were optimised resulting in improved recoveries (generally >90 %). Due to the use of ^{14}C-labelled HBCDD it was possible to establish mass balances. By using higher concentrations of HBCDD, the disappearance of the α- and β-HBCDD could also be followed. The concentration of HBCDD used in simulation study 2 was up to100-fold higher than in simulation study 1. The test guidelines accept higher concentrations in order to be able to identify metabolites, as long as the substance has no significant influence on the biological activity (OECD guidelines 307 and 308). In this case the concentrations of HBCDD used were found not to affect the biodegradability of the reference substance in the samples (sodium benzoate), *i.e.* there are no indications of an influence of HBCDD on the biological activity of the samples. Overall, the quality of simulation study 2 is very good.

Three general conclusions can be drawn from simulation study 2,

- the transformation that occurs is mediated via a step-wise reductive dehalogenation of HBCDD via tetrabromocyclododecene and dibromocyclododecadiene to 1,5,9-cyclododecatriene,
- there are no indications of further transformation of 1,5,9-cyclododecatriene
- α-HBCDD seems to degrade slowest of the diastereomers

The reductive dehalogenation also occurs in abiotic samples (although normally at a slower rate), and is faster in an anaerobic, reducing environment, than in aerobic environments.

In simulation study 2, there were no indications of any metabolism of HBCDD in aerobic soil. Even if metabolites could 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. As shown in simulation study 2, using a higher concentration of HBCDD, approximately half of the added HBCDD was transformed into the three dehalogenated metabolites within 4 months at 20 °C giving a temperature-corrected half-life for primary degradation of 191 days (at 12 °C) in aerobic sediments. For product identification the biodegradation of HBCDD in anaerobic digester sludge was also investigated at concentrations between 1 and 500 mg HBCDD/l, which could have given some clues as to the dose-dependent kinetics of HBCDD biodegradation. Unfortunately, no data are reported from this part of the study.

The DT_{50}-values in simulation study 1 (12 °C) were 21 and 61 d in two different sediments at test concentrations of 34 and 60 µg HBCDD/kg sediment, respectively. At an approx. 100 fold higher test concentration in simulation study 2 the DT_{50}-value in sediment was 191 days at 12 °C. Despite the doubts that can be cast on the results from simulation study 1, the results from the two studies indicate that the kinetics for primary degradation of HBCDD may differ between the two studies.

The concentrations of HBCDD used in simulation study 1 (34-89 µg/kg dwt in sediment) are more representative of the levels normally found in the sediment. The median concentration in freshwater sediment based on all available data (see Table 3-70) is 1.5 µg/kg dwt and the 90th percentile is 197 µg/kg. In marine sediment the median of all available data is 4.2 µg/kg dwt with a 90th percentile of 122 µg/kg dwt. However, far higher concentrations in the range of what was used simulation study 2 (4000 µg/kg dwt) have been measured in sediments associated to point sources e.g. 260 µg/kg dwt in sediment textile industry areas in Belgium, 514 µg/kg in a Spanish river Cinca. Concentrations as high as 1500 µg/kg has been detected close to a Swedish municipal STP treating waste water from textile industries. The highest concentrations in sediment (up to 174000 µg/kg dwt) have been detected in the river Skerne close to the former production site in Aycliffe in UK. Surprisingly high concentrations of approx. 8000 µg HBCDD/kg dwt have been detected in the Norwegian Åsnefjord which receives waste water from e.g. an EPS formulator.

Measurement of HBCDD in soil are scarce and mainly attributed to point sources at which the concentrations of HBCDD are comparable to or higher than those used in simulation study 2 (see Table 3-84).

The calculated half-lives in sediment and soil from simulation study 2 are considered being realistic worst-case values. These figures have been used for the EUSES-modelling. For comparison, values of DT50 from simulation study 1 have also been used. The DT_{50} values used in the modelling are compiled in Table 3-44.

Table 3-44 Calculated DT50s for HBCDD for comparative use in the EUSES modelling.

Test system	DT50 at 12 °C	
	Simulation study 1	Simulation study 2
Anaerobic freshwater sediment	2.8 days	125
Aerobic freshwater sediment	21 days	191
Soil	119 days	- (no degradation)

In addition to the biodegradation studies with HBCDD the degradability of the main degradation product t,t,t1,5,9-cyclododecatriene (CDT) has been studied in a modified ready biodegradation test according to OECD guideline 301 F (Davis, 2006b). Up to 70% carbon dioxide was formed during 70 days of incubation. This has been interpreted by the TCNES subgroup for PBT assessment as evidence that CDT is not ready biodegradable, but does not fulfil the P criterion of the TGD.

3.1.3.2 Environmental distribution

3.1.3.2.1 Adsorption

Industry has requested derogation from performing a soil adsorption/desorption test. Due to the low water solubility of HBCDD they do not believe they can successfully perform the test

and monitor for a decrease in HBCDD as a measurement of adsorption to soil. According to OECD guideline 106 determining also the concentration in the soil should perform a mass balance. Thus, the analytical difficulties could be avoided by analysing the concentration in soil instead of only trying to analyse the decrease in solution. Nevertheless, HBCDD can be predicted to adsorb to soil based on its water solubility, log K_{ow} and vapour pressure.

A log K_{oc} value can be calculated from the log K_{ow} (5.625) by applying the QSAR equation (Part III, Chapter 4,Table 4, and TGD):

Log K_{oc} = 0.81 × log K_{ow} + 0.10 → log K_{oc} = 4.66

This also indicates a very high potential to adsorb to soils and sediment and a low potential to leach through soil.

The rapporteur has the opinion that this request for derogation from testing is acceptable (Letter to ECB from KemI 1999-03-10).

3.1.3.2.2 Volatilisation

No specific studies on the volatilisation of HBCDD have been identified. HBCDD has a low vapour pressure (6.3×10^{-5} Pa at 20 °C). The Henry's law constant for HBCDD at 20-25 °C is 0.75 Pa×m^3/mol if based on the sum of the solubilities of the individual diastereomers (66 µg/l).This indicates, that HBCDD has a low potential to evaporate from aquatic surfaces. Furthermore since HBCDD can be predicted to adsorb to suspended matter and in the aquatic environment possibly end up in sediment evaporation of HBCDD seems to be a less important route of dispersion.

The volatilisation from water can be estimated with EPIWIN. Using the CAS nr, vapour pressure and water solubility of HBCDD as indata, the half-life from a model river will be 15 hours and from a model lake 16 days.

Modelling of the potential of HBCDD to be transported long range has been performed (Wania, 2003). Four models for assessment of the long-range transport potential (LRTP) were used: TaPL3-2.10, ELPOS, Chemrange-2.0 and Globo-POP-1.1. The results suggest that HBCDD has a very low potential to reach remote areas, which is dependent on the LRTP behaviour of the atmospheric particulate matter to which they sorb. The LRTP of HBCDD is similar to those of polybrominated diphenylethers.

In the IVL model of the Stockholm area (Chapter 3.1) a high amount of HBCDD was estimated to be transported out of the Stockholm area via air and therefore the potential for long-range transport was modelled (Palm *et al.*, 2002). The estimated distance for HBCDD to be transported was 2550 km in air and 2600 km in water. This distance is short compared to well known persistent substances like hexachlorobenzene and lindane, however longer than for polybrominated diphenylethers.

The results of estimations of the possible distance that HBCDD can be transported in air (***Palm et al.,*** 2002; Wania, 2003) are shown in Table 3-45 below.

Table 3-45 LRTP for HBCDD estimated with different models.

	TaLP3	ELPOS	ChemRange	Globo-POP

	Km in air	Km in air	% of earth's circumference	ACP***$_{10}$ in %
HBCDD	760*	784*	11*	2.28*
	2550**			

*Data from Wania (**Wania, 2003)**

****(Palm** *et al.*, **2002)**

***Arctic contamination potential

Furthermore, Muir (D. Muir presentation at SETAC-meeting in Lille, France 2005) has calculated half-distances for HBCDD, PBDEs and POPs in the North Pacific based on skipjack tuna monitoring (Table 3-46. Half-distance is defined as the distance X from the source where the initial mass in the air drops to 50 % (Beyer and Matthies, 2001), (van Pul *et al.*, 1998).

Table 3-46 Calculated half-distances for HBCDD, PBDEs and POPs in the North Pacific based on skipjack tuna monitoring.

Substance	Number of measured levels	Correlation coefficient	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
BDE99	5	0.87	1400±320
BDE153	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

Data from Muir, 2005.

Even though the uncertainty for the HBCDD-results is rather high the data support the above estimations of how long possible distance that HBCDD can be transported in air.

3.1.3.2.3 Elimination in Sewage Treatment Plants

The Simple Treat model which is a part of the EUSES model is used to estimate the fate of a chemical in the STP. The following input data were used for the estimation of the fate of HBCDD in the STP:

Total rate constant for degradation in STP: 0
Vapour pressure: 8.3×10^{-8} kPa (25 ºC)
Water solubility: 0.066 mg/l (20 ºC)
Molecular weight: 641.7
log K_{ow}: 5.625

Henrys Laws constant: H = 0.75

The output data on the removal in STP from EUSES are shown in Table 3-47.

Table 3-47 Elimination of HBCDD in sewage treatment plants, calculated with the Simple Treat model in EUSES.

Removal in STP	
Adsorbed to sludge	79.2 %
Released to water recipient	20.6 %
Degraded in STP	0 %
Evaporation to air	0.2 %
Removal rate	79.4 %

The calculation indicates that the overall removal of HBCDD in a STP is approximately 80 %. The major part is expected to be adsorbed to the sludge. Approximately 21 % is expected to be released to the water recipient. Very little HBCDD is expected to evaporate. Using monitoring studies on concentrations of HBCDD in influents and effluents at STPs, removal rates above 90 % can be calculated (See section 3.1.4.2.3). These monitoring studies are not designed for calculation of the removal rate for HBCDD in the STPs and the information on the circumstances that took place at the sampling are very limited. The results from these studies are therefore not possible to use for this purpose and the output data from EUSES and the Simple treat model will be used.

3.1.3.2.4 Bioaccumulation

BCF FOR FISH

The BCF for HBCDD in fathead minnow *(Pimephales promelas*) was determined during a 32-day exposure period (Veith *et al.*, 1979). Thirty fish were exposed to a mean water concentration of 6.2 µg HBCDD/l. Five fish were removed for analysis after 2, 4, 8, 16, 24 and 32 days of exposure. The test temperature was 25±0.5 °C. The steady-state BCF was determined to 18100 (log BCF 4.26).

The study, published in a scientific journal, is of acceptable quality. However the study is made before any standardised test procedures were agreed for BCF and the experimental setup is briefly described.

A flow-through bioconcentration test with rainbow trout (*Oncorhynchus mykiss*) consisting of a 35-day uptake phase followed by a 35-day depuration phase was performed (Drottar and Krueger, 2000) according to standardised guidelines (U.S. Environmental Protection Agency Series 850 – Ecological Effects Test Guidelines OPPTS Number 850.1730, ASTM Standard E1022-84, and OECD Guideline 305). The study was conducted in compliance with GLP standards.

The experimental setup and the results are presented in summary in Table 3-48 and Table 3-49 below.

Table 3-48 Experimental set-up for testing of bioconcentration of HBCDD.

Parameter	Conditions	Comments by the performing laboratory
Duration	35 days uptake phase and 35 days depuration phase.	
Test groups	*solvent control* 0.060 ml acetone/l test substance nominal concentration *0.34 µg HBCDD/l* + 0.060 ml acetone/l test substance nominal concentration *3.4 µg HBCDD/l* + 0.060 ml acetone/l	One replicate per group.
Number of animals	85 fish per test group	
Sampling	*Water samples* (5 samples per time): Pre-test day -4 and -1 Uptake day 0 (0 and 4 h), 1, 3, 7, 14, 21, 28, 35 Depuration day 1, 3, 7, 10, 14, 21, 28, 35 *Tissue samples* (control/test - 2/4 replicates): Uptake day 0 (4 h), 1, 3, 7, 14, 21, 28, 35 Depuration day 1, 3, 7, 10, 14, 21, 28, 35	
Test substance	HBCDD composite from Great Lakes Chemicals Corporation, Eurobrom b.v. and Albemarle Corporation. Diastereomer composition: α 6.4 %; β 4.5 %; γ 79.1 % Purity 90 %	
Test solution	Primary stock 56.7 mg HBCDD/l inverted to mix. Secondary stock 5.67 mg/l prepared by proportional dilution with acetone. Stock solution injected into diluter mixing chambers with well water to desired concentration. Solvent control prepared by adding acetone to the mixing chamber.	Resultant test concentrations not adjusted for the purity of the active ingredient in test substance (90 %).
Test water delivery	Approximately 9.0 volume additions of test water per test chamber per 24 hours.	
Test chambers	106 l stainless steel aquaria with approximately 80 l test solution.	
Environmental conditions	Temperature 12±1 °C Dissolved oxygen >8.8 mg/l pH 7.9-8.3	
Test organism	Rainbow trout (*Oncorhynchus mykiss)*	Acclimatisation 49 hours prior test.
Length and weight	72 mm (range 64-86) 3.46 g wet weight (range 2.33-5.52)	Results are presented on a wet weight basis.
Feeding	No feed during the acclimation period. Daily feeding during the test period.	Sampling >4 hours after feeding.

During the uptake phase, the fish were exposed in three groups: 1) a solvent control (0.06 ml acetone/l, 2) a nominal concentration of 0.34 µg HBCDD/l, or 3) a nominal concentration of

3.4 µg HBCDD/l. At the start of the depuration phase, test article exposure to the treated groups was stopped and the rainbow trout were exposed to dilution water without HBCDD for the remainder of the test. Each test chamber consisted of 85 rainbow trout at test initiation, and one replicate was tested for each treatment and solvent control. Water samples were collected during exposure and depuration and analysed for HBCDD using liquid chromatography-mass spectrometry (LC/MS) with heated nebulizer operated in the selected ion monitoring mode. Tissue samples were also collected at selected water sample collection periods and analysed for HBCDD by LC/MS. The method for the analysis of HBCDD in water and fish tissue was as follows. Water samples were extracted with dichloromethane. Edible and inedible fish tissues were separated, weighed and sodium chloride was added to give a homogenate density of 1 g tissue per 5 ml. The samples were extracted and rinsed in several steps. After evaporation all samples were reconstituted and analysed in acetonitrile and water. The LOQ for water samples was 0.025 µg HBCDD/l. For the edible and inedible tissues the LOQ was 25.0 µg HBCDD/kg at day 0 and for all other sampling times 125 µg HBCDD/kg.

The results of these analyses were used to calculate the BCF values at steady-state, uptake rates and depuration rates in edible tissue, inedible tissue and whole fish. Inedible tissue consisted of the head, fins and viscera; the remainder was considered edible tissue. Replicate tissue measurements were averaged to report the concentration of the test substance in fish tissue. Whole fish concentrations were calculated based on the sum of the edible and inedible parts. Selected fish were also collected to determine lipid content and dry weight.

The steady-state bioconcentration factor (BCF) values were determined from the tissue concentrations at apparent stead-state divided by the average water concentration. Tissue concentrations were considered to be at apparent steady-state if three consecutive sets of tissue concentrations were not significantly different ($p > 0.05$) using analysis of variance. If apparent steady-state was not achieved, Day 35 BCF values were calculated using the average tissue concentration on uptake day 35. The kinetic bioconcentration factor (BCFK), uptake rate (K1) and depuration rate (K2) were calculated for edible, inedible, and whole fish using BIOFAC software. BIOFAC is a nonlinear parameter estimation routine that estimates rate constants from a set of sequential time-concentration data. These rate constants are then used to calculate a BCFK (BCFK = K1/K2).

Table 3-49 Results of measurements of HBCDD bioconcentration in fish tissue.

Parameter	Result	Comments by the performing laboratory
Mortalities	0	3 fish unaccounted, assumed to have escaped from test chamber.
Observations	All fish appeared normal.	
Measured HBCDD in *water*: *group 1*	*<LOQ***	Day 28 and 35 of the depuration phase HBCDD was measured (0.0461 and 0.0496 µg HBCDD/l, respectively), probably due to contamination of samples.
Measured HBCDD in *water*: *group 2*	*Uptake phase*: range <LOQ (level of quantitation ≤0.025 µg HBCDD/l) -0.231 µg HBCDD/l Average *0.18 µg HBCDD/l* representing 53 % of nominal concentration *Depuration phase*: generally *<LOQ*	Day 21 new samples were taken due to low recoveries (LOQ). Even with frequent cleaning of the test chamber to remove potentially adsorptive surfaces, the constant exposure was difficult to maintain.
Measured HBCDD in	*Uptake phase*: range 0.972-2.68 µg	

Parameter	Result	Comments by the performing laboratory
water: *group 3*	HBCDD/l Average *1.8 µg HBCDD/l* representing 53 % of nominal concentration *Depuration phase*: 5 of 16 samples contained *measurable HBCDD*	
Measured HBCDD in *fish tissue*: *group 1*	*<LOQ*	Day 21 200 µg HBCDD/kg was measured in one edible fish tissue sample. Day 14 204 µg HBCDD/kg was measured in one non-edible fish tissue sample. Both results are considered to be due to probable contamination of samples.
Measured HBCDD in *fish tissue*: *group 2*	*Edible*: 1175.5 µg HBCDD/Kg *Inedible*: 3 730.75 µg HBCDD/Kg *Whole fish**: 2 355.25 µg HBCDD/Kg	Day 21 120509 µg HBCDD/kg was measured in one edible fish tissue sample, probably due to contamination of samples. Day 35 145905 µg HBCDD/kg was measured in one non-edible fish tissue sample. Both results are considered to be due to probable contamination of samples. Steady state not reached at the time for measurements day 35.
Measured HBCDD in *fish tissue*: *group 3*	*Edible*: 8 370 µg HBCDD/kg *Inedible*: 23 158 µg HBCDD/kg *Whole fish**: 16 154 µg HBCDD/kg	Steady-state edible day 14; inedible day 21; whole fish day 21.
BCF: group 2	*Edible*: 6 531 *Inedible*: 20 726 *Whole fish**: 13 085	Day 35 BCF was calculated using the average tissue concentration since steady-state was not achieved.
BCFK: group 2	*Edible*: 14 039 *Inedible*: 30 242 *Whole fish**: 21 940	The kinetic BCFK was calculated with BIOFAC.
BCF: group 3	*Edible*: 4 650 *Inedible*: 12 866 *Whole fish**: *8 974*	Steady-state BCF (average day 14-35 edible tissue; average day 21-35 inedible tissue and whole fish).
BCFK: group 3	*Edible*: 9 826 *Inedible*: 23 303 *Whole fish**: 16 450	The kinetic BCFK was calculated with BIOFAC.

*The concentration of HBCDD in whole fish has been calculated according to the following equation: Whole fish concentration = (edible weight · edible concentration) + (inedible weight · inedible concentration)/(edible weight + inedible weight).

**LOQ = limit of quantitation.

No mortality occurred during the test and no treatment-related clinical signs of toxicity were observed.

When water concentrations were measured during the uptake phase in the low concentration group, the mean measured concentration was 0.18 µg HBCDD/l (53 % of the nominal test concentration). Concentrations of HBCDD during the depuration phase were generally <LOQ. Measured water concentrations of HBCDD in the 3.4 µg HBCDD/l treatment group ranged from 0.972 to 2.68 µg HBCDD/L. When concentrations measured during the uptake

phase were averaged, the mean measured concentration was 1.8 µg HBCDD/l (53 % of the nominal test concentration). Five of the 16 samples analyzed during the depuration phase contained measurable HBCDD. However, in both groups the variation in water concentration during the exposure phase was considerable (<32 % and <47 % at low and high concentration, respectively), actually exceeding what is acceptable according to the OECD guideline. The requirement according to the OECD guideline is: ”- the concentration of the test substance in the chambers is maintained within ±20 % of the mean of the measured values during the uptake phase.”

In the low concentration group apparent steady-state was not achieved and BCF was calculated using the average tissue concentration on uptake day 35 (BCF 13085). In the 3.4 µg/l (nominal) group, HBCDD concentrations in edible tissues appeared to reach steady-state at Day 14. HBCDD concentrations in edible tissues were statistically comparable for uptake Days 14-35 ($p > 0.05$) and BCF was determined by dividing the tissue concentrations at apparent steady-state by the average water concentration (BCF 8974). However, the concentrations were somewhat higher at two time-points during the depuration phase. This value is chosen as the BCF in the test report. Considering the high variation in concentrations, the rapporteur considers the big differences in BCF-values between the two groups (low and high concentration) and between the two methods of calculating the BCF not to be surprising. The opinion of the rapporteur is that choosing one of the BCF calculations/figures or a mean value as being the most correct one will always be questionable, even if the kinetic (BIOFAC) modelling BCF values may be the most appropriate in dealing with the variation. BIOFAC estimates of the time to reach 90 % of steady-state concentrations in edible, inedible and whole fish were 63, 65 and 64 days, respectively. During the depuration phase, HBCDD was eliminated slowly with estimates of time to reach 50 % clearance of 19, 20 and 19 days for edible tissue, inedible tissue and whole fish, respectively. For the two groups, kinetic modelling gave BCF-values of 21940 and 16450, with a mean value of 19195.

CONCLUSIONS BCF FOR FISH

The study on fathead minnow (Veith *et al.*, 1979) was performed without (before any were available) agreed standardised test procedures for BCF measurements, and the experimental setup is briefly described. The quality is nevertheless considered to be of acceptable quality. The study gave a BCF of 18100.

The study on rainbow trout (Drottar and Krueger, 2000) is a guideline study performed according to GLP, and valid. However, the calculations give rather different BCF-values for the two groups (low and high concentration) and the BCF-values also depend on the choice of method to calculate the BCF. Still, all values (8974-21940), and especially the values from the kinetic modelling (mean 19195), are similar to the one obtain by Veith et al (Veith *et al.*, 1979), and can be regarded to support that value, although the tests are performed on different species.

The BCF values for rainbow trout and fathead minnow are considered to support each other. Based on both studies an overall BCF of 18100 will be used in the risk characterisation.

BCF FOR EARTHWORM

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 study is reviewed in detail in section 3.1.3.2.4.

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 deionized 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 3-50)

Table 3-50 Concentration of HBCDD in soil and earthworm tissue after 28 day of exposure and corresponding bioaccumulation factors (BAF).

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 3-51 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 3-51 Concentration of α-, β- and γ- HBCDD in soil and earthworm tissue after 28 day of exposure, and diastereomer specific bioaccumulation factors (BAF).

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

Discussion

According to TGD the BCF for earthworm relative to pore water is calculated using the following equation:

$BCF_{earthworm} = 0.84 + 0.012\ K_{ow}/RHO_{earthworm}$ (Equation 82d, revised TGD)

$RHO_{earthworm}$ default = 1 kg_{wwt}/l

The $BCF_{earthworm}$ for HBCDD using this equation becomes: $0.84 + 0.012 \times 421697 = 5061$ l/kg_{wwt} in pore water.

This BCF factor is equivalent to a soil/worm BAF (wwt/wwt) of approx. 6 which is 2 orders of magnitude higher than the BAF for total HBCDD found in this study. This indicates that EUSES may overestimate the HBCDD concentration in earthworm by a factor of 100. However, the study is not a valid BCF study. HBCDD concentrations in earthworm were only measured at one point in time and it is therefore not known if steady state was reached and it is thus not possible to make a reliable estimation of the BCF for earthworms from this study. The calculated BCF factor of 5100 relative pore water will therefore be used in the risk assessment for the time being acknowledging this probably largely overestimates the BCF for earthworms.

3.1.3.2.5 Biomagnification

Measurements of HBCDD in the environment indicating biomagnification in different food chains are presented below. The data are presented in further detail in section 3.1.7.2.3.

FRESHWATER FOOD CHAIN

The biomagnification of α- and γ-HBCDD diastereomers in a Lake Ontario food web (invertebrates: plankton, Mysis and Diporeia; forage fish: alewife, sculpin and smelt; top predator: trout) has been studied (Tomy *et al*., (2003), Tomy *et al*., (2004a)).

Whole body concentrations of α- and γ-HBCDD were highest in the top predator lake trout samples; 0.4-3.8 µg/kg (wwt) for the α- diastereomer and 0.1-0.8 µg/kg for the γ-diastereomer. There was linear relationship between the total HBCDD concentrations (wwt) and trophic level based on δ15N suggesting that HBCDD biomagnifies in the Lake Ontario fod web. The trophic magnification factor (TMF) was 6.3, derived from the slope of total HBCDD to trophic level relationship.

This TMF was higher than for p,p'-DDE, 6.1, and for sum of PCBs, 5.7. Lipid corrected biomagnification factors (BMF) for predator/prey, were variable between feeding relationships and highest for foragefish/zooplankton where it ranged from 3.5 (Sculpin/Diporeia) to 10.8 (Smelt/Mysis) for α-HBCDD.

The extent of bioaccumulation and trophic transfer of the HBCDD diastereomers in a Lake Winnipeg food web was studied by (Law *et al.*, 2006). Six species of fish, zooplankton, mussels, sediment and water were analyzed for the HBCDD diastereomers. All three diastereomers were consistently detected in all species sampled. The highest concentrations were detected in burbot (*Lota lota*) wit a mean concentration (n=5) of 65 ng sum HBCDD/g lipid wt. The only diastereomer detected in sediments was the γ-diastereomer with a mean concentration (n=4) of 0.05 µg/kg dw. In water on the other hand, the only detected diastereomer was α-HBCDD at a mean concentration (n=3) of 11 pg/l in the dissolved phase. This is consistent with the fact that this diastereomer has the highest water solubility of the three. The trophic magnification (excluding mussels) factor was calculated to 2.3, 2.3 and 4.8 for α-, β-, and γ-HBCDD, respectively.The equation used for assigning the organisms to trophic levels was applied incorrectly in the original paper leading to wrong numeric values of the trophic levels for the different species and also to wrong TMF values. This was corrected in an erratum published 2007 (Law, 2007). The corrected TMF-values were 1.4, 1.3 and 2.2 for α-, β-, and γ-HBCDD respectively. The TMF factors obtained in this study were lower than in the Lake Ontario study and the authors speculate that this partly may be explained by the fact that the zooplankton in Lake Winnipeg had three orders of magnitude higher HBCDD concentrations than Zooplankton in Lake Ontario. This was probably a result of the higher lipid content in zooplankton from Lake Winnipeg. The lipid adjusted biomagnification factors for different predator/prey relationships varied between 0.1 to 8.2 for α-HBCDD, between 0.3 and 5 for β- HBCDD and between 0.1 and 6.3 for γ-HBCDD. It shall be noted that this study has some weaknesses which has been pointed out and evaluated in more detail in the RAR for decabomodiphenyl ether REF (e.g. the number of organisms analysed is small and the sampling of the different species has not been performed at the same time, one of the fish species seems to have been assigned to a higher trophic level by the $\delta^{15}N$ method than would be expected from their ecology and feeding habits). This casts some doubts over the TMF-factors derived and the low correlation coefficient also points to a week correlation between trophic level and HBCDD concentration. Another disadvantage of the study is that the comparison is based on concentration of HBCDD in fish muscle and not on the whole body concentrations. This is probably somewhat counterbalanced by the lipid normalisation of the HBCDD concentrations but still introduces uncertainty to the analysis.

Fjeld (2006a) reported concentrations of HBCDD in European smelt (*Osmerus eperlanus*), Vendace (*coregonus albula*), and Brown Trout (*Salmo trutta trutta*) from lake Mjösa in Norway. European smelt and Vendace are important preyfish for the trout. The concentrations detected in 2005 were 466 µg HBCDD/kg lwt (8.8 µg HBCDD/kg wwt), 374 µg HBCDD/kg lwt (10.7 µg HBCDD/kg wwt), 729 µg HBCDD/kg lwt (18 µg HBCDD/kg wwt) for the European smelt, the Vendace, and the Brown trout, respectively.

Overall these three studies together indicate that the levels of HBCDD increase with increasing trophic level in the aquatic food chain from zooplankton to predatory fish.

MARINE WATER FOOD CHAIN

de Boer *et al* (2002) measured the occurrence of HBCDD in

- a North Sea food chain, comprising of benthic invertebrates (hermit crabs, whelks, and sea stars), fish (whiting) and sea mammals (harbour porpoises and harbour seals). The concentrations in the bentic invertebrates ranged from below detection limit in hermit crab (n = 9), via 29-47 µg HBCDD/kg lwt in the common whelk (n = 3), to 47-84 in the sea star (n = 3). The common whelk was sampled near the English coast, in the German Bight and in the Skagerrak. The selected samples of sea star lived close to the sites where production of HBCDD takes place (Tees-estuary, UK and Western Scheldt, The Netherlands). No HBCDD could be detected in the muscle fillet of the whiting (n = 3), which however mainly depots it lipids in the liver. The concentrations in blubber from harbour porpoise (n = 4) ranged from 880-6275 µg HBCDD/kg lwt and in blubber from harbour seal (n = 2) from 63-2055 µg HBCDD/kg lwt. The harbour porpoises and harbour seals originated from the western Wadden Sea area but as they are migrating and inhabit a much larger area, the occurrence of HBCDD in them is difficult to deduce from something special. Even though it is difficult to calculate a biomagnification factor from these data it is clear that marine mammals contain much more HBCDD than lower levels in a food chain, which indicates a biomagnification of HBCDD.

- a Western Scheldt food chain, comprising of mysid shrimp, gudgeon and common tern eggs. The concentration in mysid shrimp was below the detection limit, in gudgeon (n = 25, pooled sample) 230 µg HBCDD/kg lwt, and in common tern eggs (n = 10) ranged from 330-7100 µg HBCDD/kg lwt (median = 870 µg HBCDD/kg lwt). Even though the number of mysid shrimp and gudgeon samples analysed are too small to give reliable estimates of biomagnification factors, the data indicate a bioaccumulation of HBCDD.

Jenssen *et al*. (2004) measured brominated flame retardants (including HBCDD) in the arctic marine food web in the Svalbard area in the North-Atlantic. The concentration of HBCDD increased with increasing trophic level, except for the polar bear which may indicate a capability of metabolising the substance for the polar bear. No HBCDD was detected in the lower pelagic zooplankton species *Calanus glacialis*, *Thysanoessa inermis*, and *Parrratemisto libellula*. The levels detected in polar cod, ringed seal, and polar bear ranged from 5-25 µg HBCDD/kg lwt, 15-35 µg HBCDD/kg lwt, and 5-15 µg HBCDD/kg lwt, respectively.

FISH-MARINE BIRD FOOD CHAIN

Lundstedt-Enkel *et al*. (2005) used a statistical resampling method to calculate biomagnifications factors for a number of substances, including HBCDD, from Herring muscle and Guillemot eggs collected from the Baltic Proper. The biomagnification factor for HBCDD for the step between herring and guillemot was 9.1 calculated for lipid weight. For comparison, in the same study, the BMF for $PBDE_{sum}$ was 5.5, for PCB_{sum} 24.6 and for DDT_{sum} 36.

FISH-MARINE MAMMAL FOOD CHAIN SCENARIOS

Generally, the highest levels of HBCDD are found in marine mammals such as seals and porpoises which are predominantly exposed to HBCDD via their food.

Available monitoring data (see section 3.1.7.2.3) have been used to create the following three food chain scenarios.

Scenario: Western Europe

Overall, the median concentration ratios between marine mammals and fish on a whole wet weight basis and a lipid weight basis are 272 and 28, respectively. These figures are based on the data on HBCDD levels in marine fish and mammals from Europe presented in section 3.2.4.2.3.

Table 3-52 Ratios between the concentrations of HBCDD detected in marine mammals and fish in Western European waters

Region	Species	n	Median concentration	Concentration ratios (marine mammals/fish)	
				wwt bwt/ wwt	lwt/lwt
Western Europe	Fish	102	0.40 µg HBCDD/ kg wwt	272	28
		100	13 µg HBCDD/ kg lwt		
	Marine mammals	225	109 µg HBCDD/ kg wwt bwt		
		225	368 µg HBCDD/ kg lwt		

Scenario: Baltic Sea and Western Scheldt

As a way of reducing the influence of potential local sources, location specific comparisons have been made for the Baltic Sea and the Western Scheldt except Terneuzen (which approximately can be considered to represent a region). The median concentration ratios between marine mammals and fish on a wet weight basis were 61 and 187, for the Baltic Sea and the Western Scheldt, respectively. The corresponding ratios on a lipid weight basis were 5.8 and 11, for the Baltic Sea and the Western Scheldt, respectively.

Table 3-53 Ratios between the concentrations of HBCDD detected in marine mammals and fish from the Baltic Sea and from NL coasts and marine waters (including BE/FR coasts from Zeegers *et al*., 2005)

Region	Species	n	Median concentration	Concentration ratios (marine mammals/fish)	
				wwt bwt/wwt	lwt/lwt

Baltic Sea	Fish	42	0.31 µg HBCDD/ kg wwt	61	5.8
		38	11.5 µg HBCDD/ kg lwt		
	Marine mammals	2 (representing 20 + 30 individuals)	19 µg HBCDD/kg wwt bwt		
		2	67 µg HBCDD/kg lwt		
WesternScheldt (approx. region)	Fish	18	1.8 µg HBCDD/ kg wwt	187	11
		16	107 µg HBCDD/ kg lwt		
	Marine mammals	19	336 µg HBCDD/ kg wwt bwt		
		19	1144 µg HBCDD/ kg lwt		

Scenario: U.K. Harbour porpoise

In addition to the scenarios presented above, a specific UK-harbour porpoise using U.K. specific HBCDD concentrations in fish (Anonymous, 2006) and marine mammals, i.e. harbour porpoise, (Law *et al*., 2006b), has also been constructed.

Since harbour porpoise have coastal habitats, such a comparison between the concentrations of HBCDD in the porpoise with the concentration of HBCDD in their prey, from approximately the same region becomes informative. In this particular case, the concentration of HBCDD in harbour porpoise by-caught and stranded in the U.K. between 2002 and 2004 will be compared with the concentration in fish sampled between 2002 and 2004 and available on the U.K. market.

According to a review by Santos & Pierce (2003), the literature on porpoise diets in the Northeast Atlantic suggests that there has been a long-term shift from predation on clupeid fish (mainly herring) to predation on sandeels and gadoid fish, possibly related to the decline in herring stock since the mid-1960. The most important prey found in stomachs of 100 porpoises stranded and by-caught on the British coast from 1989 to 1994 in terms of biomass were gadoids (whiting, haddock, Norway pout *Trisopterus esmarkii* and Pollack), while sandeels and gobies were the most frequently eaten (Martin, 1996).

Based on the information presented above, not all of the fish species included in the study by Anonymous (2006) can be considered to be of equal importance in the porpoise diet, and therefore a selection of species to include was performed. The fish species used to derive the concentration in porpoise prey will thus be those constituting the largest biomass, i.e. whiting, haddock being specifically mentioned. No data were presented for the gadoid species Norway pout and Pollack. It is however decided to also include coley, cod and hake, which are all gadoid species having been reported to be eaten by porpoise. The median concentration

derived for the harbour porpoise, the porpoise fish diet, concentrations ratios etc. are presented in Table 3-54 below.

Table 3-54 Ratios between the concentration of HBCDD detected in harbour porpoise and its diet in the U.K. (using wet weight concentration for muscle for fish and whole body weight concentration for porpoise)

Region	Species	n	Median concentration	Concentration ratios (marine mammals/fish)	
				wwt bwt/wwt	lwt/lwt
U.K.	Fish (muscle conc.)	300 (5 species; each specie pooled data of 60 individuals)	0.44 µg HBCDD/ kg wwt	1859	44
		300	63 µg HBCDD/ kg lwt		
	Harbour porpoise	34	818 µg HBCDD/ kg wwt bwt		
		34	2780 µg HBCDD/ kg lwt		

These concentration ratios 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. Using the relationship between the concentration of HBCDD in muscle and liver in the gadoid species bib and whiting presented by Janák *et al*. (2005) results in a median conversion factor of 123, and a liver concentration of 54 µg HBCDD/kg wwt in the porpoise fish diet (123×0.44 µg HBCDD/kg wwt = 54 µg HBCDD/kg wwt). According to Lall (Lall, 2005) the proportion of body components of cod are as described in Table 3-55 below.

Table 3-55 Body components of cod (Lall, 2005)

Body component of Cod	Proportion (%)
Fillet (skinned)	37.4
Head	21.8
Vertebrae	14.6
Fins	10.4
Gut	7.5
Liver	5.2
Skin	3.1

Assuming the same concentration in fillet, head, vertebrae, fins, gut and skin, i.e. 0.44 µg HBCDD/kg wwt, and 54 µg HBCDD/kg wwt in the liver results in an average porpoise fish diet concentration of about 3.2 µg HBCDD/kg wwt. Even though this whole body concentration is based on a number of assumptions, it is probably more realistic than when only the muscle concentration is used. This likely results in the most realistic concentration

ratio of HBCDD between a marine mammal and its prey. Thus, the concentration ratios between harbour porpoises and its prey from the UK based on an approximate whole weight basis for both the marine mammal harbour porpoise and its fish diet, is 254 (see Table 3-56 below).

Table 3-56. Ratios between the concentration of HBCDD detected in harbour porpoise and its diet in the U.K. (using whole body weight concentration for both fish and porpoise)

Region	Specie	n	Median concentration	Ratio (Harbour porpoise/fish)
U.K.	Fish	300	3.2 µg HBCDD/kg wwt bwt	254
	Harbour porpoise	34	818 µg HBCDD/kg wwt bwt	

DIETARY ACCUMULATION STUDY

Law *et al.* (2006a) exposed juvenile rainbow trout to three diasteromers (α, ß and γ) of HBCDD via their diet for 56 d followed by 112 d of untreated food. Bioaccumulation parameters were determined by analysis of muscle tissue samples at various points of uptake and depuration phases of the experiment. Three groups were exposed to food fortified with known concentrations of an individual diastereomer, while a fourth group was fed unfortified food. No peaks from debrominated or OH-HBCDD metabolites were found in the monitored ions of either the muscle or liver tissue extracts. The BMFs for the α-, ß- and γ-diastereoisomers were calculated to be 9.2, 4.3 and 7.2, respectively.

DISCUSSION ON BIOMAGNIFICATION

The conclusion, based on the different food chains and the dietary accumulation study presented above, is that HBCDD biomagnifies. It is however not possible to determine any definite biomagnification factors that could be used to replace the default values for BMF1 and or BMF2, even though it is clear that the former should be larger than one and the latter should be larger than 10. Therefore, the default value of 10 for BMF1 and BMF2 given in the TGD for substances with BCF >5000 is used in the assessment of secondary poisoning in the aquatic and marine compartment.

3.1.3.2.6 Summary of degradation and distribution in the environment

Data on hydrolysis, biodegradation, log K_{ow}, bioaccumulation are summarised in Table 3-57.

Table 3-57 Summary of data used on the environmental fate of HBCDD.

Method	Conditions	Results	Quality of the data	Reference
Hydrolysis		Persistent		

Method	Conditions	Results	Quality of the data	Reference
Biodegradation	Closed Bottle Test carried out according to GLP	No biodegradation of HBCDD at 7.7 mg/l was observed over the 28-day test period – not readily biodegradable	OECD Guideline 301D, Council of the European Community, guideline C.4-E, and TSCA Title 40, Part 796, Section 3200	**(Schaefer and Haberlein, 1996)**
	Simulation biodegradation	Data used for comparative purposes in the EUSES-modelling: Anaerobic freshwater sediment DT50, 12 °C, 2.6 d; Aerobic freshwater sediment DT50, 12 °C, 19 d; Soil DT50, 12 °C, 111 d	OECD guideline 308 and GLP	Simulation study 1 **(Davis *et al.*, 2003a)**
		Data used for EUSES-modelling: Anaerobic freshwater sediment DT50, 12 °C, 124 d; Aerobic freshwater sediment DT50, 12 °C, 190 d;	In general according to OECD guidelines 302B for sludge and OECD guideline 308 for sediment and 307 for soil and GLP	Simulation study 2 **(Davis *et al.*, 2004, 2006)**
Partition coefficient n-octanol/water (log K_{ow})		5.625		**(MacGregor and Nixon, 1997)**
Bioaccumulation	i) Fathead minnow, flow through conditions, 25 °C ii) Rainbow trout, flow through conditions, 12 °C iii) Earthworm, calculated for pore water	i) 18100* ii) 8974 - 21940 iii) 5100	i) Scientific publication, brief description of experimental setup ii) outlined according to agreed standardised test guidelines, GLP	i) **(Veith *et al.*, 1979)** ii) **(Drottar and Krueger, 2000)** iii) Revised TGD

Method	Conditions	Results	Quality of the data	Reference

*This value will be used in the RAR.

3.1.4 Aquatic compartment (including sediment)

The local, regional and continental PECs are calculated with EUSES version 2.0.3 in the following named EUSES.

Local PECs are determined for all sites where site-specific data have been provided from Industry. In addition, also generic local PEC:s are estimated to represent sites for which no information has been submitted.

Due to the number of sites the data has been split up in four sets. The data set GEN is used to calculate the generic local PEC:s and the three data sets denoted "SS…" are used to calculate Site-Specific PEC:s. The EUSES-modelling has been split up in eight different runs, two for each set of data. Four runs are performed with the assumption that all sites are connected to a STP (EUSES files with ending –ON) and four runs with the assumption that they are not (EUSES file with the ending OFF). An overview of the EUSES files and their content is given in Table 3-58.

Table 3-58 EUSES files for the calculation of Predicted environmental concentrations (PEC) for HBCDD

EUSES file	Content	Life cycle stage
[GEN-ON] sites connected to a municipal STP [GEN-OFF] sites, not connected to a municipal STP	**Generic local sites:** EPS, Formulation XPS, Formulation Textile backcoating, Formulation EPS, Industrial use HIPS, industrial use XPS, Industrial use Textile backcoating, Industrial use	II, III
[SS1-ON] sites connected to a municipal STP [SS1-OFF] sites, not connected to a municipal STP	XPS, Industrial use, sites 1-21	III.
[SS2-ON] sites connected to a municipal STP [SS2-OFF] sites not connected to a municipal STP	XPS, Industrial use, sites 23-27 EPS, Formulation(including 4 sites also with production of HIPS)	III II
[SS3-ON] sites, connected to a municipal STP [SS3-OFF] sites not connected to a municipal STP	Production Micronising XPS, formulation Textile, Formulation Textiles, Industrial use	I II II III I II

3.1.4.1 Calculation of predicted environmental concentrations (PEC)

Local PECs are calculated based on releases figures given in section 3.1.2.

For sites where no information is available if the site is connected to a municipal STP or not, PECs both with and without connection to a municipal STP are calculated. Generic PECs are always calculated using both options.

If there is no information to what recipient the wastewater is released, PECs for both freshwater and the marine compartments are calculated. Marine PECs are shown in section 3.2.3.

Intermittent releases are common practice in industrial use of XPS. The TGD does not give any guidance on how $PEC_{sediment}$ should be calculated for sites with intermittent releases. EUSES assumes that the substance in water instantly equilibrates with suspended material. The concentration in suspended material is used as the local $PEC_{sediment}$. However, this snapshot PEC which is a worst case figure for one day only is not relevant to compare to a long term PNEC which is based on, in the case of HBCDD, a toxicity study with a 28 days exposure period. For the purpose of this risk assessment we have chosen to also calculate a 30 day average concentration in sediment for sites with intermittent releases (i.e. $PEC_{sed}/30$). This PEC is thought to be more relevant to use in the risk characterisation.

3.1.4.1.1 Calculation of PEC_{local} for production and micronising

Both production sites release their wastewater to a river via municipal STPs according to industry data. The micronising plant does not have any releases to waste water and thus, no local PECs are calculated. The PECs are summarised in Table 3-59.

Table 3-59 Local PECs for production and micronising of HBCDD for STP, surface water and sediment,

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
			(µg HBCDD/l)			(µg HBCDD/kg dwt)
ProdA	Yes	10*	560	53	53	240000
ProdB	Yes	1000**	0.21	0.028	0.028	130

*TGD default.

The dilution factor at Site B is 43000 **(Drohmann, 2005). However, according to TGD, dilution factors greater than 1000 should not be used.

3.1.4.1.2 Calculation of PEC_{local} for formulation

Local PECs for STP, surface water and sediment for formulation of compound for EPS and HIPS, formulation of XPS compound and formulation of polymer dispersions for textiles are summarised in Table 3-60 - Table 3-62. Local PECs are not calculated for sites which do not have any releases to waste water or surface water.

FORMULATION OF COMPOUND FOR EPS AND HIPS

Table 3-60 Local PECs in STP, surface water and sediment for formulation of compound for EPS and HIPS.

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
			(µg HBCDD/l)			(µg HBCDD/kg dwt)
Site A	No	156*	-	0.033	0.033	150
Site F	No	289*	-	0.11	0.070	500
Site I	No	84*	-	0.040	0.034	180
Site B	Yes	>1000**	0.042	0.028	0.028	130
Site C	No	201*	-	0.05	0.046	230
Site D	No	150*	-	0.0430.15	0.042	200
Site E	No	39*	-	0.071	0.035	320
Site G	Yes	10***	0.90	0.11	0.098	510
Site H	No	21*	-	0.11	0.050	510
Site J	No	10***	-	1.5	1.2	6600
Site K	Yes	10***	7.0	0.68	0.57	3100
Site L	Yes	10***	3.5	0.36	0.30	1600
Site P****	No	10***	-	14	0.067	65000 (2200****)
GEN_EPS_FORM	Yes	10***	7.8	0.76	0.63	3500
	No	10***	-	3.6	3.0	16000

*Site-specific information.

**Greater dilution than 1000 according to site-specific data. However, TGD specifies a maximum dilution of 1000.

***TGD default.

**** Intermittent releases. Figure within parenthesis represents PEC sediment calculated by EUSES averaged over 30 days.

FORMULATION OF XPS COMPOUND

Table 3-61 Local PECs in STP, surface water and sediment for formulation of XPS compound

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
			(µg HBCDD/l)			(µg HBCDD/kg dwt)
MasterbG	Yes	10*	0.041	0.032	0.032	150
MasterbH	Yes	10*	0.093	0.037	0.035	170
MasterbI	Yes	10*	13	1.2	1.0	5500
GEN_XPS_FORM	Yes	10*	8.9	0.86	0.72	3900
	No		-	4.1	3.4	19000

*TGD default.

FORMULATION OF POLYMER DISPERSIONS FOR TEXTILES

The sites TexForm2, TexForm3, TexForm5 and TexFormA have no releases to wastewater according to site-specific information. Local PECs are therefore not calculated for these sites.

Table 3-62 Local PECs in STP, surface water and sediment for formulation of polymer dispersions for textiles.

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
			(µg HBCDD/l)			(µg HBCDD/kg dwt)
TexForm1*	Yes	10**	0.034	0.031	0.031	140
	No		-	0.043	0.041	200
TexForm4*	Yes	10**	0.016	0.030	0.029	130
	No		-	0.035	0.034	160
TexFormB*	Yes	10**	2.9	0.30	0.25	1400
	No		-	1.4	1.1	6100
GEN_TEX_FORM	Yes	10**	33	3.1	2.6	14000
	No		-	15	12	68000

*It is not known whether or not these sites have sewage treatment. PECs are therefore calculated using both options.
**TGD default.

3.1.4.1.3 Calculation of PEC_{local} for industrial use (processing)

Local PECs for STP, surface water and sediment, for industrial use of EPS compound at the manufacture of flame retarded EPS, industrial use of HIPS, industrial use of XPS compound at the manufacture of flame retarded XPS, industrial use of HBCDD powder for flame retarded XPS and textile back-coating are summarised in Table 3-63 - Table 3-67. Local PECs are not calculated for sites which do not have any releases to waste water or surface water.

INDUSTRIAL USE OF EPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED EPS

Table 3-63 Local PECs in STP, surface water and sediment for industrial use of EPS compound at the manufacture of flame retarded EPS.

Site	Connected to municipal STP (Yes/No)	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
		(µg HBCDD/l)			(µg HBCDD/kg dwt)
GEN_EPS_IndUse	Yes	0.18	0.044	0.041	200
	No	-	0.11	0.093	490

INDUSTRIAL USE OF HIPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED HIPS

Table 3-64 Local PECs in STP, surface water and sediment for industrial use of HIPS compound at the manufacture of flame retarded HIPS.

Site	Connected to municipal STP (Yes/No)	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
		(µg HBCDD/l)			(µg HBCDD/kg dwt)
GEN_HIPS_IndUse	Yes	0.54	0.079	0.032	360
	No	-	0.27	0.048	1200

INDUSTRIAL USE OF XPS COMPOUND FOR FLAME RETARDED XPS

Site XPS 2 has no emissions to waste water or surface water according to site-specific information. Therefore, no local PECs are estimated for the aquatic compartment for this site.

Table 3-65 Summary of calculated PECs in STP, surface water and sediment, both freshwater and marine for industrial use of compound for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
			(µg HBCDD/l)			(µg HBCDD/kg dwt)
XPS 1*	Yes	1000***	15	0.042	0.029	190
	No		-	0.098	0.031	450
XPS 3*****	Yes	108**	630	1.2	0.031	5300 (180)*****
	No		-	5.5	0.043	25000 (720)*****
XPS 11*****	Yes	10****	1000	20	0.14	92000 (3100)*****
	No		-	99	0.57	450000 (15000)****
GEN_XPS_IndUse*****	Yes	10****	4000	76	0.24	350000 (12000)*****
	No		-	370	1.0	1700000 (55000)*****

*It is not known whether or not these sites have sewage treatment. PECs are therefore calculated using both options.

**Site-specific information.

***Greater dilution than 1000 according to site-specific data. However, TGD specifies a maximum dilution of 1000.

****TGD default.

***** Intermittent releases. Figure within parenthesis represents PEC sediment calculated by EUSES averaged over 30 days.

INDUSTRIAL USE OF HBCDD POWDER FOR FLAME RETARDED XPS.

Sites XPS9, XPS16, XPS 17 and XPS 18 have no releases to wastewater according to site-specific information. Local PECs are therefore not calculated for these sites as they will be the same as the regional PECs. Sites XPS 4 and XPS 14 release their waste water to the sea. Local PECs for these sites are shown in section 3.2.3.

Table 3-66 Local PECs in STP, surface water and sediment for industrial use of HBCDD powder for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
			(µg HBCDD/l)			(µg HBCDD/kg dwt)
XPS 4#	Yes	10	1100	-	-	-
XPS 5*	Yes	1000***	290	0.084	0.028	380 (13)*****
	No		-	0.30	0.030	1400 (45)*****
XPS 6*	Yes	1000***	28	0.033	0.028	150 (5.0)*****
	No		-	0.054	0.028	240 (8.1)*****
XPS 7*	Yes	1000***	1800	0.39	0.029	1800 (58)*****
	No		-	1.8	0.033	8000 (270)*****
XPS 8*	Yes	10****	0.60	0.040	0.028	180 (6)*****

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
			(µg HBCDD/l)			(µg HBCDD/kg dwt)
	No		-	0.084	0.028	380 (13)*****
XPS 10*	Yes	14**	3000	41	0.14	190000 (6300)*****
	No		-	201	0.58	910000 (30000)*****
XPS 13*	Yes	1.6**	1.4	0.056	0.028	250 (8)*****
	No		-	0.16	0.028	740 (25)*****
XPS 14#	Yes		0.48	-	-	-
XPS 20	Yes	901**	28	0.034	0.028	150 (5)*****
XPS 21*	Yes	209**	3800	3.5	0.047	16000 (530)*****
	No		-	17	0.12	77000 (2600)*****
XPS 23	Yes	10****	0.02	0.028	0.028	130 (4)*****
XPS 24	Yes	10****	0.017	0.028	0.028	130 (4)*****
XPS 26	Yes	10****	11	0.23	0.029	1100 (35)*****
XPS 27*	Yes	10****	1300	24	0.094	110000 (3700)*****
	No		-	120	0.35	530000 (18000)*****

*It is not known whether or not these sites have sewage treatment. PECs are therefore calculated using both options.

**Site-specific information. (In the EUSES modelling for site XPS 13 dilution factors of 10 and 100 are used for the aquatic and marine compartment, respectively.)

***Greater dilution than 1000 according to site-specific data. However, TGD specifies a maximum dilution of 1000.

****TGD default.

***** Intermittent releases. Figure within parenthesis represents PEC sediment calculated by EUSES averaged over 30 days.

#These sites release their waste water to the sea.

INDUSTRIAL USE OF TEXTILE BACK-COATING AGENT.

Table 3-67 Local PECs in STP, surface water and sediment for textile back-coating

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
			(µg HBCDD/l)			(µg HBCDD/kg dwt)
Backcoat.1*	Yes	10**	3.2	0.33	0.21	1500
	No		-	1.5	0.93	6800
Backcoat.2*	Yes	10**	3.2	0.33	0.073	1500
	No		-	1.5	0.24	6700

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	PEC_{stp}	PEC_{water} during emission period	PEC_{water} Annual average	$PEC_{sediment}$
			(µg HBCDD/l)			(µg HBCDD/kg dwt)
Backcoat.3*	Yes	10**	550	52	12	230000
	No		-	250	58	1100000
Backcoat.4*	Yes	10**	0.0064	0.029	0.028	130
	No		-	0.031	0.030	140
BackcoatC*	Yes	10**	1.0	0.13	0.040	570
	No		-	0.50	0.083	2300
GEN_TEX_IndUse	Yes	10**	550	51	36	230000
	No		-	250	170	1100000

*It is not known whether or not these sites have sewage treatment. PECs are therefore calculated using both options.

**TGD default.

3.1.4.1.4 Calculation of $PEC_{regional}$ and $PEC_{continental}$ for surface water and sediment

Regional and continental PECs for surface water and sediment have been calculated with EUSES 2.03.

Table 3-68 Regional and continental PECs for surface water and sediment.

PEC	Surface water	Sediment
	(µg HBCDD/l)	(µg HBCDD/kg dwt)
Regional	0.028	81
Continental	0.0005	1.4

3.1.4.2 Measured levels in the aquatic compartment (including sediment)

3.1.4.2.1 Surface water and suspended particles

OVERVIEW

In general, measurements of HBCDD in water and suspended particles are rare. The few available in freshwater environment are associated with measurements within and/or in the vicinity (upstream & downstream) of a production facility in the U.K.

Concentrations of HBCDD measured in filtered freshwater span nearly two orders of magnitude, from approximately 0.02 µg HBCDD/l (lowest reported is 0.016 µg HBCDD/l

concentration; half detection limit of samples below the detection limit is 0.025 µg HBCDD/l) to about 1.5 µg HBCDD/l detected in a small tributary receiving surface water from a production facility estate. Concentrations in suspended solids ex water samples upstream & downstream of a production facility in the U.K. range from 0.025 µg HBCDD/l (half detection limit) to 1.3 µg HBCDD/l.

INDIVIDUAL STUDIES

HBCDD has been analysed with GC-MS in samples (filtered water samples and suspended solids) at different localities in Aycliffe, close to the river Tees, UK (Deuchar, 2002). The concentrations detected ranged almost two orders of magnitude, from approximately 0.02 µg HBCDD/l (there also exists two samples below the detection limit <0.05 µg HBCDD/l) to about 1.5 µg HBCDD/l. A new survey of the environmental concentrations of HBCDD in the River Skerne and River Tees catchments were performed in Autumn 2005 ((U.K. Environment Agency, 2006)). The samples were taken close to production site A, which ceased production of HBCDD in December 2003 and was demolished by July 2004. A total of six locations from the 2002 survey were re-sampled in the 2005 survey. The 2005 data show that the concentration of HBCDD was below the detection limit (0.4 µg HBCDD/l) in all samples, except for the sample from Howden Beck, upstream of the Aycliffe STP. The level found in 2005 of 0.88 µg HBCDD/l is around half that was found in the 2002 survey at this location (1.52 µg HBCDD/l as dissolved concentration). The sample analysed in the 2005 survey was reportedly taken from a concrete-lined channel and so it may not be representative of the concentrations throughout the Howden Beck. It should be noted that the 2005 data represent total concentrations while the 2002 results are for dissolved concentrations. However, very little suspended matter was present in the samples taken in 2005, so they may represent concentrations close to dissolved concentrations. No details of the analytical method used in the 2005 survey were given. A comparison of the levels found in water at the other sites sampled in 2005 with the levels found in 2002 is not meaningful owing to the higher detection limit for the 2005 samples.

Water from a local water supply, used as process water in a production facility was analysed for HBCDD using LC/MS and GC/MS (Anonymous, 2000b). The concentration of HBCDD was reported to be 0.16 µg HBCDD/l, using LC/MS, and below the detection limit of 0.5 µg HBCDD/l (half detection limit will be used), using GC/MS.

Environmental monitoring was performed on a major European manufacturing plant, producing bromine based flame-retardants including HBCDD (Anonymous, 2000e). Release points during the production of HBCDD were identified, however there were no direct releases of HBCDD from the site to controlled waters (i.e. rivers and streams). Samples were analysed with HPLC. Concentrations detected ranged from 4.8-16 µg HBCDD/l from a borehole water supply to site, and from about 5 to 10 µg HBCDD/l in the site mains inlet.

Peters (2003) detected 1.8 µg HBCDD/l in rainwater at Terneuzen, NL.

Table 3-69 Measured levels of HBCDD in water within the EU.

Location	Level of HBCDD	Comment	Reference
Surface water	µg HBCDD/l		
NL		2000-01-07	**(Anonymous, 2000b)**
Terneuzen		Process water, originating from the local water supply	
	0.16	LC/MS	
	0.25*	GC/MS	
UK		1999-11-30	**(Anonymous, 2000e)**
Aycliffe	16, 14, 4.81	Borehole water supply to site, normal operation	
	10, 6.61, 4.97	Site mains water inlet	
UK		2002-03-21	**(Deuchar, 2002)**
Aycliffe		Receiving water course immediately upstream of Aycliffe sewage treatment plant input. A key point to identify 'background' concentrations.	
River Skerne upstream Demons Beck.		Filtered water sample	
	0.057	Suspended solids ex water sample	
	0.087 (wt. 0.011 g)		
River Skerne at Aycliffe Village Bridge.		Receiving water course approximately 1 km downstream of Aycliffe sewage treatment plant. A key point to identify immediate impact after dilution.	
		Filtered water sample	
	0.025*	Suspended solids ex water sample	
	0.215 (wt. 0.009 g)		
Demons Beck upstream of Aycliffe STW		A small tributary which receives surface water from half of Aycliffe Industrial Estate before accepting Aycliffe sewage treatment plant. Used to identify other sources into the water course.	
		Filtered water sample	
	0.080	Suspended solids ex water sample	
	0.025* (wt. 0.004 g)		
Howden Beck upstream of Aycliffe STW		Small tributary which receives surface water from half of Aycliffe Industrial Estate before combining with Demons Beck and Aycliffe sewage treatment plant effluent. Also the receiving water course for any fugitive release to surface water from the Great Lakes site. Used to identify other sources into the water course.	
		Filtered water sample	

Location	Level of HBCDD	Comment	Reference
Surface water	µg HBCDD/l		
River Skerne at South Park, Darlington	1.52	Suspended solids ex water sample	
	1.31 (wt. 0.010 g)	Remote sites on River Skerne to the south of Darlington (approximately 17 km downstream), but upstream of Darlington sewage treatment plant to avoid confusion.	
	0.210	Filtered water sample	
		Suspended solids ex water sample	
River Tees at Victoria Bridge, Stockton	0.025* (wt. 0.009 g)	Very remote site (approximately 40 km downstream) at the most downstream non-tidal monitoring point before the Tees Barrage and estuary. Used to identify the extent of any continuing input from the catchment to the estuary.	
	0.025*	Filtered water sample	
	0.025* (wt. 0.006 g)	Suspended solids ex water sample	
River Skerne upstream Demons Beck.	0.2*	The reported concentrations represent total concentrations (however, very little suspended matter was present in the samples). For comments on the various sites, see above	(U.K. Environment Agency, 2006)
River Skerne at Aycliffe Village Bridge.	0.2*		
Demons Beck upstream of Aycliffe STW	0.2*		
Howden Beck upstream of Aycliffe STW	0.884		
River Skerne at South Park, Darlington	0.2*		
River Tees at Victoria Bridge, Stockton	0.2*		
Precipitation	µg HBCDD/l		
NL		2003-02 and four weeks duration,	**(Peters, 2003)**
Terneuzen	1.835	Deposition from rainwater in an open air sampler.	

*For concentration values below the detection limit, the concentration is assumed to be half the detection limit. In case the concentration only is presented for the individual diastereomers, the sum of the different diastereomers is used, and if the concentration for one or several of the individual diastereomers is below the detection limit, the concentration for that/those diastereomer(s) is/are assumed to be half the detection limit.

3.1.4.2.2 Sediment

OVERVIEW

Concentrations of HBCDD measured in freshwater sediments span more than five orders of magnitude, from <0.1 µg HBCDD/kg dwt in unpolluted sediments to more than 30000 µg HBCDD/kg dwt in sediments polluted by industrial activities (production of HBCDD, and textile industry using HBCDD). Concentrations measured in water systems in Belgium (Scheldt Basin), Switzerland, Spain, Ireland, Norway, Sweden, and United Kingdom are presented in Table 3-70, below. The high concentrations measured in BE, SE, and UK are associated with production/industrial use (primarily textile industry) of HBCDD. The type chemical industry associated with the high concentrations of HBCDD detected in ES is presently unknown. Values below detection limit are set at half detection limit.

Table 3-70 Concentrations of HBCDD in freshwater sediment in European water systems. Values below detection limit are set at half detection limit. The percentiles were calculated using Weighted Average at X(n+1)p.

Location	n	Concentration (µg HBCDD/kg dwt)				
		Median	Mean ± SD	90P	Min	Max
BE	19	1.4	29±69	170	0.1	260
CH	1	2.5	2.5		2.5	2.5
ES	4	45	151±246	514	0.05	514
IRL	5	16	15±13	32	1.4	32
NL	6	4.55	8.5±11	30	0.25	30
NO	40	0.71	2.8±4.7	9.0	0.09	21
SE	21	1.41	94±346	181	0.05	1591
UK*	88	14	665±3743	424	1.5	33350
Total	183	1.6	338±2690	270	0.05	33500
Total**	162	1.5	31±78	100	0.05	511

*Only one value is used for each site, i.e. the results from the 2002 survey (Deuchar, 2002) and 2005 survey (U.K. Environment Agency, 2006) are combined (using median value) when more than one measurement is available for the same location.

**Not including measurements considered affected by local point sources: BE: 170, 260; ES: 90,514; NO: 11.27, 16.82, 8.99, 21.34, 7.56, 8.76; SE: 75.2, 270, 1591; UK: 307 (median of 147, 779 and 307), 414 (median of 387 and 441), 981, 1163 (median of 25 and 2300), 1678, ,2037 (median of 1826, 2290, 2037), 2657 (median of 2410 and 2903), 33350 (median of 174400, 71910, 679, 33350 and 11310)

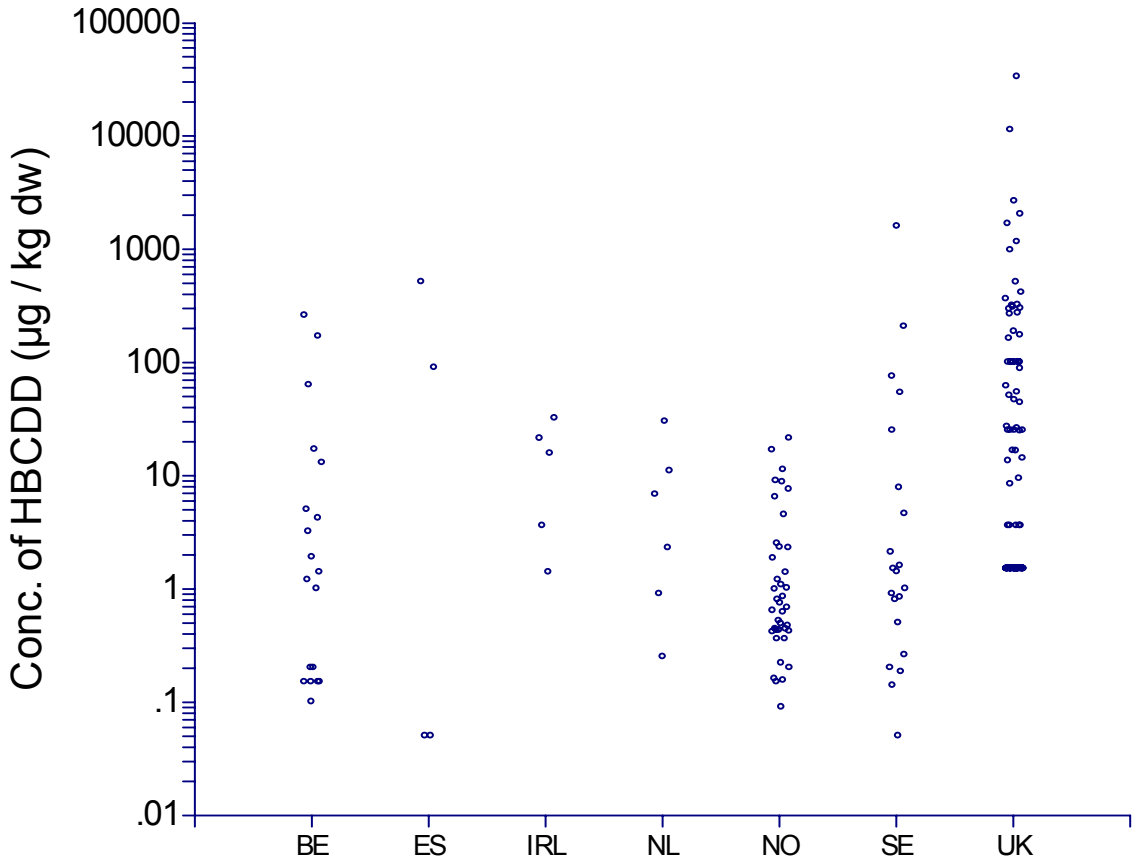


Figure 3-4 Concentrations of HBCDD in freshwater sediments in European water systems. Values below the detection limit are set at half detection limit.

INDIVIDUAL STUDIES

de Boer (de Boer *et al.*, 2002a) analysed sediments from the Scheldt basin in Belgium (close to/on the border to The Netherlands), from The Netherlands (mainly from the southern part), UK and Ireland including the North Sea. The Scheldt basin study was initiated since there had been indications of high HBCDD concentrations in biota and sediments, where many textile industries upstream are located. A substantial number of these industries may use HBCDD as a flame retardant in their products. The processes in which HBCDD is impregnated in the materials could lead to losses to the aquatic environment. In addition to that, a production plant of HBCDD is situated at Terneuzen, at the border of the Western Scheldt. Also here, HBCDD losses to the aquatic environment could take place. Indications of occurrence of HBCDD in STPs initiated the sampling of STPs in The Netherlands, UK and Ireland. Landfills were suspected to be a possible source of HBCDD contamination and therefore included in the study. A selected number of sediment samples still available after earlier studies were analysed for screening of the occurrence of HBCDD in several freshwater locations in The Netherlands and UK. Sediment samples were composed of 10 sub-samples of surface sediment.

Analysis of HBCDD were performed in a pilot study on sediment samples taken of the Elbe River, from the Czech-German border to the inner estuary, including the tributaries Schwarze Elster, Mulde and Saale (Heemken *et al.*, 2002). The samples were freshly deposed sediments, which were collected over a period of one month in January and February 2001. The concentration of HBCDD was below the limit of detection in all samples. No information on the size of the detection limit for HBCDD was provided.

In a study made in the UK the three diastereomers of HBCDD were analysed separately by LC-MS in freshwater sediments (Anonymous, 2002a). Most concentrations were below the detection limit (1.5 µg HBCDD/kg dwt was used). The highest concentration detected was 981 µg HBCDD/kg dwt, which was measured in River Weaver at Northwich.

Sediments from several locations along the Swedish River Viskan, sampled in 1995, were analysed for HBCDD (Sellström *et al.*, 1998). The samples were collected up- and down-stream from several possible point sources (textile industries and sewage treatment plants) for PBDE and HBCDD. One of the locations (no 1) was situated up-stream the point sources and another location (no 9), River Häggån, was used as reference. HBCDD was found in sediment samples from locations 2-8. A GC-MS method was used to analyse the samples in duplicate. Recovery efficiencies were checked with spiked samples and were shown to be acceptable. At locations 4, 5 and 6 concentrations were high in sediment samples, 2700, 4300 and 7000 ng/g ignition loss. Recalculation of these values from the given average ignition loss of the samples give approximate dry weight concentrations of 54, 1591 and 207 µg HBCDD/kg dwt, respectively.

Sternbeck *et al.,* (2001) measured HBCDD in samples of sediments taken from several locations of the River Viskan as it passes through the city of Borås, which is a center of textile industry. The concentration detected ranged from below detection limit (half detection limit is used, which for this sample means 0.05 µg HBCDD/kg dwt) from the most upstream sample, to 25 µg HBCDD/kg dwt, which was detected in the center of Borås. The most downstream measurement was 7.8 µg HBCDD/kg dwt.

Sediment samples from the southeast of Norway in 2003 were analysed (with LC/MS) (Schlabach *et al.*, 2004). Detectable concentrations of α-HBCDD and γ-HBCDD were found in the Drammens River.

Measurements of HBCDD has been performed in the River Skerne, UK, u/s and d/s of a production facility (Deuchar, 2002). Four, out of five samples were below the limit of detection (25 µg HBCDD/kg dwt will be used). However, the fifth value was 11310 µg HBCDD/kg dwt, and was measured at Howden Beck u/s of Aycliffe STW. A new survey of the environmental concentrations of HBCDD in sediments in the River Skerne and River Tees cathments were performed in Autumn 2005 ((U.K. Environment Agency, 2006)). The samples were taken close to production site A, which ceased production of HBCDD in December 2003 and was demolished by July 2004. Four sites were sampled in both the 2002 and 2005 survey. In the 2002 survey only the total levels were reported, whereas the separate levels of α-, β-, γ- diastereomers were reported in the 2005 survey. Higher concentrations of HBCDD were measured at the Aycliffe village bridge in River Skerne in 2005 (2300 µg HBCDD/kg dwt) compared with 2002 (25 µg HBCDD/kg dwt, which was half detection limit in that study). Four sediment samples were taken at Howden Beck, upstream of Aycliffe STP and the concentrations show a large degree of variability, which could be due to the presence of localised hotspots. Three, out of four sample concentrations (174 400, 71 910 and 33 350 µg HBCDD/kg dwt) have higher HBCDD concentrations than the single sample taken in 2002 (11310 µg HBCDD/kg dwt). The remaining samples has a lower HBCDD concentration

compared to the samples taken in 2002. The 2005 survey also determined the levels of HBCDD in a number of further ad hoc sediment samples from the River Skerne catchment. No details of the sampling locations are available, but all HBCDD concentrations were below the limit of detection. In addition to the samples from the River Skerne and River Tees catchment, the 2005 survey also analysed a number of sediments taken at locations close to textile processing sites in the U.K. over the period January to April 2005. These concentrations range from below detection limit (half detection limit = 100 µg HBCDD/kg dwt is used) to 2903 µg HBCDD/kg dwt measured in a sample from River Roach.

HBCDD has been measured in sediment in lake Ellasjøen at the arctic Bear Island, north of Norway (Christensen *et al.*, 2004). The α- and γ- diastereomers of HBCDD were detected in sediment at 1-2 cm depth, *i.e*. from the period 1973-1987. HBCDD was not found in the layers from the period 1987-2001 nor from the period 1934-1973. The β- diastereomer was not at all found.

A screening of the occurrence of HBCDD in the Norwegian environment was performed by Fjeld *et al*. (2005). Sediment samples were taken from the freshwater environment from 6 localities in the southern Norway. From each sampling station 5-8 samples were taken from the upper layer 0-2 cm.

Fjeld and co-workers (2006b) measured HBCDD in surface sediments in Lake Mjøsa in Norway. Elevated concentrations of HBCDD (8-21 µg HBCDD/kg dwt) were found outside of the town of Lillehammer and the Vingrom station, as compared to the commonly found levels (0.5-2 µg HBCDD/kg dwt). These elevated concentrations were considered to reflect that a textile factory in Lillehammer used HBCDD in their production in recent years. Only slightly elevated concentrations (2-6.5 µg HBCDD/kg dwt) were found at a few other urban sediment stations. The dated sediment core at the Vingrom station showed an evident increase in the HBCDD concentration from the late 1990s, with a maximum level in the surface layer. The other dated cores showed only a small increase in the HBCDD concentration towards the sediment surface.

Kohler *et al*. (2006) reported a historical record for HBCDD from a dated sediment core taken from Greifensee, which is a small and shallow lake on the Swiss plateau east of Zürich. The method blank concentration was 0.5 µg HBCDD/kg dwt. The concentrations reported for the different years (given in parenthesis) were about 0.5 (1974), 0.4 (1982), 1.3 (1989), 1.8 (1995), and 2.5 µg HBCDD/kg dwt (2001), with the latter constituting the surface sediment.

Eljarrat and co-workers (2004) analysed surficial sediments (0-2 cm) from four places along the Spanish River Cinca for PBDEs and HBCDD. Samples were collected up- and downstream from the heavily industrialized town Monzón with important chemical industry. HBCDD was detected only downstream of the source at concentrations 514 µg HBCDD/kg dwt at the site near the chemical industry and 90 µg HBCDD/kg dwt further downstream of the river.

Table 3-71 Levels of HBCDD in freshwater sediments within the EU, and Norway

Location	Level of HBCDD (µg/kg dwt)	Comment	Reference
Rivers, water courses			
BE:			**(de Boer *et al.*, 2002a)**

Location	Level of HBCDD (µg/kg dwt)	Comment	Reference
River Scheldt basin			
Warmebeek, Achel-Kluis	0.15*		
Moervaart, Dacknam	0.15*		
Benede Nete Duffel	1.4		
Grote Beverdijk Lo-Reninge	0.2*		
Ijzer Nieuwpoort	3.2		
Durne Lokeren	1.2		
Leie Wervik	4.2		
Leie Wevelgem	0.15*		
Leie Oeselgem	0.1*	Textile industry area	
Leie St Martens	170	Textile industry area	
Scheldt Doel	0.2*		
Scheldt Grens	63		
Scheldt Oudenaarde	260	Textile industry area	
Antwerp Kruisschansbr.	13		
Scheldt Kastel	5.0		
Scheldt Kennedyt.	1.9		
Dender Appels	1.0		
Dender Ninove	0.15*		
Vrasenedoc Beveren	17		
DE		2001-01 – 2001-02	**(Heemken *et al.*, 2002)**
River Elbe	<detection limit	No information available on the size of the detection limit	
ES		2002-10	**(Eljarrat *et al.*, 2004)**
River Cinca	0.05*	Site 1: 20 km upstream of Monzón, which is heavily industrialised town with important chemical industry	
	0.05*	Site 2: 12 km upstream of Monzón	
	514	Site 3: Just downstream of Monzón	
	90	Site 4: 30 km downstream of site 3	
IRL			**(de Boer *et al.*, 2002a)**
River Nore, KilkennyRiver Bregagh, Kilkenny	21*, 32* 3.6*, 16		
River Liffey, Dublin	1.4		
NL:			**(de Boer *et al.*, 2002a)**
River Rhine			
Waal Tiel	2.3		
Lobith	30		
Nieuwe Merwede (canal) River Meuse	11		

Location	Level of HBCDD (µg/kg dwt)	Comment	Reference
Eijsden	0.25*		
Keizersveer	6.8		
River Roer			
Vlodrop	0.9*		
NO			**(Schlabach *et al.*, 2004)**
Drammen River	1.2*, 2.51*, 0.44*, 1.39*, 0.49*, 0.64*, 0.15*		
SE		Original values given as ignition loss	**(Sellström *et al.*, 1998)**
River Viskan	75	Downstream of the city of Borås, which have several textile industries	
River Viskan	1591	Further downstream	
River Viskan	54	Upstream of STP	
River Viskan	207	Downstream of STP	
River Häggån	1.41*	Upstream textile industry	
	0.26*	Downstream of textile industry	
SE			**(Sternbeck *et al.*, 2001)**
River Viskan	0.05*	Borås (textile ind.), sed 1 (most upstream)	
	0.2	Borås (textile ind.), sed 2	
	25	Borås (textile ind.), sed 3	
	1	Borås (textile ind.), sed 4	
	4.6	Borås (textile ind.), sed 5	
	7.8	Borås (textile ind.), sed 6 (most downstream)	
UK			**(de Boer *et al.*, 2002a)**
River Tees	317, 363, 14.2*, 273, 295, 511, 62, 88, 3.6*, 3.6*		
River Tyne	17*, 322		
River Skerne	1678, 174		
River Humber	8.4*		
River Mersey	3.6*, 3.6*, 25, 3.6*, 55		
River Clyde	9.4*, 187		

Location	Level of HBCDD (µg/kg dwt)	Comment	Reference
UK			**(Anonymous, 2002a)**
Belper River, Belper	1.5*, 1.5*, 1.5*, 1.5*	Upstream of outfall, 100 m d/s, 300 m d/s, d/s	
River Weaver, Northwich	1.5*, 1.5*, 981*, 1.5*, 13.5*, 1.5*	u/s bridge, 0.5 km d/s of works, 0.6 km d/s of works, 0.8 km d/s of works, d/s railway bridge, d/s on A559	
River Hull, Beverly	1.5*, 1.5*, 1.5*, 1.5*, 1.5*, 1.5*	100m u/s canal, 600m d/s of discharge, 1.7km d/s, 2.0km d/s, d/s	
River Blackwater, Blackburn	1.5*, 1.5*, 1.5*, 1.5*	200m u/s of confluence, 100m d/s of STW, 300m d/s, Confluence , d/s 2 roach bridge, 3.0km d/s	
	44		
Langollen Canal, Ellesmere	1.5*, 1.5*, 1.5*, 1.5*, 1.5*, 1.5*	0.5km w of outfall, 0.5km w of outfall, 300m u/s above STW, 100m d/s of STW, 300m d/s of STW, Confluence of River Gowry	
River Idle, Retford	1.5*, 1.5*, 46.5*, 16.5*, 1.5*	U/s bridge s. of Thrumpton, 100m d/s of outfall, 300m d/s of outfall, d/s of Tiln farm, d/s bridge Chain Bridge Lane	
River Roach, Rochdale	1.5*, 206*, 346*, 609*, 1.5*	100m u/s from road bridge, 100m d/s of outfall, 300m d/s of oufall, d/s bridge Roe Acre drying, d/s bridge at Hooley Brow	
River Tweed, Berwick upon Tweed	267*, 1.5*, 1.5*	Canty bridge, between bridges, Union bridge	
River Lark, Bury St. Edmunds	26*, 27*, 51*, 1.5*	400m u/s of outfall, 100m d/s of outfall, 300m d/s of outfall, 1.2km d/s Mill Farm	
River Chess, Rickmansworth	1.5*, 1.5*, 1.5*, 1.5*, 1.5*, 1.5*	1km u/s of STW, STW outfall, 100m d/s of STW, 300m d/s of STW, 700m d/s of STW, 1.8km d/s at Inn	
UK			**(Deuchar, 2002)**
River Skerne	25*	u/s Demons Beck	
	25*	Aycliffe Village bridge	
	25*	Demons Beck u/s of Aycliffe STW	
	11310	Howden Beck u/s of Aycliffe STW	
	25*	South Park, Darlington	
River Tees	441	Victoria Bridge, Stockton	

Location	Level of HBCDD (µg/kg dwt)	Comment	Reference
UK			(U.K. Environment Agency, 2006)
River Skerne	2300*	Aycliffe Village bridge	
	174400, 71910, 679*, 33350	Howden Beck u/s of Aycliffe STW	
	300*	South Park, Darlington	
	300*	Ad hoc sample	
	300*	Ad hoc sample	
	300*	Ad hoc sample	
	300*	Ad hoc sample	
River Tees	387*	Victoria Bridge, Stockton	
		Measurements of HBCDD performed downstream of textile processing sites	
River Calder	1826, 2290, 2037	Methley Bridge	
	100*, 100*	Gawthorpe Hall	
River Soar	100*, 100*, 303*	Sileby Mill	
River Aire	100*, 110*, 100*	Rodley Bridge, downstream of Esholt STP	
River Roch	2410, 2903	Above Crimble Mill, Heywood	
River Erewash	147*, 779, 307*	Downstream of Toton	
River Clydach	100*	Neath Abbey	
River Frome	100*, 100*, 100*	Downstream of Strachans	
Black Brook	100*, 100*	Green Lane Bridge	
River Irwell	100*, 100*	Downstream of Rossendale STP	
Lakes			
CH			**(Kohler *et al.*, 2006)**
Lake Greifensee	2.5	2001 (surface sediment)	
	1.8	1995	
	1.3	1989	
	0.25*	1982	
	0.25*	1974	
NO			**(Christensen *et al.*, 2004)**
Lake Ellasjøen, Bjørnøya (arctic region)	0.155*	0-1cm (1987-2001)	
	4.34*	1-2cm(1973-1987)	
	0.095*	2-3cm (1959-1973)	
	0.11*	3-4cm(1946-1959)	
	0.07*	4-5cm(1934-1946)	

Location	Level of HBCDD (µg/kg dwt)	Comment	Reference
NO			**(Fjeld *et al.*, 2005)**
Hardangervidda	0.42		
Haldenvassdraget	1.86		
Glomma (Brandval)	0.2		
Glomma (Skinnerflo)	0.415*		
Glomma (Øyeren sør)	0.745*		
Hurdalssjøen	4.5*		
NO		0-1 cm	(Fjeld *et al.*, 2006b)
Lake Mjøsa	11.27	Vingnes Skibl. A (VIN-A)	
	16.82	Vingnes Skibl. B (VIN-B)	
	0.16*	Vingnes Skibl. C (VIN-C)	
	0.85*	Vingnes Skibl. D (VIN-D)	
	8.99*	Lillehammer overvann (LIL-1)	
	21.34*	Lillehammer Vingnesbr. Sør (LIL-2)	
	7.56	Lillehammer camping. sør (LIL-3)	
	0.99*	Lillehammer RA (LRA)	
	1.01	Moelv (MOE)	
	0.68*	Gjøvik. Redalen (RED)	
	2.32*	Gjøvik. Skibl. (GSK)	
	0.62*	Gjøvik. havna (GHA)	
	1.08*	Gjøvik. Hunnselv (GHU)	
	2.30*	Gjøvik. RA (GRA)	
	0.36*	Furnesfj. hovedst. (FHO)	
	0.22*	Hamar. Rosenlund (HRO)	
	6.45*	Hamar. Politihus (HPO)	
	0.52*	Hamar. Bukta (HBU)	
	0.36*	Hamar. RA (HRA)	
	0.44*	Morskogen (MOR)	
	0.09*	Minnesund (MIN)	
		Vingrom (VIR)	
	8.76*	0-1cm (2003)	
	7.49*	1-2cm(1999)	
	0.90*	2-3cm (1994)	
	0.49*	3-4cm(1898)	
	0.22*	4-5cm(1985)	
	0.36*	5-6cm(1981)	

Location	Level of HBCDD (µg/kg dwt)	Comment	Reference
		Gjøvik dypområde (GDY)	
	0.47	0-1cm (2005)	
	0.57*	1-2cm(2003)	
	0.58*	2-3cm (1999)	
	0.20*	3-4cm(1996)	
	0.19*	4-5cm(1994)	
	0.28*	5-6cm(1992)	
		Furnesfj. Brummund (FBR)	
	0.43*	0-1cm (2003)	
	0.63*	1-2cm(1999)	
	0.23*	2-3cm (1994)	
	0.29*	3-4cm(1988)	
	0.52*	4-5cm(1982)	
	0.20*	5-6cm(1978)	
		Hamar hovedstasjon (HHO)	
	0.43	0-1cm	
	0.11*	1-2cm	
	0.03*	2-3cm	
	0.30*	3-4cm	
	0.06*	4-5cm	
	0.09*	5-6cm	
		(The core from HHO was taken from unstable sediments and could therefore not be dated)	
		Skreia (SKR)	
	0.80*	0-1cm (2005)	
	0.63*	1-2cm(2004)	
	0.19*	2-3cm (2001)	
	0.09*	3-4cm(1999)	
	0.11*	4-5cm(1997)	
	0.24*	5-6cm(1995)	
SE			**(Sellström *et al.*, 1998)**
Lake Marsjön	0.84*	Upstream of first textile industry	
Lake Öresjön	0.14*	Downstream of first textile indusrty	
Lake Skäresjön	0.185*	Reference water to textile indusrty	

Location	Level of HBCDD (µg/kg dwt)	Comment	Reference
SE			**(Sternbeck *et al.*, 2001)**
Lake Mälaren, Årstaviken	0.8	(2-4 cm), Urban environment	
	0.2	(30-32 cm)	
Lake Saltsjön, Fjäderholmarna	0.9	(2-4 cm), Urban environment	
	0.5	(30-22 cm)	
Lake Ältasjön	1.5	(2-4 cm), Urban environment	
	0.5	(30-22 cm)	
Lake Mälaren, Riddarfjärden	1.6	Sediment trap (Urban environment)	
Lake Mälaren, Klubben	0.5*	Sediment trap (Urban environment)	
Lake Mälaren, Slussen	2.1	Sediment trap (Urban environment)	

*One, two, or all of the individual measurements (duplicate/triplicate, or individual diastereomers) resulting in the presented value was/were below the detection limit. For values below the detection limit half detection limit is used. If the analysis was performed using LC, and the values for the individual diastereomers were reported, the * can also mean that one, two or all three of the diastereomers were below the detection limit, and half detection limit is used for that/those diastereomers.

3.1.4.2.3 STP and leachate water from landfill

OVERVIEW

STP

Concentrations of HBCDD measured in water in STP influents close downstream of production facilities range from about 0.93-26 µg HBCDD/l, and in effluents from formulator/compounder on-site treatment plants from 3-46 µg HBCDD/l. Concentrations in influents to general municipal STPs range from 0.0193 (half detection limit) -0.024 µg HBCDD/l and in effluents from 0.0006 µg HBCDD/l -0.0225 µg HBCDD/l (half detection limit in that study).

Concentrations of HBCDD in sludge from municipal STPs range from 0.3 µg HBCDD/kg dwt (half detection limit) -9120 µg HBCDD/kg dwt. In general, the concentrations observed in Ireland (median = 1439 µg HBCDD/kg dwt, n = 3) and UK (median = 1256 µg HBCDD/kg dwt, n = 5) are more than one orders of magnitude higher than those observed in The Netherlands (median = 21 µg HBCDD/kg dwt, n = 9), Norway (median = 35 µg HBCDD/kg dwt, n = 4) and Sweden (median = 24 µg HBCDD/kg dwt, n = 50). Values below the detection limit are set at half detection limit.

Landfill

Concentrations of HBCDD measured in water in landfill leachates range for, untreated leachate water incl. particles, from 0.00035 µg HBCDD/l to 0.15 µg HBCDD/l. The concentration detected in suspended particles range from about 6 (half detection limit) -67700 µg HBCDD/kg dwt, and in sludge range from 0.05 (half detection limit) -10 µg HBCDD/kg dwt.

INDIVIDUAL STUDIES

Measurements of HBCDD in effluents and sludge were performed on a wastewater treatment plant close to production plant B (Anonymous, 2000b). The concentrations detected in the effluents ranged from 10.8-24.3 µg HBCDD/l, measured with LC/MS and from 11.8-26.0 µg HBCDD/l measured with GC/MS. The concentrations detected in sludge ranged from 728000-942000 µg HBCDD/kg dwt.

Fjeld and co-workers (2005) analysed effluent water and sludge from four urban STPs. The concentration detected in the effluent water ranged from about 0.0005 µg HBCDD/l from Bekkelaget to about 0.025 µg HBCDD/l from Høvringen. The concentrations in sludge ranged from parts of µg HBCDD/kg dwt at Bekkelaget to about fifty in sludge from Høvringen. The authors also analysed leachate water and sludge from landfills. The concentrations of HBCDD in untreated leachate water, and sludge ranged from 0.00036-0.149 µg HBCDD/l, and 0.16-9.95 µg HBCDD/kg dwt The highest concentrations were measured at the Djupvik landfill. For the three leachate waters where both the untreated and the rinsed concentration were reported, the concentration in the rinsed sample was 34-67 % of that in the untreated water samples.

The concentrations of HBCDD detected by Sternbeck *et al.* (2001) in outgoing wastewater from a textile laundry, in sludge from STP receiving input from textile industry, and in sludge from STPs in urban environments (Stockholm), were 0.031 µg HBCDD/l, 3-33 µg HBCDD/kg dwt, 0.05 µg HBCDD/kg dwt (digested sludge; half detection limit used), and 6.9 µg HBCDD/kg dwt (primary sludge), respectively. The authors also reported measurements of HBCDD in drain water from a construction waste deposit and in sediment from a sedimentation basin to the construction waste deposit. The concentrations were 0.003-0.009 µg HBCDD/l, and 0.05 µg HBCDD/kg dwt (half detection limit used), respectively.

Filtered STW influent and effluent at Aycliffe contained 0.934 µg HBCDD/l and 0.025 µg HBCDD/l (half detection limit), respectively (Deuchar, 2002). Corresponding values in suspended solids were 216 µg HBCDD/l and 1.26 µg HBCDD/l, respectively. The concentration detected in a sewage sludge cake from Aycliffe STW was 9547 µg HBCDD/kg dwt.

An arithmetic mean concentration of HBCDD of 3200 µg HBCDD/l in waste water effluents to the Northumbrian Water treatment plant has been reported (Allen, Great Lakes, Letter to KemI February 2003).

HBCDD was measured in site effluent treatment plant inlet and outlet, in off-site treatment works outlet, and in the receiving water for off site effluent treatment works at a HBCDD production facility (Anonymous, 2000e). The concentrations detected ranged from 79100-

83200 µg HBCDD/l, 5100-81700 µg HBCDD/l, 8.44-16.3 µg HBCDD/l, and 0.53-0.74 µg HBCDD/l, respectively.

Concentrations of HBCDD reported (Anonymous, 2000d) reported for a formulator/compounder plant for the pre-dispersion manufacture treatment plant inlet was 172-334 µg HBCDD/l, for the HBCDD dispersion manufacture on site treatment plant inlet 313-1890 µg HBCDD/l, and for the HBCDD dispersion manufacture on site treatment plant outlet 3.03-46 µg HBCDD/l.

de Boer and colleagues (2002) analysed influent and effluent water from STPs in the UK, STP sludge and leachate water from landfills from STPs in Ireland, the Netherlands and the UK. HBCDD was detected in the influent dissolved phase (range 0.0193-0.0236 µg HBCDD/l) of four out of five, and in the influent particulate phase (range 2.3-29.4 µg HBCDD/kg dwt) in two out of five STPs in the UK. All concentrations in the effluent phase (dissolved and particulate) in the STPs in the UK were below the limit of detection. The concentrations of HBCDD in STP sludge from both the UK (median 1256 µg HBCDD/kg dwt, range 531-2683 µg HBCDD/kg dwt) and Ireland (median 1439 µg HBCDD/kg dwt, range 211-8315 µg HBCDD/kg dwt), are considerably higher than those detected in the Netherlands (median 14 µg HBCDD/kg dwt, range 0.3-1320 µg HBCDD/kg dwt). No HBCDD was detected in leachate water (dissolved or particulate phase) from landfills in Ireland or UK (half detection limit of 23 µg HBCDD/l and 5.85 µg HBCDD/kg dwt, is used). The concentrations of HBCDD in the particulate phase of lichgate water from landfills in the Netherlands ranged from below the detection limit (15 µg HBCDD/kg dwt, half detection limit used) to 22000 µg HBCDD/kg dwt.

Sellström and co-workers (1999) reported of concentrations ranging from 19-54 µg HBCDD/kg dwt in sewage sludge from three STPs in Stockholm.

In an investigation by Nylund *et al.* (*Nylund et al.*, 2002) HBCDD was found in sludge from all 50 investigated STP in Sweden. The mean value for all the STPs was 45±94 µg HBCDD/kg dwt. Four STPs have higher levels than the other plants. These plants with higher levels are Slottshagen in Norrköping, Gässlösa in Borås, Öna in Mora and Loudden in Stockholm. In both Norrköping and Borås there are industries that use or have used HBCDD. The high levels found in Mora and Stockholm indicates that there might be some point sources at these places. The levels found in sludge from all other STPs are in the range 4-78 µg HBCDD/kg dwt regardless of the size of the STP, thus indicating a leakage from products and compounds in the society.

Schlabach *et al.* (2002) measured HBCDD in sedimentation basins for leachate waters from six landfills in southern Norway. The concentrations ranged from below the detection limit in Drammen to 84 ng HBCDD/kg wwt in the landfill from Kristiansand.

HBCDD has also been found in the furnace bottom ash from the municipal incinerator at a concentration of 0.3 µg HBCDD/kg dwt, Turku, Finland (Peltola, 2002). According to the author this incinerator was not a well functioning plant, which can explain the occurrence of HBCDD.

Table 3-72 Levels of HBCDD in samples from STP influent and effluent water, STP suspended particles/solids and STP sludge within the EU and Norway

Location	Level of HBCDD	Comment	Reference

Location	Level of HBCDD	Comment	Reference
Water	µg HBCDD/l		
NL Terneuzen	 15 20 11 12 15 13 24 26	STP of Broomchemie B.V. Effluent water 2000-01-03 LC/MS GC/MS 2000-01-04 LC/MS GC/MS 2000-01-05 LC/MS GC/MS 2000-01-06 LC/MS GC/MS	**(Anonymous, 2000b)**
NO Arendal Bekkelaget Høvringen Ladehammeren	 0.00425, 0.00328 0.00047, 0.00059 0.02579 0.00187	2004 Effluent water from STP (sampled w 39, w41) (sampled w 36, w37)	**(Fjeld *et al.*, 2005)**
SE Rimbo	 0.031	2000-10 Outgoing wastewater from textile laundry	**(Sternbeck *et al.*, 2001)**,
UK Aycliffe	 0.934 216[a] (wt. 0.36 g) 0.025* 1.26 (wt. 0.006 g)	2002-03-21 STW influent Filtered water samples Suspended solids ex water sample STW effluent Filtered water samples Suspended solids ex water sample	**(Deuchar, 2002)**
UK Aycliffe	 Arith. mean = 3200 Min = 0.0 Max = 13600 n = 12	2002-03 – 2002-06 Aycliffe wastewater effluent to Northumbrian Water treatment plant. Total release of HBCDD per annum 405.6 kg.	Nigel Allen, Great Lakes, Letter to Keml February 2003
UK Aycliffe	 86100, 79100, 83200	1999-11-30 Site effluent treatment plant inlet, normal operation.	**(Anonymous, 2000e)**

Location	Level of HBCDD	Comment	Reference
		Site effluent treatment plant outlet, normal operation	
		1999-11-19	
	81700	1999-11-21	
	20700	1999-11-23	
	5100		
		1999-11-30	
		Off-site treatment works outlet, normal operation.	
	16, 15, 16, 8.85, 10, 8.44		
		Receivivng water for off site effluent treatment works, normal operation	
	0.55, 0.53, 0.74		
UK		Formulator/compounder plant	**(Anonymous, 2000d)**
		HBCDD pre-dispersion manufacture	
		1999-12-06	
	172, 334	On site effluent treatment plant inlet	
		HBCDD dispersion manufacture	
		1999-12-07	
	1890, 780, 313	On site effluent treatment plant inlet	
	3.03, 4.15, 5.40	On site effluent treatment plant outlet	
		1999-12-08	
	505, 832, 1120	On site effluent treatment plant inlet	
	24, 46, 36	On site effluent treatment plant outlet	
UK			**(de Boer *et al.*, 2002a)**
Burnham		Population 10000	
	0.0236	Influent dissolved phase	
	0.0225*	Effluent dissolved phase	
Latchingdon		Population 4750	
	0.0241*	Influent dissolved phase	
	0.0225*	Effluent dissolved phase	
Wickford		Population 35000	
	0.0196*	Influent dissolved phase	
	0.0225*	Effluent dissolved phase	
S. Woodham Ferrers		Population 20000	
	0.0225*	Influent dissolved phase	

Location	Level of HBCDD	Comment	Reference
	0.0225*	Effluent dissolved phase	
Chelmsford		Population 143000	
	0.0193*	Influent dissolved phase	
	0.0225*	Effluent dissolved phase	
Particulate phase/Sludge	µg HBCDD/kg dwt		
IRL		Sludge from STW	**(de Boer *et al.*, 2002a)**
Clonmel	154*, 268		
Cork	7510, 9120		
Portlaoise	1233, 1645		
NL		Sludge from STW	**(de Boer *et al.*, 2002a)**
STP1		Treatment capacity 200000-750000 pe	
	729*	Influent residue	
	0.5*	Effluent residue	
	3.45*	Sludge	
STP2		Treatment capacity 200000-750000 pe	
	3847	Influent residue	
	18*	Effluent residue	
	49	Sludge	
STP3		Treatment capacity 200000-750000 pe	
	56*	Influent residue	
	5.5*	Effluent residue	
	21	Sludge	
STP4		Treatment capacity 200000-750000 pe	
	270*	Influent residue	
	0.5*	Effluent residue	
	1320	Sludge	
STP5		Treatment capacity 100000 pe	
	165*	Influent residue	
	1.6	Effluent residue	
	n.a.	Sludge	
STP6		Treatment capacity 150000 pe	
	28	Sludge	

Location	Level of HBCDD	Comment	Reference
STP7		Treatment capacity 750000 pe	
	7.9*	Sludge	
STP8		Treatment capacity 150000 pe	
	21*	Sludge	
STP9		Treatment capacity 200000-750000 pe	
	0.3*	Sludge	
STP10		Treatment capacity 400000 pe	
	1.55*	Sludge	
NL		Activated sludge of the wastewater treatment plant of Broomchemie B.V.	**(Anonymous, 2000c)**
Terneuzen		1999-12-13	
	728000	1999-12-14	
	942000	1999-12-15	
	901000		
NO		Sludge from STW	**(Fjeld *et al.*, 2005)**
Arendal	47, 23*	(sampled w. 39, 41)	
Bekkelaget	2.33, 0.48	(sampled w. 36, 37)	
Høvringen	51		
Ladehammeren	44		
SE		2000-10	**(Sternbeck *et al.*, 2001)**,
Rimbo	30, 33	Sludge from STP receiving input from textile industry	
SE		2000-12-13	**(Sternbeck *et al.*, 2001)**,
Bromma	0.05*	Digested sludge from STP in Stockholm, urban environment	
Henriksdal	0.05*		
Loudden	0.05*		
SE		2000-12-13	**(Sternbeck *et al.*, 2001)**,
Henriksdal	6.9	Primary sludge from STP in Stockholm, urban environment	
SE		Sewage sludge from STPs in Stockholm	**(Sellström *et al.*, 1999)**
Bromma	54		
Loudden	19		
Sickla	21		
SE		Sludge from STP	**(Nylund *et al.*, 2002)**
		Number of connected pers. equivalents	
Henriksdal (Stockholm)	26	621000	
Ryaverket (Göteborg)	30	584451	

Location	Level of HBCDD	Comment	Reference
Käppala (Lidingö)	58	380819	
Bromma (Bromma)	36	272100	
Sjölunda (Malmö)	9.3	264000	
Himmerfjärden (Grödinge)	51	245000	
Slottshagen (Norrköping)	100	127397	
Borås Gässlösa (Borås)	200	79194	
Klagshamn (Malmö)	13	59700	
Simsholmen (Jönköping)	32	57360	
Arvidstorp (Trollhättan)	12	48000	
Trelleborg (Trelleborg)	9.2	27000	
Loudden (Stockholm)	140	26100	
Tuolluvaara (Kiruna)	35	23250	
Mariestad (Mariestad)	17	17000	
Lybyverket (Hörby)	54	15600	
Arvika (Arvika)	28	15000	
Prästbordet (Svedjeholm)	18	14535	
Strävliden (Stenungsund)	78	13800	
Bålsta (Bålsta)	24	13700	
Klippan (Klippan)	8.9	13500	
Svedala (Svedala)	27	11800	
Åstorp (Helsingborg)	22	11000	
Kil (Kil)	26	10800	
Ljusdal (Ljusdal)	32	10400	
Hönö (Öckerö)	57	10000	
Flen (Flen)	24	9480	
Borgholm (Mörbylånga)	6.5	8000	
Ådalen (Järpen)	22	8000	
Gonäs (Ludvika)	57	6000	
Mellerud (Mellerud)	4.6	5600	
Emmaboda (Emmaboda)	13	5374	
Rimbo (Norrtälje)	16	5000	
Broby (Broby)	12	4500	
Veberöd (Lund)	28	4284	
Nordmaling (Nordmaling)	23	3400	
Övertorneå (Övertorneå)	19	3400	
Bengtsfors (Bengtsfors)	29	3100	
Gimo (Östhammar)	11	3100	
Årjäng (Årjäng)	14	3100	
Grästorp Forshall (Grästorp)	24	3000	

Location	Level of HBCDD	Comment	Reference
Robertsfors	42	2700	
Öna (Mora)	650	2400	
Bräkne-Hoby (Ronneby)	15	2300	
Rimforsa (Kisa)	13	2190	
Billingsfors (Bengtsfors)	11	2050	
Stöde (Sundsvall)	24	1450	
Håkantorp (Vara)	3.8	1000	
Råda (Hagfors)	19	900	
Skärplinge (Skärplinge)	24	889	
UK			**(de Boer *et al.*, 2002a)**
Burnham		Population 10000	
	33*	Influent particulate phase	
	5.85*	Effluent particulate phase	
	1256	STP sludge	
Latchingdon		Population 4750	
	6.2*	Influent particulate phase	
	5.85*	Effluent particulate phase	
	958	STP sludge	
Wickford		Population 35000	
	5.85*	Influent particulate phase	
	5.85*	Effluent particulate phase	
	531	STP sludge	
S. Woodham Ferrers		Population 20000	
	5.85*	Influent particulate phase	
	5.85*	Effluent particulate phase	
	1578	STP sludge	
Chelmsford		Population 143000	
	5.85*	Influent particulate phase	
	5.85*	Effluent particulate phase	
	2683	STP sludge	
UK		Sewage sludge cake from Aycliffe STW	**(Deuchar, 2002)**
Aycliffe	9547		

[a]Estimate only. High concentration causes compounds to precipitate out of solution on standing.

n.a. Not available.

*For concentration values below the detection limit, the concentration is assumed to be half the detection limit. In case the concentration only is presented for the individual diastereomers, the sum of the different diastereomers is used, and if the concentration for one or several of the individual diastereomers is below the detection limit, the concentration is assumed to be half the detection limit.

Table 3-73 Levels of HBCDD in samples from landfill within the EU and Norway

Location	Level of HBCDD	Comment	Reference
Water	µg HBCDD/l		
IRL		Leachate water from landfill, Dissolved phase	**(de Boer *et al.*, 2002a)**
Dumore #1, #2	0.0225*		
Kinsale Road #1, #2	0.0225*		
Kyletalesha #1, #2	0.0225*		
NO		Leachate water (incl. particles) from landfill.	**(Fjeld *et al.*, 2005)**
Grønmo	0.00349	Untreatead	
	0.0012	Rinsed	
Spillhaug	0.00036	Untreatead	
	0.00024	Rinsed	
Esval	0.00453	Untreatead	
	0.00249	Rinsed	
Røyken	0.00035	Untreatead	
Bølstad	0.00115	Untreatead	
Djupvik A	0.00089	Untreatead	
Djupvik B	0.14919	Untreatead	
Mile	0.00036	Untreatead	
Bremnes	0.0018	Untreatead	
Trollmyra	0.00307	Untreatead	
SE		Drain water from deposit for construction waste	**(Sternbeck *et al.*, 2001)**
Vallentuna			
	0.009	2000-08-30	
	0.003	2000-09-14	
UK		Landfill leachate, dissolved phase	**(de Boer *et al.*, 2002a)**
Ockendon	0.0225*		
Pitsea	0.0225*		
Rainham	0.0225*		
Suspended particles/ solids/sludge/sediment	µg HBCDD/kg dwt, unless otherwise specified		
IRL		Leachate water from landfill, particulate phase	**(de Boer *et al.*, 2002a)**
Dumore #1, #2	5.85*		
Kinsale Road #1, #2	5.85*		

Location	Level of HBCDD	Comment	Reference
Kyletalesha #1, #2	5.85*		
NL			**(de Boer *et al.*, 2002a)**
Landfill 1	2.1 120	Sewage sludge Leachate water, particulate phase	
Landfill 2	0.15* 70	Sewage sludge Leachate water, particulate phase	
Landfill 3	110	Leachate water, particulate phase	
Landfill 4	85	Leachate water, particulate phase	
Landfill 5	21	Leachate water, particulate phase	
Landfill 6	22000	Leachate water, particulate phase	
Landfill 7	660	Leachate water, particulate phase	
Landfill 8	15*	Leachate water, particulate phase	
Landfill 9	67700	Leachate water, particulate phase	
NO		Sedimentation basin for leachate water from landfill	**(Schlabach *et al.*, 2002)**
Arendal	0.0369 µg/kg wwt		
Drammen	0, 00026 µg/kg wwt		
Fredrikstad	0.0029, 0.0054 µg/kg wwt		
Kristiansand	0.034, 0.084 µg/kg wwt		
Oslo	0.0144, 0.0407 µg/kg wwt		
NO		Sludge from sedimentation basin for leachate water from landfill	**(Fjeld *et al.*, 2005)**
Bølstad	0.19		
Djupvik	9.95		
Esval	6.18		
Grønmo	0.60		
Mile	0.40		
Røyken	0.16		
Spillhaug	0.36		
SE		Sediment from a sedimentation basin at a deposit for construction waste	**(Sternbeck *et al.*, 2001)**
Vallentuna	0.05*, 0.05*		
UK Ockendon	5.85*	Landfill leachate, particulate phase	**(de Boer *et al.*, 2002a)**
Pitsea	5.85*		
Rainham	5.85*		

*For concentration values below the detection limit, the concentration is assumed to be half the detection limit. In case the concentration only is presented for the individual diastereomers, the sum of the different diastereomers is used, and if the concentration for one or several of the individual diastereomers is below the detection limit, the concentration is assumed to be half the detection limit.

3.1.4.3 Comparison between predicted and measured levels

SURFACE WATER

Calculated PECs of HBCDD in surface water are found in Table 3-59 to Table 3-67 and measured levels in Table 3-69.

The measured level of HBCDD in filtered water at Howden Beck upstream of Aycliffe STW, the receiving water course for any fugitive release to surface water from the production site A, was 1.52 µg HBCDD/l, which is about 35 times lower than the corresponding calculated PEC, which is 53 µg HBCDD/l. The HBCDD concentrations in water courses further from the plant vary from below the detection limit (0.05 µg/l) to 0.2 µg/l.

It is not possible to make any relevant comparisons between regional and continental PECs for freshwater and marine waters calculated by EUSES and measured data, due to the rarity/absence of monitoring data. The only available measured data that is comparable to the calculated PECs indicate that EUSES overestimates the concentrations in surface water.

However, due to the scarcity of data it is not possible to draw firm conclusions regarding the accuracy of the EUSES modelling. The calculated PECs will therefore be used in the risk characterisation.

SEDIMENT

Calculated PECs of HBCDD in sediment are found in Table 3-59 to Table 3-67 and measured levels in sediments are found in Table 3-71.

The measured level of HBCDD in rivers in the northeast of England close to production plant A varied in River Skerne between 25 µg HBCDD/kg dwt (half detection limit) and 11310 µg HBCDD/kg dwt, (median value of 25 µg HBCDD/kg dwt, arithmetic mean of 1895 µg HBCDD/kg dwt) , and in River Tees between 3.6 µg HBCDD/kg dwt (half detection limit) and 363 µg HBCDD/kg dwt (median value of 180 µg HBCDD/kg dwt, arithmetic mean of 193 µg HBCDD/kg dwt). The calculated PEC for this site, which is 240000 µg HBCDD/kg dwt, can be compared to the highest measured concentration (11310 µg HBCDD/kg dwt) originating from the same sampling site close to the plant that also have the highest measured concentration in surface water. EUSES overpredicts the HBCDD concentration in sediment at this site approx. 20 times. However, a new survey of environmental concentrations of HBCDD in the River Skerne and River Tees catchments were performed in 2005. Sampling in the same site, that in the 2002 survey resulted in a concentration of 11310 µg HBCDD/kg dwt, resulted in concentrations ranging from 679-174400 µg HBCDD/kg dwt (median = 52630 µg HBCDD/kg dwt; n = 4). When comparing the EUSES estimate with the median and max. concentrations from the 2005 survey EUSES overpredicts the HBCDD concentration approx. 4.56 and 1.38 times, respectively (*note: measurements were performed in August 2005; production site A ceased production in December 2003 and was demolished by July 2004*).

In the Scheldt basin and The Western Scheldt there are many data on measured levels of HBCDD, possibly reflecting that there are many known facilities that use HBCDD both for production and industrial use in the area. The highest measured value is 151 µg/kg dwt at Terneuzen, The Netherlands, close to production plant B. The calculated PEC in freshwater

sediment for production plant B is 130 µg HBCDD/kg dwt Measured and calculated levels show quite good correspondence for this site.

In Sweden, river Viskan, the measured levels in river sediment downstream textile industries varied between 1 and 1591 µg HBCDD/kg dwt. In the U.K. the measured levels in river sediments downstream textile processing sites varied from below the limit of detection (half detection limit = 100 µg HBCDD/kg dwt is used) to 2903 µg HBCDD/kg dwt (median value = 2657; n = 2) measured in River Roach. This could be compared to the calculated generic PEC (Table 3-67) for industrial use of HBCDD as textile back-coating agent in fresh-water sediment which is 230000 or 1100000 µg HBCDD/kg dwt with or without connection to STP, respectively, *i.e.* the calculated levels are much higher than the measured levels.

The regional calculated PEC in freshwater sediment is 85 µg HBCDD/kg dwt, which is above the median values for BE, NL, and UK (which may be considered as regions), which are 1.4, 4.6, and 1.5, respectively. As regards the 90P, it is above the 90P for NL, but below the 90P for BE, and UK, which are 30, 170, and 424 respectively.

The continental calculated PEC in freshwater sediment is 2.1 µg HBCDD/kg dwt, which is very similar in size to the median value of all the measured freshwater sediment concentrations which is 1.5 µg HBCDD/kg dwt This median value is however strongly influenced by the large number of values from the UK which were below the limit of detection in that study (resulting in the use of the half detection limit of 1.5 µg HBCDD/kg dwt). The calculated PEC is also roughly about a factor of 10 higher than the lowest detected level of HBCDD. In addition to that there also exists data where the level of HBCDD were below the limit of detection (half detection limit was used) which resulted in even lower values.

Due to the scarcity of data on the different scenarios it is not possible to draw any firm conclusions regarding the accuracy of the EUSES modelling in general, except for the scenarios for the textile industries, where the calculated levels are much higher than the measured. For freshwater sediments, the calculated regional PEC is in the range of measured 90P for NL and BE (which can be considered as regions), and the calculated continental PECs is in the range of reported background levels. To conclude, the calculated PECs will therefore be used in the risk characterisation.

STP AND LEACHATE WATER FROM LANDFILL

Calculated PECs of HBCDD in STP are found in Table 3-59 to Table 3-67, measured levels of HBCDD in STPs are found in Table 3-72 and in leachate water from landfills in Table 3-73.

The measured level in filtered water sample from Aycliffe STW influent, close to production plant A was 0.934 µg HBCDD/l, with a corresponding concentration of 0.025 µg HBCDD/l (half detection limit) in the filtered water sample STW effluent. Concentration of HBCDD measured in the Aycliffe wastewater effluent to Northumbrian water treatment plant ranged from 0.0 µg HBCDD/l to 13600 µg HBCDD/l, with an arithmetic mean value of 3200 µg HBCDD/l (n = 12). The calculated PEC for microorganisms in the STP is 560 µg HBCDD/l and is in the range of the measured levels in Aycliffe wastewater effluent to Northumbrian water treatment plant.

The measured level in effluent water from the wastewater treatment plant of Broomchemie at Terneuzen, The Netherlands, close to production plant B was 11-26 µg HBCDD/l and the calculated PEC is 0.21 µg HBCDD/l, which is somewhat lower than the measured level.

There are measured levels from a formulator/backcoater plant in UK for a site water treatment plant inlet of 313-1890 µg HBCDD/l and from the outlet of 3-46 µg HBCDD/l (Table 3-72). The calculated generic PEC for microorganisms in STP is 33 µg HBCDD/l (Table 3-62). Measured levels at the outlet and calculated levels correspond well.

Based on the limited measured data available, and its correspondence with the predicted concentrations, it is decided to use the calculated PECs in the risk characterisation.

There also exists measured levels in leachate water from landfill; however there are no calculated PECs in the EUSES for leachate water.

3.1.5 Terrestrial compartment

3.1.5.1 Calculation of PEC_{local}

Calculations of PECs are based on release figures presented in Chapter 3.1.2.

STP sludge is considered to be the major source for emissions of HBCDD to agricultural soil. Therefore, local PECs are not calculated for sites not connected to a municipal STP or having no emissions to wastewater or where it is known that the sludge is not spread on agricultural soil. The emissions to air are in most cases to small to give a significant contribution to the local PECs for soil.

Intermittent releases are common in industrial use of XPS. TGD does not give any guidance on how to handle the estimation of PEC_{soil} for sites with intermittent releases. The HBCDD concentration in sewage sludge for sites with intermittent releases to waste water is calculated by EUSES under the assumption that the chemical released that day is adsorbed onto the sludge produced that day. However, the sludge retention time in the default waste water treatment plant is 9.2 days and thus it can be assumed that the sludge is diluted by a factor of 9.2. Therefore, for the calculation of PEC_{soil} for sites with intermittent releases the same approach as proposed by the UK in the risk assessment of medium-chained chlorinated paraffins has been used (UK Environment Agency, 2000). This means that the concentration in sewage sludge calculated by EUSES for the sites with intermittent releases is divided by a factor of 9.2

PRODUCTION AND MICRONISING OF HBCDD

Both production sites are connected to municipal STPs according to information from industry. It can be assumed that the sludge is spread on agricultural land. The micronising company has no emissions to waste water and therefore no local PEC is calculated. The PECs are summarised in Table **3-74**.

Table 3-74 Local PECs in agricultural soil and grassland for production of HBCDD .

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland}$. 180 day average
	(mg HBCDD/kg dwt)		
ProdA	91	91	36
ProdB	0.035	0.035	0.014

3.1.5.1.1 Calculation of PEC_{local} for formulation

Local PECs for agricultural soil and grassland for formulation of compound for EPS and HIPS, formulation of XPS compound for the manufacture of flame retarded XPS and formulation of polymer dispersions for textiles are summarised in Table 3-77.

FORMULATION OF COMPOUND FOR EPS AND HIPS

Sites A, C, F, I, D, E, H, J and P are not connected to a municipal STP. Local PECs for soil are therefore not calculated for these sites.

Table 3-75 Local PECs in agricultural soil and grassland for formulation of compound for EPS (and HIPS).

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland}$. 180 day average
	(mg HBCDD/kg dwt)		
Site B	0.0075	0.0075	0.0034
Site G	0.15	0.15	0.059
Site K	1.1	1.1	0.45
Site L	0.57	0.57	0.23
GEN_EPS_FORM	1.3	2.3	0.50

FORMULATION OF XPS COMPOUND FOR THE MANUFACTURE OF XPS

Table 3-76 Local PECs in agricultural soil and, grassland for formulation of XPS compound for the manufacture of XPS.

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland}$. 180 day average
	(mg HBCDD/kg dwt)		
MasterbG	0.0074	0.0074	0.0034
MasterbH	0.016	0.016	0.0067

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland}$. 180 day average
	(mg HBCDD/kg dwt)		
MasterbI	2.1	2.1	0.82
GEN_XPS_FORM	1.4	1.4	0.57

FORMULATION OF POLYMER DISPERSIONS FOR TEXTILES

TexForm3, TexForm5 and TexFormA have no releases to wastewater according to site-specific information. Local PECs for soil are therefore not calculated for these sites.

Table 3-77 Local PECs in agricultural soil and grassland for formulation of polymer dispersions for textiles.

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland}$. 180 day average
	(mg HBCDD/kg dwt)		
TexForm1	0.0062	0.0062	0.0029
TexForm4	0.0032	0.0032	0.0017
TexFormB	0.47	0.47	0.19
GEN_TEX_FORM	5.3	5.3	2.1

3.1.5.1.2 Calculation of PEC_{local} for industrial use

Local PECs for agricultural soil, grassland, and groundwater, for industrial use of EPS-compound at the manufacture of flame retarded EPS, industrial use of HIPS, industrial use of compound for flame retarded XPS, industrial use of HBCDD powder for flame retarded XPS and industrial use of textile back-coating agent are summarised in Table 3-78 -Table 3-82.

INDUSTRIAL USE OF EPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED EPS

Table 3-78 Local PECs for HBCDD for agricultural soil and grassland for industrial use of EPS compound at the manufacture of flame retarded EPS.

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland}$. 180 day average
	(mg HBCDD/kg dwt)		
GEN_EPS_IndUse	0.029	0.029	0.012

INDUSTRIAL USE OF HIPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED HIPS

Table 3-79 Local PECs for HBCDD for agricultural soil and grassland for industrial use of EPS compound at the manufacture of flame retarded HIPS.

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland}$. 180 day average
	(mg HBCDD/kg dwt)		
GEN_HIPS_IndUse	0.088	0.088	0.036

INDUSTRIAL USE OF COMPOUND FOR FLAME RETARDED XPS

Site XPS 2 has no emissions to waste water according to site-specific information. Therefore, no local PECs for soil are estimated for this site.

Table 3-80 Local PECs in agricultural soil and grassland for industrial use of compound for flame retarded XPS.

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland}$. 180 day average
	(mg HBCDD/kg dwt)		
XPS 1	2.5	2.5	1.0
XPS 3	2.3	2.3	0.92
XPS 11	3.8	3.8	1.5
GEN_XPS_IndUse	14	14	5.7

INDUSTRIAL USE OF HBCDD POWDER FOR FLAME RETARDED XPS.

XPS9, XPS16, XPS 17 and XPS 18 have no releases to wastewater according to site-specific information. Local PECs are therefore not calculated for these sites.

Table 3-81 Local PECs in agricultural soil and grassland, for industrial use of HBCDD powder for flame retarded XPS.

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland}$. 180 day average
	(mg HBCDD/kg dwt)		

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland\ .}$ 180 day average
	(mg HBCDD/kg dwt)		
XPS 4	4.0	4.0	1.6
XPS 5	1.0	1.0	0.42
XPS 6	0.10	0.10	0.42
XPS 7	6.7	6.7	2.7
XPS 8	0.0031	0.0031	0.0020
XPS 10	11	11	4.3
XPS 13	0.0061	0.0061	0.0031
XPS 14	0.0025	0.0025	0.0015
XPS 20	0.10	0.10	0.041
XPS 21	14	14	5.4
XPS 23	0.00075	0.00075	0.00073
XPS 24	0.00075	0.00075	0.00074
XPS 26	0.039	0.039	0.016
XPS 27	4.5	4.5	1.8

INDUSTRIAL USE OF TEXTILE BACK-COATING AGENT

Table 3-82 Local PECs in agricultural soil and grassland textile back-coating

Site	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{agricultural\ soil}$ 180 day average	$PEC_{grassland\ .}$ 180 day average
	(mg HBCDD/kg dwt)		
Backcoat.1	0.52	0.52	0.21
Backcoat.2	0.52	0.52	0.21
Backcoat.3	89	89	35
Backcoat.4	0.0017	0.0017	0.0011
BackcoatC	0.17	0.17	0.067
GEN_TEX_IndUse	88	88	35

3.1.5.1.3 Calculation of $PEC_{regional}$ and $PEC_{continental}$

Table 3-83 Regional and continental PECs soil.

PEC	Agricultural soil	Natural soil	Industrial soil

	(mg HBCDD/kg dwt)		
Regional	0.23	0.00066	0.00066
Continental	0.0030	0.000085	0.000085

3.1.5.2 Measured levels in soil

OVERVIEW

Measurements of HBCDD in soil are rare. Those available include railway embankments and a XPS producing industry in Sweden, and a formulator/compounder industry, and a backcoater industry in the UK, with the highest concentrations found within site boundaries of the two latter. The concentration detected at the formulator/backcoater site ranged from 19-90 mg HBCDD/kg dwt and for the backcoater site from 0.835-61 mg HBCDD/kg dwt. The concentrations measured decreased with increasing distance from a XPS producer from 1.3 mg HBCDD/kg dwt, 300 m from the facility, to 0.14 mg HBCDD/kg dwt, 700 m from the facility.

INDIVIDUAL STUDIES

Levels of HBCDD detected in shallow soil samples from within the site boundary of a formulator/compounder (Anonymous, 2000d) and a backcoater (Anonymous, 2000e) and ranged from 19-90 mg HBCDD/kg (mean 62.8 mg HBCDD/kg), and 0.835-61 mg HBCDD/kg (mean 20 mg HBCDD/kg), respectively.

Sternbeck et al. (2001) analysed soil from the ground at a XPS producing industry (Sternbeck *et al.*, 2001) for HBCDD. The concentrations decreased with increasing distance from the plant, from 1.3 mg HBCDD/kg dwt at 300 m to 0.14 mg HBCDD/kg dwt at 700 m.

The Swedish Banverket has made an investigation of the occurrence of HBCDD in railway embankments (Jansson, 2004). Cellular plastic is used to diminish the effects of frost damages. Earlier cellular plastics with flame retardants were used to protect from fire during transport and storage. The sampling was made by drilling through the soil in the railway embankments. During drilling the plastics was crushed. Even though fragments of cellular plastics were taken away, contamination of the soil samples can not be excluded. The concentrations in soil beneath the plastic decreased with increasing depth, from 0.4-45 mg HBCDD/kg dwt directly beneath the plastics to 0.01-1.6 mg HBCDD/kg at 0.5 m beneath the plastics.

Table 3-84 Measured levels of HBCDD in soil within the EU.

Location	Level of HBCDD (mg HBCDD/kg dwt, unless otherwise stated)	Comment	Reference

Location	Level of HBCDD (mg HBCDD/kg dwt, unless otherwise stated)	Comment	Reference
UK	Arithm. mean = 63 Min = 19 Max = 90 n = 4	1999-12-01 Formulator/compounder plant, shallow soil samples from within the site boundary	**(Anonymous, 2000d)**
UK	Arithm. mean = 20 Min = 0.835 Max = 61 n = 4	1999-12-08 Backcoater shallow soil samples from within the site boundary	**(Anonymous, 2000e)**
SE Norrköping	1.3 1.0 0.14	XPS-producing industry; samples taken at increasing distance from the plant 300 m South 500 m South East 700 m North West	**(Sternbeck *et al.*, 2001)**
SE Railway embankments in the middle and eastern Sweden	0.4-45 0.01-1.6	The soil samples were taken below the plastics which were located around 1 m under the ground surface (Cellular plastics: 9000-24600 mg/kg dwt) Directly under the plastics. Around 0.5 m under the plastics.	**(Jansson, 2004)**

3.1.5.3 Comparison of predicted and measured levels

Measured levels are found in Table 3-84 and calculated PECs in Table 3-74 to Table 3-82.

There is one measurement of the occurrence of HBCDD in shallow soil from within the site boundary of a formulator/compounder of HBCDD for the manufacture of XPS in the UK. The measured arithmetic mean concentration was 63 mg HBCDD/kg dwt (n = 4, min = 19 mg HBCDD/kg dwt, max = 90 mg HBCDD/kg dwt) (Table 3-84) to be compared with the generic PEC (Table 3-76) for terrestrial ecosystem of 1.4 mg HBCDD/kg dwt The measured level is probably not relevant for the terrestrial ecosystem since the sample was taken within the site boundary.

The measured levels in soil 300-700 m from an XPS-producing industry were 0.14-1.3 mg HBCDD/kg dwt (Table 3-84), which can be compared with the generic PEC (Table 3-80) for terrestrial ecosystems of 0.44 mg HBCDD/kg dwt The estimated value, which is assumed to

be found in a surrounding area within 1000 m from the point source (20 cm below the surface), is in the range of the detected concentrations.

There are also measurements of HBCDD in shallow soil from within the site boundary of a backcoater facility in the U.K. The measured arithmetic mean concentration was 20 mg HBCDD/kg dwt (n = 4, min = 0.835 mg HBCDD/kg dwt, max = 61 mg HBCDD/kg dwt) (Table 3-84), which can be compared with the generic PEC (Table 3-82) for terrestrial ecosystem of 88 mg HBCDD/kg dwt As discussed above, the measured level is probably not relevant for the terrestrial ecosystem since the sample was taken within the site boundary.

To conclude, there are not enough relevant measured data to verify the EUSES estimates, which therefore will be used.

3.1.6 Atmosphere

3.1.6.1 Calculation of PEC

Calculations of local, regional and continental PECs are based on release figures presented in Chapter 3.1.1.2.

EUSES calculates local PEC_{air} both for the site itself, based on the direct emissions from the site, and a PEC_{air} for the local STP. The larger of the two PECs is used as PEC_{local}. This means that for some sites having large emissions to waste water compared to the emissions to air, EUSES give slightly differing PEC_{air}-values depending on whether the model is run with or without local STP connection. This is applicable to a few sites dealing with formulation of dispersions for textile coating and a few sites dealing with textile back coating.

3.1.6.1.1 Calculation of PEC_{local} for production and micronising of HBCDD

Calculated local PECs in air for local sites with production and micronising of HBCDD are summarised in Table 3-85.

Table 3-85 Local PECs in air for production and micronising of HBCDD

Site	Annual average local PEC in air
	(ng HBCDD/m^3)
ProdA	2600
ProdB	1.6
Micronising	0.24

3.1.6.1.2 Calculation of PEC_{local} for formulation

Local PECs in air for formulation of compound for EPS and HIPS, formulation of XPS compound ,and formulation of polymer dispersions for textiles are summarised in Table 3-86 - Table 3-88.

FORMULATION OF COMPOUND FOR EPS AND HIPS

Table 3-86 Local PECs in air, for formulation of compound for EPS and HIPS.

Site	Annual average local PEC in air
	(ng HBCDD/m³)
Site A	0.33
Site F	1.6
Site I	1.7
Site B	0.84
Site C	1.6
Site D	1.2
Site E	0.23
Site G	1.4
Site H	0.23
Site J	0.69
Site K	0.86
Site L	0.78
Site P	0.047
GEN_EPS_FORM	1.7

FORMULATION OF XPS COMPOUND

Table 3-87 Local PECs in air, for formulation of XPS compound.

Site	Annual average local PEC in air
	(ng HBCDD/m³)
MasterbG	2.0
MasterbH	0.94
MasterbI	2.6
GEN_XPS_FORM	2.0

FORMULATION OF POLYMER DISPERSIONS FOR TEXTILES

Table 3-88 Local PECs in air, for formulation of polymer dispersions for textiles.

Site	Annual average local PEC in air
	(ng HBCDD/m³)
TexForm1	1.1
TexForm3	0.053
TexForm4	0.53
TexForm5	0.86
TexFormA	0.035
TexFormB*	0.038
GEN_TEX_FORM*	1.0

3.1.6.1.3 Calculation of PEC_{local} for industrial use

Local PECs in air for industrial use of EPS compound at the manufacture of flame retarded EPS, industrial use of XPS compound for flame retarded XPS, industrial use of HBCDD powder for flame retarded XPS and textile back-coating are summarised in Table 3-91 - Table 3-93.

INDUSTRIAL USE OF EPS COMPOUND

Table 3-89 Local PECs in air, for industrial use EPS compound at the manufacture of flame retarded EPS.

Site	Annual average local PEC in air
	(ng HBCDD/m³)
GEN_EPS_IndUse	0.41

INDUSTRIAL USE OF HIPS COMPOUND

Table 3-90 Local PECs in air, for industrial use HIPS compound at the manufacture of flame retarded HIPS.

Site	Annual average local PEC in air
	(ng HBCDD/m³)
GEN_HIPS_IndUse	0.15

INDUSTRIAL USE OF XPS COMPOUND FOR FLAME RETARDED XPS

Table 3-91 Local PECs in air, for industrial use of compound for flame retarded XPS.

Site	Annual average local PEC in air
	(ng HBCDD/m^3)
XPS 1	0.26
XPS 2	14
XPS 3	11
XPS 11	7.1
GEN_XPS_IndUse	13

INDUSTRIAL USE OF HBCDD POWDER FOR FLAME RETARDED XPS.

Table 3-92 Local PECs in air, for industrial use of HBCDD powder for flame retarded XPS.

Site	Annual average local PEC in air
	(ng HBCDD/m^3)
XPS 4	1.2
XPS 5	1.1
XPS 6	2.8
XPS 7	1.2
XPS 8	0.88
XPS 9	0.58
XPS 10	0.44
XPS 13	0.56
XPS 14	0.14
XPS 16	0.33
XPS 17	1.4
XPS 18	1.4
XPS 20	0.96
XPS 21	1.2
XPS 23	0.48
XPS 24	0.72
XPS 26	2.9
XPS 27	0.20

INDUSTRIAL USE OF TEXTILE BACK-COATING AGENT

EUSES give slightly differing PEC $_{air}$-values for two sites, Backcoat 3 and the generic local site (GEN_TEX_IndUse) depending on whether the model is run with or without local STP connection.

Table 3-93 Local PECs in air, for industrial use of textile back-coating agent.

Site	Annual average local PEC in air
	(ng HBCDD/m^3)
Backcoat.1	0.078
Backcoat.2	0.038
Backcoat.3*	0.72
Backcoat.4	0.037
BackcoatC	0.025
GEN_TEX_IndUse*	2.1

3.1.6.1.4 Calculation of PECregional and PECcontinental

Table 3-94 Regional and continental PECs for air

PEC	Air ng HBCDD/m^3
Regional	0.025
Continental	0.0032

3.1.6.2 Measured levels in air

OVERVIEW

Measurements of HBCDD in air include outdoor air in middle and northern Scandinavia, HBCDD producing industry, textile industry, XPS producing industry, deposition in urban and at remote sites, and EU urban indoor dust.

The concentrations of HBCDD in air span more than seven orders of magnitude range, from 0.0005 ng HBCDD/m^3 (half detection limit) in remote areas in northern Sweden to 1070 ng HBCDD/m^3, sampled 10 m from a ventilator at a XPS producing facility. Concentrations of HBCDD in office dusts from parliment buildings span 4 orders of magnitude and range from

2 µg HBCDD/kg dust (half detection limit) in Italy to 58000 µg HBCDD/kg dust detected in one of the offices in the EU Parliament in Brussels.

INDIVIDUAL STUDIES

High volume air samples (48 hours) were collected in Sweden during 1990 and 1991 at locations far from known point sources. One location was situated in the northern most part of Sweden (Ammarnäs, 66°N) and the other at Hoburgen, on the southern part of the Baltic island Gotland. Both sites can be considered to represent background locations. The particles were trapped on a glass fibre filter and the compounds in the gas phase were adsorbed on polyurethane foam plugs (PUF). Filters and PUFs were extracted and analysed separately using GC/MS-NCl. HBCDD. Most HBCDD found was particle bound. On the filters 5.7 and 5.1 pg/m^3 were found at Ammarnäs and Hoburgen, respectively. On the PUF 0.2 and 0.4 pg/m^3 was found at the two sites, respectively (Bergander *et al.*, 1995).

Waindzioch (2000) measured 280 ng HBCDD/m^3 HBCDD in ambient air in the vicinity of a HBCDD production plant.

Sternbeck and co-workers measured HBCDD in air and deposition samples from the point sources, urban environment and in remote areas (Sternbeck *et al.*, 2001). The concentrations in air range from 0.0005 ng HBCDD/m^3 (half detection limit) in remote areas (Rörvik) to 1070 ng HBCDD/m^3, sampled 10 m from a ventilator at a XPS producing facility. The deposition spanned four orders of magnitude and was lowest in remote areas 0.02 ng HBCDD/m^2 d (Rörvik) and highest 366 ng HBCDD/m^2 d in urban environments (Stockholm city).

During 2000 samples of dust from offices were analysed for HBCDD (Leonards *et al.*, 2001). Levels of HBCDD were shown in 5 samples from offices in parliament buildings in 8 countries and from 2 offices at internet/computer provider in 1 country. The levels ranged between 8.6 and 3700 µg HBCDD /kg dust in parliament buildings and 840-1400 at the internet/computer provider.

Santillo *et al*. ((2003a)) measured HBCDD in dust in houses in different regions of the UK. HBCDD was found in all samples, with a median concentration of 3250 µg HBCDD/m^3 and a range of 940-6900 µg HBCDD/m^3. In another study by Santillo and co-workers ((2003b)) HBCDD was also measured in household dust from France (median = 485 µg HBCDD/m^3, range 77-1600 µg HBCDD/m^3), Germany (1200 µg HBCDD/m^3), Italy (250 µg HBCDD/m^3), and Spain (median = 225 µg HBCDD/m^3, range 190-850 µg HBCDD/m^3), with concentrations generally lower than those detected in the UK.

Dust samples were collected from homes of 51 volunteers in and around eight Belgian towns and in a village in the Ardennes, which was used as a control in distinguishing, to some extent, the potential contribution of outdoor contamination, since the region is commonly regarded as having low levels of outdoor contamination (Al-Bitar, 2004). Samples were also collected in offices in the European Parliament building, and from the press briefing room. Additional individual samples were also collected from homes or offices of politicians. HBCDD was detected in 6 out of 22 samples, with the six detected values ranging from 11000-58000 µg HBCDD/m^3 (method detection limit = 20 µg HBCDD/m^3). Conclusions

from the study were that no region in Belgium was systematically more contaminated than others, and that homes and offices were equally contaminated.

Table 3-95 Measured levels of HBCDD in air within the EU.

Location	Level of HBCDD	Comment	Reference
Outdoor air	ng HBCDD/m^3		
FIN		Long distance transport, air	**(Sternbeck *et al.*, 2001)**,
Pallas	0.003	2000-09-25 – 2000-10-02 (HVS 3648 m3)	
	0.002	2001-01-04 – 2001-01-11 (HVS 3694 m3)	
NL		1999-12	**(Waindzioch, 2000)**
Terneuzen	280	Production plant, ambient air opposite warehouse western outside left from main entrance at Broomchemie B.V.	
SE		High volume air samples (48 hours) were collected on filters background locations.	**(Bergander *et al.*, 1995)**
Hoburgen, on the southern part of the Baltic island Gotland	0.0051		
SE		Textile industry	**(Sternbeck *et al.*, 2001)**,
Borås	0.019	2000-09-26 – 2000-10-25 (LVS 527 m^3)	
	0.74	2001-01-29 – 2001-02-28 (LVS 528 m^3)	
SE		Production of XPS, sample 10 m from the ventilator	**(Sternbeck *et al.*, 2001)**,
Norrköping	1070		
SE		Urban air 2 locations in city	**(Sternbeck *et al.*, 2001)**
Stockholm	0.076	2000-08-21 – 2000-09-18 (LVS 748 m3)	
	0.61	2001-01-05 – 2001-02-02 (LFS 722 m3)	
SE		Deposit for construction waste	**(Sternbeck *et al.*, 2001)**,
Vallentuna	0.013	2000-08-30 – 2000 -10-04 (LVS 883 m^3)	
	0.18	2001-01-12 – 2001-02-16 (LVS 413 m^3)	
SE		High volume air samples (48 hours) were collected on filters background locations.	(Bergander et al., 1995)
Ammarnäs, 66°N	0.0057		
SE		Long distance transport, air	**(Sternbeck *et al.*, 2001)**,
Aspvreten	0.28	2000-08-19 – 2000-09-18 (LVS 675 m3)	
	0. 25	2001-01-29 – 2001-02-12 (LVS 615 m3)	
Rörvik	0.005	2000-08-28 – 2000-09-18 (LVS 4951 m3)	
	0.0005*	2001-01-29 – 2001-02-12 (LVS 5157 m3)	
Deposition	ng HBCDD/m^2 d		
FIN		Long distance transport, air deposition	**(Sternbeck *et al.*, 2001)**,
Pallas	13	2000-0-25 – 2000-10-16p precipitation 21 L/m2, equal to 21 mm	

Location	Level of HBCDD	Comment	Reference
	5.1	2001-01-09 – 2001-01-23 precipitation 0.4 l/m2, equal to 0.4 mm	
SE Stockholm	366 5.5	Urban air, 2 locations in city 2000-08-21– 2000-09-18 precipitation 32 mm 2001-01-05 – 2001-02-02 precipitation 25 mm	**(Sternbeck *et al*., 2001)**
SE Rörvik	1.6 0.02	Long distance transport, air deposition 2000-08-28 – 2000-09-18 precipitation 46 mm 2001-01-29 – 2001-02-12 precipitation 32 mm	**(Sternbeck *et al.*, 2001)**,
Indoor air/dust	µg HBCDD/kg dust		
AT	1800, 1800	Dust (<1 mm) in urban environment Parliament buildings	**(Leonards *et al.*, 2001)**
BE Hainaut Namur Liège Gros-Fays (in the Ardennes) Brabant Wallon/Vlaamse Brabant Antwerpen Gent Oostende Limburg (normal homes) Limburg (ecological homes) Brussels-European Parliament building Offices Press briefing room	10* (n = 5) 10* (n = 5) 10* (n = 5) 10* (n = 5) 10* (n = 11) 10* (n = 5) 10* (n = 5) 16000 (n = 5) 11000* (n = 3) 12000 (n = 2) 21000 (n = 6) 13000 (n = 1)	Dust (<2 mm) sampled with a vacuum cleaner. Pooled samples from different houses at the selected locations. Samples were also taken from offices at the European Parliament Building and in the press briefing room. In addition, 12 individual samples from homes and offices of politicians.	**(Al Bitar, 2004)**

Location	Level of HBCDD	Comment	Reference
Homes and offices of politicians	10*, 10*, 10*, 10*, 10*, 10*, 10*, 10*, 10*, 58000, 10*,		
DE		Dust (<1 mm) in urban environment	**(Leonards *et al.*, 2001)**
	940, 820	Parliament buildings	
DE		Dust (<2 mm) sampled in houses in 3 regions with a vacuum cleaner. Samples were pooled before analyses,	**(Santillo *et al.*, 2003b)**
Berlin, Leipzig, Regensburg	1200		
DK		Dust (<1 mm) in urban environment	**(Leonards *et al.*, 2001)**
	20, 19	Parliament buildings	
DK	1000	Samples were full or partially filled dust filter bags from routine cleaning donated by 3 individual volunteers	**(Santillo *et al.*, 2003b)**
ES		Dust (<2 mm) sampled in houses in 4 regions region with a vacuum cleaner. Samples were pooled per region before analyses,	**(Santillo *et al.*, 2003b)**
Asturias/Leon	200 (n = 2)		
Granada	190 (n = 3)		
Madrid	850 (n = 2)		
Valencia	250 (n = 3)		
FIN		Dust (<1 mm) in urban environment	**(Leonards *et al.*, 2001)**
	6.5*	Parliament buildings	
FIN	790	Samples were full or partially filled dust filter bags from routine cleaning donated by 3 individual volunteers	**(Santillo *et al.*, 2003b)**
FR		Dust (<2 mm) sampled in 5 cities with a vacuum cleaner. Samples were also collected from other areas in France. Samples were pooled before analyses.	**(Santillo *et al.*, 2003b)**
Lille	1600 (n = 7)		
Lyon	470 (n = 7)		
Nantes	660 (n = 7)		
Paris	240 (n = 7)		
Toulouse	830 (n = 7)		
		Other selected locations around France	
	77, 500, 160		
IT		Dust (<1 mm) in urban environment	**(Leonards *et al.*, 2001)**
	8.6, 2*	Parliament buildings	
IT		Dust (<2 mm) sampled in 3 houses in the city of Rome with a vacuum cleaner. Samples were pooled before analyses,	**(Santillo *et al.*, 2003b)**
Rome	250 (n = 3)		
NL		Dust (<1 mm) in urban environment	**(Leonards *et al.*, 2001)**
	300	Parliament buildings	
	840, 1400, 1.5*	Internet/computer provider	
SE		Dust (<1 mm) in urban environment	**(Leonards *et al.*, 2001)**
	45	Parliament buildings	
UK		Dust (<1 mm) in urban environment	**(Leonards *et al.*, 2001)**

Location	Level of HBCDD	Comment	Reference
	540, 1650, 3700, 980	Parliament buildings	
UK		Dust (<2 mm) sampled in 10 houses per region with a vacuum cleaner. Samples were pooled before analyses, HBCDD was found in all pooled samples.	**(Santillo *et al.*, 2003a)**
Scotland	3800		
North East	940		
North West	1400		
East Midlands	1000		
West Midlands	1640		
East Anglia	4700		
Wales	4700		
London	2700		
South East	3800		
South West	6900		

*For values below the limit of detection, half detection limit is used

3.1.6.3 Comparison between predicted and measured levels

For production plant B (Table 3-64) the calculated level is 1.5 ng HBCDD/m^3, compared to 280 ng HBCDD/m^3 measured at the production plant (Table 3-95). However, the measured value is taken before an "absolute filter" was installed whereas the EUSES calculation is based on release figures after the installation of the filter. For production site A EUSES calculates a PEC_{air} of 2600 ng HBCDD/m^3 but there are no measured data available for this site.

The calculated level, for industrial use of XPS ranges from 0.26-14 ng HBCDD/m^3 (generic scenario 13 ng HBCDD/m^3) for sites using compound (Table 3-91) and from 0.18-23 ng HBCDD/m^3 for sites using powder (Table 3-92) compared to 1070 ng HBCDD/m^3 measured 10 m from the ventilator at a production plant for XPS, Norrköping in Sweden (Table 3-95

For the generic industrial use of textile back-coating the calculated (Table 3-93) value (0.12 ng HBCDD/m^3) is within the range of what have been measured (0.02 and 0.7 ng HBCDD/m^3) at a location for textile industry, Borås in Sweden (Table 3-95).

Measured concentrations of HBCCD in air from the Stockholm area range from 0.013-0.6 ng HBCDD/m^3. This is in fairly good agreement with the calculated regional PEC (0.028 ng HBCDD/m^3).

The measured levels (Table 3-95) from the remote area Pallas, Finland of around 0.003 ng HBCDD/m^3 as well as for the background locations Ammarnäs and Hoburgen, both in Sweden, around 0.006 ng HBCDD/m^3 are very similar to the calculated continental PECs of 0.004 ng HBCDD/m^3 .

There are deposition data from urban and remote areas and a number of measurements of HBCDD in indoor dusts. However, there are no suitable values calculated by EUSES to compare them with.

Based on the limited available local data and the relatively good correspondence between measured and predicted regional and continental concentrations, it is decided to use the values predicted by EUSES in the calculations.

3.1.7 Secondary poisoning

3.1.7.1 Calculation of PEC for secondary poisoning

Calculations of PECs are based on data presented in section 3.1.2.

PECs for secondary poisoning, both in the aquatic and terrestrial environment, are calculated based on the assumption that the predator receives 50 % of its food from the local area and 50 % from the regional area. Thus, for sites having no local emissions the PEC reflects the regional concentration only. $PEC_{oral\ predator,fish}$ for sites having no emissions to water are therefore not presented in the tables below. The same approach is taken for $PEC_{oral\ predator,\ earthworm}$ for sites not connected to STP. These PECs only reflects the regional concentration in earthworms.

3.1.7.1.1 Calculation of PEC for production and micronising of HBCDD

PECs in food of predators in the aquatic and terrestrial food chains for production and micronising of HBCDD are summarised in Table 3-96.

Table 3-96 PECs in food of predators in the aquatic and terrestrial for production and micronising of HBCDD.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
ProdA	Yes	4800	230
ProdB	Yes	5.1	0.68

3.1.7.1.2 Calculation of PEC for formulation

FORMULATION OF COMPOUND FOR EPS AND HIPS

PECs in food of predators in the aquatic and terrestrial food chains for formulation of compound for EPS and HIPS are summarised in Table 3-97.

Table 3-97 PECs in food of predators in the aquatic and terrestrial food chains, for formulation of compound for EPS and HIPS.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
Site A+M	No	5.5	-
Site F+N	No	8.6	-
Site I+O	No	5.6	-
Site B	Yes	5.1	0.61
Site C	No	6.7	0.71

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
Site D	No	6.3	-
Site E	No	5.7	-
Site G	Yes	11	0.96
Site H	No	7.1	-
Site J	No	110	-
Site K	Yes	54	3.5
Site L	Yes	30	2.0
Site P	No	11	-
GEN_EPS_FORM	Yes	60	3.86.2
	No	270	-

FORMULATION OF XPS COMPOUND FOR THE MANUFACTURE OF XPS

Calculated PECs in food of predators in the aquatic and terrestrial food chains, in local sites with formulation of polystyrene compound containing HBCDD for the manufacture of XPS are summarised in Table 3-98.

Table 3-98 PECs in food of predators in the aquatic and terrestrial food chains for formulation of XPS compound.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
MasterbG	Yes	5.4	0.61
MasterbH	Yes	5.7	0.63
MasterbI	Yes	94	5.8
	No	440	-
GEN_XPS_FORM	Yes	67	4.3
	No	310	-

FORMULATION OF POLYMER DISPERSIONS FOR TEXTILES

Calculated PECs in food of predators in the aquatic and terrestrial food chains, in local sites with formulation of polymer dispersions for textiles are summarised in Table 3-99.

Table 3-99 PECs in food of predators in the aquatic and terrestrial food chains, for formulation of polymer dispersions for textiles.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
TexForm1	Yes	5.3	0.60
	No	6.2	-
TexForm3	Yes/No*	5.1	0.59
TexForm4	Yes	5.2	0.60
	No	5.6	-
TexFormB	Yes	25	1.8
	No	100	-
GEN_TEX_FORM	Yes	230	14
	No	1100	-

*STP connection not known, however no emissions to wastewater.

3.1.7.1.3 Calculation of PEC for industrial use HBCDD

INDUSTRIAL USE OF EPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED EPS

Calculated PECs in food of predators in the aquatic and terrestrial food chains for the generic site with industrial use of polystyrene beads containing HBCDD at the manufacture of flame retarded EPS are summarised in Table 3-100.

Table 3-100 PECs in food of predators in the aquatic and terrestrial food chains, for industrial use of EPS compound at the manufacture of flame retarded EPS.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
GEN_EPS_IndUse	Yes	6.3	0.66
	No	11	-

INDUSTRIAL USE OF HIPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED HIPS

Calculated PECs in food of predators in the aquatic and terrestrial food chains, for the generic site with industrial use of polystyrene beads containing HBCDD at the manufacture of flame retarded HIPS are summarised in Table 3-101.

Table 3-101 PECs in food of predators in the aquatic and terrestrial food chains, for industrial use of HIPS compound at the manufacture of flame retarded HIPS.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
GEN_HIPS_IndUse	Yes	5.5	0.81
	No	6.9	-

INDUSTRIAL USE OF COMPOUND FOR FLAME RETARDED XPS

Calculated PECs in food of predators in the aquatic and terrestrial food chains, in local sites with Industrial use of compound for flame retarded XPS are summarised in Table 3-102.

Table 3-102 PECs in food of predators in the aquatic and terrestrial food chains, for industrial use of compound for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
XPS 1	Yes	5.1	6.9
	No	5.3	-
XPS 3	Yes	5.4	6.4
	No	6.4	-
XPS 11	Yes	15	10
	No	55	-
GEN_XPS_IndUse	Yes	24	37
	No	96	-

INDUSTRIAL USE OF HBCDD POWDER FOR FLAME RETARDED XPS.

Calculated PECs in food of predators in the aquatic and terrestrial food chains, in local sites with industrial use of HBCDD powder for flame retarded XPS are summarised in Table 3-103. Sites XPS 9, XPS16, XPS 17 and XPS 18 have no releases to water according to site specific information. Local PECs are therefore not calculated for these sites as they will be the same as the regional PEC.

Table 3-103 PECs in food of predators in the aquatic and terrestrial food chains for industrial use of HBCDD powder for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
XPS 4*	Yes	-	11

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
	No	-	-
XPS 5	Yes	5.1	3.2
	No	5.2	-
XPS 6	Yes	5.1	2.9
	No	5.1	-
XPS 7	Yes	5.2	18
	No	5.5	-
XPS 8	Yes	5.1	0.6
	No	5.1	-
XPS 10	Yes	15	28
	No	55	-
XPS 13	Yes	5.1	0.6
	No	5.1	-
XPS 14*	Yes	-	0.59
XPS 20	Yes	5.1	0.84
XPS 21	Yes	6.8	35
	No	13	-
XPS 23	Yes	5.1	0.59
XPS 24	Yes	5.1	0.59
XPS 26	Yes	5.1	0.69
XPS 27	Yes	11	12
	No	34	-

*XPS4 and XPS14 are known to have releases to the sea.

INDUSTRIAL USE OF TEXTILE BACK-COATING AGENT

Calculated PECs in food of predators in the aquatic and terrestrial food chains, in local sites with Industrial use of textile back-coating agent are summarised in Table 3-104.

Table 3-104 PECs in food of predators in the aquatic and terrestrial food chains, for industrial use of textile back-coating .

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
Backcoat.1	Yes	22	1.9
	No	87	-
Backcoat.2	Yes	9.1	1.9

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ predator\ .\ fish}$	$PEC_{oral\ predator.earthworm}$
		(mg HBCDD/kg wwt)	
	No	25	-
Backcoat.3	Yes	1100	230
	No	5200	-
Backcoat.4	Yes	5.1	0.59
	No	5.2	-
BackcoatC	Yes	6.1	1.0
	No	10	-
GEN_TEX_IndUse	Yes	3200	230
	No	16000	-

3.1.7.2 Measured levels of HBCDD in biota in the aquatic compartment

3.1.7.2.1 Introduction

Concentrations of HBCDD in Table 3-107 - Table 3-110 below are always presented in the form the values originally appeared (µg HBCDD/kg lwt, µg HBCDD/kg wwt, and/or µg HBCDD/kg dwt). If not given in the original article, concentrations on a wet weight basis were also calculated by the rapporteur and added to the table(s) when possible. In order to perform the calculation, information on lipid concentration and water content presented in the original study were used whenever available. If missing, the following values on lipid and water content were used (Table 3-105):

Table 3-105 Literature data on the lipid content on wet weight of fish and shell fish and water content

Specie	Lipid content (% of wwt)	Water content (%)	Reference
Low fat freshwater fish (e.g. pike)	0.71		**(Lind *et al.*, 2002)**
Eel	19.9		
Perch	0.61	79	http:///www.slv.se
Trout	3.3	75	
Whitefish	0.6	80	

3.1.7.2.2 Overview of the concentrations of HBCDD measured in freshwater biota

FRESHWATER INVERTEBRATES

HBCDD has only been reported for freshwater invertebrates in one study which was performed in Lake Mjøsa in Norway (Fjeld, 2006a). The concentrations detected ranged from below the limit of detection (half detection limit, 0.025 µg HBCDD/kg wwt will be used) for zooplankton species (mainly *Cladocera* but also *Copepoda*), up to 28 µg HBCDD/kg wwt detected in *Gracantus loricatus* (see Figure 3-5 below).

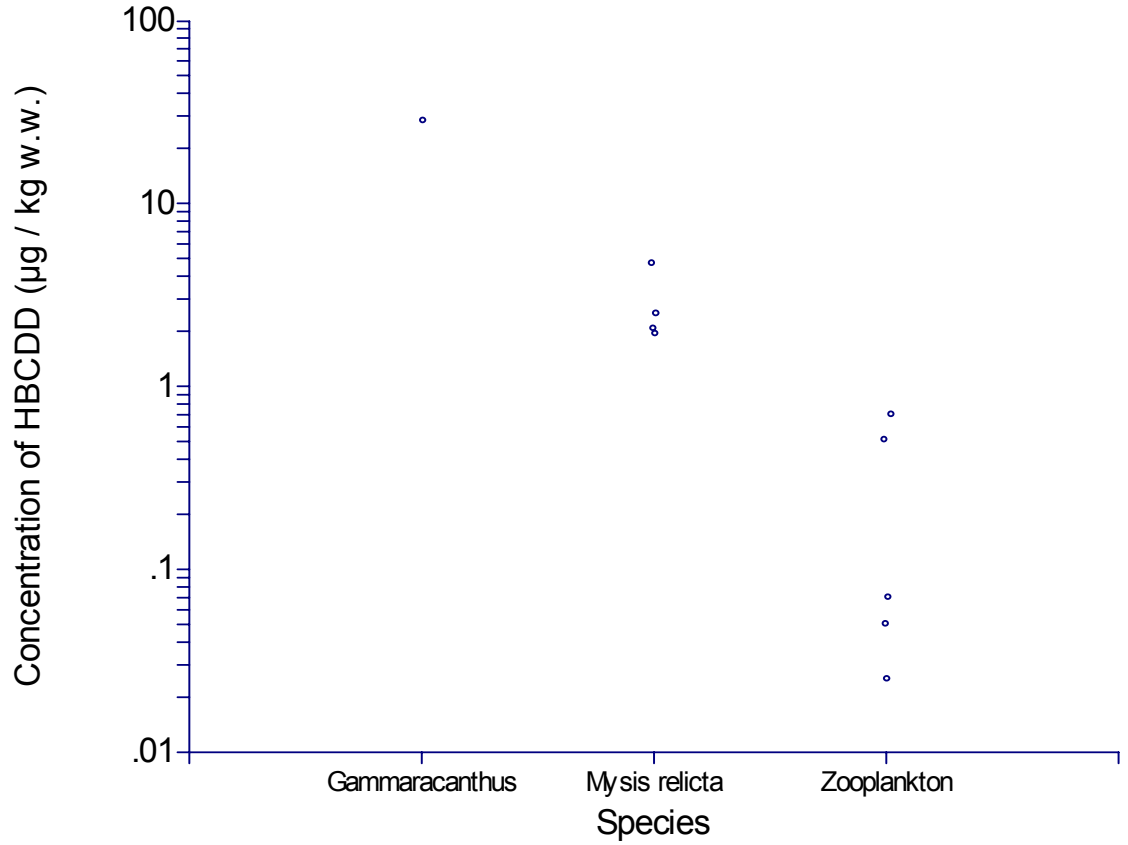


Figure 3-5 Dot plot of all measurements of HBCDD in freshwater invertebrates.

FRESHWATER FISH

HBCDD has been detected in freshwater fish with the measured concentrations spanning more than five orders of magnitude, with the lowest (0.03 µg HBCDD/kg wwt) concentrations found in perch in the Swedish lake Stensjön, and the highest found in eel (9432 µg HBCDD/kg wwt) and Brown trout (6758 µg HBCDD/kg wwt) caught downstream in the River Skerne in the UK. The highest concentrations of HBCDD were measured downstream a HBCDD production in the UK, and in areas with textile industries in Belgium and Sweden. The distribution of all values, including measurements affected by local point sources, is presented in Figure 3-6. Descriptive statistics are presented in Table 3-106.

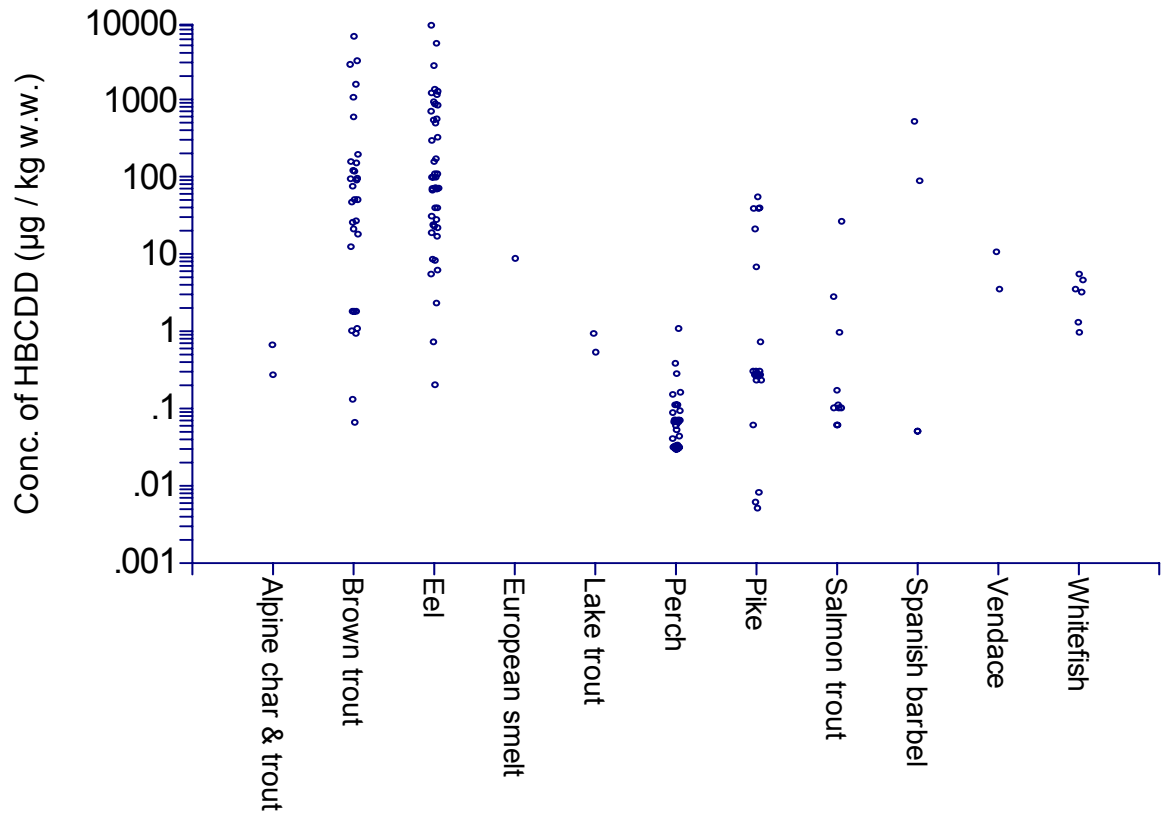


Figure 3-6 Dot plot of all measurements of HBCDD in muscle of freshwater fish. In the EU and Norway

Table 3-106 Descriptive statistics of HBCDD concentrations in muscle of freshwater fish in the EU and Norway, all values included. The percentiles were calculated using Weighted Average at X(n+1)p.

	Conc.	n	Median	Geometric mean	Arithmetic mean ± SD	90P	Min	Max
All values	µg HBCDD/kg wwt	151	5.5	4.64	321 ± 1130	834	0.005	9432
	µg HBCDD/kg lwt	151	120	171	5223 ± 18745	7927	0.52	160905

3.1.7.2.3 Individual studies

FRESHWATER INVERTEBRATES

An overview of the levels of HBCDD detected in freshwater invertebrates was given in section 3.1.7.2.2 above.

The only study, to the knowledge of the rapporteur, where HBCDD has been measured in freshwater invertebrates is the study by Fjeld (2006a). Analysis were performed on *Mysis relicta*, zoo planctonic species (mainly *Cladocera* but also *Copepoda*), and *Gracantus loricatus* sampled in Lake Mjøsa in Norway. The reported levels ranged from below the

detection limit (half detection limit is used) -0.51 µg HBCDD/kg wwt for the zoo planctonic species, 1.94-4.71 µg HBCDD/kg wwt for the *Mysis*, up to 28 µg HBCDD/kg wwt for *Gracantus.* The reason for the high concentration in *Gracantus* was suggested to be due to its spatial distribution (bottom living) and that it lives on preys like the *Mysis*, which feeds on even smaller zoo planctonic species.

Table 3-107 Measured levels of HBCDD in freshwater invertebrates in Norway

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
Gracantus loricatus	NO: Lake Mjøsa Year 2004 (Gjøvik)	-	 28	**(Fjeld, 2006a)**
Mysis relicta	NO: Lake Mjøsa Year 2003 (Helgøya) Year 2005 (Brøttum, Furnesfjord, Skreia)	-	 4.71 1.94, 2.07, 2.5	**(Fjeld, 2006a)**
Zoo planctonic species (mainly *Cladocera* but also *Copepoda)*	NO: Lake Mjøsa Year 2004 (Skreia) Year 2005 (Brøttum, Brøttum , Furnesfjord, Skreia)	-	 0.51 0.07, 0.025*, 0.05, 0.7	**(Fjeld, 2006a)**

*For concentration values below the detection limit, the concentration is assumed to be half the detection limit

FRESHWATER FISH

An overview of the levels of HBCDD detected in freshwater fish was given in section 3.1.7.2.2 above. Results from the different studies included are presented below.

In a study by the UK Food Standard Agency (Anonymous, 2004; Gems, 2006**)** HBCDD was analysed in samples of trout and eel caught in 2002 at various locations in the Skerne-Tees River system. For both trout and eel, the highest levels of HBCDD were found in the samples caught closest downstream to the manufacturing plant, 9432 µg HBCDD/kg wwt (27705 µg HBCDD/kg lwt) and 6758 µg HBCDD/kg wwt (160905 µg HBCDD/kg lwt), for eel and trout, respectively. The levels fell with distance further downstream. The production at the manufacturing plant of HBCDD ceased on 23 December 2003. Most of the different HBCDD diastereomers were detected in the majority of samples of trout and eels analysed.

de Boer and colleagues (de Boer *et al.*, 2002a) measured HBCDD in eels caught in NL rivers and in the Scheldt basin. The highest concentrations were found in Belgium in areas, such as Oeselgem and St Martens at the river Leie, and Oudenaarde at the river Scheldt, associated with textile industry where concentrations of 890 µg HBCDD/kg wwt (7100 µg HBCDD/kg lwt), 1300 µg HBCDD/kg wwt (4700 µg HBCDD/kg lwt) and 5500 µg HBCDD/kg wwt (33000 µg HBCDD/kg lwt), respectively, were detected in muscle of eel.

Sternbeck *et al*. (2001) found increased levels of HBCDD in muscle of eel and pike downstream textile industry, 1392 µg HBCDD/kg wwt (1808 µg HBCDD/kg lwt) and 6.8 µg HBCDD/kg wwt (974 µg HBCDD/kg lwt), respectively, in the watersystem of river Viskan in Sweden. The concentrations detected upstream were 0.72 µg HBCDD/kg wwt (65 µg HBCDD/kg lwt) and 0.72 µg HBCDD/kg wwt (120 µg HBCDD/kg lwt) for eel and pike, respectively.

Sternbeck and colleagues (2004) detected HBCDD in muscle in perch in Swedish lakes, with concentrations ranging from about 0.03 µg HBCDD/kg wwt (4.8 µg HBCDD/kg lwt) to 0.15 µg HBCDD/kg wwt (24 µg HBCDD/kg lwt)

Fjeld *et al*. (2005) measured the concentration of HBCDD in muscle of perch, pike, and Brown trout in Norwegian lakes. The concentrations in perch ranged from about 0.2-1.1 µg HBCDD/kg wwt (33-271 µg HBCDD/kg lwt), the concentration in pike was 0.06 µg HBCDD/kg wwt (50 µg HBCDD/kg lwt), and Brown trout ranged from about 0.9-51 µg HBCDD/kg wwt (9.8-1008 µg HBCDD/kg lwt).

Fjeld (2006a) reported of HBCDD measured in muscle of European smelt, Vendace, and Brown in Lake Mjøsa in Norway. The analysis on European smelt and Vendace were performed on pooled samples from 20 individuals each. The concentrations for 2005 were 8.8, 11, and 18 µg HBCDD/kg wwt for the European smelt, Vendace, and Brown trout, respectively.

Sellström and co-workers (1998) analysed muscle of pikes caught upstream and downstream of textile industries in the water systems of river Viskan in Sweden. The concentrations detected increased with the number of textile industries (and STP) connected to the river, from parts of µg HBCDD/kg wwt upstream (and in a reference water) to about 55 µg HBCDD/kg wwt (7000 µg HBCDD/kg lwt).

Measurements of HBCDD in pike and salmon trout from in Finnish lakes and rivers are presented by Peltola (2002). All concentrations measured in pike were below detection limit (half detection limit will be used), as were half of the measurements in salmon trout (i.e. half detection limit will be used). As regards the salmon trouts with detectable concentrations of HBCDD, the levels ranged from about 0.1-27 µg HBCDD/kg wwt (7-490 µg HBCDD/kg lwt).

Gerecke and colleagues (2003) measured HBCDD in muscle in Whitefish in Swiss lakes. The concentrations detected ranged from about 1-5.5 µg HBCDD/kg wwt.

Schmid and co-workers (2004) detected HBCDD in fish from remote alpine lakes, situated between 2062 and 2637 m above sea level, in the Grisons in Switzerland. With the exception of the lake Laghetto Moesola, which is situated adjacent to a mountain pass road, input from local anthropogenic emissions was considered excluded for the lakes. The analyses were performed on pooled muscle tissues (approx. 300 g) of 2 to 19 individuals per lake. The concentrations found ranged from 5 (half detection limit) to 36 µg HBCDD/kg lwt.

Eljarrat and co-workers (2004) analysed fish (Spanish barbel) from four places along the Spanish River Cinca for PBDEs and HBCDD. Fish were collected up- and downstream from the heavily industrialized town Monzón with important chemical industry. HBCDD was only detected in the fish sampled downstream of the source at concentrations of 215-1127 µg HBCDD/kg wwt in muscle and 161-1172 µg HBCDD/kg wwt in liver at the site near the chemical industry and at concentrations of 42-135 µg HBCDD/kg wwt in muscle and 49-180 µg HBCDD/kg wwt in liver further downstream of the river. The barbel belongs to the carp

family, which is considered to have a moderate fat content together with species like Atlantic salmon, European sardines, blue fin tuna, coho salmon, and sockeye salmon. Using the lipid content for Baltic Sea salmon of 9.55 % lipid content (of wwt) results in the approximate corresponding lipid weight concentrations of 2251-11801 µg HBCDD/kg lwt in muscle close downstream and 440-1414 µg HBCDD/kg lwt in muscle at the site further downstream.

Table 3-108 Measured levels of HBCDD in freshwater fish in the EU, Norway, and Switzerland

Specie	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
Apline char (Salvelinus alpinus) and Brown trout (*Salmo trutta*) (muscle)	CH: Lake Lai da Tuma	 33 (pooled sample of n = 2-19)	 0.66	**(Schmid *et al.*, 2004)**
Apline char (Salvelinus alpinus) and Brook trout (*Salvelinus fontinalis*) (muscle)	CH: Lake Surettasee (Untersee)	 14 (pooled sample of n = 2-19)	 0.27	**(Schmid *et al.*, 2004)**
Brown trout (*Salmo trutta*) (muscle)	CH: Lake Lagh del Teo Lake Lai Grond (Macun) Lake Laghetto Moesola	 5* (pooled sample of n = 2-19) 10* (pooled sample of n = 2-19) 36 (pooled sample of n = 2-19)	 0.065* 0.13* 1.01	**(Schmid *et al.*, 2004)**
Brown trout (*Salmo trutta*) (muscle)	UK: River Skerne Ricknall Grange (upstream HBCDD production plant, control for Skerne) Haughton Road (downstream HBCDD production plant) River Tees Middleton in Teesdale (control for River Tees) Low Coniscliffe Croft-on-Tees (where River Skerne confluences with River Tees)	 9530, 6914, 885, 7413, 7637 38357, 116000, 27591, 91000, 5693, 160905, 26024 4244, 309, 1562 2590, 225*, 180* 4517, 8729, 22253, 4145, 38125	 95, 97, 21.2, 119, 76 1611, 3248, 607, 2912, 159, 6758, 1093 51, 1.8*, 12.5 26, 1.8*, 1.8' 27, 122, 198, 91, 153	**(Anonymous, 2004)**; **(Gems, 2006)**
Brown trout (*Salmo trutta*) (muscle)	NO: Lake Blånuttjern Lake Mjøsa Lake Losna	 9.8 778, 1008 23	 0.93 47, 51 1.08	**(Fjeld *et al.*, 2005)**
Brown trout (*Salmo trutta*)	NO:			**(Fjeld, 2006a)**

Specie	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
(muscle)	Lake Mjøsa Year 2005	729	18	
Eel (*Anguilla anguilla*) (muscle)	UK: River Skerne Ricknall Grange (upstream HBCDD production plant, control for Skerne) Oxenfield Bridge (downstream HBCDD production plant) River Tees Croft-on-Tees (where River Skerne confluences with River Tees) Low Moor (downstream Croft-on-Tees) Tees Barrage (downstream Croft-on-Tees)	 711 3904, 9979, 4359, 1760, 27705 1277, 3496, 919 948, 1795, 361, 204, 39168 3160, 292, 4082	 159 1241, 2814, 1177, 570, 9432 173, 503, 720 300, 552, 100, 68, 862 329, 40, 951	**(Anonymous, 2004)**; **(Gems, 2006)**
Eel (*Anguilla anguilla*) (muscle; homogenates of 25 individuals, except for the samples from the river Scheldt which consisted of homogenates from ca. 10 individuals each))	NL: River Rhine Waal Tiel Lobith Nieuwe Merwede (canal) Lake Ijssel Zeughoek River Meuse Eijsden Keizersveer River Roer Vlodrop River Lek Culemborg	 570 470 140 12 71 120 850 495	 100 73 28 2.3 6.2 19 110 71	**(de Boer *et al.*, 2002a)**

Specie	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
	BE:			
	River Scheldt basin			
	Warmebeek, Achel-Kluis	32	5.5	
	Moervaart, Dacknam	180	31	
	Benede Nete Duffel	190	22	
	Grote Beverdijk Lo-Reninge	0.85*	0.2*	
	Ijzer Nieuwpoort	210	40	
	Durne Lokeren	51	8.6	
	Leie Wervik	78	17	
	Leie Wevelgem	80	23	
	Leie Oeselgem (textile industry area)	4700	1300	
	Leie St Martens (textile industry area)	7100	890	
	Scheldt Doel	360	72	
	Scheldt Grens	300	49	
	Scheldt Oudenaarde (textile industry area)	33000	5500	
	Antwerp Kruisschansbr.	120	24	
	Scheldt Kastel	390	100	
	Scheldt Kennedyt.	570	70	
	Dender Appels	1300	110	
	Dender Ninove	35	8.3	
Eel (*Anguilla anguilla*) (muscle)	SE:			**(Sternbeck *et al.*, 2001)**
	River Viskan-Lake Öresjön			
	upstream textile ind	65 (n = 17)	7.8 (n = 17)	
	River Viskan-Lake Guttasjön			
	Downstream textile ind	1808 (n = 3)	1392 (n = 3)	
European smelt (*Osmerus eperlanus*) (muscle)	NO:			**(Fjeld, 2006a)**
	Lake Mjøsa			
	Year 2005	466 (n = 20, pooled sample)	8.8 (n = 20, pooled sample)	
Lake trout (*Salvelinus namaycush*) (muscle)	CH:			**(Schmid *et al.*, 2004)**
	Lake Lägh dal Lunghin	32 (pooled sample of n = 2-19)	0.93	
	Lake Lej da Diavolezza	14 (pooled sample of n = 2-19)	0.53	
Perch (*Perca fluviatilis*)	SE:			**(Sternbeck *et al.*,**

Specie	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
(muscle)	Lake Bysjön	24, 16, 11, 11, 11, 11, 15, 15	0.15, 0.11, 0.069, 0.087, 0.070, 0.070, 0.11, 0.11	**2004)**
	Lake Hjärtsjön	14, 10, 10, 4.3, 4.2, 7.7, 6.2, 10	0.092, 0.059, 0.066, 0.033, 0.029, 0.065, 0.043, 0.069	
	Lake Stensjön	5.7, 9.0, 4.8, 6.5, 5.6, 5.3, 5.5, 5.7	0.031, 0.052, 0.030, 0.040, 0.029, 0.031, 0.032, 0.032	
Perch (*Perca fluviatilis*)	NO:			**(Fjeld *et al.*, 2005)**
(muscle)	River Glomma	44	0.28	
	Lake Øyeren	33	0.16	
	Lake Hurdalssjøen	271	0.38	
	Lake Femsjøen	235	1.08	
Pike (*Esox lucius*)	SE:			**(Sellström *et al.*, 1998)**
(muscle)	River Viskan			
	Upstream textile ind.	50*, 40*, 50*	0.28*, 0.26*, 0.23*	
	Down-stream textile ind	50*, 50*, 50*, 50*	0.3*, 0.27*, 0.23*, 0.3*	
	Downstream further textile ind and a STP	8000, 7000, 7000	39.2, 39.9, 39.2	
	Further downstream additional textile ind.	7000, 4000	55, 21.2	
	Lake Skaresjön (reference water)	45*, 25*, 40*	0.30*, 0.27*, 0.26*	
Pike (*Esox lucius*)	SE:			**(Sternbeck *et al.*, 2001)**
(muscle)	River Viskan-Lake Öresjön	120 (n = 8)	0.72	
	upstream textile ind	974 (n = 12)	6.8	
	River Viskan-Lake Guttasjön			
	Downstream textile ind			
Pike (*Esox lucius*)	FIN:			**(Peltola, 2002)**
(muscle)	Lake Ahvenkoskenlahti	2.5*	0.006*	
	River Kokemäenjoki	2.5*	0.008*	
	Lake Hirvilampi	2.5*	0.005*	
Pike (*Esox lucius*)	NO:			**(Fjeld *et al.*, 2005)**
(muscle)	River Glomma	50	0.06	
Salmon trout (*Salmo salar*) (muscle)	FIN:			**(Peltola, 2002)**
	River Kymijoki	2.5*, 2.5*, 7, 17	0.06*, 0.06*, 0.10, 0.17	
	River Simojoki	490, 2.5*, 2.5*, 2.5*, 15, 94	26.6, 0.11*, 0.10*,0.10*, 0.96, 2.8	
Spanish barbel (*Barbus*	ES:			**(Eljarrat *et al.*, 2004)**

Specie	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
graellsi)	River Cinca			
(liver)	20 km upstream of Monzón		0.05* (n = 4)	
	12 km upstream		0.05* (n = 8)	
	Close downstream			
	Young fish		484 (mean, n = 3)	
	Adult fish		625 (mean, n = 3)	
	Total		554 (mean, n = 6)	
	30 km downstream		432 (mean, n = 5)	
Spanish barbel (*Barbus graellsi*)	ES:			**(Eljarrat *et al.*, 2004)**
(muscle)	River Cinca			
	20 km upstream of Monzón	0.52* (n = 4)	0.05* (n = 4)	
	12 km upstream	0.52* (n = 8)	0.05* (n = 8)	
	Close downstream			
	Young fish	3236 (mean, n = 3)	309 (mean, n = 3)	
	Adult fish	7853 (mean, n = 3)	750 (mean, n = 3)	
	Total	5550 (mean, n = 6)	530 (mean, n = 6)	
	30 km downstream	937 (mean, n = 5)	90 (mean, n = 5)	
Vendace (*Coregonus albula*)	NO:			**(Fjeld, 2006a)**
(muscle)	Lake Mjøsa			
	Year 2004	166 (n = 20, pooled sample)	3.5 (n = 20, pooled sample)	
	Year 2005	374 (n = 20, pooled sample)	11 (n = 20, pooled sample)	
Whitefish (*Coregonus sp.*)	CH:			**(Gerecke *et al.*, 2003)**
(muscle)	Lake Zug	210 (n = 2)	5.5	
	Lake Zürich	130 (n = 2)	4.6	
	Lake Greifen	85 (n = 2)	3.2	
	Lake Sempach	64 (n = 1)	0.96	
	Lake Neuenberg	48 (n = 2)	3.5	
	Lake Geneva	25 (n = 2)	1.3	

*For concentration values below the detection limit, the concentration is assumed to be half the detection limit. In case the concentration only is presented for the individual diastereomers, the sum of the different diastereomers is used, and if the concentration for one or several of the individual diastereomers is below the detection limit, the concentration is assumed to be half the detection limit.

3.1.7.2.4 Measured levels of HBCDD in biota in the terrestrial compartment

OVERVIEW

Measurements of HBCDD in terrestrial biota are rare and only include HBCDD detected in moss samples, eggs of peregrine falcons and muscle tissues of sparrow hawk.

PLANTS

HBCDD has only been reported for plants in one study, which involved measurements in stair-step moss in Norway. The concentrations detected span almost four orders of magnitude from below the limit of detection (half detection limit, 1.5 µg HBCDD/kg wwt will be used) to 11114 µg HBCDD/kg wwt. The highest concentrations were detected on the south-southwest coast and in general decreased from south to north.

BIRDS

HBCDD have been analyzed in different of terrestrial birds, mainly eggs. The levels of HBCDD in eggs of peregrine falcons range from 0.002 µg/kg wwt (below the detection limit, half detection limit used) in Greenland/DK, to 160 µg/kg wwt in Sweden. No time trends could be discerned.

INDIVIDUAL STUDIES

PLANTS

Schlabach and co-workers (2002) detected α-HBCDD in 6 out of 10 moss samples, and γ-HBCDD in 2 out of 10 samples. In general, the highest concentrations were detected in moss samples from monitoring stations Ualand and Risør at the south-southwest coast with 11114 and 1339 µg HBCDD/kg wwt, respectively (see Figure 3-7 and Table 3-109 below). The detected concentrations decreased further north along the Norwegian coastline, from 586 and 291 in Stord and Fore, respectively, to below the detection limit (using half detection limit of 1.5 µg HBCDD/kg wwt) at Molde and Roan. Except for the moss sample in Nannestad which was below the level of detection (half detection limit of 1.5 µg HBCDD/kg wwt), the levels inland decreased in a south to north direction from Narbuvol, with 325 µg HBCDD/kg wwt, via Limningen, with 27 µg HBCDD/kg wwt, to being below the limit of detection ((half detection limit of 1.5 µg HBCDD/kg wwt is used) at Skoganvarre.

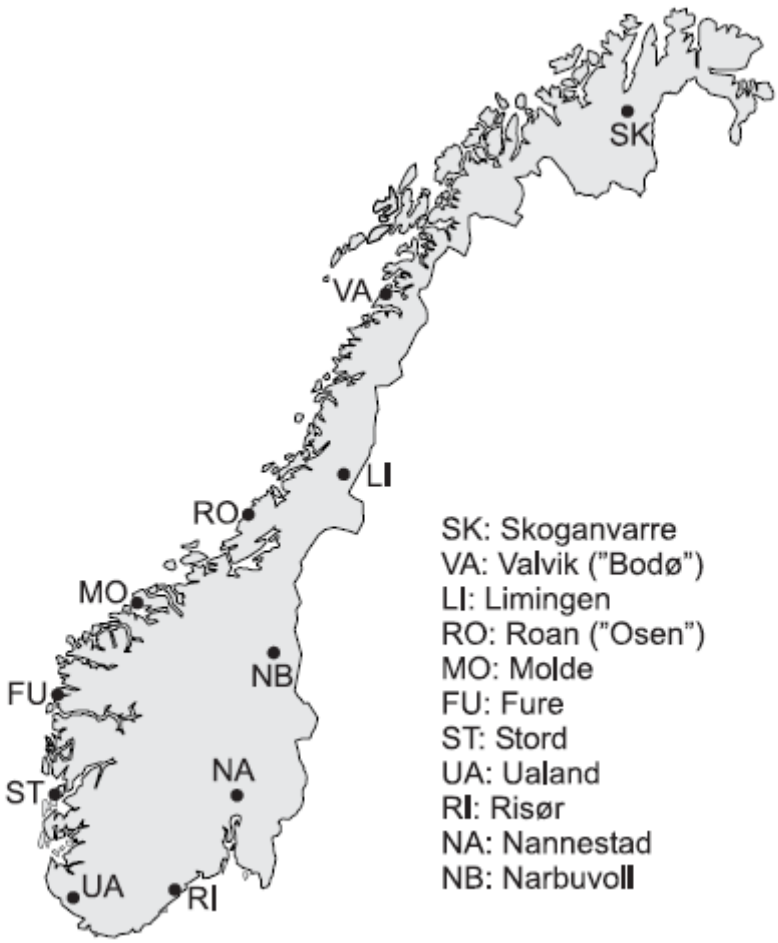


Figure 3-7 Locations of sampling of stair-step moss in Norway by Schlabach *et al.* (2002).

Table 3-109 Concentrations of HBCDD in stair-step moss in Norway by Schlabach *et al.* (2002)..

Species	Location	Concentration (µg HBCDD/kg wwt)	Reference
Stair-step moss (*Hylocomium splendens*)	NO:		**(Schlabach *et al.*, 2002)**
	Coast (from south to north)		
	Risør	1339*	
	Ualand	11114	
	Stord	586*	
	Fure	291*	
	Molde	1.5*	
	Roan	1.5*	
	Inland (from south to north)		
	Nannestad	1.5*	

	Narbuvoll	325*	
	Limingen	27*	
	Skoganvarre	1.5*	

*For concentration values below the detection limit, the concentration is assumed to be half the detection limit. In case the concentration only is presented for the individual diastereomers, the sum of the different diastereomers is used, and if the concentration for one or several of the individual diastereomers is below the detection limit (often the γ- diastereomer), the concentration is assumed to be half the detection limit. This asterisk thus indicate that the entire value, or part of it was below detection limit, and therefore that half the detection limit was used for that fraction(s).

BIRDS

Lindberg *et al.* (2004); (Sellström *et al*., 2001) analysed HBCDD in peregrine falcon eggs collected 1988-1999. The eggs represented females from three different breeding populations, northern Sweden, south-western Sweden, and a captive breeding population. The northern wild population feeds on waders and ducks, while the southern wild population feeds on birds in the terrestrial food web. The captive population was raised on a controlled diet of domestic chickens. The concentrations detected in both the north and the south wild populations were significantly higher than the levels found in the captive breeding population. The concentrations detected in eggs range from a few µg HBCDD/kg wwt in the captive population (all values below the detection limit) to 11.5 (median; range 5.36-112) µg HBCDD/kg wwt, and 6.8 (median; range 2.19-160) µg HBCDD/kg wwt in the Southern and Northern populations, respectively. No temporal trends could be discerned in either the wild or the captive population.

Peregrine falcon (Falco *peregrinus)* eggs (1973-2002) and sparrow hawk (*Accipiter nisus*) muscle tissue samples (1975-2001) were selected for a time trend study (Leslie *et al*., 2004). In the majority of samples, no HBCDD could be detected for neither of the individual diastereomers. HBCDD was detected in 12 of the 51 of the peregrine eggs and in 8 of the 66 samples of sparrow hawk muscle tissue. Concentrations in the eggs ranged from 4.1 µg HBCDD/kg wwt (which is below the limit of detection; half detection limit for the individual diastereomers is used) to 55 µg HBCDD/kg wwt (all diastereomers detected), which was in an egg from 1995. The concentrations in muscle from sparrow hawk ranged from 4.8 µg HBCDD/kg wwt (which is below the limit of detection; half detection limit for the individual diastereomers is used) to 190 µg HBCDD/kg wwt (all diastereomers detected), which was from a sample from 1995. All three diastereomers of HBCDD were detected in both species, although the patterns of occurrence varied from sample to sample The detection of α, β and γ-HBCDD in *F. peregrinus* eggs and *A. nisus* muscle tissue collected in the UK indicates that, at least some individuals of these species are exposed to HBCDD sources and are unable to metabolise the residues faster than they are accumulated. All samples were collected between 1973 and 2002 in the UK, but no time trend could be observed in sum HBCDD concentrations. The data were analysed further by statistical analyses (Verdonck and Vangheluwe, 2004). By box-whisker plots it could be shown that the α-diastereomer of HBCDD was more dominant in the peregrine falcon. In the sparrowhawk no obvious dominant diastereomer could be determined. Even with further statistical analyses it was not possible to identify any time trends.

In a study on the occurrence of HBCDD from the south of Greenland, eggs from peregrine falcons were analysed (Sørensen *et al.*, 2004). Due to low levels it was not possible to detect individual diastereomers of HBCDD, instead total HBCDD was analysed with GC-MS. The

majority of samples were above the detection limit, 20/33. The levels detected ranged from 0.002 (half detection limit) to 5.3 µg HBCDD/kg wwt, with a median value of 0.175 µg HBCDD/kg wwt.

Jaspers *et al.* (2004) analysed eggs of Belgian little owl (*Athene noctua*) for brominated flame retardants, including HBCDD. However, only a limited number of eggs were analysed for HBCDD (it is not clear from the article exactly how many that were analysed). HBCDD could only be detected in 2 out of the subsample used for HBCDD, at levels of 20 and 50 µg HBCDD/kg lwt (detection limit 5 µg HBCDD/kg lwt).

Table 3-110 Measured levels of HBCDD in terrestrial birds in the EU

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
Little owl (*Athene noctua*) (eggs)	BE	20, 50, 2.5* (unknown number measurement below the limit of detection)		**(Jaspers *et al.*, 2004)**
Peregrine falcon (*Falco peregrinus*) (eggs)	SE:			**(Lindberg *et al.*, 2004)**; **(Sellström *et al.*, 2001)**
	Captive population			
	9-2-2			
	1988	4*	0.35*	
	132-1			
	1998	3.5*	0.25*	
	377-1-1			
	1998	2*	0.1*	
	1999	3.5*	0.2*	
	Southern Sweden			
	A			
	1999	390, 280	24, 13	
	B			
	1995	120	8.1	
	C			
	1994	79	5.4	
	D			
	1993	390	20	
	1995	48	2.9	
	E			
	1996	2400	110	
	K			
	1999	98	6.1	
	Q			
	1999	370	21	

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
	Northern Sweden			
	R			
	1991	63	4.2	
	S			
	1991	34	2.2	
	Y			
	1996	210	11	
	AD			
	1999	490	28	
	AE			
	1999	120	6.8	
	AH			
	1999	150	7.2	
	AI			
	1999	110	6.2	
	AK			
	1999	590	160	
Peregrine falcon	UK:			**(Leslie *et al.*, 2004)**
(*Falco peregrinus,*)	1973	109*, 84*, 75*, 79*, 68', 30*, 58.5*, 502*, 150*	6.35*, 6.95*, 5*, 8.8*, 3.6*, 4.05*, 3.3*, 9.15*, 8.25*	
(eggs)	1980	73*, 65*, 104.5*,, 578*, 75*, 107*, 50*, 668*	7.65*, 11*, 8.55*, 41*, 3.3*, 5.25*, 3.9*, 75*	
	1986	210*, 75*, 113*, 134*/83*, 318*, 106.5*, 1450*, 36*	5.2*, 5.7*, 7.7*, 6.95*/3.6*, 24*, 3*, 11*, 4.8*	
	1991	94*, 64*, 73*, 208* 45*	6.2*, 5.4*, 5.1*, 15*, 3.6*, 5.25*	
	1992	107*,	5.4*, 5.55*, 8*, 9*, 30.1*, 3.45*, 55, 3.15*, 11*, 23*,	
	1995	117*, 88*, 112*, 141*, 549*, 59*, 780, 63, 171*, 360*	4.1*, 5.75*, 30*/22*, 3.6*	
	2001	63*, 82*, 709*/445*, 47*	4.55*, 5.7*, 3.6*	
	2002	82*, 96* , 63*		
Peregrine falcon	DK/Greenland			**(Sørensen *et al.*, 2004)**
(*Falco peregrinus,*)	Bagerfalken			
(eggs)	1991	10	0.68	
	Enoraq			
	2002	1.6	0.11	
	2003	27	1.1	

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
	Eqaluit			
	1992	1.2	0.11	
	1994	2.4	0.18	
	2001	0.4*	0.03*	
	Havnen			
	1994	77, 67, 0.05*, 2.1	5.2, 5.3, 0.003*, 0.08	
	Hosp. Dal			
	1991	22	1.1	
	Igaliko			
	1986	0.4*	0.03	
	1987	34	3.2	
	1990	9	0.57	
	1995	4*	0.26*	
	Kirkeruin			
	1991	0.55*	0.04*	
	Lejren			
	1995	3.2	0.20	
	1999	0.6*	0.036*	
	Qanisartut			
	2002	0.5*	0.03*	
	Skyggesø			
	1989	7.8	0.83	
	1990	230, 4.1	n.a., 0.26	
	1992	26, 32, 0.55*,	1.8, 1.8, 0.03*	
	2003	0.05*	0.003*	
	Upernaviarsuk			
	1998	4*	0.30*	
	1999	0.05*, 4.5*	0.002*, 0.17*	
	2000	0.05*, 2.7	0.002*, 0.11	
	Sdr. Igaliko			
	1998	2.6	0.15	
	2000	14	0.50	
Sparrow hawk (*Accipiter nisus)* (muscle)	UK			**(Leslie *et al.*, 2004)**
	1975	590*, 460*, 2570*, 150*, 210	14*, 14*, 65*, 5.7*, 12*	
	1980	265*, 315*, 330*, 310*, 180*, 126*, 165*, 180*, 210*, 137.5*	15*, 14*, 14*, 14*, 6.15*, 7.05*, 9.3*, 13*, 13*, 7.15*	
	1985	2150*, 465*, 325*, 1530*, 2145*, 1010*,	17*, 13*, 14*, 13*, 14*, 14*, 6.3*, 29*, 22*, 6.45,	

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
		375*, 210*, 375*, 300*	8.7*, 12*, 15*	
	1990	580*, 575*, 605*, 555*, 410*, 510*, 143*180*, 360*, 390*, 320*, 360*, 375*	10*, 11*, 10*, 13*, 8.15*, 11*, 9*, 5.1*, 4.8*, 6.15*, 9*, 4.8*, 9.75*	
	1995	525*/255*, 249*, 600*, 925*, 365*, 570*, 660*, 375*, 18900, 1380*, 340*, 340*, 500*	11*/6.6*, 7.6*, 11*, 12*, 9.9*, 9.15*, 11*/5.55*, 7.95*, 5.7*, 189, 11*, 5.25*, 5.25*, 14*	
	2001	385*, 1145*, 210*, 825*, 955*, 485*, 1400*, 375*, 63*, 7050*, 149*, 385*, 1145*, 210*, 825*, 955*, 485*	9.95*, 11.45*, 6.35*, 9*, 8.6*, 12*, 6.15*, 14*, 6*, 5.4*, 4.95*, 5.4*	

*For concentration values below the detection limit, the concentration is assumed to be half the detection limit. In case the concentration only is presented for the individual diastereomers, the sum of the different diastereomers is used, and if the concentration for one or several of the individual diastereomers is below the detection limit (often the γ- diastereomer), the concentration is assumed to be half the detection limit. This asterisk thus indicates that the entire value or part of it was below detection limit, and therefore that half the detection limit was used for that fraction(s).

3.1.7.2.5 Trends and relative proportions of the individual diastereomers of HBCDD in biota

In the following section trends and relative proportions of the individual diastereomers based on measured HBCDD concentrations in freshwater, terrestrial and marine biota are discussed. The data from the marine environment is presented in detail in section 3.2.4.2.

SPATIAL TREND

The levels of HBCDD are higher close to local sources, e.g. production and industrial use, and decrease with increasing distance from source(s).

The results by Schlabach and co-workers (2002), which included measurements of HBCDD in stair-step moss in Norway, indicate a spatial trend, since the concentrations in general decreased from south to north.

Bytingsvik and co-workers (2004) analysed livers from atlantic cod caught at Hvaler at the estuary of river Glomma, Froan on the Atlantic west coast, and livers from polar cod caught at Bear Island in the arctic. The concentrations decreased significantly from south to north (both on wwt and lwt basis).

de Wit *et al.* (2006) suggested, based on what was considered sufficient data, that the concentrations of HBCDD in the European arctic are higher as compared with the North American arctics. The authors proposed that this implied that some combination of atmospheric and oceanic transport from western Europe and eastern North America must be important pathways for transporting HBCDD to the marine food webs in the arctic.

To conclude, HBCDD display a spatial trend, where the concentrations decrease with increasing distance from source(s). Such trends have been observed for both stair-step moss and cod livers in Norway, with decreasing concentrations in a south-north direction. The concentrations of HBCDD measured in the European arctic are reported to be higher than those measured in the North American arctic.

TEMPORAL TREND

In a recent report Law and co-workers (2006b) presented concentrations of HBCDD in blubber of 85 harbour porpoises stranded or dying due to physical trauma in the U.K. during the period 1994-2003. α-HBCDD dominated over the other diastereomers and was detected in all samples at concentrations ranging from 11 to 21342 µg/kg lipid. The study shows increasing concentrations which was not confounded by age (length), sex, nutritional status, or location. The median concentration in the blubber increased from below 100 µg/kg lwt in the mid 1990-ies to 9400 µg/kg lwt in year 2003.

Results from Roos (2006) also indicate an increase of HBCDD over time in seals from the Baltic Sea. Median levels in the 1980-ies ranged between 16 and 35 µg/kg lwt with a median concentration of 28 µg/kg lwt (n = 7). In the 1990-ies the levels ranged between 34 and 177 g/kg lwt with a median of 73 µg/kg lwt. (n = 12). From 2000 and onwards data from only one seal is available having a HBCDD concentration of 64 µg/kg lwt. However, in another study Lundstedt-Enkel (2006) analysed blubber from 30 Grey seals during the period 2000-2002. The HBCDD concentration ranged from 31-554 µg/kg lwt with a mean of 101±98 µg/kg lwt (mean±SD). The median concentration is not available but the results indicate that the levels have not decreased.

Sellström *et al.* (2003) presented results of measurements of HBCDD in eggs from Guillemot from St. Karlsö in the Baltic Sea from 1969 to 2001. The concentration of HBCDD approximately doubled during the study period from 8 µg/kg wwt in the early 1970-ties to approx. 16 µg/kg wwt in the late 1990-ties. The increase appears according to the authors to have levelled out since the mid-1990s.

Knudsen *et al.* (2005) analysed eggs from Atlantic puffins, Herring gull, and Kittywake from northern Norway (Hornøya and Røst) from 1983, 1993, and 2003. The HBCDD levels have raised from 1.1-2.9µg/kg wwt 1983 to 6.1-17 µg/kg wwt 2003.

Bytingsvik and co-workers (2004) reported a temporal trend for HBCDD in Atlantic cod (liver) caught at the estuary of river Glomma, as the concentration increased significantly, 8 or 3-4 times from 1998 to 2003, when expressed on a wwt or lwt basis, respectively.

In a recent study (Stapleton *et al*., (2006) blubber samples from male California sea lions were analysed for PBDEs and HBCDD (n = 26). A significant trend was observed between HBCDD and date of stranding, but not between HBCDD and mass, length, lipid or age of the sea lions. When based on wet weight the levels of HBCDD seemed to have increased in an almost exponential way from 0.7 µg/kg wwt to 12 µg/kg wwt between 1993 and 2003. However, when based on lipid weight the increase was less obvious.

To conclude, HBCDD display a temporal trend, where the concentration in brackish/marine biota has increased with trophic level and time.

RELATIVE PROPORTION OF THE DIFFERENT HBCDD-CONGENERS

The relative proportion of the individual HBCDD-congeners in abiotic samples e.g. sediment resembles that of the technical product with the γ-diastereomer having the highest concentrations. However, reports of sediment samples where the α-diastereomer contributes to a considerable fraction exist:

Marvin *et al.* (2006) analysed the distribution of HBCDD diastereomers in suspended sediments from the River Detroit using liquid chromatography tandem mass spectrometry (LC/MS/MS). The individual diastereomers were measured at concentrations ranging from <0.025 to 1.9 µg/kg (dry weight) for the α-diastereomer, <0.025 to 0.28 µg/kg for the β-diastereomer, and <0.025 to 2.3 µg/kg for the γ-diastereomer. Concentrations of total HBCDD ranged from <0.075 to 3.7 µg/kg. Roughly two-thirds of HBCDD profiles in suspended sediments were dominated by the γ-diastereomer, and were similar to profiles of commercial technical mixtures. Profiles in the remaining samples were dominated by the α-diastereomer. The β-diastereomer, which is a minor constituent in the commercial technical product, was consistently detected at substantially lower levels than the other two diastereomers. Seasonal sampling showed significant shifts in the relative ratios of the HBCDD diastereomers. The spatial distribution of HBCDD in the Detroit River was similar to persistent organic pollutants (e.g. PCBs), and showed a strong association with urban/industrial activities in the watershed. However, the highest HBCDD concentrations (2.6-3.7 µg/kg) were associated with areas of contemporary industrial activity, and were much lower than maximum concentrations of PCBs (2.2 mg/kg) found in areas of the Detroit River associated with historical industrial activity.

Schlabach and co-workers (2004) analysed sediments and muscle tissue from fish from Drammens River and the Drammenfjord, which are located in an industrialized area in the southeast of Norway. The sediments were collected from seven stations in Drammens river and four in the Drammensfjord in Norway. The fish samples (muscle or liver tissue from pooled sample of 5-20 individuals) constituted a number of fish species (Brown trout, Perch, Orfe, Cod, Flounder, and Eel) caught in the Drammenfjord. In one of the sediment samples from Drammen river all diastereomers were below the limit of detection, but for the remaining six samples, the ratio between α- and γ-diastereomers were 1.2, 0.25, 0.32, 0.6, 1.8, and 2.4. For the only sediment sample from the Drammenfjord, for which not all diastereomers were below the limit of detection, a location reported to be close to an industrial site, the ratio between the α and γ-diastereomer was 3.0. Except for the trout (muscle), for which all diastereomers were below the limit of detection, only the α-diastereomer was detected in the different species. The concentrations detected ranged from 4.7 µg/kg lwt (eel, muscle) to 22 µg/kg lwt (perch, muscle).

As regards the two studies described above, being exceptions to what is normally found regarding the proportion of the individual diastereomers, the source of the HBCDD detected in the sediments may not have been the technical product itself, but instead the production of heat-treated polymer materials or textiles. This since temperatures may reach 160 °C and above when producing certain polymers, which results in the thermal conversion of γ- to α -diastereomer. Increased proportion of the α-diastereomer in sediments was actually suggested by de Boer *et al.* (2002) as indications of textile or plastic industry. It was observed that HBCDD in sediments from the Scheldt basin, which normally predominantly was present as the γ-diastereomer, in some cases, e.g. at Oudenaarde and St. Martens (which are in textile industry areas) with high total HBCDD concentrations, had elevated levels of the α-diastereomer.

In biota (e.g. fish, birds and mammals) on the other hand, the α-diastereomer (<10 % of the technical product) is detected at the highest levels (de Boer *et al*., 2002; Schlabach *et al*., 2002; Gerecke *et al*., 2003; Tomy *et al*., 2004; Janák *et al*., 2005; Zeegers *et al*., 2005; Law *et al*., 2006b). The reasons for this difference are not known, but factors that may contribute to this are:

i) Differences in metabolism between the diastereomers.

Zeegers *et al*. (2005) reported on differences in metabolism between the individual diastereomers in microsomal preparations of livers from laboratory rats and harbour seal. The *in vitro* assays showed that β- and γ- diastereomers metabolized significantly when incubated in the presence of NADPH as electron donor, compared to a set of reference samples which were identical except for the addition of NADPH. In contrast, the peak of the α- diastereomer did not decrease significantly in the presence of NADPH. In separate microsomal assays with the β- and γ- diastereomers, new peaks of brominated compounds (indicative of metabolites) were only formed when NADPH was added, which confirms a cytochrome P450 mediated biotransformation. Based on the results of the analysis the metabolites were considered to be hydroxylated. A peak with a retention time similar to one of the two γ- metabolites from the in vitro experiments were also found in residues in some of the blubber samples, indicating that at least one of these metabolites may accumulate to a certain degree in lipid-rich tissues of marine mammals. Eventhough the rat and the harbour seal belong to different families of the mammalian than the cetaceans, the authors proposed that the biotransformation by the cytochrome P450 system is the most likely process to explain the exclusive accumulation of the α- diastereomer found in blubbers of 10 harbour porpoise and 9 common dolphin analysed by LC/MS.

Janák and co-workers (2005b) reported on enantiospecific biotransformation for β-HBCDD in microsomal preparations of livers from fish (*Limanda limanda*). The *in vitro* assays showed that (+)β- isomer disappeared almost totally from the solution when incubated in the presence of NADPH as electron donor, while the reference solution maintained in 3 out of 4 a racemic composition. A corresponding enatioselective metabolism was not found for the other HBCDD-enatiomers. For γ-HBCDD, both biotransformation and reference solutions had still a racemic composition after incubation. The α-HBCDD was least biotransformed, with assays resulting in faster decrease of (+) α-HBCDD resulting in less (+) relative to (-), which is similar to what was found in blubber from marine mammals (common dolphins and harbour porpoise). However, the reference assay without NADPH resulted in a similar proportion between (+) and (-), which could not be explained by the authors.

ii) Bioisomerization of the isomers.

Law and co-workers (2006a) studied bioisomerization in juvenile rainbow trout. The fish were held in three separate tanks and given food fortified with known concentrations of an individual diastereoisomer (α, ß and γ) for 56 d, followed by a depuration period of 112 d, in which all fish were fed unfortified food. A control group in a fourth tank was fed unfortified food throughout the experiment. Muscle tissues were taken during the course of the experiment to examine the diastereoisomeric profiles in order to examine the possibility of bioisomerization. At the termination of the experiment (i.e. after 168 d), it was observed that in the muscle tissues from the fish in the:

α- diastereoisomer group, the individual concentrations of the α-, ß- and the γ-diastereoisomers were 0.5±0.08, <0.01, and 0.07±0.08 nmol, respectively. (values are arithmetic mean of n = 4±standard error)

ß- diastereoisomer group, the individual concentrations of the α-, ß- and the γ-diastereoisomers were 0.3±0.07, 0.3±0.07, and 0.1±0.003 nmol, respectively.

γ- diastereoisomer group, the individual concentrations of the α-, ß- and the γ-diastereoisomers were 0.2±0.03, <0.01, and 0.17±0.01 nmol, respectively.

The above indicate that juvenile rainbow trout have the cabability to bioisomerize the ß- and the γ-diastereoisomers, but that the α-diastereoisomer appears recalcitrant to bioisomerization. It is presently unclear which is/are the responsible enzyme system(s) for these observations.

To conclude, the γ-diastereomer dominates in abiotic samples, whereas the α-diastereomer dominates in biological samples. The reasons for this may e.g. be differences in bioavailability/uptake, differences in metabolism and/or bioisomerization of the different diastereomers.

3.1.7.3 Comparison between predicted and measured levels

Calculated PECs ($PEC_{oral\ predator,\ fish}$, $PEC_{oral\ predator,\ earthworm}$) are found in Table 3-96 to Table 3-104 and measured levels of HBCDD in freshwater fish in Table 3-108, respectively. Besides fish, HBCDD has also been detected in other biota. Concentrations of HBCDD detected in Western European biota are presented in Table 3-107 to Table 3-110. No concentrations of HBCDD in terrestrial biota directly comparable to $PEC_{oral\ predator,\ earthworm}$ exists, and it is therefore not possible to compare those modelled data with the monitoring data.

The measured level of HBCDD in fish in the northeast of England close to production plant A in the River Skerne varied for brown trout between 76 µg HBCDD/kg wwt upstream (median 97 µg HBCDD/kg wwt, arithmetic mean of 120 µg HBCDD/kg dwt, n = 5) and 6758 µg HBCDD/kg wwt (median 1611 µg HBCDD/kg wwt, arithmetic mean of 2341 µg HBCDD/kg dwt, n = 7) downstream, and for eel between 159 µg HBCDD/kg wwt upstream (n = 1) and 9432 µg HBCDD/kg wwt (median 1241 µg HBCDD/kg wwt, arithmetic mean of 3047 µg HBCDD/kg dwt, n = 5) downstream. The calculated PEC for this site, which is 4800000 µg HBCDD/kg dwt, is thus an over prediction of about 500 times, as compared to the highest concentration measured.

There exist a number of measurements of HBCDD in fish from the River Scheldt basin, which includes regions of textile production. The concentrations in eel ranged more than three orders of magnitude, from below the detection limit (half detection limit is used) to 5500 µg HBCDD/kg dwt (median = 36 µg HBCDD/kg wwt, arithmetic mean = 459 µg HBCDD/kg dwt, n = 18), where the highest value was measured in Scheldt Oudenaarde, which is located in textile industry areas. In river Viskan in Sweden, measurements of HBCDD in fish were made upstream and downstream of textile industries. Concentrations upstream and downstream were below 1 up to 212 µg HBCDD/kg wwt in pike, respectively, and about 8 to 1392 µg HBCDD/kg wwt in eel, respectively. The generic calculated PEC for textile production is 230000 or 1100000 µg HBCDD/kg wwt, respectively, depending on whether the site is connected to municipal STP or not. Assuming that the textile industry is connected

to a municipal STP, EUSES then over predicts 40 times, as compared to the highest concentration measured.

When assessing secondary poisoning via the aquatic food chain the predicted concentration of HBCDD in fish are calculated as described below:

$PEC_{oral,\ predator} = (PEClocal_{freshwater} + PECregional_{freshwater}) \times 0.5 \times BCF_{fish} \times BMF_1$

Using the following values for HBCDD:

$PEClocal_{freshwater}$: 0.03-53 µg HBCDD/l

$PECregional_{freshwater}$: 0.03 µg HBCDD/l

BCF_{fish} : 18100

BMF_1: 10

results in $PECoral_{,\ predator}$ = 5430-4799215 µg HBCDD/kg wwt, depending on concentration of HBCDD in the water at the specific local site.

The regional part of $PEC_{oral,predator}$ is calculated by EUSES to be 5321 µg/kg wwt ($PEC_{reg\ surface\ water} \times BCF \times BMF1$). This value can be compared with the median value of 20 µg HBCDD/kg wwt for fish (n = 52) selected to represent regional concentrations of HBCDD in fish (see Table 3-111).

Table 3-111 Descriptive statistics of HBCDD concentrations in muscle of freshwater fish in the EU and Norway, selected to represent regional values. The percentiles were calculated using Weighted Average at X(n+1)p.

	Conc.	n	Median	Geometric mean	Arithmetic mean ± SD	90P	Min	Max
Freshwater fish	µg HBCDD/kg wwt	52	20	13	38±45	107	0.06	182
(muscle)	µg HBCDD/kg lwt	52	230	275	1031±2133	3748	12	9530

Selected values:

µg HBCDD/kg wwt: 95.3, 96.8, 21.2, 119, 76, 50.9, 1.8, 12.5, 25.9, 1.8, 1.8, 47.44, 51, 182, 159, 100, 73, 28, 2.3, 6.2, 19, 110, 71, 5.5, 31, 22, 40, 8.6, 17, 23, 72, 49, 24, 100, 70, 110, 8.3, 7.8, 8.8, 0.28, 0.16, 0.38, 1.08, 0.06, 3.5, 10.7, 5.5, 4.6, 3.2, 0.96, 3.5, 1.3

µg HBCDD/kg lwt: 9530, 6914, 885, 7413, 7637, 4244, 309, 1562, 2590, 225, 180, 778, 1008, 729, 711, 570, 470, 140, 12, 71, 120, 850, 495, 32, 180, 190, 210, 51, 78, 80, 360, 300, 120, 390, 570, 1300, 35, 65, 466, 44, 33, 271, 235, 50, 166, 374, 210, 130, 85, 64, 48, 25

In the risk characterisation the $PEC_{reg\ surface\ water}$ will be adjusted to 1.1×10^{-4} µg HBCDD/l so that the regional part of $PEC_{oral\ predator}$ becomes 20 µg HBCDD/kg wwt. The resulting PECs are presented in Appendix 1.

3.2 MARINE EXPOSURE ASSESSMENT

3.2.1 General Discussion

3.2.2 Degradation

There are no degradation studies performed in the marine environment, instead data from freshwater has to be used see further chapter 3.1.3.

PECs for the sites with releases to surface water, and sites with no releases to water at all are not included in this chapter.

3.2.3 Predicted Environmental Concentrations (PEC) in the marine environment

3.2.3.1.1 Calculation of PEC_{local} for production and micronising

Both production sites release their wastewater to a river via municipal STPs according to industry data. However, production site B releases its waste water close to where the river enters the Western Scheldt and therefore it is considered relevant to calculate marine PECs for this site. The micronising plant does not have any releases to waste water and thus, no local PECs are calculated. The PECs are summarised in Table 3-112.

Table 3-112 PECs in marine surface water and sediment for production of HBCDD

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			µg HBCDD/l		µg HBCDD/kg dwt
ProdB	Yes	1000	0.0030	0.0030	13

3.2.3.1.2 Calculation of PEC_{local} for formulation

Local PECs for marine surface water and sediment for formulation of compound for EPS and HIPS, formulation of XPS compound and formulation of polymer dispersions for textiles are summarised in Table 3-113 - Table 3-115. Local PECs are not calculated for sites which do not have any releases to waste water or marine surface water.

FORMULATION OF COMPOUND FOR EPS AND HIPS

Table 3-113 PECs in marine surface water and sediment for formulation of compound for EPS and HIPS

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)
Site C	No	201	0.025	0.021	110
Site G	Yes	Default (100)	0.011	0.0097	51
Site J	No	Default (100)	0.15	0.12	660
Site K	Yes	Default (100)	0.068	0.057	310
Site L	Yes	Default (100)	0.036	0.030	160
Site P*	No	Default (100)	1.4	0.0067	6500 (220)*
GEN_EPS_FORM	Yes	Default (100)	0.076	0.063	350
	No		0.36	0.30	1600

* Intermittent releases. Figure within parenthesis represents PEC sediment calculated by EUSES averaged over 30 days.

FORMULATION OF XPS COMPOUND

Table 3-114 PECs in marine surface water and sediment for formulation of XPS compound .

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)
MasterbG	Yes	Default (100)	0.0031	0.0031	14
MasterbI	Yes	Default (100)	0.12	0.10	550
GEN_XPS_FORM	Yes	Default (100)	0.086	0.071	390
	No		0.41	0.34	1900

FORMULATION OF POLYMER DISPERSIONS FOR TEXTILES

The sites TexForm2, TexForm3, TexForm5 and TexFormA have no releases to wastewater according to site-specific information. Local PECs are therefore not calculated for these sites.

Table 3-115 PECs in marine surface water and sediment for formulation of polymer dispersions for textiles.

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)
TexForm1	Yes	Default (100)	0.0031	0.0030	14
	No		0.0043	0.0040	20

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)
TexForm4	Yes	Default (100)	0.0029	0.0029	13
	No		0.0035	0.0033	16
TexFormB	Yes	Default (100)	0.030	0.025	140
	No		0.14	0.11	610
GEN_TEX_FORM	Yes	Default (100)	0.31	0.26	1400
	No		1.5	1.2	6800

3.2.3.1.3 Calculation of PEC_{local} for industrial use (processing)

Local PECs for STP, surface water and sediment, for industrial use of EPS compound at the manufacture of flame retarded EPS, industrial use of HIPS, industrial use of XPS compound at the manufacture of flame retarded XPS, industrial use of HBCDD powder for flame retarded XPS and textile back-coating are summarised in Table 3-116 - Table 3-120. Local PECs are not calculated for sites which do not have any releases to waste water or marine surface water.

INDUSTRIAL USE OF EPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED EPS

Table 3-116 PECs in marine surface water and sediment for industrial use of EPS compound at the manufacture of flame retarded EPS.

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)
GEN_EPS_IndUse	Yes	Default (100)	0.0044	0.0041	20
	No		0.011	0.0093	49

INDUSTRIAL USE OF HIPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED HIPS

Table 3-117 PECs in marine surface water and sediment for industrial use of HIPS compound at the manufacture of flame retarded HIPS.

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)
GEN_HIPS_IndUse	Yes	Default (100)	0.0078	0.0032	36
	No		0.027	0.0048	120

INDUSTRIAL USE OF XPS COMPOUND FOR FLAME RETARDED XPS

Site XPS 2 has no emissions to waste water or marine surface water according to site-specific information. Therefore, no local PECs are estimated for the aquatic compartment for this site.

Table 3-118 PECs in marine surface water and sediment for industrial use of compound for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)
XPS 1	Yes	1000	0.017	0.0034	78
	No		0.073	0.0056	330
XPS 3*	Yes	108	1.1	0.0059	5200 (170)*
	No		5.5	0.018	25000 (830)*
XPS 11*	Yes	Default (100)	2.0	0.014	9200 (310)*
	No		9.9	0.057	45000 (1500)*
GEN_XPS_IndUse*	Yes	Default (100)	7.6	0.024	35000 (1200)*
	No		37	0.10	170000 (5500)*

* Intermittent releases. Figure within parenthesis represents PEC sediment calculated by EUSES averaged over 30 days.

INDUSTRIAL USE OF HBCDD POWDER FOR FLAME RETARDED XPS.

Sites XPS9, XPS16, XPS 17 and XPS 18 have no releases to wastewater or marine surface water according to site-specific information. Local PECs are therefore not calculated for these sites.

Table 3-119 PECs in marine surface water and sediment for industrial use of HBCDD powder for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)
XPS 4*	Yes	Default (100)	2.1	0.014	9600 (320)*
	No		10	0.059	47000 (1600)*
XPS 8*	Yes	Default (100)	0.0039	0.0028	18 (0.60)*

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)
	No		0.0084	0.0028	38 (1.3)*
XPS 14*	Yes	Default (100)	0.0037	0.0028	17 (0.60)*
XPS 23*	Yes	Default (100)	0.0028	0.0028	13 (0.43)*
XPS 24*	Yes	Default (100)	0.0028	0.0028	13 (0.43)
XPS27*	Yes	Default (100)	2.4	0.0094	11000 (370)*
	No		12	0.035	53000 (1800)*

*Intermittent releases. Figure within parenthesis represents PEC sediment calculated by EUSES averaged over 30 days.

INDUSTRIAL USE OF TEXTILE BACK-COATING AGENT

Table 3-120 PECs in marine surface water and sediment for textile back-coating.

Site	Connected to municipal STP (Yes/No)	Dilution in the recipient	$PEC_{seawater.\ during\ emission}$	$PEC_{seawater.\ ann.\ average}$	$PEC_{marine\ sediment}$
			(µg HBCDD/l)		(µg HBCDD/kg dwt)
Backcoat.1	Yes	Default (100)	0.033	0.021	150
	No		0.15	0.093	680
Backcoat.2	Yes	Default (100)	0.033	0.0072	150
	No		0.15	0.024	670
Backcoat.3	Yes	Default (100)	5.2	1.2	23000
	No		25	5.8	110000
Backcoat.4	Yes	Default (100)	0.0028	0.0028	13
	No		0.0031	0.0029	14
BackcoatC	Yes	Default (100)	0.012	0.0039	57
	No		0.050	0.0083	230
GEN_TEX_IndUse	Yes	Default (100)	5.1	3.6	23300
	No		25	17	110000

3.2.3.1.4 Calculation of $PEC_{regional}$ and $PEC_{continental}$

Table 3-121 Regional and continental PECs for marine surface water and sediment.

PEC	Sea water	Marine sediment
	(µg HBCDD/l)	(µg HBCDD/kg dwt)
Regional	0.0028	3.5
Continental	0.000010	0.013

3.2.3.2 Measured levels in the marine compartment (including sediment)

3.2.3.2.1 Marine surface water and suspended particles

OVERVIEW

In general, measurements of HBCDD in water and suspended particles are rare, and most of the few available are associated with freshwater. The only available for marine/brackish waters are taken in the vicinity of a production facility in the Netherlands.

The concentrations of HBCDD in suspended particles sampled in the vicinity of a production facility in the Netherlands ranged from 74-472 µg HBCDD/kg dwt.

INDIVIDUAL STUDIES

Bouma *et al*. (2000) detected HBCDD in suspended particles in waters sampled in the vicinity of Terneuzen, NL. The concentrations detected in the Gent/Tern canal and in the Western Scheldt were 472 and 74 µg HBCDD/kg dwt, respectively.

Table 3-122 Measured levels of HBCDD in suspended particles/solids within the EU.

Location	Level of HBCDD (µg HBCDD/kg dwt)	Comment	Reference
NL			(Bouma *et al.*, 2000)
Terneuzen			
Western Scheldt	74		
Gent/Tern Canal	472		

3.2.3.2.2 Estaurine/brackish/marine sediment

OVERVIEW

Concentrations of HBCDD measured in estuarine/brackish/marine sediments span more than four orders of magnitude, from below the detection limit (values set at half detection limit, resulting in a lowest value of 0.25 µg HBCDD/kg dwt) in unpolluted sediments to almost 8.5 mg HBCDD/kg dwt in heavily polluted sediments. Concentrations measured in water systems at/outside the coasts of Ireland, the Netherlands, and Norway are presented in Table 3-123, and Figure 3-8.

Table 3-123 Concentrations of HBCDD in estuarine/brackish/marine sediments in Europe. Values below detection limit are set at half detection limit. The percentiles were calculated using Weighted Average at X(n+1)p.

Location	n	Concentration (µg HBCDD/kg dwt)				
		Median	Mean ± SD	90P	Min	Max
IRL	8	1.4	4.3±4.98	13	0.55	13
NL	31	5.2	23.5±40.4	110	0.25	151
NO	14	2.3	603±2137	4127	0.28	8024
Total	53	4.2	174±1100	122	0.25	8024
Total*	45	2.8	11±26	21.8	0.25	128

*Not including measurements considered affected by local point sources: NL; 25.4, 63.5, 70.1, 151; NO: 8024, 230.3, 24.4, 124.02

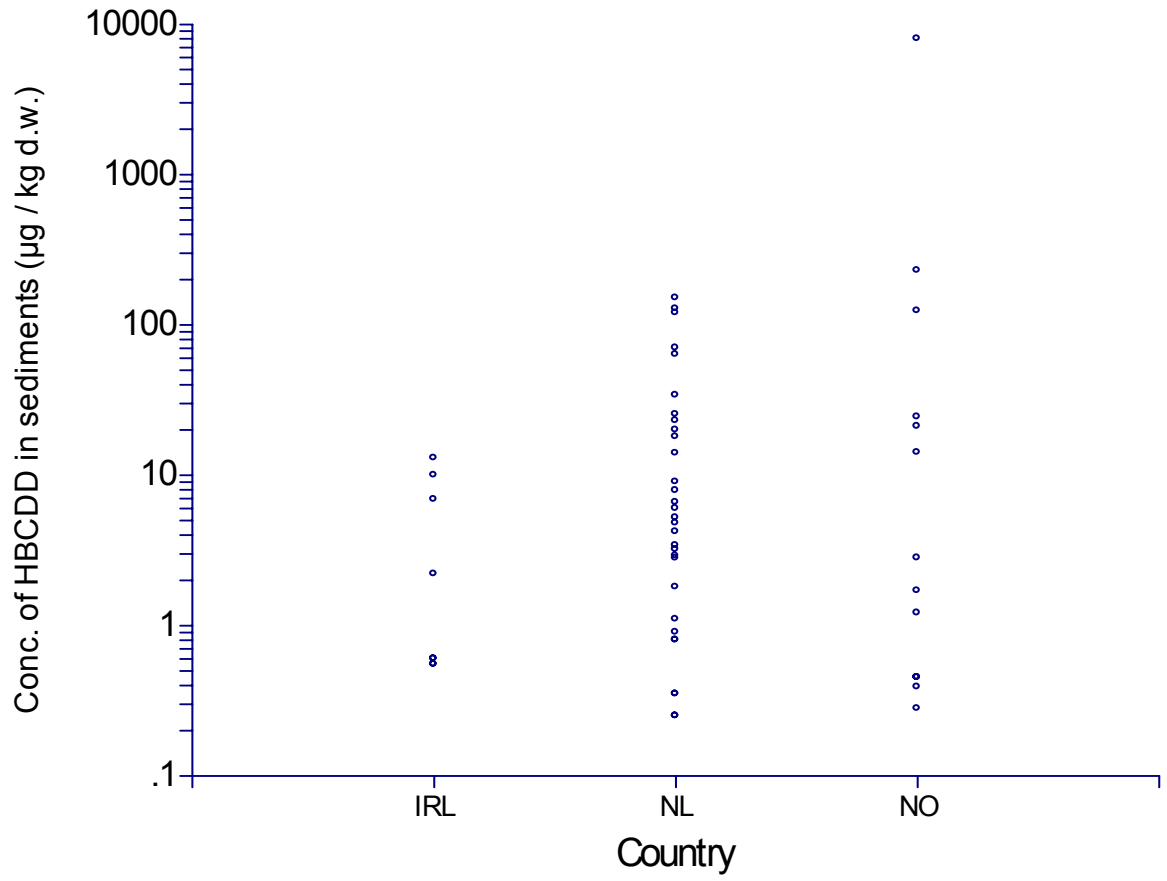


Figure 3-8 Concentrations of HBCDD in estuarine/brackish/marine sediments in European water systems. Values below detection limit are set at half detection limit.

INDIVIDUAL STUDIES

de Boer (de Boer *et al.*, 2002a) analysed sediments from the Scheldt basin in Belgium (close to/on the border to The Netherlands), from The Netherlands (mainly from the southern part), UK and Ireland including the North Sea. The Scheldt basin study was initiated since there had been indications of high HBCDD concentrations in biota and sediments, where many textile industries upstream are located. A substantial number of these industries may use HBCDD as a flame retardant in their products. The processes in which HBCDD is impregnated in the materials could lead to losses to the aquatic environment. In addition to that, a production plant of HBCDD is situated at Terneuzen, at the border of the Western Scheldt. Also here, HBCDD losses to the aquatic environment could take place. Indications of occurrence of HBCDD in STPs initiated the sampling of STPs in The Netherlands, UK and Ireland. A selected number of sediment samples still available after earlier studies were analysed for screening of the occurrence of HBCDD in the marine environment, mainly the North Sea. Sediment samples were composed of 10 sub-samples of surface sediment.

At the workshop on brominated flame retardants in Stockholm 2001 monitoring results from the North Sea outside of the coast of the Netherlands were presented (Klamer *et al.*, 2001). The concentrations were higher close to the NL coast, with maximum values of about 6 µg

HBCDD/kg dwt, as compared to further out at sea where the were below 1 µg HBCDD/kg dwt.

Due to a decrease in number of colonies of the common tern in Terneuzen, The Netherlands a study was performed of the occurrence of HBCDD (and other chemicals) in environmental samples, including sediments (Bouma *et al.*, 2000). The concentrations in sediment samples ranged from 151 µg HBCDD/kg dwt, sampled at the Gent/Tern Channel, to 25 µg HBCDD/kg dwt, sampled in the Western Scheldt.

In Norway a screening study was performed on the occurrence of brominated flame retardants (Schlabach *et al.*, 2002). The analyses of HBCDD were performed with HPLC. Further sediment samples were analysed (with LC/MS) from the southeast of Norway in 2003 (Schlabach *et al.*, 2004). In the Drammensfjord, detectable concentrations of all three diastereomers were found at one station close to an industrial area.

A screening of the occurrence of HBCDD in the Norwegian environment was performed by Fjeld *et al*. (2005). Sediment samples were taken from the marine environment along the Norwegian coast up until around middle of Norway on the west coast from 11 localities. From each sampling station 5-8 samples were taken from the upper layer 0-2 cm. Very high levels were observed in Åsnefjord, which indicate a point source. At least one source has later been identified as a producer of EPS beads (Kaland, 2006).

Table 3-124 Levels of HBCDD in sediments in estuarine/brackish/marine waters within the EU, and Norway

Location	Level of HBCDD (µg/kg dwt)	Comment	Reference
IRL			**(de Boer *et al.*, 2002a)**
Dublin Bay	10, 6.9, 13, 2.2, 0.55*, 0.55*, 0.6*, 0.6*	Closest first, then with increasing distance	
NL			**(Bouma *et al.*, 2000)**
Terneuzen	25	Westerschelde	
	64	Westbuitenhaven	
	70	Oostbuitenhaven	
	151	Gent/Tern Canal	
NL			**(Klamer *et al.*, 2001)**
Southern part of NL section of the North Sea Continental Shelf			
Close to the NL coast	3.2, 4.2, 4.8, 5.2, 6, 6.6		
Further out at sea	0.8, 0.8, 0.9		

Location	Level of HBCDD (µg/kg dwt)	Comment	Reference
NL			**(de Boer *et al.*, 2002a)**
Western Scheldt	2.9, 0.35*, 9, 7.9*, 0.35*, 20, 0.25*, 23, 128, 0.25*, 18, 120, 1.8, 2.8, 1.1		
Hollands Diep	34	Estaury of Rhine and Meuse River	
Haringvliet West	14	Estuary	
Harlingvliet East	3.4		
NO			**(Schlabach *et al.*, 2004)**
Drammen fjord	0.45*	Main basin	
	14.14, 0.45*, 0.45*	Close to industrial site	
NO			**(Fjeld *et al.*, 2005)**
Trondheims fjord	1.7		
Hvaler	1.21		
Oslo fjord (inner)	0.39		
Bømlo	21		
Ulstein, Møre og Ro	2.81		
Oslofjord (outer)	0.28		
Åsnefjord	8024, 230, 24, 124	(Arithm mean of 8482.8 & 7565 which are the values for St 1 (core C), St 1 (core D); St 2, St 2b, St 3)	

*One, two, or all of the individual measurements (duplicate/triplicate, or individual diastereomers) resulting in the presented value was/were below the detection limit. For values below the detection limit half detection limit is used. If the analysis was performed using LC, and the values for the individual diastereomers were reported, the * can also mean that one, two or all three of the diastereomers were below the detection limit, and half detection limit is used for that/those diastereomers.

3.2.3.3 Comparison between predicted and measured levels

SURFACE WATER

Calculated PECs are found in **Error! Reference source not found.** to Table 3-121, but there exists no measured levels of HBCDD in seawater. It is therefore not possible to conclude on the accuracy of the EUSES modelling, and the calculated PECs will therefore be used in the risk characterisation.

SEDIMENT

Calculated PECs are found in **Error! Reference source not found.** to Table 3-121 and measured levels in Table 3-124.

In the Western Scheldt there are many data on measured levels of HBCDD, possibly reflecting that there are many known facilities that use HBCDD both for production and industrial use in the area. The concentrations detected at Terneuzen, The Netherlands, close to production plant B range from 25 (sampled in the Western Scheldt) to 151 µg/kg dwt (sampled at the Gent/Tern Canal). The calculated PEC in marine sediment for production plant B is 14 µg HBCDD/kg dwt which is about ten times lower than the value from the Gent/Tern Canal and about half of the lowest concentration.

The calculated generic PECs for EPS formulation in marine sediments is 610 or 2900 µg HBCDD/kg dwt, depending whether or not it is connected to a STP. This is in the range of what has been measured in the Norwegian Åsnefjord, 24-8024 µg HBCDD/kg dwt, where a manufacturer of EPS beads is situated.

The regional calculated PEC in marine sediment is 3.7 µg HBCDD/kg dwt, which is close to the median value, but clearly lower than the 90P for NL (which may be considered as a region), which are 5.2, and 110 µg/kg dwt, respectively. The calculated PEC is almost identical to the median value for all the 52 reported sediments values, which is 3.8 µg HBCDD/kg dwt.

The continental calculated PEC in marine sediment is 0.015 µg HBCDD/kg dwt, which is about 20 times lower than the lowest values reported in Table 3-71; 0.25 µg HBCDD/kg dwt (Western Scheldt; below detection limit → half detection limit used), and 0.28 µg HBCDD/kg dwt (measured in the outer Norwegian Oslo fjord). The median value in marine sediments (when values likely affected by local point sources are excluded) is 2.8 µg HBCDD/kg dwt and the arithmetic mean is 11 µg HBCDD/kg dwt. The median and the arithmetic mean are about 10 to 40 times higher than the lowest detected concentrations. Since the continental PEC should represent a value, not directly influenced by industrial activities, the calculated continental PEC for marine sediments is lower than what can be expected to be measured in background concentrations.

Relevant measurements to compare with the estimated PECs are rare, but the few available are in reasonable agreement with the corresponding PECs. The calculated regional PEC is in the range of measured median values, but the calculated continental PEC is lower than lowest reported background levels. Based on this the calculated PECs will be used in the risk characterisation.

3.2.4 Secondary poisoning

3.2.4.1 Calculation of PEC for secondary poisoning

Calculations of PECs are based on data presented in section 3.1.2.

PECs for secondary poisoning are calculated based on the assumption that the predator receives a certain % of its food from the local area and the remaining % from the regional area. For the marine predator the distribution is 50 % from the local area and 50 % from the regional, and for the marine top predator the distribution between the two are 10 % from the local area and 90 % from the regional. Thus, for sites having no local emissions the PEC reflects the regional concentration only, which becomes especially pronounced for the marine

top predator. $PEC_{oral\ marine\ predator\ (fish)}$ and $PEC_{oral\ marine\ top\ predator}$ for sites having no emissions to water are therefore not presented in the tables below.

3.2.4.1.1 Calculation of PEC for production and micronising of HBCDD

PECs in food of marine predators and marine top predators in the marine food chain for production and micronising of HBCDD is presented in Table 3-125.

Table 3-125 PECs in food of marine predators and marine top predators in the marine food chain for production of HBCDD

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ marinepredator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
		(mg HBCDD/kg wwt)	
ProdB	Yes	0.52	5.0

3.2.4.1.2 Calculation of PEC for formulation

FORMULATION OF COMPOUND FOR EPS AND HIPS

PECs in food of marine predators and marine top predators in the marine food chain for formulation of compound for EPS and HIPS are summarised in Table 3-126.

Table 3-126 PECs in food of marine predators and marine top predators in the marine food chain for formulation of compound for EPS and HIPS

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ marinepredator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
		(mg HBCDD/kg wwt)	
Site C	No	2.1	8.3
Site G	Yes	1.1	6.2
Site J	No	11	26
Site K	Yes	5.4	15
Site L	Yes	3.0	9.9
Site P	No	0.85	5.7
GEN_EPS_FORM	Yes	6	16
	No	27	58

FORMULATION OF XPS COMPOUND FOR THE MANUFACTURE OF XPS

Calculated PECs in food of marine predators and marine top predators in the marine food chains, in local sites with formulation of polystyrene compound containing HBCDD for the manufacture of XPS are summarised in Table 3-127.

Table 3-127 PECs in food of marine predators and marine top predators in the marine food chain for formulation of XPS compound .

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ marinepredator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
		(mg HBCDD/kg wwt)	
MasterbG	Yes	0.53	5.0
MasterbI	Yes	9.4	23
GEN_XPS_FORM	Yes	6.7	17
	No	31	65

FORMULATION OF POLYMER DISPERSIONS FOR TEXTILES

Calculated PECs in food of marine predators and top predators in the marine food chains, in local sites with formulation of polymer dispersions for textiles are summarised in Table 3-128.

Table 3-128 PECs in food of marine predators and top predators in the marine food chain for formulation of polymer dispersions for textiles.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ marinepredator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
		(mg HBCDD/kg wwt)	
TexForm1	Yes	0.52	5.0
	No	0.61	5.2
TexForm4	Yes	0.51	5.0
	No	0.60	5.1
TexFormB	Yes	2.5	9.0
	No	10	25
GEN_TEX_FORM	Yes	23	51
	No	110	230

3.2.4.1.3 Calculation of PEC for industrial use HBCDD

INDUSTRIAL USE OF EPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED EPS

Calculated PECs in food of marine predators and top predators in the marine food chains for the generic site with industrial use of polystyrene beads containing HBCDD at the manufacture of flame retarded EPS are summarised in Table 3-129.

Table 3-129 PECs in food of marine predators and marine top predators in the marine food chain for industrial use of EPS compound at the manufacture of flame retarded EPS.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ marinepredator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
		(mg HBCDD/kg wwt)	
GEN_EPS_IndUse	Yes	0.62	5.2
	No	1.1	6.2

INDUSTRIAL USE OF HIPS COMPOUND AT THE MANUFACTURE OF FLAME RETARDED HIPS

Calculated PECs in food of marine predators and top predators in the marine food chains, for the generic site with industrial use of polystyrene beads containing HBCDD at the manufacture of flame retarded HIPS are summarised in Table 3-130.

Table 3-130 PECs in food of marine predators and top predators in the marine food chain for industrial use of HIPS compound at the manufacture of flame retarded HIPS.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ marinepredator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
		(mg HBCDD/kg wwt)	
GEN_HIPS_IndUse	Yes	0.54	5.1
	No	0.68	5.4

INDUSTRIAL USE OF COMPOUND FOR FLAME RETARDED XPS

Calculated PECs in food of marine predators and marine top predators in the marine food chains, in local sites with industrial use of compound for flame retarded XPS are summarised in Table 3-131.

Table 3-131 PECs in food of marine predators and top predators in the marine food chain for industrial use of compound for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ marinepredator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
		(mg HBCDD/kg wwt)	
XPS 1	Yes	0.55	5.1
	No	0.76	5.5
XPS 3	Yes	0.78	5.6
	No	1.9	7.7
XPS 11	Yes	1.5	7.0
	No	5.4	15
GEN_XPS_IndUse	Yes	2.4	8.8
	No	9.6	23

INDUSTRIAL USE OF HBCDD POWDER FOR FLAME RETARDED XPS.

Calculated PECs in food of marine predators and top predators in the marine food chains, in local sites with industrial use of HBCDD powder for flame retarded XPS are summarised in Table 3-132.

Table 3-132 PECs in food of marine predators and top predators in the marine food chain for industrial use of HBCDD powder for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ marinepredator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
		(mg HBCDD/kg wwt)	
XPS 4	Yes	1.6	7.1
	No	5.6	15
XPS 8	Yes	0.50	5.0
	No	0.50	5.0
XPS 14	Yes	0.5	5.0
XPS 23	Yes	0.50	5.0
XPS 24	Yes	0.50	5.0
XPS27	Yes	1.1	6.2
	No	3.4	11

INDUSTRIAL USE OF TEXTILE BACK-COATING AGENT

Calculated PECs in food of predators in the aquatic terrestrial and marine food chains, in local sites with Industrial use of textile back-coating agent are summarised in Table 3-133.

Table 3-133 PECs in food of marine predators and top predators in the marine food chain for industrial use of textile back-coating.

Site	Connected to municipal STP (Yes/No)	$PEC_{oral\ marinepredator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
		(mg HBCDD/kg wwt)	
Backcoat.1	Yes	2.2	8.4
	No	8.7	21
Backcoat.2	Yes	0.9	5.8
	No	2.5	8.9
Backcoat.3	Yes	110	220
	No	520	1050
Backcoat.4	Yes	0.50	5.0
	No	0.51	5.0
BackcoatC	Yes	0.60	5.2
	No	1.0	6.0
GEN_TEX_IndUse	Yes	320	650
	No	1600	3100

3.2.4.2 Measured levels of HBCDD in biota in the marine environment

3.2.4.2.1 Introduction

Concentrations of HBCDD in tables Table 3-138 - Table 3-142 below are always presented in the form the values originally appeared (µg HBCDD/kg lwt, µg HBCDD/kg wwt, and/or µg HBCDD/kg dwt). If not given in the original article, concentrations on a wet weight basis were also calculated by the rapporteur and added to the table(s) when possible. In order to perform the calculation, information on lipid concentration and water content presented in the original study were used whenever available. If missing, the values on lipid and water content presented in Table 3-134 were used.

Table 3-134 Literature data on the lipid content on wet weight of fish and shell fish and water content

Specie	Lipid content (% of wwt)	Water content (%)	Reference
Low fat marine fish (e.g. cod)	0.74		**(Lind *et al.*, 2002)**
Herring	12.3		
Baltic sea salmon	9.55		
Eel	19.9		
Shellfish	1.04		
Baltic herring	9.3	65	http://www.slv.se

Specie	Lipid content (% of wwt)	Water content (%)	Reference
Bass, perch	0.61	79	
Bib	0.8	79	
Cod	0.7		
Haddock	0.6		
Halibut	4.8		
Hake	0.4		
Herring/sprat	18.5	65	
Mackerel	15		
Orfe, bream	4	78	
Plaice	0.61	77	
Saithe	0.9		
Salmon	12		
Sole, flounder	2.2	79	
Trout	3.3	75	
Turbot	1.7		
Whiting	0.6	78	
Crayfish	0.5		
Mussels	2.2	79	
Oysters	2		
Prawn	0.8	79	

In addition, for the marine mammals Common dolphin, Grey seal, Harbour porpoise, and Harbour seal concentrations on whole weight basis were calculated using the approximation that about 1/3 of their body weight consist of fat (Bäcklin, 2005). The whole weight concentration is calculated by dividing the concentration in the blubber (on wwt basis) with three. In case the original article only presents the concentration on a lwt basis, an approximation of a fat content of 88 % is used, based on the median concentration of fat in blubber of 85 Harbour porpoises from Law *et al*. (2006b).

3.2.4.2.2 Overview of the concentrations of HBCDD measured in brackish and marine biota (including marine mammals, polar bears and marine birds)

BRACKISH AND MARINE INVERTEBRATES

Concentrations detected in invertebrates in brackish and marine waters span more than four orders of magnitude, from parts of μg HBCDD/kg wwt (and below) in shrimp in the Western Scheldt and in mussels caught at the Norwegian coast to 329 μg HBCDD/kg wwt, measured in mussels sampled in the polluted Norwegian Åsne fjord. The distribution of all values,

including measurements affected by local point sources, is presented in Figure 3-9, and the corresponding descriptive statistics, in Table 3-135.

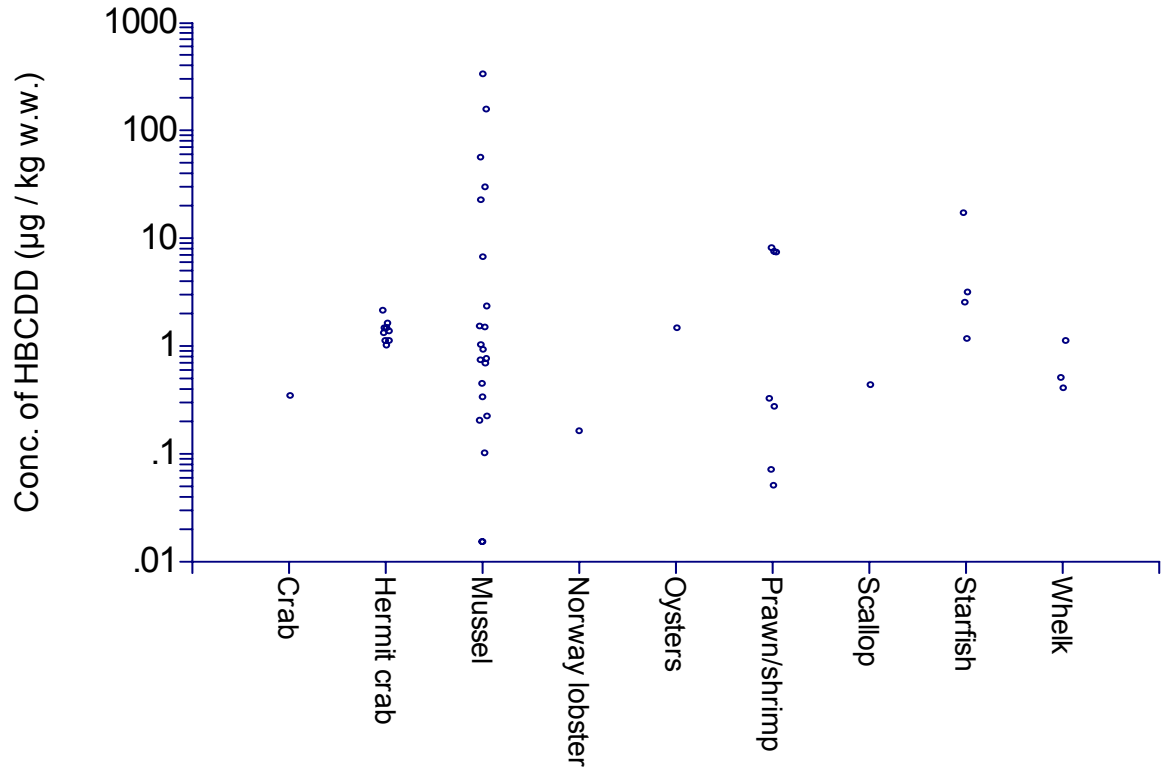


Figure 3-9 Dot plot of all measurements of HBCDD in brackish and marine invertebrates species.

Table 3-135 Descriptive statistic of measurements of HBCDD in brackish and marine invertebrates. All concentrations are in µg HBCDD/kg wwt The percentiles were calculated using Weighted Average at X(n+1)p.

	Conc.	n	Median	Mean±SD	90P	Min	Max
All values[1]	µg HBCDD/kg wwt	51	1.2	14±52	24	0.015	329
All values[1]	µg HBCDD/kg lwt	44	43	823±2913	1586	0.5	17337

[1]All values, including measurements considered affected by local point sources

BRACKISH AND MARINE FISH

Fish caught in brackish and marine waters contain HBCDD in the range of 0.001 µg HBCDD/kg wwt (half detection limit) in muscle of perch in the Baltic Sea to almost 50 µg HBCDD/kg wwt in muscle of eel (48 µg HBCDD/kg wwt) and gudgeon (49 µg HBCDD/kg wwt) caught in the Western Scheldt. Concentrations in liver range from 0.3 µg HBCDD/kg

wwt measured in cod caught in the outer parts of the Norwegian Oslo fjord to 89 µg HBCDD/kg wwt in livers of Sole caught in the Western Scheldt Estuary. The distribution of all values of HBCDD in fish muscle, including measurements affected by local point sources, is presented in Figure 3-10 and the corresponding descriptive statistics is presented in Table 3-136 below.

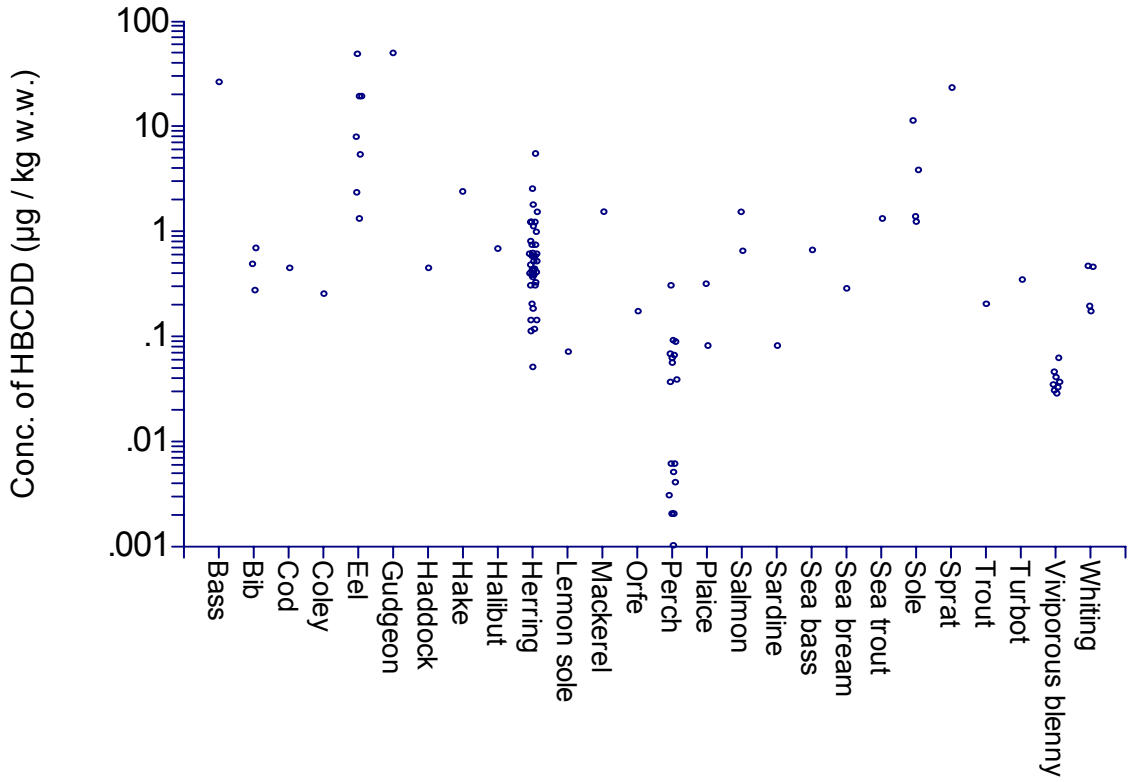


Figure 3-10 Dot plot of all measurements of HBCDD in muscle in brackish and marine fish species.

Table 3-136 Descriptive statistics of HBCDD concentrations in muscle of marine and brackish fish in the EU and Norway, all values included. The percentiles were calculated using Weighted Average at X(n+1)p.

	Conc.	n	Median	Geometric mean	Arithmetic mean±SD	90P	Min	Max
Marine fish	µg HBCDD/kg wwt	102	0.40	0.31	2.6±7.9	4.8	0.001	49
(muscle)	µg HBCDD/kg lwt	100	13	13	51±135	116	0.1	1113

MARINE MAMMALS AND POLAR BEARS

Marine mammals

HBCDD levels in European marine mammals (seal, porpoise, dolphin) range from 0.5 to 6400 µg/kg wwt (1.7-21345 µ/kg based on lipid weight), with a median wet weight concentration of 109 µg/kg wwt whole weight basis (368 µg HBCDD/kg based on lipid weight). The

distribution of all values is presented in Figure 3-11, with the corresponding descriptive statistics in Table 3-137.

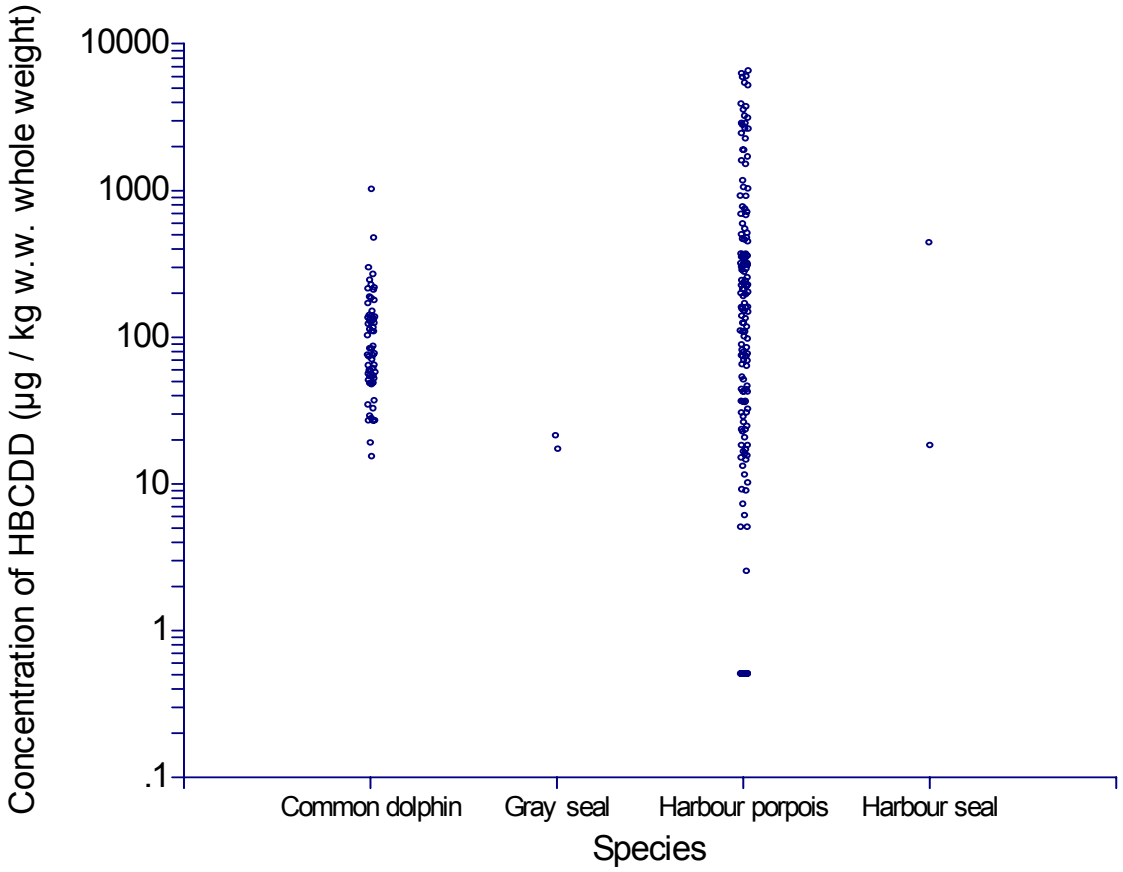


Figure 3-11 Dot plot of all measurements of HBCDD marine mammals presented on an approximate whole weight basis (conc. in blubber/3).

Table 3-137 Descriptive statistics of HBCDD concentrations in marine mammals in the EU. The percentiles were calculated using Weighted Average at X(n+1)p.

	Conc.	n	Median	Geometric mean	Arithmetic mean±SD	90P	Min	Max
Marine mammals	µg HBCDD/kg wwt, whole weight basis	225	109	91	498±1121	1517	0.5	6404
	µg HBCDD/kg lwt	225	368	311	1696±3781	5171	1.7	21345

Polar bears

HBCDD has been detected in adipose tissue from polar bears from Svalbard in the arctic region. In one of the two available studies, the arithmetic mean value was 26 µg HBCDD/kg wwt, with a range of 9.7-45 µg HBCDD/kg wwt (all of the 15 measurements were above the limit of detection). In the other, the reported concentration ranged between 5-15 µg HBCDD/kg lwt.

MARINE BIRDS

HBCDD have been analysed in different tissues of marine birds, mainly eggs. The levels of HBCDD in marine bird eggs range from a few µg/kg wwt, at remote islands in northern Norway, to around 100 µg/kg wwt, close to a production facility in the Netherlands.

A trend with increasing concentrations of HBCDD in eggs, regardless of specie and location of sampling, can be seen in Figure 3-12, below. The mean concentrations measured in Atlantic puffin, Herring gull, and Kittywake in the North of Norway have increased with a factor of about 5-8 over 20 years, from about 1-3 µg HBCDD/kg wwt in the 1983 to between 6-17 µg HBCDD/kg wwt in 2003, depending on species and location of sampling. The mean concentration measured in eggs from Guillemot from St. Karlsö in the Baltic Sea has approximately doubled from 8 µg HBCDD/kg wwt in the early 1970-ties to about 16 µg HBCDD/kg wwt in the late 1990-ties. However, according to the authors the increase appears to have levelled out since the mid-1990-ties.

No descriptive statistic will be presented since the concentrations reported are a result of measurements performed over time at the same location, with increasing concentrations reported over time. However, in order to display the highest concentrations measured in bird eggs, i.e. in eggs from Common tern sampled at Terneuzen, Figure 3-13 is also included below.

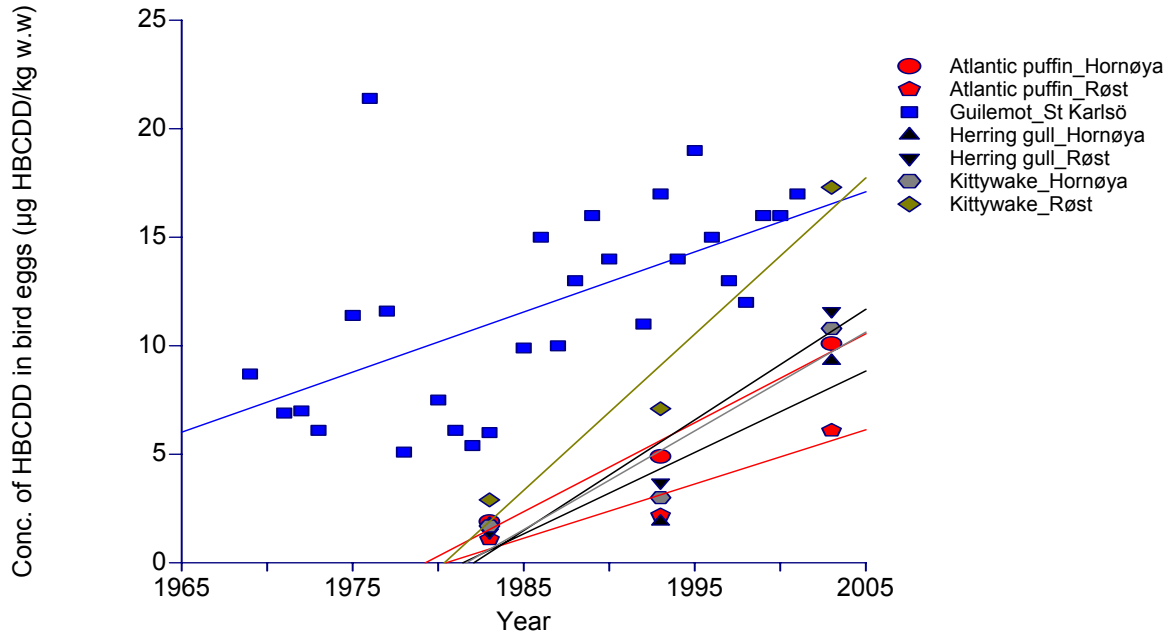


Figure 3-12 Mean/pooled concentrations of HBCDD in eggs from marine birds measured at different locations at time intervals.

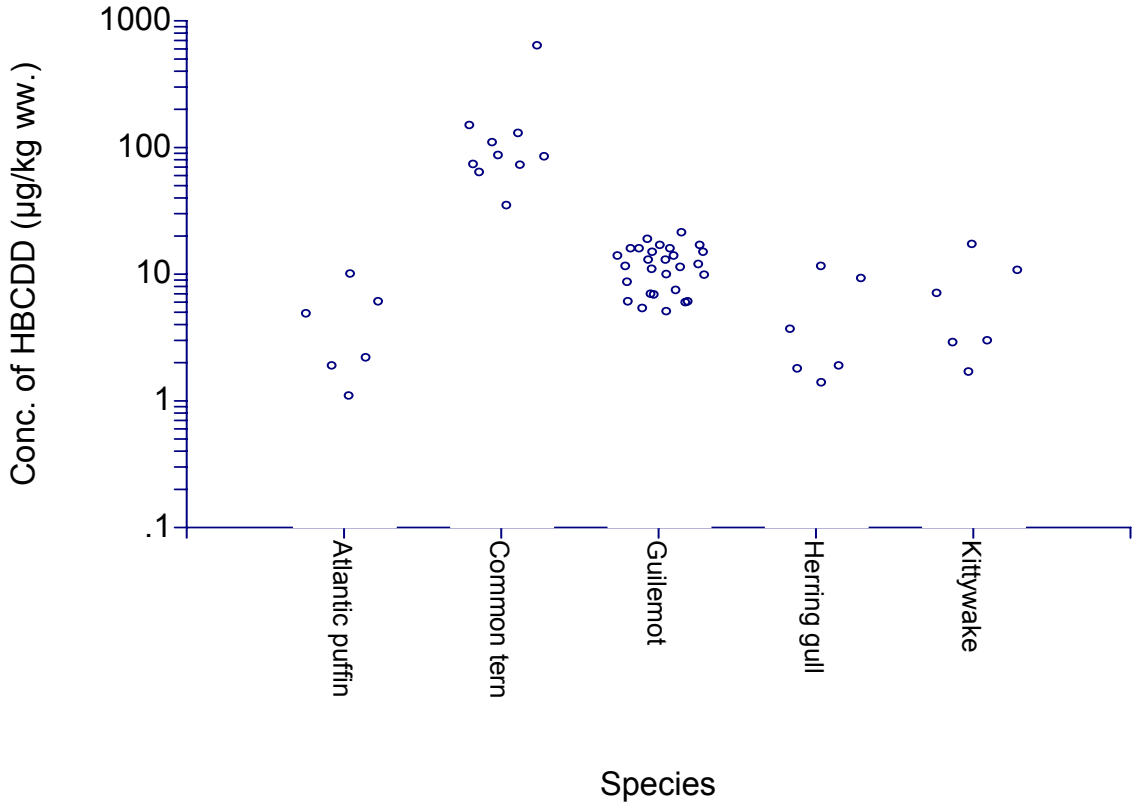


Figure 3-13 Concentration of HBCDD in eggs from marine birds.

3.2.4.2.3 Individual studies

BRACKISH AND MARINE INVERTEBRATES

An overview of the levels of HBCDD detected in invertebrates in brackish and marine waters was given in section 3.2.4.2.2 above. Results from the different studies included are presented below and in Table 3-138.

de Boer and co-workers (2002a) measured HBCDD in Hermit crabs (abdomen), Shrimps (whole), Starfish (whole and pyloric caeca), and Common Whelk (soft parts) caught in the North Sea, and in Starfish (whole) caught in the estuary of the river Tees in the UK. The concentrations detected ranged from 0.05 µg HBCDD/kg wwt (representing half detection limit) for the shrimp, to about 17 µg HBCDD/kg wwt that was detected in Starfish, sampled in the Tees estuary.

Bouma *et al*. (2000) detected 26 and 37 µg HBCDD/kg wwt in mussels sampled in the Western Scheldt Estuary in the vicinity of Terneuzen.

Schlabach and colleagues (2002) collected mussels in the Norwegian Oslo fjord, and on the Norwegian coast of Skagerrak. The concentrations detected ranges from parts of µg HBCDD/kg wwt to 1.5 µg HBCDD/kg wwt, with the highest value detected in the inner part of the Oslo fjord.

Göransson *et al.* (2004) detected HBCDD in mussels on the Swedish coast of Kattegatt. The concentrations ranged from about 0.4 µg HBCDD/kg wwt to 6.6 µg HBCDD/kg wwt, with the highest detected outside of a STP.

Fjeld and co-workers (2005) sampled mussels along the Norwegian coast and in Norwegian fjords. Most values ranged from about 0.2-2.3 µg HBCDD/kg wwt However, concentrations from 55-329 µg HBCDD/kg wwt were detected in the Åsne fjord, where a manufacturer of EPS beads is situated.

Verslycke *et al.* (2005) detected concentrations of about 7-8 µg HBCDD/kg wwt in mysid shrimps sampled in the Scheldt estuary, with higher concentrations upstream sites of the harbour of Antwerp. This indicated that the source might be spills during use of HBCDD in textile industries along river Scheldt near Antwerp or further upstream.

Janák and colleagues (2005a) measured about 0.3 µg HBCDD/kg wwt in shrimps collected in the Western Scheldt Estuary.

Jenssen *et al*. (2004) analysed a number of different species at different trophic levels in the arctic marine food web in the Svalbard area north of Norway in the arctic region, among them the lower pelagic zooplankton species *Calanus glacicalis*, *Thysanoessa inermis* and *Paratemisto libellula*. No HBCDD was detected, and no detection limit was given.

Most fish and shellfish species commonly available on the U.K. market, resulting in 24 species of fresh wild fish, seven of framed fish, seven of fresh shellfish and ten of canned or processed fish and shellfish were sampled between 2002 and 2004 (Anonymous, 2006). The samples that were analysed comprised composites consisting of 30 or 60 individual samples. The samples were analysed for brominated chemicals, including HBCDD which was analysed using HPLC-MS. Only species present in European waters are included. The concentrations detected in shellfish ranged from 0.04 µg HBCDD/kg wwt in prawns, which is below the detection limit (half detection limit is used) to 1.45 µg HBCDD/kg wwt in oysters.

Table 3-138 Measured levels of HBCDD in invertebrates in brackish and marine waters in the EU, and Norway

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
Crab (*Cancer pagurus*) (contents of displayed carapace)	Probably U.K. waters (specimens available on U.K. market)		0.34* (n = 60, pooled sample)	**(Anonymous, 2006)**
Hermit crab (abdomen)	North Sea	15*, 15*, 15*, 15*, 15*, 15*, 15*, 15*	1.1*, 1.0*, 1.3*, 1.6*, 1.35*, 1.1*, 1.45*, 2.1*, 1.45*	**(de Boer *et al.*, 2002a)**
Mussel (*Mytilus edulis*)	Western Scheldt Estuary, Terneuzen – The Netherlands	125, 177 µg HBCDD/kg dwt 1313[1], 1859[1] µg HBCDD/kg lwt	26[1], 37[1]	**(Bouma *et al.*, 2000)**
Mussel (*Mytilus edulis*) (drained contents of shell)	Probably U.K. waters (specimens available on U.K. market)	46[1]	1.01 (n = 60, pooled sample)	**(Anonymous, 2006)**
Mussel (*Mytilus edulis*)	Oslo fjord - Norway (highest value measured in the inner of	0.68[1], 0.68[1], 68[1]	0.015*, 0.015*, 1.5	**(Schlabach *et al.*, 2002)**

	the Oslo fjord) Skagerrak – Norweigian coast	 5.45[1], 10[1]	 0.12, 0.22	
Mussel (*Mytilus edulis*)	Kattegatt – Swedish coast (highest value measured outside of STP)	20, 33, 67, 300	0.44, 0.73, 1.47, 6.6	**(Göransson *et al.*, 2004)**
Mussel (*Mytilus edulis*)	Oslo fjord – Norway Vrengensundet, St. 3 Leangbukta, St. 2 Hvaler, St. 2 Atlantic - Norweigian coast/fjords Ulsteinvik havn, St. 1 Espevær Austvik Åsnefjord, St. 4 Åsnefjord, St. 5 Åsnefjord, St. 6	 52 84 411 131 76 3125 8807 17337	 0.33 0.91 2.3 0.22 0.68 0.75 55 155 329	**(Fjeld *et al.*, 2005)**
Mysid shrimp (*Neomysis integer*)	Scheldt Estuary Schaar van Warde Overloop van Valkenisse Bath	 569 562 727	 7.4, 7.3 8.0	**(Verslycke *et al.*, 2005)**
Norway lobster (*Nephros norvegicus*.) (muscle from tail)	Probably U.K. waters (specimens available on U.K. market)	32[1]	0.16 (n = 60, pooled sample)	**(Anonymous, 2006)**
Oysters (*Ostrea edulis and Crassostrea gigus*) (drained contents of shell)	Probably U.K. waters (specimens available on U.K. market)	73[1]	1.45 (n = 60, pooled sample)	**(Anonymous, 2006)**
Prawns, cold water (*Pandalid spp.*) (muscle from tail)	Probably U.K. waters (specimens available on U.K. market)	8.75[1]	0.07* (n = 60, pooled sample)	**(Anonymous, 2006)**
Scallops (*Pecten and Chlamys spp.*) (abductor muscle + roe)	Probably U.K. waters (specimens available on U.K. market)	20[1]	0.43 (n = 60, pooled sample)	**(Anonymous, 2006)**
Shrimp	Western Scheldt Estuary	0.5*	0.05*	**(de Boer *et al.*, 2002a)**
Shrimp (whole)	Western Scheldt Estuary	39, 46	0.27, 0.32	**(Janák *et al.*, 2005a)**
Starfish (pyloric caeca)	North Sea	84, 15*, 47	1.15*, 2.5, 3.1	**(de Boer *et al.*, 2002a)**

Starfish (whole)	Atlantic – UK river Tees estuary		17	**(de Boer *et al.*, 2002a)**
Whelk (soft parts)	North Sea	47, 30, 29	0.4, 0.5, 1.1	**(de Boer *et al.*, 2002a)**
Zooplankton *Calanus glacicalis* *Thysanoessa inermis* *Paratemisto libellula*	North Atlantic, Svalbard area (arctic region)	Not detected (no detection limit given)	Not detected (no detection limit given)	**(Jenssen *et al.*, 2004)**

*For concentration values below the detection limit, the concentration is assumed to be half the detection limit. In case the concentration only is presented for the individual diastereomers, the sum of the different diastereomers is used, and if the concentration for one or several of the individual diastereomers is below the detection limit, the concentration is assumed to be half the detection limit.
[1]Recalculated using values in Table 3-105.

BRACKISH AND MARINE FISH

An overview of the levels of HBCDD detected in fish in brackish and marine waters was given in section 3.2.4.2.2 above. Results from the different studies included are presented below and in Table 3-139.

Bouma *et al.* (2000) measured 26 and 23 µg HBCDD/kg wwt in muscle (?) of bass and sprat, respectively, sampled in Western Scheldt.

Janák and co-workers (2005a) detected HBCDD in muscle and liver from bib, eel (muscle only), plaice, sole, and whiting caught in the Western Scheldt Estuary. The concentrations detected in muscle ranged from of 0.2-0.3 µg HBCDD/kg wwt (bib and whiting, respectively) to 11 µg HBCDD/kg wwt (sole), and in liver from about 5 (whiting) to 89 µg HBCDD/kg wwt detected in liver of Sole.

Schlabach and colleagues (2002) presented concentrations of 0.3-9.9 µg HBCDD/kg wwt in livers of cod caught in the Norwegian Oslo fjord, with the highest values coming from the most inner parts of the fjord.

Fjeld *et al*. (2004); (2005) sampled cods from Norwegian coasts and fjords, and measured the concentration of HBCDD in their livers. The values ranged from 0.68-23 µg HBCDD/kg wwt, with the highest values detected in the inner parts of the Oslo and Sør fjords.

Bytingsvik and co-workers (2004) analysed livers from Atlantic cod caught at Hvaler at the estuary of river Glomma, Froan on the Atlantic west coast, and livers from polar cod caught at Bear Island in the arctic. The concentrations decreased significantly from south to north (both on wwt and lwt basis). A temporal trend for HBCDD in Atlantic cod caught at Hvaler was reported, as the concentration had increased significantly, 8 or 3-4 times from 1998 to 2003, when expressed on a wwt or lwt basis, respectively.

Schlabach, Fjeld, and Borgen (2004) analysed cod (liver), eel (muscle), flounder (liver), orfe (muscle), perch (muscle), trout (muscle) caught in the Drammen fjord in Norway for HBCDD. The concentrations detected in muscle range from about 0.2 µg HBCDD/kg wwt measured in orfe and trout (half detection limit) to 1.3 µg HBCDD/kg wwt measured in eel.

de Boer and co-workers (2002a) measured HBCDD in eel caught in the Western Scheldt Estuary and gudgeon in the Western Scheldt Estuary in the vicinity of Terneuzen. The

concentrations detected were 19-48 µg HBCDD/kg wwt and 49 µg HBCDD/kg wwt, in eel and gudgeon, respectively.

Sternbeck *et al.* (2001) analysed muscle samples from herring caught along the Swedish coast. The highest concentrations were found in the northern part of the Baltic Sea, however a direct comparison between the different locations is difficult since the age of the fishes differed. The detected concentrations ranged from about 0.6-5.4 µg HBCDD/kg wwt.

Nylund (2006) reported of concentrations of HBCDD in herring sampled at six sites along the Swedish coast, from the east coast to the west coast. The detected concentrations ranged from 0.05 to 0.38 µg HBCDD/kg wwt, with the highest and lowest concentrations detected on the east coast in the Baltic Sea.

Sternbeck and colleagues (2004) measured HBCDD in muscle of herring, perch, and viviparous blenny caught in lakes and along the Swedish coast at Swedish background locations. No obvious difference between limnic and marine/brackish locations could be observed. The concentrations detected ranged from 0.2-1.2 µg HBCDD/kg wwt, 0.002 (half detection limit) -0.09 µg HBCDD/kg wwt, and 0.03-0.06 µg HBCDD/kg wwt, for herring, perch, and viviparous blenny, respectively.

Lundstedt-Enkel (2006) reported of measurements of HBCDD in muscle of herring caught in the Baltic Sea November-December 2000. The arithmetic mean concentration was 0.51 µg HBCDD/kg wwt and the range was 0.02-1.9 µg HBCDD/kg wwt.

Jenssen *et al*. (2004) analysed a number of different species at different trophic levels in the arctic marine food web in the Svalbard area north of Norway in the arctic region, among them polar cod. The concentrations detected in liver (not clear from the study report) in polar cod ranged from 5-25 µg HBCDD/kg lwt.

Most fish and shellfish species commonly available on the U.K. market, resulting in 24 species of fresh wild fish, seven of framed fish, seven of fresh shellfish and ten of canned or processed fish and shellfish were sampled between 2002 and 2004 (Anonymous, 2006). The samples that were analysed comprised composites consisting of 30 or 60 individual samples. The samples were analysed for brominated chemicals, including HBCDD which was analysed using HPLC-MS. Only species present in European waters are included. The concentrations detected in fish ranged from 0.08 µg HBCDD/kg wwt in plaice which is below the detection limit (half detection limit is used) to 5.29 µg HBCDD/kg wwt in eel.

Table 3-139 Measured levels of HBCDD in fish in marine and brackish waters in the EU and Norway

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt (unless otherwise stated)	µg HBCDD/kg wwt	
Bass (muscle?)	Westerschelde	124 µg HBCDD/kg dwt	26[1]	**(Bouma *et al.*, 2000)**
Bib (*Trisopterus luscus*) (muscle)	Western Scheldt Estuary	55*, 96, 136	0.27*, 0.48, 0.68	**(Janák *et al.*, 2005a)**
Bib (*Trisopterus luscus*) (liver)	Western Scheldt Estuary	78, 110, 155	42, 59, 84	**(Janák *et al.*, 2005a)**
Cod (*Gadus morhua*) (liver)	Skagerrak, Oslo fjord - Norway		0.3, 3.2, 7.3, 9.9	**(Schlabach *et al.*, 2002)**

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt (unless otherwise stated)	µg HBCDD/kg wwt	
	(two highest values measured in the inner of the Oslo fjord)			
Cod (*Gadus morhua*) (liver; homogenate from 5-20 individuals)	Skagerrak, Drammen fjord – Norway	9.9*	4.3*	**(Schlabach *et al.*, 2004)**
Cod (*Gadus morhua*) (α-diastereomer only; liver; homogenate from 7 individuals/value)	Skagerrak, Drammen fjord – Norway	9.3	4.05	**(Fjeld *et al.*, 2004)**
	Outer of Oslo fjord, Færder	9.3	0.68	
	Atlantic - Norweigian coast/fjords			
	Ullerøy-area/Lista	7.7	4.45	
	Karihavet	2.4	1.15	
	Lofoten, Svolvær	6.6	4.91	
	Varangerfjorden	7.7	4.0	
Cod (*Gadus morhua*) (liver; homogenate from 7 individuals/value)	Skagerrak, Hvaler – Norway 1998	n.d.-23 (min-max) n = 11	n.d.-2.7 (min-max) n = 11	**(Bytingsvik *et al.*, 2004)**
	Hvaler – Norway 2003	n.d.-51 (min-max) n = 14	n.d.-13 (min-max) n = 14	
	Atlantic Froan – Norway 2003	n.d.-51 (min-max) n = 14	n.d.-13 (min-max) n = 14	

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt (unless otherwise stated)	µg HBCDD/kg wwt	
Cod (*Gadus morhua*) (liver)	Skagerrak, Oslo fjord – Norway Færder Inner of Oslo fjord Inner of Oslo fjord Atlantic - Norweigian coast/fjords Ulsteinvik, St. 3 Svolvær St. 2 Bømlo/Sotra, St. 2 Lista, St. 2 Inner of Sørfjord		 5.8 23 23 2.5 2.8 7.2 7.6 20	**(Fjeld *et al.*, 2005)**
Cod (*Gadus morhua*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	63[1]	0.44 (n = 60, pooled sample)	**(Anonymous, 2006)**
Coley/Saithe(*Pallohius virens*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	28[1]	0.25* (n = 60, pooled sample)	**(Anonymous, 2006)**
Eel (*Anguilla anguilla*) (muscle)	Western Scheldt Estuary	86, 100, 310	19, 19, 48	**(de Boer *et al.*, 2002a)**
Eel (*Anguilla anguilla*) (muscle; homogenate from 5-20 individuals)	Skagerrak, Drammen fjord – Norway	6.55*	1.3	**(Schlabach *et al.*, 2004)**
Eel (*Anguilla anguilla*) (muscle)	Western Scheldt Estuary	9, 30	2.3, 7.8	**(Janák *et al.*, 2005a)**
Eel (*Anguilla spp*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	27[1]	5.29 (n = 60, pooled sample)	**(Anonymous, 2006)**
Flounder (*Platichtys flesus*) (liver; homogenate from 5-20 individuals)	Skagerrak, Drammen fjord – Norway	8.95*	0.69*	**(Schlabach *et al.*, 2004)**
Gudgeon (*Gobio gobio*) (muscle; muscle homogenate from 25 individuals)	Western Scheldt-Terneuzen (year 2001)	230	49	**(de Boer *et al.*, 2002a)**
Haddock (*Melanogammus aeglefinus*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	73[1]	0.44 (n = 60, pooled sample)	**(Anonymous, 2006)**
Hake (*Merluccius*	Probably U.K. waters	588[1]	2.35* (n = 60, pooled	**(Anonymous, 2006)**

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt (unless otherwise stated)	µg HBCDD/kg wwt	
merluccius) (muscle))	(specimens available on U.K. market)		sample)	
Halibut (*Hippoglossus hippoglossus*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	14[1]	0.67* (n = 60, pooled sample)	**(Anonymous, 2006)**
Herring (*Clupea harengus*) (muscle)	Baltic Sea – Swedish coast (year 2004)			**(Nylund, 2006)**
	Harufjärden	1.18[1]	0.11±0.026 (mean±SD) n = 12	
	Ängskärsklubb	0.54[1]	0.05±0.032 (mean±SD) n = 12	
	Landsort	4.09[1]	0.38±0.27 (mean±SD) n = 12	
	Utlängan	1.24[1]	0.115±0.082 (mean±SD) n = 12	
	Kattegatt – Swedish coast			
	Fladen	0.76[1]	0.14±0.034 (mean±SD) n = 12	
	Skagerack – Swedish coast			
	Väderöarna	0.97[1]	0.18±0.035 (mean±SD) n = 20	
Herring (*Clupea harengus*) (muscle; each value represent a homogenate from 10 individuals)	Baltic Sea – Swedish coast			**(Sternbeck *et al.*, 2001)**
	Bergöfjärden	180	5.4	
	Ängskärklubb	58	1.5	
	Landsort	34	1.2	

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt (unless otherwise stated)	µg HBCDD/kg wwt	
	Utlängan	58	2.5	
	Kattegatt – Swedish coast Fladen	26	0.57	
	Skagerack – Swedish coast N Skagen	21	1.2	
Herring (*Clupea harengus*) (muscle)	Baltic Sea – Swedish coast			**(Sternbeck *et al.*, 2004)**
	Harufjärden	8.8, 17, 18, 11, 12, 14, 5.8, 13	0.51, 0.42, 0.40, 0.30, 0.42, 0.32, 0.14, 0.36	
	Utlängan	26, 31, 38, 36, 21, 26, 35, 26	0.97, 0.79, 1.1, 1.2, 0.73, 0.60, 0.73, 0.57	
	Kattegatt – Swedish coast Fladen	6.6, 8.8, 8.6, 9.1, 6.2, 5.9, 6.6, 7.6	0.38, 0.39, 0.43, 0.47, 0,20, 0.30, 0.61, 0.60	
Herring (*Clupea harengus*) (muscle)	Baltic Sea – Swedish coast			**(Lundstedt-Enkel, 2006)**
	Landsort, Gotland, Utlängan	13±7.6 (mean±SD) 1.4-36 (min-max) n = 60	0.51±0.41 (mean±SD) 0.02-1.9 (min-max) n = 60	
Herring (*Clupea harengus*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	9.51[1]	1.76* (n = 30, pooled sample)	**(Anonymous, 2006)**
Lemon sole (*Microstomus kitt*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	3.5[1]	0.07* (n = 60, pooled sample)	**(Anonymous, 2006)**
Mackerel (*Scomber scombrus*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	10[1]	1.51* (n = 60, pooled sample)	**(Anonymous, 2006)**
Orfe (*Leuciscus idus*) (muscle; homogenate from 5-20 individuals)	Skagerrak, Drammen fjord – Norway	19*	0.17*	**(Schlabach *et al.*, 2004)**
Perch (*Perca fluviatilis*) (muscle)	Baltic Sea			**(Sternbeck *et al.*, 2004)**
	Holmöarna	9.8, 6.6, 7.4, 4.5, 5.3, 7.2, 4.8, 7.7	0. 087, 0.067, 0.061, 0.038, 0.055, 0.065, 0.036, 0.090	

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt (unless otherwise stated)	µg HBCDD/kg wwt	
	Kvädöfjärden	0.8*, 0.1*, 0.6*, 0.4*, 0.65*, 0.3*, 0.85*, 1.2*	0.005*, 0.001*, 0.003*, 0.002*, 0.004*, 0.002*, 0.006*, 0.006*	
Perch (*Perca fluviatilis*) (muscle; homogenate from 5-20 individuals)	Skagerrak, Drammen fjord – Norway	25	0.3*	**(Schlabach *et al.*, 2004)**
Plaice (*Pleuronectes platessa*) (muscle)	Western Scheldt Estuary	39*	0.31*	**(Janák *et al.*, 2005a)**
Plaice (*Pleuronectes platessa*) (liver)	Western Scheldt Estuary	27, 29, 32	9.2, 9.9, 10.9	**(Janák *et al.*, 2005a)**
Plaice (*Pleuronectus platessa*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	13[1]	0.08* (n = 60, pooled sample)	**(Anonymous, 2006)**
Polar cod (*Boreogadus saida*) (liver?)	North Atlantic, Svalbard area (arctic region)	5-25 (range)		**(Jenssen *et al.*, 2004)**
Polar cod (*Boreogadus saida*) (liver)	North-East Atlantic Froan – Norway 2003	Median: 13.8 Arithm. mean: 14.7 7.67-23 (min-max) n = 6	Median: 1.99 Arithm. mean: 2.39 0.53-5.66 (min-max) n = 6	**(Bytingsvik *et al.*, 2004)**
Salmon (*Salmo salar*) (muscle)	Baltic Sea Gotland	15±4.4 (mean±SD) 8.7-29 (min-max) n = 20	1.5±0.7 (mean±SD) 0.6-2.7 (min-max) n = 20	**(Lundstedt-Enkel, 2006)**
Salmon (*Salmo salar*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	5.33[1]	0.64* (n = 30, pooled sample)	**(Anonymous, 2006)**
Sardine (*Sardina pilchard*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	0.43[1]	0.08* (n = 60, pooled sample)	**(Anonymous, 2006)**
Sea bass (*Dicentrarchus labrax*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	107	0.65 (n = 60, pooled sample)	**(Anonymous, 2006)**
Sea bream (*Sparus aurata*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	28[2]	0.28 (n = 60, pooled sample)	**(Anonymous, 2006)**
Sea trout/	Probably U.K. waters	39	1.30 (n = 60, pooled	**(Anonymous, 2006)**

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt (unless otherwise stated)	µg HBCDD/kg wwt	
(*Oncorhynchus mykiss*) (muscle)	(specimens available on U.K. market)		sample)	
Sole (*Solea vulgaris*) (muscle)	Western Scheldt Estuary	121, 136, 377, 1113	1.21, 1.36, 3.77, 11	**(Janák *et al.*, 2005a)**
Sole (*Solea vulgaris*) (liver)	Western Scheldt Estuary	100*, 118, 151*, 681*	13*, 15, 20*, 89*	**(Janák *et al.*, 2005a)**
Sprat (*Sprattus sprattus*) (muscle?)	Westerschelde	66 µg HBCDD/kg dwt	23	**(Bouma *et al.*, 2000)**
Trout (*Salmo trutta*) (muscle; homogenate from 5-20 individuals)	Skagerrak, Drammen fjord – Norway	1.7*	0.2*	**(Schlabach *et al.*, 2004)**
Turbot (*Psetta maxima*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	20[1]	0.34 (n = 60, pooled sample)	**(Anonymous, 2006)**
Viviporous blenny (*Zoarces viviparous*) (muscle)	Skagerrak – Swedish coast Väderöarna	5.4, 4.8, 7.2, 5.0, 5.5, 9.2, 6.2, 5.3	0.036, 0.032, 0.040, 0.046, 0.030, 0.061, 0.034, 0.028	**(Sternbeck *et al.*, 2004)**
Whiting (*Merlangius merlangus*) (muscle)	Western Scheldt Estuary	47*, 113, 116	0.19*, 0.45, 0.46	**(Janák *et al.*, 2005a)**
Whiting (*Merlangius merlangus*) (liver)	Western Scheldt Estuary	18*, 179, 275	5.25*, 54, 83	**(Janák *et al.*, 2005a)**
Whiting (*Merlangius merlangus*) (muscle)	Probably U.K. waters (specimens available on U.K. market)	28	0.17* (n = 60, pooled sample)	**(Anonymous, 2006)**

*For concentration values below the detection limit, the concentration is assumed to be half the detection limit. In case the concentration only is presented for the individual diastereomers, the sum of the different diastereomers is used, and if the concentration for one or several of the individual diastereomers is below the detection limit (often the γ-diastereomer), the concentration is assumed to be half the detection limit. This asterisk thus indicate that the entire value, or part of it was below detection limit, and therefore that half the detection limit was used for that fraction(s).

[1]Recalculated using values in Table 3-105.

[2]Recalculated using an approximative fat content of 1 percentage (**(Grigorakis *et al.*, 2002)**).

MARINE MAMMALS AND POLAR BEARS

Marine mammals

An overview of the levels of HBCDD detected in fish in marine mammals was given in section 3.2.4.2.2 above. Results from the different studies included are presented below and in Table 3-140.

In a recent report Law and co-workers (2006b) presented concentrations of HBCDD in blubber of 85 female and male harbour porpoises stranded or dying due to physical trauma in the U.K. during the period 1994-2003. α-HBCDD dominated over the other diastereomers and was detected in all samples at concentrations ranging from 10 to 19210 µg/kg wwt (3-6403 µg HBCDD/kg wwt on whole weight basis). Investigation of possible trends indicated a sharp increase in HBCDD concentrations from about 2001 onward, which was not confounded be age (length), sex, nutritional status, or location.

Zegers *et al* (2005) reported levels of HBCDD in blubber of female 43 harbour porpoise and 61 common dolphin from different European seas. The highest concentrations were measured in harbour porpoises stranded on the Irish and Scottish coasts of the Irish Sea (median: 971 µg HBCDD/kg wwt (whole weight basis); range 310-2929 µg HBCDD/kg wwt (whole weight basis) and the northwest coast of Scotland (median: 1685 µg HBCDD/kg wwt whole weight basis); range 336-3197 µg HBCDD/kg wwt whole weight basis). The lowest concentrations were observed for the mammals stranded at the Galician coast, median values 56 and 47 µg HBCDD/kg wwt whole weight basis for dolphins and harbour porpoise, respectively.

Roos *et al*. (2001) analysed blubbers from twenty juvenile male grey seals from the Baltic Sea for HBCDD. The seals were divided into three groups: healthy seals collected 1980-1985 and two groups from the 1990s: one having no or slight intestinal ulcers, and one having moderate, severe or fatal intestinal ulcers. No obvious differences in concentrations were found between the three groups that could be connected with the disease other than the concentration of HBCDD were lower in the 1980s. A different presentation of the same data (Roos, 2006) indicated an increase of HBCDD from the 1980-ties (median value 8.2 µg HBCDD/kg wwt whole weight basis; n = 7) to the 1990-ties (median value 21 µg HBCDD/kg wwt whole weight basis; n = 12). It was however not possible to say if the increase continued beyond the 1990-ties into the 2000-ties as only one value is available (19 µg HBCDD/kg wwt whole weight basis; n = 1).

Lundstedt-Enkel (2006) reported of measurements of HBCDD in blubber and muscle of female and male grey seals from the Baltic Sea from 2000-2002. The median concentration was 25 µg HBCDD/kg wwt whole weight basis (n = 30).

Twenty five porpoise blubbers collected through the UK national marine mammals stranding programme were analysed for presence of HBCDD (Anonymous, 2002a). The levels ranged from below detection (half detection limit is used) -306 µg HBCDD/kg wwt whole weight basis, with most values being below the limit of detection.

de Boer and colleagues (2002a) measured HBCDD in blubber and liver from nine harbour porpoise and two harbour seal from the North Sea/UK east coast/West Wadden Sea. The concentration detected ranged from 7.5 µg HBCDD/kg wwt whole weight basis (half detection limit) in harbour porpoise stranded on the UK east coast to 1667 µg HBCDD/kg wwt whole weight basis in harbour porpoise that had drowned in fishing nets. The

concentrations measured in Harbour seal from the West Wadden Sea were 18 and 433 µg HBCDD/kg wwt whole weight basis.

Jenssen *et al*. (2004) analysed a number of different species at different trophic levels in the arctic marine food web in the Svalbard area north of Norway in the arctic region, among them ringed seal. The concentrations detected in blubber (?) in ringed seal ranged from 15-35 µg HBCDD/kg lwt.

Table 3-140 Measured levels of HBCDD in marine mammals in the EU, and calculated on a whole weight basis assuming that about 1/3 of the body weight consists of fat and the wet weight is 88 % of the lipid weight

Species	Location	Concentration			Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	µg HBCDD/kg wwt, whole weight basis	
Common dolphin (*Delphinus delphis*) Female (blubber)	ES Atlantic Ocean (Galician north and west coast)	64, 281, 251, 184, 259, 291, 90, 368, 205, 454, 109, 160, 91, 171, 52, 124, 216, 162, 163, 116, 247, 94, 90, 182	56, 247, 221, 162, 228, 256, 80, 324, 180, 400, 96, 141, 80, 150, 45, 109, 190, 143, 144, 102, 217, 82, 79, 160	19, 82, 74, 54, 76, 85, 27, 108, 60, 133, 32, 47, 27, 50, 15, 36, 63, 48, 48, 34, 72, 27, 26, 53	**(Zegers *et al.*, 2005)**
	IRL Atlantic ocean (Irish west coast)	727, 411, 193, 3416, 1591, 998	640, 362, 170, 3006, 1400, 878	213, 121, 57, 1002, 467, 293	
	FR Western English channel (between Normandy and Brest)	175, 630, 279, 98, 180, 418, 344, 199, 389, 899, 461, 717, 188, 197, 215, 253, 236, 443, 619, 472, 439, 597, 418, 366, 569, 468, 761, 703, 821, 504, 378	154, 555, 246, 86, 159, 368, 303, 175, 342, 791, 405, 631, 166, 174, 189, 223, 207, 390, 545, 416, 386, 526, 368, 322, 501, 412, 669, 618, 722, 443, 332	51, 185, 82, 29, 53, 123, 101, 58, 114, 264, 135, 210, 55, 58, 63, 74, 69, 130, 182, 139, 129, 175, 123, 107, 167, 137, 223, 206, 241, 148, 111	
Grey seal (*Halicoerus grypus*) Male (blubber)	SE Data presented in Roos et al., 2001 Baltic Sea	median: 59 range (min-max): 16-177 n: 20 geometric. mean group 1: 26 geometric. mean group 2: 61	median: 52 range (min-max): 14-156 n: 20 geometric. mean group 1: 23 geometric. mean group 2: 54	median: 17 range (min-max): 4.7-52 n: 20 geometric. mean group 1: 7.6 geometric. mean group 2: 18	**(Roos *et al.*, 2001)**

	Same data as above presented in Roos, 2006 Baltic Sea 1980-ties 1990-ties Year 2000	geometric. mean group 3: 91 median: 28 aritm. mean: 24 range (min-max): 16-35 n: 7 median: 73 aritm. mean: 83 range (min-max): 34-177 n: 12 64	geometric. mean group 3: 80 median: 28 aritm. mean: 24 range (min-max): 14-31 n: 7 median: 64 aritm. mean: 73 range (min-max): 30-156 n: 12 56	geometric. mean group 3: 27 median: 8.2 aritm. mean: 7.0 range (min-max): 4.7-10 n: 7 median: 21 aritm. mean: 24 range (min-max): 10-52 n: 12 19	**(Roos, 2006)**
Grey seal (*Halicoerus grypus*) (blubber) (muscle)	SE Baltic Sea (year 2000-2002)	median: 75 101±98 (mean±SD) 31-554 (min-max) n = 30 51±20 (mean±SD) 30-87 (min-max) n = 30	median: 62.4 67±34 (mean±SD) 14-152 (min-max) n = 30 1±0.8 (mean±SD) 0.36-2.5 (min-max) n = 30	median: 21 22±11 (mean±SD) 5-51 (min-max) n = 30	**(Lundstedt-Enkel, 2006)**
Harbour porpoise (*Phocoena phocoena*) (blubber)	UK: Cardigan Bay Dyfed Humber	1.7*,1.7* 1.7* 1.7*, 102, 1.7*, 1043, 1.7*, 1.7*, 1.7* 1.7*	1.5*,1.5* 1.5* 1.5*, 89, 1.5*, 917, 1.5*, 1.5*, 1.5* 1.5*	0.5*0.5* 0.5* 0.5*, 30, 0.5*, 306, 0.5*, 0.5*, 0.5* 0.5*	**(Anonymous, 2002a)**

	Mounts Bay Tyne/Tees Swansea Bay Camarthen Bay Channel West Liverpool Bay Southern Bight Channel East Thames Celtic Sea	365, 61 1.7* 1.7* 1.7* 1.7*, 1.7* 1.7*, 1.7* 17, 1.7* 34	322, 54 1.5* 1.5* 1.5* 1.5*, 1.5* 1.5*, 1.5* 16, 1.5* 30	107, 18 0.5* 0.5* 0.5* 0.5*, 0.5* 0.5*, 0.5* 5, 0.5* 10	
Harbour porpoise (*Phocoena phocoena*) (blubber)	NL North Sea UK East coast	3584, 729, 6275, 880 1043, 365, 61, 102, 8.5	3100, 670, 5000, 750 917, 322, 54, 89, 7.5*	1033, 223, 1667, 250 306, 107, 18, 30, 2.5*	**(de Boer *et al.*, 2002a)**
(liver)	NL North Sea	3925	1600		
Harbour porpoise (*Phocoena phocoena*) Female (blubber)	ES Atlantic Ocean (Galician north and west coast)	79, 143, 142	70, 125, 125	23, 42, 42	**(Zegers *et al.*, 2005)**
	IRL Celtic Sea (Irish south coast)	2269, 1158, 710	1997, 1019, 625	666, 340, 208	
	NL/BE/FR: Southern North Sea	679, 1219, 1233, 538, 666, 1676, 1160, 816, 1597, 1065, 1144, 2312, 754	597, 1072, 1085, 473, 586, 1475, 1021, 718, 1405, 937, 1007, 2034, 664	199, 357, 362, 158, 195, 492, 340, 239, 468, 312, 336, 678, 221	
	UK Irish Sea (Scottish west coast south of Campbeltown and Irish east coast)	931, 6301, 5343, 466, 3441, 1981, 8786, 2383	819, 5545, 4702, 410, 3028, 1743, 7732, 2097	273, 1848, 1567, 137, 1009, 581, 2577, 699	
	Northern North Sea (Scottish east coast)	449, 781, 2593, 770, 497, 1054, 1564, 1068, 744, 393, 638	395, 688, 2282, 678, 437, 928, 1377, 940, 655, 346, 561	132, 229, 761, 226, 146, 309, 459, 313, 218, 115, 187	

	Northern North Sea (Northwest coast of Scotland)	1503, 9590, 6325, 1009, 5056	1322, 8440, 5566, 888, 4450	441, 2813, 1855, 296, 1483	
Harbour porpoise (*Phocoena phocoena*) Female, male (blubber)	UK 1994 E. England 1995 Scotland 1996 E. England Scotland W. England and Wales 1997 Scotland 1998 E. England Scotland 1999 E. England Scotland W. England and Wales 2000 E. England Scotland W. England and Wales 2001 Scotland W. England and	 78* 16*, 52* 151*, 117* 37*, 29*, 24*, 122 45 85*, 492* 99* 240, 30*, 19*, 74*, 55* 96* 248*, 529*, 330* 56* 52*, 55*, 129*, 291, 305, 231* 221* 67*, 274*, 55*, 276*, 263*, 121* 326, 409, 144, 684, 204*, 170*, 12892, 647, 2924, 234* 1036, 936*, 1794*, 1694*, 255*	 69* 15*, 45* 130*, 108* 34*, 27*, 22*, 106 39 78*, 443* 85* 221, 27*, 18*, 67*, 51 73 219*, 471*, 287* 49* 43*, 47*, 108*, 250, 262, 130* 204* 61*, 233*, 47*, 241*, 218*, 107* 297, 368, 125, 622, 188*, 152*, 10958, 576, 2690, 206 912, 711, 1615, 1508, 227	 23* 5*, 15* 43*, 36* 11*, 9*, 7*, 35 13 26*, 148* 28* 74, 9*, 6*, 22*, 17* 24* 73, 157*, 96* 16* 14*, 15*, 36*, 83, 87, 43* 68* 20*, 78*, 16*, 80*, 73*, 36* 99, 123, 42, 207, 63*, 51*, 3653, 192, 897, 69* 304, 237*, 538*,	**(Law *et al.*, 2006b)**

	Wales 2002 E. England Scotland W. England and Wales 2003 E. England Scotland W. England and Wales	 945*, 3775* 551*, 927*, 362, 213, 106*, 20249, 11475, 407, 551, 8809, 1541*, 1198 1238, 3005*, 20013*, 17209 12577*, 9112*, 9910*, 21345*, 18111, 10401*, 1242*, 7818, 2594*, 9616* 18928*, 539* 181*, 154*, 1176*, 10441*	 860*, 3436* 501*, 844*, 326, 192, 96*, 18427, 10442, 366, 468, 7223, 1357*, 1054 1089, 2705*, 17612*, 15316 11445*, 7745*, 8225*, 19211*, 15938, 9465, 1081*, 6645, 2205, 8463 17414*, 458* 158*, 137*, 1035*, 9189*	503*, 76* 287*, 1145* 167*, 281*, 109, 64, 32*, 6142, 3481, 122, 156, 2408, 452*, 351 393, 902*, 5871*, 5105 3815*, 2582*, 2742*, 6404*, 5313, 3155*, 360*, 2215, 735*, 2821* 5805*, 153* 53*, 46*, 345*, 3063*	
Harbour seal (*Phocoena phocoena*) (blubber) (liver)	NL Western Wadden Sea (North Sea) Western Wadden Sea (North Sea)	2055, 63 9.5*, 18*, 21*	1300, 53 0.6*, 0.35*, 0.45*	433, 18	**(de Boer *et al.*, 2002a)**
Ringed seal (*Phoca hispida*) (blubber?)	NO North Atlantic, Svalbard area (arctic region)	15-35 (min-max)			**(Jenssen *et al.*, 2004)**

*For concentration values below the detection limit, the concentration is assumed to be half the detection limit. In case the concentration only is presented for the individual diastereomers, the sum of the different diastereomers is used, and if the concentration for one or several of the individual diastereomers is below the detection limit (often the γ-diastereomer), the concentration is assumed to be half the detection limit. This asterisk thus indicate that the entire value, or part of it was below detection limit, and therefore that half the detection limit was used for that fraction(s).

Polar bears

A presentation of the levels of HBCDD detected in polar bears was given in the introduction in section 3.2.4.2.2 above. Results from the different studies included are presented below and in Table 3-141.

Gabrielsen *et al.* (2004) measured halogenated organic contaminants, including HBCDD, in adipose tissue of Polar Bears from Svalbard north of Norway in the arctic region. The

arithmetic mean value was 25.6 µg HBCDD/kg wwt, with a range of 9.7-45 µg HBCDD/kg wwt (all of the 15 measurements were above the limit of detection).

Jenssen *et al*. (2004) analysed a number of different species at different trophic levels in the arctic marine food web in the Svalbard area in northern Norway, among them polar bear. The concentrations detected in adipose tissue (?) in polar bear ranged from 5-15 µg HBCDD/kg lwt.

Table 3-141 Measured levels of HBCDD in Polar Bears in Norway.

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
Polar bear (*Ursus maritimus*) (fat)	NO: Svalbard (arctic region)		26±9.0 min-max: 9.7-45 n = 15 (all values above detection limit)	**(Gabrielsen *et al.*, 2004)**
Polar bear (*Ursus maritimus*) (fat?)	NO: Svalbard	5-15 (min-max)		**(*Jenssen et al.*, 2004)**

MARINE BIRDS

An overview of the levels of HBCDD detected in eggs from marine birds was given in section 3.2.4.2.2 above. Results from the different studies included are presented below and in Table 3-142.

HBCDD have been analysed in different tissues, primarily eggs, of marine birds. A trend with increasing concentrations of HBCDD in eggs, regardless of specie and location of sampling, can be seen in Table 3-142, below. The mean concentrations measured in Atlantic puffin, herring gull, and kittywake in the North of Norway have increased with a factor of about 5-8 over 20 years, from about 1-3 µg HBCDD/kg wwt in the 1983 to between 6-17 µg HBCDD/kg wwt in 2003, depending on species and location of sampling. No descriptive statistic will be presented since the concentrations reported are a result of measurements performed over time at the same location, with increasing concentrations reported over time.

Knudsen *et al.* (2005) analysed bird eggs from Atlantic puffin, glaucous gull, herring gull, and kittiwake sampled in northern Norway at different intervals. The concentrations measured increased with time for all species at all of the different locations. Bouma *et al.* (2000) found about ten times higher concentrations of HBCDD in eggs of common tern from Terneuzen, as compared to eggs from a reference area.

Sellström *et al.* (2003) presented results of measurements of HBCDD in eggs from guillemot from St. Karlsö in the Baltic Sea from 1969 to 2001. The concentration of HBCDD approximately doubled during the study period from 8 µg/kg wwt in the early 1970-ties to approx. 16 µg/kg wwt in the late 1990-ties. The increase appears according to the authors to have levelled out since the mid-1990s.

Lundstedt-Enkel *et al.* (2001) reported of HBCDD measured in eggs from guillemot from St. Karlsö in the Baltic Sea from 1996-2000. The median concentration was 122 µg HBCDD/kg

lwt (arithmetic mean = 124 µg HBCDD/kg lwt, geometric mean = 117 µg HBCDD/kg lwt). It was from this material not possible to observe any temporal trends. Median concentration for year 2000 (Lundstedt-Enkel, 2006) was 66 µg HBCDD/kg lwt (arithmetic mean = 70 µg HBCDD/kg lwt, range 25-148 µg HBCDD/kg lwt), the corresponding concentrations presented on a wet weight basis are a median concentration of 1.83 µg HBCDD/kg wwt (arithmetic mean 2.07 µg HBCDD/kg lwt, range 0.72-4.3 µg HBCDD/kg wwt).

Cormorant livers from CEFAS archive were analysed for presence of HBCDD (Anonymous, 2002a). The levels ranged from below detection (half detection limit is used) -917 µg HBCDD/kg wwt, with most values being below the limit of detection.

de Boer *et al*. (2002) analysed livers from cormorants sampled mainly from the south of UK. All values were above detection limit, ranging from 2.2-35 µg HBCDD/kg wwt.

Gabrielsen and co-workers (2005) measured HBCDD in livers of northern fulmars from Bjørnøya north of Norway in the arctic region. HBCDD was detected in 14 out of 15 samples with an arithmetic mean concentration of 0.65 µg HBCDD/kg wwt, and a range of 0.2-2.22 µg HBCDD/kg wwt (the sample below the detection limit was excluded in the presented mean and range by the authors).

Verreault *et al*. (2004) presented measurements of HBCDD in eggs (n = 10) and in blood plasma of male (n = 20) and female (n = 20) glaucous gull from Bjørnøya north of Norway in the arctic region. The concentration of HBCDD in eggs ranged from 2-70 µg HBCDD/kg wwt, with a reported arithmetic mean of 13 µg HBCDD/kg wwt. The arithmetic mean concentration of HBCDD in blood plasma in male and female fulmars was 0.51 µg HBCDD/kg wwt (range 0.1-1.5 µg HBCDD/kg wwt) and 0.70 µg HBCDD/kg wwt (range 0.2-2 µg HBCDD/kg wwt), respectively.

Murvoll and co-workers (2006) analysed yolk sac from newly hatched chicks of the European shag from the island Sklinna, 50 km of the coast of mid-Norway. HBCDD was detected in all specimens, with a mean concentration of 29 µg HBCDD/kg wwt, or 417 µg HBCDD/kg lwt. The concentration of HBCDD was higher than any of the PBDE congener.

Table 3-142 Measured levels of HBCDD in marine birds in the EU, and Norway

Species	Location	Concentration		Reference
		µg HBCDD/kg lwt	µg HBCDD/kg wwt	
Atlantic puffin (*Fratercula arctica*) (eggs)	NO: Hornøya Year 1983		1.9±0.4 (mean±SD) 2.0 (median) 1.2-2.3 (min-max) n = 5	**(Knudsen *et al*., 2005)**
	Year 1993		4.9±1.0 (mean±SD) 5.2	

			(median) 4.0-5.6 (min-max) n = 5	
	Year 2003		10±2.0 (mean±SD) 11 (median) 6.3-13 (min-max) n = 5	
	Røst (Hekkingen) Year 1983		1.1±0.2 (mean±SD) 1.0 (median) 0.8-1.7 (min-max) n = 5	
	Year 1993		2.2±0.5 (mean±SD) 2.1 (median) 1.6-2.9 (min-max) n = 4	
	Year 2003		6.1±1.2 (mean±SD) 5.0 (median) 3.5-11 (min-max) n = 5	
Common tern (*Sterna hirundo*) (eggs)	NL: Maasvlakte (reference area)	46 µg HBCDD/kg dwt (n = 10, pooled samples)	4.9[a]	**(Bouma *et al.*, 2000)**
	Terneuzen	533 µg HBCDD/kg dwt (n = 10, pooled samples)	56[a]	

Common tern (*Sterna hirundo*) (eggs)	NL: Terneuzen	1200, 930, 1200, 680, 590, 1500, 810, 7100, 670, 330	130, 87, 110, 74, 64, 150, 85, 640, 73, 35	**(de Boer *et al.*, 2002a)**
Cormorant (*Phalacrocorax carbo*) (liver)	UK: Shropshire Surrey Lancashire Cumbria Hampshire Warwickshire Staffordshire Northumberland Cheshire Middlesex Monmouthshire		 1.5*, 1.5* 7.75*, 1.5* 1.5*, 1.5*, 1.5* 1.5*, 1.5*, 1.5* 9.27, 1.5*, 1.5*, 1.5*, 35, 1.5* 1.5*, 1.5* 1.5*, 1.5*, 1.5*, 1.5*, 1.5* 1.5* 1.5* 1.5* 26, 1.5*	**(Anonymous, 2002a)**
Cormorant (*Phalacrocorax carbo*) (liver)	UK: South west (Monmouthshire) South east (Hampshire) South east (Hampshire) South east (Surrey) Central (Staffordshire)		 26 9.3 35 7.8 2.2	**(de Boer *et al.*, 2002a)**
European shag (P*halacrocorax aristotelis*) (yolk sac from newly hatched chick)	NO: Sklinna	417 ± 208 (mean ± SD) n = 30	29 ± 19 (mean ± SD) n = 30	**(Murvoll *et al.*, 2006)**
Glaucous gull (*Larus hyperboreus*) (eggs)	NO: Bjørnøya (arctic region) Year 1997 Year 2002		 2.3±0.2 (mean±SD) 2.3 (median) 1.7-2.9 (min-max) n = 3 12±3.3 (mean±SD) 7.5 (median) 5.2-23	**(Knudsen *et al.*, 2005)**

			(min-max) n = 4	
Glaucous gull (*Larus hyperboreus*) (eggs) (blood plasma) Males Females	NO: Bjørnøya (arctic region)	 142±72 (mean±SE) 20-774 (min-max) n = 10 37±9.55 (mean±SE) 6.13-108 (min-max) n = 20 52 ± 9.0 (mean±SE) 19-122 (min-max) n = 20	 13±6.42 (mean±SE) 2.00-70 (min-max) n = 10 0.51±0.13 (mean±SE) 0.10-1.50 (min-max) n = 20 0.70±0.16 (mean±SE) 0.20-2.00 (min-max) n = 20	**(Verreault *et al.*, 2004)**
Guillemot (*Uria aalge*) (eggs)	SE: Baltic Sea, St. Karlsö Year 1996-2000	 124±40 (mean±SD) 122 (median) 117 (geometric mean) n = 49		**(Lundstedt-Enkel *et al.*, 2001)**
Guillemot (*Uria aalge*) (eggs)	SE: Baltic Sea, St. Karlsö Year 1969 1971 1972 1973 1975 1976	 78 (pooled of n = 10) 66 (pooled of n = 10) 58 (pooled of n = 10) 52 (pooled of n = 10) 100 (pooled of n = 10) 140 (aritm. mean of n = 10)	 8.7 6.9 7.0 6.1 11 21	**(Sellström *et al.*, 2003)**

		94-220		
		(min-max)		
	1977	95	12	
		(pooled of n = 10)		
	1978	45	5.1	
		(pooled of n = 10)		
	1980	47	7.5	
		(pooled of n = 10)		
	1981	34	6.1	
		(pooled of n = 10)		
	1982	45	5.4	
		(pooled of n = 10)		
	1983	48	6.0	
		(pooled of n = 10)		
	1985	76	9.9	
		(pooled of n = 10)		
	1986	120	15	
		(pooled of n = 10)		
	1987	83	10	
		(pooled of n = 10)		
	1988	98	13	
		(pooled of n = 10)		
	1989	130	16	
		(aritm. mean of n = 9)		
		85-260		
		(min-max)		
	1990	110	14	
		(pooled of n = 10)		
	1992	81	9.9	
		(pooled of n = 10)		
		97	11	
		(aritm. mean of n = 10)		
		63-120		
		(min-max)		
	1993	150	17	
		(aritm. mean of n = 10)		
		69-250		
		(min-max)		
	1994	130	14	
		(aritm. mean of n = 10)		

		79-150 (min-max) 1995 170 (aritm. mean of n = 10) 100-300 (min-max) 1996 130 (aritm. mean of n = 10) 54-180 (min-max) 1997 110 (aritm. mean of n = 10) 57-170 (min-max) 1998 110 (aritm. mean of n = 10) 61-180 (min-max) 1999 130 (aritm. mean of n = 9) 76-200 (min-max) 2000 140 (aritm. mean of n = 10) 80-180 (min-max) 2001 140 (aritm. mean of n = 10) 64-220 (min-max)	19 15 13 12 16 16 17	
Guillemot (*Uria aalge*) (muscle)	SE: Baltic Sea, St. Karlsö Year 2000	78, 72, 74, 62, 39, 67, 51, 96, 60, 90, 25, 49, 148, 40, 65, 53, 83, 118, 91, 41	1.75, 2.93, 2.44, 1.64, 1.12, 2.05, 1.38, 2.95, 1.64, 2.34, 0.72, 1.1, 4.29, 1.05, 1.91, 1.44, 2.22, 3.85, 3.28, 1.23	**(Lundstedt-Enkel, 2006)**
Herring gull (*Larus argentatus*) (eggs)	NO: Hornøya Year 1983		1.8±0.4 (mean±SD) 2.0	**(Knudsen *et al.*, 2005)**

			(median) 0.4-3.1 (min-max) n = 5	
	Year 1993		1.9±0.4 (mean±SD) 1.9 (median) 1.6-2.2 (min-max) n = 5	
	Year 2003		9.3±1.49 (mean±SD) 6.7 (median) 5.7-18 (min-max) n = 5	
	Røst (Hekkingen) Year 1983		1.4±0.3 (mean±SD) 0.8 (median) 0.6-2.7 (min-max) n = 5	
	Year 1993		3.7±0.7 (mean±SD) 3.1 (median) 2.4-5.3 (min-max) n = 5	
	Year 2003		12±2.3 (mean±SD) 11 (median) 6.5-17 (min-max) n = 5	
Kittiwake (*Rissa tridactyla*)	NO: Hornøya			**(Knudsen *et al.*, 2005)**

(eggs)	Year 1983		1.7±0.3 (mean±SD) 1.5 (median) 1.2-2.4 (min-max) n = 5	
	Year 1993		3.0±0.7 (mean±SD) 2.7 (median) 2.4-3.7 (min-max) n = 5	
	Year 2003		11±2.2 (mean±SD) 12 (median) 7.9-13 (min-max) n = 5	
	Røst (Hekkingen)			
	Year 1983		2.9±0.6 (mean±SD) 2.0 (median) 0.7-7.9 (min-max) n = 5	
	Year 1993		7.1±1.4 (mean±SD) 5.7 (median) 4.6-11 (min-max) n = 5	
	Year 2003		17±3.5 (mean±SD) 17 (median) 12-27 (min-max)	

			n = 5	
Northern Fulmar (*Fulmarus glacialis*) (liver)	NO: Bjørnøya (arctic region)		0.65±0.7 (mean±SD) 0.2-2.22 (min-max) n = 14*	**(Gabrielsen *et al.*, 2005)**

*In total 15 samples, but one sample was below the detection limit and was not included.

[a]The conversion between dwt and wwt was made using a conversion factor of 9.48, which represents the median values of the ratios between dwt and wwt for eggs of common tern from Terneuzen by de Boer et al., 2002.

3.2.4.3 Comparison between predicted and measured levels

Calculated PECs ($PEC_{oral\ marine\ predator\ (fish)}$, $PEC_{oral\ marine\ top\ predator}$) are found in Table 3-125 to Table 3-133, and measured levels of HBCDD in marine fish and marine mammals in Table 3-139 and Table 3-140, respectively. Besides marine fish and marine mammals, HBCDD has also been detected in other biota. Concentrations of HBCDD detected in Western European biota, associated with the marine environment, are presented in Table 3-138 to Table 3-142.

In the Western Scheldt there are many data on measured levels of HBCDD, possibly reflecting that there are many known facilities that use HBCDD both for production and industrial use in the area. The highest concentration measured in fish in this area is 49µg HBCDD/kg wwt and comes from a gudgeon caught at Terneuzen. Besides this value, the levels in fish varies ranges from 0.19 to 48 µg HBCDD/kg wwt (median 1.8 µg HBCDD/kg wwt, arithmetic mean of 9.2 µg HBCDD/kg dwt, n = 18). The calculated $PEC_{oral\ marine\ predator\ (fish)}$ for production plant B is 540 µg HBCDD/kg dwt, and is thus an over prediction of about 11 times, as compared to the highest concentration measured.

When assessing secondary poisoning via the marine food chain for marine predators and top-predators the predicted concentration of HBCDD is calculated as described below:

$$PEC_{oral,\ predator} = (PEClocal_{seawater} + PECregional_{seawater}) \times 0.5 \times BCF_{fish} \times BMF_1$$

$$PEC_{oral,\ toppredator} = (0.1 \times PEClocal_{seawater,ann} + 0.9 \times PECregional_{seawater}) \times BCF_{fish} \times BMF_1 \times BMF_2$$

Using the following values for HBCDD:

$PEClocal_{seawater}$: 0.003-19 µg HBCDD/l

$PECregional_{seawater}$: 0.003 µg HBCDD/l

BCF_{fish}: 18100

BMF_1: 10

BMF_2: 10

results in $PEC_{oral,\ predator}$ = 543-1719772 µg HBCDD/kg wwt, and $PEC_{oral,\ toppredator}$ = 5430-3443887 µg HBCDD/kg wwt, depending on concentration of HBCDD in the water at the specific local site. These concentrations may be compared with the highest concentrations detected in marine fish and mammals, which are 49 µg HBCDD/kg wwt and 6404 µg

HBCDD/kg wwt (whole weight basis), respectively. It is thus obvious that the predicted values are large overpredictions. As a consequence of this, in the risk characterisation when calculating the risk characterisation ratios for:

- marine predators the $PECregional_{marine\ water}$ is set to 9.9 x 10^{-6} µg/l. Using this concentration the regional part of $PEC_{oral,\ marinepredator}$ becomes 1.8 µg HBCDD/kg wwt. This is equal to the median value of measured concentrations in marine fish in the Western Scheldt which is considered as being representative for the region (see Table 3-53).

- marine top predators the $PECregional_{marine\ water}$ is set to 1.86 x 10^{-4} µg/l. Using this concentration the regional part of $PEC_{oral,\ top\ predator}$ becomes 336 µg HBCDD/kg wwt. This is equal to the median value of measured concentrations in marine mammals in the Western Scheldt which is considered as being representative for the region (see Table 3-53).

The resulting PECs are presented in Appendix 1.

3.3 EFFECTS ASSESSMENT

3.3.1 Aquatic compartment (including sediment)

3.3.1.1 The maximum water solubility of HBCDD in the test systems

HBCDD is not easily dissolved in water, and the different diastereomers have different solubility. Depending on what method have been used and which diastereomers that have been analysed, the concentrations in the water solubility tests ranges from 0.0024 to 0.066 mg/l at 20 °C (see chapter 1.3.6.1). The value of 0.066mg/l is the sum of the individual water solubilities of the three diastereomers. Ideally diastereomer specific EUSES calculations should be performed. This is however not possible due to lack of data. Therefore, for EUSES calculations, the value 0.066 mg/l has been used as a compromise. When it is possible to derive how a toxicity test solution is made, that information can be used as a basis for a discussion on whether a test is performed above water solubility or not. For most toxicity studies, HBCDD has been added as a solution in acetone. It is probable that the total concentration of dissolved diastereomers will increase up to a concentration of 66 µg/l (hereafter called maximum water solubility) when 610 µg/l (or more) of technical substance is added. At the same time, not dissolved (mainly γ-) HBCDD will start to appear at additions of total concentrations higher than 2.4 µg/l. Thus, it is not possible to set a clear threshold for when shown effects should not be attributed to the dissolved substance, because concentrations above the water solubility is used, and a more pragmatic approach is therefore needed. When toxic effect are observed to increase dose-dependently up to a total concentrations of around 610 µg/l of added technical HBCDD (corresponding to the "maximum water solubility" of 66 µg/l), the effects can be induced either by the actually increasing concentrations of β- and α-HBCDD, or by the non-dissolved (γ) HBCDD, or by both. The dissolved fraction is clearly bioavailable, whereas the bioavailability of non-dissolved (γ) HBCDD is unknown. Physical effects of non-dissolved HBCDD cannot be ruled out, but it can only be speculated about the nature and dose-response for such potential effects. As the dissolved substance is more likely to cause the effects than the non-dissolved fraction, the most rational approach is to consider effects occurring up to a total concentration of the technical substance of around 610 µg/l as potentially relevant. However, toxicity tests on the isolated diastereomers (e.g., α-HBCDD) would be needed to really confirm the assumption that the dissolved diastereomers, mainly α- (and to lesser degree β- and γ-HBCDD), causes the toxicity. Even if the non-dissolved fraction would contribute to the toxic effects, it seems not possible to find an appropriate lower value to use as the threshold for relevant effects, as then the effects caused by the dissolved fraction at higher concentrations would be ignored.

The reasoning above implies that the relative toxicity of the different diastereomers is about the same.

In conclusion, 66 µg/l total dissolved diastereomers (the "maximum water solubility") corresponding to 610 µg/l of technical substance will be used as threshold for relevant effects, if nothing is known about the composition of the dissolved diastereomers in the test solutions.

3.3.1.2 Toxicity results for aquatic organisms

3.3.1.2.1 Fish

ACUTE TOXICITY

Three studies on the acute toxicity of HBCDD are available.

Study 1

The static acute toxicity of HBCDD (CAS No.: 3194-55-6) to bluegill sunfish, *Lepomis macrochirus* Rafinesque, was carried out according to US EPA, 1975 (Calmbacher, 1978). A stock solution was prepared by dissolving 1000 mg/l Hexabromid with 200 mg/l Cremophor RH 40.

No abnormal behaviour was observed in any of the concentrations during the test.

Results (nominal conc.): EC_{50} (96h) >100 mg/l

The EC_{50}-values were based on nominal concentrations. A white flocculate was formed on the surface of the water in all test concentrations upon addition of the stock. There were some further deviations from the recommendations in the OECD guideline 203, e.g. the solvent concentration was doubled compared to what is recommended; replicate concentrations were not used; the fish were taken of feed already 48 hours before initiating the test (according to guideline that should be at 24 h); the fish were only acclimated to the test water for 24 hours prior to testing (fish must be held in water of the quality to be used in the test for at least 7 days before testing. If this was done it was not clear.); dissolved oxygen, pH and temperature were measured every 48 hour and not with 24 hour intervals. EC_{50} is above the water solubility for HBCDD. The study is considered to have a low reliability.

Study 2

A static acute toxicity test on golden orfe, *Leuciscus idus L.*, golden variety, was carried out (Kirsch and Munk, 1988), according to DIN 38 412 of June 1982 (Testverfahren mit Wasserorganismen (gruppe L). Allgemeine Hinweise zur Planting, Durchfeurung und Ausvertung biologischer Testverfahren (L1) und Bestimmung der Wirkung von Wasserinhaltsstoffen auf Fische - Fischetest (L15)).

Results (nominal conc.): EC_{50} (96h) >10000 mg/l

No abnormal behaviour was observed in any of the concentrations during the test.

The test procedure is very shortly described. The EC_{50}-value were based on nominal concentrations. There were some further deviations from the recommendations in the OECD guideline 203, e.g. i) the biological loading was larger than recommended, ii) replicate concentrations were not used, iii) the fish were only adapted to the test water for three days prior to testing (fish must be held in water of the quality to be used in the test for at least seven days before testing. If this was done it was not clear.), iv) dissolved oxygen and pH were not measured with 24 hour intervals, and v) the size of the test fish was not given. EC_{50} is well above the total concentration possibly resulting in the ”maximum water solubility” for HBCDD. The study is considered to have a low reliability.

Study 3

The acute toxicity of HBCDD to rainbow trout, *Oncorhynchus mykiss*, was studied in a 96 h flow through test according to standardised guidelines (TSCA Title 40, Part 797, Section 1400; OECD guideline No 203, and ASTM Standard E729-88a) 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. There was no information on the identity and properties of the remaining 6.4 %. A stock solution was prepared by dissolving HBCDD in dimethylformamide. The concentration of dimethylformamide in the solvent control and in the HBCDD treatment groups was 0.1 ml/l. The stock solution was inverted and stirred with a glass rod.

It is stated in the report that the dimethylformamide have a potential to slightly increase the water solubility of HBCDD and thus the chosen nominal test concentrations 1.5-6.8 µg/l is well within the water solubility for HBCDD.

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. Concentrations were measured every day from day 0 to day 2 for negative control <LOQ, solvent control <LOQ. Test concentrations were measured every day from day 0 to day 4 and also repeated day 4 resulting in the following mean measured test concentrations (range): 1.5 (<0.571-6.76), 2.2 (0.732-6.97), 3.2 (1.04-20.6), the highest value is said to represent co-eluting artefacts at the retention time of HBCDD, in that case it would be 4.20 for the highest value), 4.6 (2.13-9.65, the highest value is said to be an outlier, in that case the highest value would be 5.72), and 6.8 (1.70 -4.59) µg/l. The mean measured values are calculated to 0.75, 1.5, 2.3, 2.3, and 2.5. Thus the concentrations in the water phase were almost halved.

The temperature during the test was 12±2 °C. The pH ranged from 8.2 to 8.3. Dissolved oxygen concentrations ≥78 % of saturation were observed throughout the test (according to guideline it should not be less than 80 %). Specific conductance, hardness, alkalinity and pH of the well water during the 4-week period preceding the test were 300-310 µmho/cm, 132-136 mg/l as $CaCO_3$, 178-180 mg/l as $CaCO_3$, and 8.3-8.4, respectively.

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

LONG-TERM TOXICITY

Sublethal effects

The effects of HBCDD on biomarkers in juvenile rainbow trout (*Oncorhynchus mykiss*) were studied by Ronisz *et al.*, 2004 (Ronisz *et al.*, 2004). Fish were injected once intraperitoneally with HBCDD, 50 or 500 mg/kg, solved in peanut oil. However, the high concentration HBCDD-dose was not possible to fully dissolve in peanut oil. The experiments lasted for 5 and 28 days. Bile analysis after the 28-day experiment showed that the concentration of HBCDD in the bile was 0.14-0.7 µg/g for the 50 mg dose and 7-15 µg/g for the 500 mg dose. Different hepatic enzyme activities (catalase, glutathione S-transferase, glutathione reductase, and ethoxyresorufin-O-deethylase (EROD)), the liver somatic index, the occurrence of DNA-adducts in the liver, and plasma vitellogenin levels were measured. No consistent effects was observed after 5 days. At 28 days, a 40 % increase in the liver somatic index was the only effect observed.

The possible endocrine effects of HBCDD on European flounder (*platichthys flesus*) were investigated by ((Kupier *et al.*, 2007)).The flounders were 940 days old at the start of the study. They were weighed in groups of 10 and their average body weight was 86 ± 26 g (8 groups). The animals were kept in 160 l aquaria at a temperature of 15^{o}C with a continuous flow through of Eastern Scheldt water (renewal twice a week). The aquaria contained 15 kg of sediment (sandy sediment, 78.35% dry matter, 0.3% TOC). The flounders were exposed to HBCDD for 78 days via both the food and sediment. The fish were fed three times a week at rate of 1% of the total bodyweights at the start of the experiment, increased to 2% after 21 days until 5 days before the end of the study, when feeding was discontinued. The test substance was a technical mixture with an average composition of 10.28%, 8.72% and 81.01% of the α-, β- and γ-diastereomers, respectively. The exposure levels are shown in Table3-143

Table3-143 HBCDD concentrations in European flounder after 78 days of exposure to HBCDD via sediment and food

Nominal HBCDD exposure		**HBCDD in muscle median (µg/g lipid)**[a]		
Sediment (µg/g TOC)	**Food** (µg/g lipid)	**α**	**β**	**γ**
Reference A	0	<2.2	<2.2	<0.9
Reference B[b]	0	<2.1	<2.1	2.1**
0.08	0.3	<4.3	<4.3	1.4**
0.8	3.0	<0.7	<0.7	2.0**
8	30	0.9	<0.4	12.5**
80	300	18.3*	4.5*	106.4**
800	3000	44.2*	57.3*	172.7**
8000	0	33.0*	2.8*	36.7**

[a]10 animals analyzed except groups 800/3000 and 8000/0 µg/g TOC/lipid, respectively (9 fish in each group)

[b] Once exposed to food with 30µg HBCDD/g lipid during the second week of the experiment

*/** Mean significantly increased compared to reference A at p<0.05/0.01, respectively

The fish were euthanized with MS222A after the exposure period. Blood was sampled from the caudal vein for analysis of thyroid hormones (T_3 and T_4).The liver was taken for

preparation of microsomes and subsequent measurements of microsomal enzyme activities (EROD, BROD, PROD). The gonads were excised and weighed and parts of them were used for preparation of microsomes and subsequent measurement of aromatase activity. Flank muscles were excised for analysis of HBCDD. In addition to the biochemical parameters histopathological examinations of internal organs was performed and endpoints such as e.g. behaviour, weight, length, liver weight, gonad weight, GSI and were recorded.

The HBCDD concentrations in fish muscle are shown in Table3-143 above. Worth noticing is the difference in proportion of α-HBCDD and γ-HBCDD when exposed via food and sediment in combination and via sediment alone. The reason for this is unknown but may be related to the higher water solubility of the α-HBCDD making it more bioavailable. No abnormal behaviour was noted during the exposure and no statistically significant differences in weight, length, liver or gonad weights, GSI etc. were observed. EROD activity was not induced by HBCDD and BROD and PROD activities did not show exposure related changes except for a weak negative correlation between the decreasing proportion of γ-HBCDD at the three highest exposure levels and a slightly increasing PROD activity. The aromatase activity in the gonads was not affected by HBCDD and neither were the thyroid hormones T_3 and T_4. There were no histopathological changes in internal organs including liver, spleen, kidney gonads and thyroid gland related to HBCDD exposure. Overall exposure to HBCDD did not affect the reproductive system of flounder in the study to a major extent. The findings indicate limited potential for in vivo endocrine disruption of the reproductive and thyroid hormonal system in immature flounder up to a reported maximum internal concentration of 446 µg HBCDD / g l.w).

Early life-stage toxicity test

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 conducted in compliance with GLP. The test guidelines used were US EPA Series 850 – Ecological effects test guidelines, OPPTS 850.1400 and OECD Guideline 210.

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, which is well below the maximum water solubility of HBCDD. 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. Four replicate test chambers were used containing two incubation cups with 15 embryos per incubation cup. After swim-up (when the larvae began swimming up from the bottom) the number of rainbow trout larvae was thinned to be 15 in each test chamber.

The test was performed under flow-through conditions in the darkness until one week after hatching. After that 16 hours of light and 8 hours of darkness per day was held. The test temperature was 12 °C. During the test, temperature, pH, dissolved oxygen, hardness, alkalinity, and specific conductance were measured.

Daily observations were made to evaluate the numbers of individuals exhibiting clinical signs of toxicity or abnormal behaviour.

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 test substance was dissolved in acetone and then diluted in dilution water with the aid of a

diluter mixing chamber. The concentration of acetone in the solvent control and in the HBCDD treatment groups was 0.1 ml/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 (range): negative control <LOQ, solvent control <LOQ, 0.25 (0.161-0.365), 0.47 (0.369-0.684), 0.83 (0.385-1.03), 1.8 (1.11-158 (this might be a decimal point missing, thus it would be maximum 2.82), and 3.7 (2.78-4.77) µg/l.

Thus the concentrations in the water phase were almost halved.

The viability of the eggs was controlled at day 11 and the mean percent of fertilisation was determined to 99 %.

The outcome of the hatching, which occurred between day 23 and 33, is presented in Table 3-144 below.

Table 3-144 Hatching success for rainbow trout (*Oncorhynchus mykiss*)

Mean measured concentration of HBCDD µg/l	Negative control	Solvent control	0.25	0.47	0.83	1.8	3.7
% hatching success	75	85	91	89	89	83	84

There was no statistically difference ($p > 0.05$) between the control groups. The hatching success ≥83 % was not statistically different ($p > 0.05$) from the pooled controls. There was no apparent difference in time between the control groups and the HBCDD treated groups. NOEC for hatching success was ≥3.7 µg/l.

The swim-up occurred between day 13 and 22 post-hatch. There was a tendency that the time to swim-up was slightly longer in the highest concentration of HBCDD. However there were no statistically significant ($p > 0.05$) reductions in the numbers of fish swimming up in any HBCDD treatment group compared to the pooled control groups. NOEC for time to swim-up was ≥3.7 µg/l.

Survival was analysed for two time periods post-hatch: 1) day 1 to day 22 (reduction of number of larvae) and 2) day 22 to day 61. Mean survival prior to the reduction of number of larvae (thinning) was 97 and 98 % in the control groups and 97-99 % in the HBCDD treatment groups. After the reduction of number of larvae the mean survival was 98 % in the control groups and 97-100 % in the HBCDD treatment groups. There was no significantly difference ($p > 0.05$) between the different groups. NOEC for larvae and fry survival was ≥3.7 µg/l.

All fish were observed daily to evaluate the numbers of mortalities and the numbers showing sublethal signs of toxicity. All fish appeared normal and healthy during the test.

Growth of the fish was evaluated day 29 post-hatch to day 61 (end of test) as total length and wet and dry weight. There was no significant difference ($p > 0.05$) between the different groups. Hence, NOEC for growth was ≥3.7 µg/l.

3.3.1.2.2 Aquatic invertebrates

ACUTE TOXICITY

Data from two studies on the acute toxicity of HBCDD to *Daphnia magna* are available.

Study 1

An acute flow through toxicity study on *Daphnia magna* (neonates) was performed according to procedures outlined in TSCA Title 40 of the Code of Federal Regulations, Part 797, Section 1300; Part I of OECD Guideline for Testing of Chemicals, 202; and ASTM Standard E729-88a. (Graves and Swigert, 1997a). The test substance consisted of a composite of HBCDD samples from three manufacturers. The composite contained 8.5 % β diastereomer, 6.0 % α- diastereomer and 79.1 % γ- diastereomer (total HBCDD 93.6 %). There was no information on the identity and properties of the remaining 6.4 %. A stock solution was prepared by dissolving HBCDD in dimethylformamide (0.068 mg HBCDD/ml DMF). The stock solution was inverted and stirred with a glass rod. The concentration of dimethylformamide in the solvent control and in the HBCDD treatment groups was 0.1 ml/l.

It is concluded in the report that the dimethylformamide slightly increases the water solubility of HBCDD. The chosen nominal test concentrations 1.5-6.8 µg/l are well below the maximum water solubility for HBCDD.

The test was performed with duplicates for each test concentration with 10 animals per replicate, at 20±2 °C.

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. There is no explanation to the differences between nominal and measured concentrations.

At day 2 (48 h) there was 1 dead animal in the nominal concentration 4.6 µg/l (measured concentration 1.55 µg/l).

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

Study 2

In one further study the acute toxicity of Hexabromid S, CAS no 3194-55-6, (1, 2, 5, 6, 9, 10-hexabromocyclododecane) on *Daphnia magna* (Jatzek, 1988) was studied according to Guideline for testing of chemicals EG-1 of Jan. 1982 issued by the EPA, Office of Toxic Substances 75-009 (1975). The test vessels were small reagent tubes with flat bottom, thus the test seems to have been performed under static conditions. The purity of the substance was stated to be ca 92 %. A stock solution was prepared by dissolving 1000 mg/l Hexabromid with 100 mg/l Tween 80. The solution was stirred for 17 hours at 20 °C. The nominal test concentrations used were 0.01-1000 mg/l, *i.e.* both below and above the water solubility for HBCDD.

Results (calculated from nominal concentrations): EC_{50} (48h) 146 mg/l

Confidence limits 95 % 146.25 - 146.43 mg/l

NOEC (48h) 1 mg/l

EC_{100} (48h) >1000 mg/l

Since there were no measured data, the study is considered to have a low reliability.

LONG-TERM TOXICITY

Life-cycle toxicity test with Daphnia

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 was conducted in compliance with GLP. The test guidelines used were OECD Guideline 202 and TSCA Title 40 of the Code of Federal Regulations Part 797, Section 1330.

The test substance consisted of a composite of HBCDD samples from three manufacturers. The composite contained 8.5 % β- diastereomer, 6.0 % α- diastereomer and 79.1 % γ- diastereomer (total HBCDD 93.6 %). There was no information on the identity and properties of the remaining 6.4 %. A stock solution was prepared by dissolving HBCDD in dimethylformamide. The concentration of dimethylformamide in the solvent control and in the HBCDD treatment groups was 0.1 ml/l.

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.

The results of the test are presented in Table 3-145, Table 3-146 and Table 3-147 below.

Table 3-145 Observations of mortality and other effects, presented as cumulative.

Mean measured concentration of HBCDD (µg/l)	**% mortality** **Number of animals with other effects**					
	24 h	48 h	96 h	7 d	14 d	21 d
Negative control	0 40 AN*	0 40 AN	0 40 AN	0 40 AN	2.5 39 AN	5.0 38 AN
Solvent control	0 40 AN	0 40 AN	0 40 AN	0 40 AN	0 40 AN	2.5 39 AN
0.87	0 40 AN	0 40 AN	0 40 AN	0 40 AN	0 40 AN	2.5 39 AN

Mean measured concentration of HBCDD (µg/l)	% mortality Number of animals with other effects					
1.6	0 40 AN	0 40 AN	0 40 AN	0 40 AN	2.5 39 AN	2.5 39 AN
3.1	0 40 AN	0 38 AN; 2 C**	0 40 AN	0 40 AN	0 40 AN	0 40 AN
5.6	0 40 AN	0 40 AN	0 40 AN	0 40 AN	0 40 AN	7.5 37 AN
11	0 40 AN	0 40 AN	0 40 AN	2.5 39 AN	5.0 38 AN	12.5 35 AN

*AN = all organisms appeared normal and no unusual behaviour was observed

**C = lethargy

Table 3-146 Reproduction of *Daphnia magna* during the life-cycle toxicity test.

Mean measured concentration of HBCDD (µg/l)	Mean number of young per reproductive day (± SD)
Negative control	3.62 (± 0.056)
Solvent control	3.85 (± 0.0894)
Pooled control group	3.74 (± 0.14)
0.87	3.72 (± 0.068)
1.6	3.86 (± 0.069)
3.1	3.83 (± 0.14)
5.6	3.51 (±0.34)
11	2.84 (± 0.63)*

*Significant difference from the pooled control group with the Bonferroni t-test (p ≤0.05).

Table 3-147 Length and dry weight of surviving first-generation daphnids.

Mean measured concentration of HBCDD (µg/l)	Mean total length (mm) (± SD)	Mean dry weight (mg) (± SD)
Negative control	4.09 (± 0.035)	0.69 (± 0.016)
Solvent control	4.07 (± 0.0083)	0.68 (± 0.00078)
Pooled control group	4.08 (± 0.024)	0.68 (± 0.0098)
0.87	4.08 (± 0.024)	0.70 (± 0.0044)
1.6	4.05 (± 0.014)	0.67 (± 0.019)
3.1	4.04 (± 0.0053)	0.68 (± 0.034)
5.6	4.01 (± 0.014)*	0.66 (± 0.025)
11	3.87 (± 0.054)*	0.56 (± 0.037)*

*Significant difference from the pooled control group with the Bonferroni t-test (p ≤0.05).

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.

QSAR values

The toxicity of HBCDD to aquatic organisms has been obtained by using an appropriate QSAR equation from Chapter 4 of the TGD. Considering the relevant domain for the models only one equation was assessed to be valid, i.e. *Daphnia magna* 48 h. The results are given below:

***Daphnia magna*:**

48h-EC_{50} = 140 µg/l

The QSAR estimate is about 1000 times less than in study 2 on acute toxicity for Daphnia magna. However, that study is considered to have a low reliability since the results are based on nominal values, far above the water solubility. The QSAR result is also above the maximum water solubility of HBCDD, but might be seen as an indicator that the LOEC from the study on chronic toxicity to *D. magna*, 5.6 µg/l, is reasonable. The result is below the water solubility of HBCDD.

3.3.1.2.3 Algae

Data are available from five algal growth inhibition studies.

Study 1

The growth inhibition of Hexabromid S (CAS 3194-55-6) to *Scenedesmus subspicatus* CHODAT DSM 86.81 was studied according to OECD guideline No 201 (Siebel-Sauer and Bias, 1987). The biomass was determined by measuring the chlorophyll a fluorescence. A stock solution was prepared by dissolving 1000 mg/l Hexabromid with 200 mg/l Cremophor RH 40. Seven different nominal test concentrations between 7.81 and 500 mg/l Hexabromid S were used in the test. No effects on growth were observed in the highest tested nominal concentration compared to the controls: EC_{50} (96h) >500 mg/l, which is well above the maximum water solubility of HBCDD.

Study 2

The toxicity of HBCDD to the freshwater alga, *Selenastrum capricornutum,* was studied in a static 96 h growth inhibition test in accordance with the procedures outlined in TSCA Title 40, Part 797, Section 1050; and OECD guideline No 201 (Roberts and Swigert, 1997). 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 %). There was no information on the identity and properties of the remaining 6.4 %. A stock solution was prepared by dissolving HBCDD in dimethylformamide, 0.068 mg HBCDD/ml.

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, all well below maximum water solubility of HBCDD) compared to control and solvent control. 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. The measured concentration of HBCDD in the 6.8 µg/l treatment group (nominal concentration) after filtration of the algae was <1.94 µg/l (this value was stated to reflect a non quantifiable level due to limited sample availability). The temperature during the test was 24±2 °C. The pH ranged from 7.4 to 7.5 on day 0 and ranged from 8.0 to 8.4 on day 4. The growth was exponential at least up to 72 hours.

The author concluded that no statistically significant effect concentration could be established in the test, neither for growth rate nor for 'area under the curve'. Cell densities were compared with Dunett's test and 'areas under the growth curves' were compared with Bonferroni's "t" test.

The rapporteur tested the results in the study, using statistic procedures recommended by OECD (OECD, 2003): The solvent control and non-solvent control were compared using Wilcoxon rank sum test. Since the controls did not differ, the two controls were combined into one control group. The combination of an expected monotone dose response and more than two doses resulted in the choice of a step-down procedure, using the Jonckheere-Terpstra test, in order to determine the NOEC. However, no NOEC could be determined. Use of only the solvent control did not change that result, i.e. no NOEC could be determined.

The maximum measured concentration on day 4 was 2.5 µg/l. Thus, the 72-hour EC_{50} is >2.5 µg/l and the LOEC is >2.5 µg/l.

The study was in general carried out in good agreement with the given guidelines.

Study 3

The algal growth inhibition of HBCDD was also studied in six marine media (Walsh *et al.*, 1987) according to a marine algal bioassay method by Walsh and Alexander, 1980 (Walsh and Alexander, 1980).

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 were derived by straight line graphical interpolation without calculation of confidence intervals. According to the test procedure (Walsh and Alexander, 1980) algae should be added to the test system to a cell concentration of 80 cells/ml for *S. costatum* and 40 cells/ml for the other algal species. However, in the equation given for the calculation of growth rate in Walsh *et al.* (1987), with a reference to Fogg, 1965, N_0 is defined as "number of cells at beginning of test (4.7×10^4 cells/ml)". The test substance HBCDD was obtained from one manufacturer, Great Lakes Chemical Inc. A stock solution was prepared by dissolving HBCDD in acetone, thereafter introducing 0.05 ml

of that into 51 ml of growth medium with algae. The highest concentration to be used in the test was determined by adding the substance slowly to growth medium and observing the highest concentration at which crystals did not form. The estimated concentration for saturation was 1.5 mg/l. The six growth media were prepared from natural seawater collected from an inshore site on the Gulf of Mexico and from five commercial sea salt formulations (salinity 30 ‰). The pH is 7.6-8.2 and the phosphate content is 13.8-21.4 µg/l. The concentration of HBCDD in the stock solution and exposure media was confirmed by capillary column gas-liquid chromatography. Percentage recovery in the spiked media: average >90 %. Tests were performed in two replicates for each medium.

Range of results in different marine growth media, derived by straight line graphical interpolation:

*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

The results for *S. costatum* and partly for *T. pseudonana* are well below the maximum water solubility for HBCDD, whereas for *Chlorella* the result is above.

No NOEC was determined in the test.

Although growth media differed greatly in composition, growth rates of each species were similar in all media without test substance. On the other hand, the toxicity response of HBCDD was, according to the authors, related to the chemical properties of the growth medium. However, there was little variation in response of *S. costatum* among the test media. No growth rates or curves were presented. The results indicate a high to very high toxicity of HBCDD to *T. pseudonana* and S. *costatum*, respectively, and a low toxicity to C*hlorella sp.* in marine media.

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 4

Another growth inhibition study was performed with *Skeletonema costatum.* The test protocol was outlined based on OECD guideline 201, ISO 10253:1995 and EU Directive 92/69/EEC – Method C.3 (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. Only one test concentration has been used. However, according to OECD test guideline at least five test concentrations should be used arranged in a geometric series and the lowest should have no observed effect on the growth rate of the algae.

The results of the study are presented in Table 3-148.

Table 3-148 Measured level of HBCDD in the algal growth inhibition test

	Measured HBCDD µg/l*			**Sum of measured HBCDD µg/l**
Sampling time h	**α**	**β**	**γ**	**Total HBCDD**
0	35.4	10.6	1.73	47.7
	31.2	9.19	1.88	42.3
	36.4	10.7	1.68	48.8
Arithmetic mean 0 h	34.3	10.2	1.76	46.3
72	28.2	7.87	1.55	37.6
	26.0	7.52	1.42	34.9
72	25.8	7.15	1.42	34.4
Arithmetic mean 72 h	26.7	7.51	1.46	35.6
Geometric mean 0 and 72 h	30.3	8.74	1.61	**40.6****

*HBCDD level measured in the medium including algae (personal information René Hunziker, BASF)

**Level of HBCDD that will be used for evaluation of the study.

In the final test report an arithmetic mean concentration of HBCDD was calculated from measured concentration at 0 and 72 h and was used to calculate the NOEC values. For the RAR the mean geometric measured concentration at 0 and 72 h will be used, *i.e.* 40.6 µg/l.

The composite HBCDD sample was composed of a mixture from three manufacturers and was assigned Wildlife International, Ltd. Identification number 5850. The composition was 7.67 % α-, 5.15 % β- and 83.04 % γ- diastereomer of HBCDD. Separate analytical standards of the α-, β- and γ- were used in the analytical procedure. Passing saltwater algal medium through a generator column saturated with HBCDD produced the single test concentration. In this way the composition of HBCDD in the test differed from that of the technical product. The concentration of the different diastereomers became the same as their respective solubility. The composition became 74.6 % α-, 21.5 % β- and 3.97 % γ- diastereomer of HBCDD in the saltwater algal medium.

The nominal concentration of algal cells was approximately 77000 cells/ml in six replicates for test substance, negative control and media control, respectively. Cell counts were performed using an electronic particle counter. Test temperature was maintained at 20±2 °C. The pH of the test increased from 8.0 to 8.3 in test substance chambers and from 7.9 to 8.4 for the controls. The light intensity ranged from 4130 lux to 4660 lux.

Mean cell density, biomass and growth rate were calculated at 72 hours. After 72 hours of exposure there were no signs of adherence of cells to the test chambers or aggregation/flocculation. Neither were there any noticeable changes in cell morphology. Growth rate and percent inhibition are presented in Table 3-149.

Table 3-149 Growth rate over 72 hours.

HBCDD µg/l	**Mean Growth rate** (mean ± standard deviation)			
	0-24 h	**24-48 h**	**48-72 h**	**0-72 h**
Mean pooled control	0.0371 ± 0.0029	0.0465 ± 0.0035	0.0514 ± 0.0040	0.0450 ± 0.0014

Mean 40.6	0.0271 ± 0.0038	0.0468 ± 0.0037	0.0489 ± 0.0022	0.0409 ± 0.0023
Percent inhibition compared to pooled control	27	-0.6	4.9	9.1

There is an effect at the measured test concentration of HBCDD 40.6 µg/l. The major effect on the growth rate occurs during the first 24 h. Nevertheless for the RAR the whole growth curve is chosen for the calculation of inhibition, which gives around 10 % inhibition of the growth rate. NOEC is <40.6 µg HBCDD/l and EC_{50} >40.6 µg HBCDD/l.

Study 5

This study consists of two toxicity tests with HBCDD (i) using a co-solvent, and (ii) performed at saturated solution (Desjardins *et al.*, 2005). Both studies are performed based on ISO 10253:1995 and the OECD Test Guideline 201. Both the biomass and the growth rate were derived. The toxicity of HBCDD to the marine diatom alga *Skeletonema costatum* was measured during 72 hours.

The test substance HBCDD used in the test was a 1:1:1 composite of three samples received from three different manufacturers. 10,000 cells/ml were tested in the test volume prepared according to ASTM 1218-90E (pH 8 and salinity 30 ppt). Cell count was conducted using a haemocytometer and a microscope at 24, 48 and 72 hr. Six replicates per concentration were used.

Study with a co-solvent

The primary stock solution was prepared in dimethylformamide (DMF) at a nominal concentration of 0.10 mg HBCDD/ml, which was diluted to secondary stock solutions. Aliquots of the appropriate stock solutions were diluted with saltwater medium in order to prepare the following nominal concentrations: 0.64, 1.6, 4.0 and 10 µg HBCDD/l, which were used in the toxicity test. 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.

Treatment groups were compared to the pooled control (media and solvent control) replicates as there were no statistically significant differences between the media control and the solvent control. The biomass inhibition at 72-hr was 1.3, 5.2, 0.19 and 1.4 %, respectively. The growth rate inhibition at 72-hr was 1.6, 0.56, 3.3, and 1.2 %, respectively. These effects are not statistically significant. It is probable that the concentrations were almost the same in all these tests, i.e. about the solubility of γ-HBCDD. 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.

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.

However, according to OECD test guideline at least five test concentrations should be used arranged in a geometric series and the lowest should have no observed effect on the growth rate of the algae. Since the report consists of both one part with 4 test concentrations and one part with a limit test the test concentrations taken together are nevertheless considered as enough.

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. At the beginning of the test the following measured concentrations of the diastereomers were found: 3.54 µg γ-HBCDD /l, 15.2 µg β-HBCDD /l and 35.8 µg α-HBCDD /l. Growth rate and percent inhibition are presented in Table 3-150.

Table 3-150 Growth rate over 72 hours.

HBCDD µg/l	**Mean Growth rate** (mean ± standard deviation)		
	0 - 24 h	**0 - 48 h**	**0 - 72 h**
0; Media control	0.0764 ± 0.0061	0.0646 ± 0.0016	0.0589 ± 0.0011
0; Column control*	0.0886 ± 0.0091	0.0666 ± 0.0017	0.0593 ± 0.0024
54.5	0.0737 ± 0.0122	0.0472 ± 0.0052	0.0288**± 0.0069
Percent inhibition compared to media control	3.5	27	51
Percent inhibition compared to column control	17	29	51

* media that was passed through a generator column.

**Statistically significant difference ($p < 0.05$) at 72 hours from the column control using Dunnett's test.

The growth rate inhibition raised during the study and was 17% compared to the column control after 24 hours, 29 % after 48 hours and 51% after 72 hours (see table Table 3-150). 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 value of 52 µg/l for 50% reduction in growth rate is taken forward to the risk characterisation.

The EC_{50} of 52 µg/l determined in is in line with the result obtained with the saturated solution (*i.e.* study 4) where EC_{10} was around 40.6 µg/l. The study is considered valid and the EC_{50} will be used in the RAR.

Summary of algal toxicity

There are five studies on algal toxicity. Study 2, 4 and 5 are performed according to standard guidelines and are clearly described. In study 2 no effects were observed for *Selenastrum capricornatum* at the highest tested concentration, 2.5 µg/l of HBCDD. Study 4 is unfortunately only a limit test with *Skeletonema costatum* that results in about 10 % inhibition at 40.6 µg/l of HBCDD. Study 5 is the best-performed study, including four lower concentrations as well as a saturated solution of HBCDD. *Skeletonema costatum,* used in this study, appears to be a sensitive algal species, which is supported by the results in study 3. The

results in study 5 are therefore used to obtain an EC_{50} for algae based on growth rate, *i.e.* 52 µg/l of HBCDD.

From study 4 and 5 the 72-hr NOEC for *Skeletonema costatum* could be determined to 10µg/l <NOEC ≤40 µg/l.

3.3.1.3 Predicted no effect concentration for water ($PNEC_{water}$)

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.

3.3.1.4 Toxicity results for micro-organisms

Oxygen consumption test with *Pseudomonas*

An oxygen consumption test using *Pseudomonas putida* was carried out according to a test method of Robra (Robra, K.H., GWF Wasser-Abwasser 117, 80-86, 1976) (Siebel-Sauer, 1990). It is stated that this method is suitable for insoluble to sparingly soluble substances. The decline in the concentration of dissolved oxygen was measured in a flow cell. 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. Therefore the results from this study have not been considered relevant to base a $PNEC_{STP}$ on.

Respiration inhibition test with activated sludge

An activated sludge respiration inhibition test has been performed (Schaefer and Siddiqui, 2003). The test was performed according to OECD guideline 209 (OECD, 1984).

The test substance was a composite sample from three manufactures of hexabromocyclododecane suppliers 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. An EC_{50} of 5.2 mg/l was determined for the 3,5-dichlorophenol reference substance which was within the normal range (5 to 30 mg/l). The respiration rates of the two controls after 3 hours were 60.5 and 55.5 mg O_2/l/hour and the variability between the two controls (9 %) was within the 15 % limit specified in the test guidelines. 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 apart from a deviation from the guideline. Only one concentration was used instead of three assuming a limit test. However, there was a significant inhibition. Due to the 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 in the RAR for calculation of PNEC.

3.3.1.5 Predicted no effect concentration for micro-organisms ($PNEC_{STP}$)

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.

3.3.1.6 Toxicity result for sediment organisms

Prolonged sediment toxicity tests with *Hyalella azteca*

Two toxicity tests were performed on the amphipod *Hyalella azteca* to determine the effects of sediment-incorporated HBCDD during a 28-day exposure period under flow-through conditions. One study used spiked sediment with 2 % total organic carbon content and the other used spiked sediment with 5 % total organic carbon content (Thomas *et al.*, 2003a-b).

The protocols were based on the ASTM E 1706-95b Guideline: Standard Test Methods for Measuring the Toxicity of Sediment-Associated Contaminants with Fresh Water Invertebrates and the OPPTS 850.1735 Guideline: Whole Sediment Acute Toxicity Invertebrates,

Freshwater and also Methods for Measuring the toxicity and Bioaccumulation of Sediment-Associated Contaminants with Freshwater Invertebrates.

Non-GLP exploratory range-finding studies were performed with three freshwater species associated with sediment: oligochaetes (*Lumbriculus variegatus*), chironimids (*Chironomus riparius*) and amphipods (*Hyalella azteca*). The three species were tested at 50, 100, 500 and 1000 mg/kg dwt in two types of sediment, one with 2 % organic carbon content and the other with 5 %. According to the present test reports on the prolonged toxicity tests with Hyalella the amphipods were found to be the most sensitive species in both sediment types, with clear effects in the 500 mg/kg treatment group. More definitive GLP studies were therefore conducted with *Hyalella azteca* in both 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 survivorship and growth as determined by dry weight measurements.

The test substance consisted of a composite of HBCDD samples from three manufacturers. Samples from the composite when used in the study with 5 % organic carbon content were analysed and found to contain 5.8 % α- diastereomer, 19.3 % β- diastereomer and 74.9 % γ- diastereomer. The study with 2 % organic carbon content was performed at a later date and samples from the composite when used in this study were found to have a purity of 95.9 % and contain 7.7 % α- diastereomer, 5.2 % β- diastereomer, and 83.0 % γ- diastereomer. Artificial test sediment, similar to that described in OECD Guideline 207 but with α-cellulose as its source of organic matter instead of peat moss, was used in both studies. The test substance was added directly to the sediment and mixed for approximately 22-23 hours on a rotating mixer.

In the study with 2 % organic carbon content none of the treatment groups were found to differ statistically significant from the control group and there was no apparent concentration dependent response. In the study with 5 % organic carbon content statistically significant mortality was observed in the middle test concentrations (63, 125 and 250 mg/kg dwt). However, survival in the 31, 500 and 1000 mg/kg treatment groups was similar to the control group and differences were not statistically significant. Since there was no concentration dependent response the mortality in the middle groups was not considered treatment related. There were no apparent effects on growth observed at the end of the test.

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.

Prolonged toxicity tests with *Lumbriculus variegatus* and *Chironomus riparius*

The impact of chemicals to organisms in sediment by using three different chemicals (HBCDD, 3,4-DCA and DODMAC) has been studied (Oetken *et al.*, 2001). 28d-sediment bioassays were carried out with *Lumbriculus variegatus* and *Chironomus riparius* in spiked and aged artificial sediment. The details on e.g. the spiking of the sediment were not

completely described in the report. Therefore, one of the authors, M. Oetken, has provided some clarifications, on request.

Methods

In contrast to the OECD Draft Guideline 218, artificial sediment with a coarser grain size (100-2000µm) and other carbon sources (stinging-nettle and leaves of alder) was developed. This modified artificial sediment provided sufficient organic matter from the beginning of the experiment, so that it was not necessary to feed the organism with uncontaminated food during the experiment (as specified in the OECD Draft Guideline 218). The organic matter content was 0.7 g per 20 ml of dried sediment, 20 ml of dry sediment corresponding to 40 g dwt (M. Oetken personal communication). Accordingly, the organic carbon content was about 1.8 % of the dry sediment.

The nominal test concentrations were: 0.05; 0.5; 5; 50 and 500 mg HBCDD/kg dwt for both test organisms. Dried whole sediment was spiked with HBCDD dissolved in ethyl acetate (e.g. 70 ml to 250 g sediment in the case of the C. riparius test), separately for each test beaker. After the solvent had evaporated overnight, the sediment was mixed for 3 minutes using a glass stirrer, and dechlorinated tap water was added to the beakers (M. Oetken, personal communication). The test systems were allowed to equilibrate (age) for 14 days before the 28d-bioassays were started.

The test with *L. variegatus* was conducted in 250 ml glass beakers containing 20 ml (spiked, dry) sediment and 160 ml dechlorinated tap water at 20 ºC, 16:8 light: dark photoperiod, and gentle aeration. Organisms of the same developmental and physiological status were obtained by cutting stock organisms. After 14 days the posterior part of the organisms had regenerated a new head, and these worms were used in the test. Ten worms were added to each beaker after the 14 days of equilibration of the sediment - water systems (in total 28 beakers: control, solvent control, five test concentrations, 4 replicates of each). At the termination of the test, worms were removed by sieving, and the following toxicological endpoints were monitored: total number of worms, number of large and small worms, as well as deformations and biomass.

The test with *C. riparius* was conducted in 2000 ml glass beakers containing 2 cm sediment (corresponding to 250 g) and 8 cm dechlorinated tap water at 20 ºC, 16:8 light: dark photoperiod, and gentle aeration. After the equilibration of the systems (14 d) 20 first instar larvae were added to each beaker (in total 28 as above). The success of emergence, the developmental time, the total number of fully emerged male and female midges, and the numbers of eggs per clutch of the F_1 were determined.

In both tests dissolved oxygen, pH, and conductivity were measured in all test beakers at the end of the experiment. Samples of the sediment and the overlaying water (only from one replicate per nominal concentration) were analysed for HBCDD at the start (day 0) and the end (day 28) of the bioassays (i.e. 14 and 42 days after the spiking of the sediments). Pore water concentrations of HBCDD were only measured at the end of the experiments. In samples of overlaying water and pore water HBCDD concentrations were enriched by using solid phase extraction techniques (SPE-C18), while sediment samples were extracted with acetone and acetone/hexane, before measurement of HBCDD with GC/MS. The detection limits were 2.5-12.5 µg/l in water and 25-125 µg/kg dwt in sediment, depending on the sample volume.

Results

The measured treatment concentrations in the test with *L. variegatus*, at the start (day 0) and end of exposure (day 28), are summarised in Table 3-151 below. The mean concentrations (geometric mean of day 0 and 28) have also been calculated. No HBCDD was detected in the overlaying water or in the pore water.

Table 3-151 Levels of HBCDD in the test with *L. variegatus*.

Exposure conc. (mg HBCDD/kg dwt)			
nominal	day 0	day 28	geom. mean (day 0 and 28)
solv. contr.	n.d.	n.d.	-
0.05	n.d.	n.d.	-
0.5	0.4	0.1	0.2
5	4.1	2.4	3.1
50	34.8	23.7	28.7
500	382.1	240.6	303.2

The HBCDD concentrations at the start of the exposure were 70-82 % of the nominal concentrations. The mean pH was 8.7±0.15, dissolved oxygen was 7.5±0.81 mg/l, and conductivity was 1026±199 µs/cm (mean of all beakers at the end of experiment).

The results for the endpoints total number of worms and biomass are presented in Table 3-152.

Table 3-152 Result for the endpoints total number of worms and biomass of *L. variegatus*.

Treatment (nominal)	Total number of worms (mean ± SD)	Biomass (mean ± SD)
Control	61.0±14.4	41.7±8.5
Solv. control. .	56.5±13.9	34.0±2.2
0.05	41.0±10.2	29.0±9.8
0.5	40.0±7.2	23.6±11.4
5	46.0±7.4	34.8±4.9
50	31.8±2.5	24.3±5.2
500	7.5±5.3	5.3±3.0

Significant reduction (one-way ANOVA; Duncan´s multiple range test.) of the total number of worms was observed in the nominal 50 and 500 mg/kg dwt treatment. The reduction was 43 % and 87 % respectively compared to the solvent control. For this endpoint a NOEC of 3.1 mg/kg dwt and a LOEC of 28.7 mg/kg dwt were calculated (geometric mean of measured concentrations at exposure days 0 and 28).

The number of large worms as well as small worms decreased similarly to the total number of worms. However, for both these endpoints the reduction was significant (Kruskal-wallis-H-Test and Nemenyi-Test) only at the nominal concentration of 500 mg/kg dwt, resulting in a NOEC of 28.7 mg/kg dwt and a LOEC of 303 mg/kg dwt (geometric mean concentration). For the endpoint mean biomass a significant reduction was calculated (one-way ANOVA;

Duncan´s multiple range test) at the highest HBCDD treatment, resulting in the same NOEC and LOEC values as for the endpoints large and small worms. No deformations were observed.

The measured treatment concentrations in the test with *C. riparius*, at the start (day 0) and end of the exposure (day 28), are summarised in Table 3-153 below. The mean concentrations (geometric mean of day 0 and 28) have also been calculated. No HBCDD was detected in the overlaying water or in the pore water.

Table 3-153 Levels of HBCDD in the test with *C. riparius*.

Exposure conc. (mg/kg dwt)			
nominal	**day 0**	**day 28**	**geom. mean (day 0 and 28)**
solv. contr.	n.d.	n.d.	-
0.05	n.d.	n.d.	-
0.5	n.d.	n.d.	-
5	1.59	0.87	1.2
50	18.9	9.8	13.6
500	157.6	160.3	159

In this test the recovery of the spiked HBCDD (about 35 %) in the sediment was much less than in the *L. variegatus* tests. The reason for this is not known but it might be a possibility that a larger amount of HBCDD adsorbed to the surface of the beaker in the chironomid test compared to the lumbriculus test (M. Oetken, personal communication).

The results of the emergence test, covering the life cycle from first instar larvae to emerging adults, showed very low total emergence and large variations in the solvent control. Of the added 20 larvae, 11.8±7.7 emerged in the solvent control while the result for the ordinary control was 16.5±1.4. Therefore, neither the results for the total emergence nor for the emergence rate were considered valid for the purpose of the risk assessment, although the authors defined a LOEC for the rate of emergence at the highest tested concentration.

For the production of eggs of the F1 generation, the mean number of eggs was significantly reduced at the highest treatment concentration, 159 mg/kg dwt (geometric mean of measured concentrations at exposure days 0 and 28).

In conclusion, the results from the *L. variegatus* test are considered valid for the purpose of the risk assessment. The overall LOEC for this species is 28.7 mg HBCDD/kg dwt and the corresponding NOEC is 3.1 mg/kg dwt. Since the organic carbon content in standard sediment

$$NOECsed_{standard,i} = NOECsed_i \cdot \frac{Foc_{sed}}{Foc_{sed,exp}}$$

is 5 %, test sediment with other organic carbon content has to be normalized. (EUSES 2.0 background report, equation 243):

The organic carbon content in the test was 1.8 % and therefore the normalized NOEC will be 8.61 mg/kg dwt.

The emergence results from the *C. riparius* test are considered invalid due to the low total emergence and large variation in the solvent control. Such variation was not found in the solvent control for the endpoint 'number of eggs from the F1 generation'. Therefore, the result for this endpoint is considered valid and the LOEC of 159 mg HBCDD/kg dwt is taken as the LOEC for *C. riparius*. The corresponding NOEC is 13.6 mg/kg dwt. The normalized NOEC is 37.8 mg/kg dwt.

This study has been questioned due to the source for organic matter in the test sediment. It has been argued that the *Urtica* and *Alnus* leaves might have caused an eventual increase of ammonia in the test system, which could have affected the test results. Krueger and Thomas (Krueger and Thomas, 2005) evaluated the effectiveness of different sediment amendments on water quality in chironomid test systems, with different sources of organic material (and food source). The organic material in the different sediments either consisted of leaves of *Urtica* and *Alnus* or of α-cellulose and Tetramine (fish food), at an organic carbon content of 2 %. In this study enhanced ammonia concentrations were found in pore water and overlaying water in static systems with *Urtica* and *Alnus* compared to static systems with α-cellulose and Tetramine, in measurements 5 and 11 days after the start of the experiment, but not consistently after day 11 when ammonia concentrations decreased in the *Urtica*/*Alnus* system. In addition, oxygen concentrations were lower in the *Urtica*/*Alnus* system, minimum 2.3 mg/l, compared to the α-cellulose/Tetramine system, during the 11 first days. The percent emergence of adult midges was decreased in the *Urtica*/*Alnus* system compared to the α-cellulose/Tetramine system, 60 compared to 99 % (emergence of adult midges was first noted at day 11 of the test).

In the tests by Oetken *et al*. (*Oetken et al.*, 2001) the ammonia was not measured. However, the sediment was aged for 14 days before exposure of the test organisms started, and according to the results of Krueger and Thomas (described above), a possible peak in ammonia concentrations due to the use of *Urtica*/*Alnus* could have diminished by this time.

Furthermore, the populations of the controls increased with a factor of 6 within 28 days. On day 28 all individuals of the control and the solvent control appeared fit and vital, so there was no indication for any kind of stress (M. Oetken, personal communication). Therefore, it is not considered likely that excess ammonia affected the results in this study.

3.3.1.7 Predicted no effect concentration for sediment dwelling organisms ($PNEC_{sediment}$)

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. One study used spiked sediment with 2 % total organic carbon content and the other used spiked sediment with 5 % total organic carbon content. When calculating $PNEC_{sed}$ the two tests are taken as one since they are almost similar. The NOEC for Hyalella is then 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 %). 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.*

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.

The PNEC sediment can also be provisionally calculated with the equilibrium partition method. The parameters used for the calculations are listed in Table 3-154 below.

Table 3-154 Equations, conversion factors and data used for calculation of PNECsed

Equations/conversion factor/data	Result/data	Reference/data source
PNECsed = Ksusp-water × PNECwater × 1000 / RHOsusp	0.306 mg/kg wwt	Eq. 70 rev TGD
Ksusp-water = Fwater susp + F solid susp × (Kp susp/1000) × RHOsolid	1136 m^3/m^3	Eq. 24 rev TGD
Kp susp = Foc susp × Koc	4540 l/kg	Eq. 23 rev TGD
RHOsusp = Fsolid susp × RHO solid + Fwater susp × RHO water	1150 kg/m^3	Eq. 18 and calculated at page 44, rev TGD
F solid susp	0.1 m^3/m^3	Table 5 rev TGD
Fwater susp	0.9 m^3/m^3	Table 5 rev TGD
Foc susp	0.1 kg_{oc}/kg_{solid}	Table 5 rev TGD
Koc	4.54×104 l/kg	data set Ch.4; EUSES
RHOsolid	2500 kg/m^3	Table 5 rev TGD
PNECwater	3.1×10-4 mg/l	RAR

##¤¤Thus $PNEC_{sed}$ = 0.306 mg/kg wwt in suspended matter. The standard volume fraction of solids in suspended matter is 0.1 (TGD). The weight fraction suspended solid will then become 0.1×2500/1150 = 0.217. The dry weight based $PNEC_{sed\ (=susp.\ matter)}$ calculated with the equilibrium partition is then 0.306/0.217 = 1.41 mg/kg dwt. Standard sediments contains a weight fraction of 0.05 organic carbon (kg_{oc}/kg_{solid}) according to TGD (table 5) and the EUSES manual. Hence, the calculated $PNEC_{sed\ (=susp.\ matter)}$ is corrected for this organic content, resulting in a PNECsed of 1.41×0.1/0.05 = 2.82 mg/kg dwt.

For substances with log K_{ow} greater than 5, the equilibrium-partitioning method is used in a modified way. In order to take uptake via ingestion of sediment into account, the $PEC_{sed}/PNEC_{sed}$ ratio is increased by a factor of 10 (according to the revised TGD).

For comparison with the experimentally derived $PNEC_{sed}$ this increasing of the PEC/PNEC ratio by a factor 10 is similar to dividing the $PNEC_{sed}$ with the same factor. Such comparison would result in a $PNEC_{sed,}$ calculated with the equilibrium partition method, of 0.28 mg/kg dwt. This arithmetical operation confirms the experimentally derived $PNEC_{sed}$ of 0.86 mg/kg dwt.

The $PNEC_{sed}$ 0.86 mg/kg dwt is used for the calculations of PEC/PNEC. The value is experimentally derived and normalized to 5 % organic carbon content in the sediment.

3.3.2 Terrestrial compartment

3.3.2.1 Toxicity results for terrestrial organisms

3.3.2.1.1 Plant seedlings

A seedling emergence test with six plant species was performed (Porch *et al.*, 2002) according to standardised guidelines (Proposal for revision of OECD Guideline 308: Terrestrial non-target plant tests, U.S. Environmental Protection Agency Series 850 –OPPTS Number 850.4100 and 850.4225, public drafts). The study was conducted in compliance with GLP standards.

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

Experimental test conditions and test results are presented in Table 3-155 and Table 3-156, respectively.

Table 3-155 Experimental conditions for the test of effects of HBCDD on seedling emergence.

Parameter	Conditions
Test groups	Control without HBCDD Test substance nominal concentrations 40, 105, 276, 725, 1904 and 5000 mg HBCDD/kg
Number of seeds	Ten seeds per pot
Soil	53 % sand, 30 % silt and 17 % clay with an organic content of 1.9 %. Soil pH was 7.5.
Test chambers	Plastic pots 16 cm in diameter and 12 cm deep.
Environmental conditions	Mean temperature 22.4-26.2 °C. Mean relative humidity 32-66 %. Artificial lighting and natural sunlight with a 14 h photoperiod.
Test substance	HBCDD composite from Great Lakes Chemical Corporation, Eurobrom BV and Albemarle Corporation. Diastereomer composition: α 5.8 %; β 19.3 %; γ 74.9 %
Incorporation of HBCDD into soil	Mixing in batches per concentration
Test duration	21 days
Sampling	At termination of the test

Table 3-156 Results of measurements of effects of HBCDD on seedling emergence.

Parameter	Results
Mean measured test levels	0 (control), 31.3, 97.8, 297, 764, 2230, and 6200 mg HBCDD/kg dry soil
Seedling emergence	NOEC >5000 mg HBCDD/kg soil for corn (*Zea mays*), cucumber (*Cucumis*

Parameter	Results
Survival Shoot dry weight Height	*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.

3.3.2.1.2 Earthworms

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 (Aufderheide *et al.*, 2003) according to standardised guidelines (OECD guideline 207: Earthworm acute toxicity tests; Proposed OECD Guideline: Earthworm reproduction test (*Eisenia fetida/andrei*), U.S. Environmental Protection Agency Series 850 –OPPTS Number 850.6200). The study was conducted in compliance with GLP standards.

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. In addition to the toxicity studies also measurements of the HBCDD concentration in the worms after 28 days of exposure were performed. The results from these measurements are presented in section 3.1.3.2.4 bioaccumulation in earthworms.

Experimental test conditions are presented in Table 3-157 below.

Table 3-157 Experimental conditions for the test of effects of HBCDD on survival and reproduction of earthworm.

Parameter	Conditions
Test groups	Control without HBCDD Test substance nominal concentrations 78.5, 157, 313, 625, 1250, 2500 and 5000 mg HBCDD/kg dry soil
Number of animals	Ten worms per jar
Soil	80 % sand, 8 % silt and 12 % clay with organic matter (carbon) content 7.4 (4.3) %. Soil pH was 6.5±0.5.
Test chambers	1.8 l glass jars, 21.2 cm high, 12 cm diameter with 500g dry soil

Environmental conditions	19.4-22.7 °C Soil pH 6.55-6.67 day 0, 5.50-5.68 day 56 Soil moisture 18.9-23.3 % day 0, 34.4-42.3 % day 56 16 hr light
Test substance	HBCDD composite from Great Lakes Chemical Corporation, Eurobrom BV and Albemarle Corporation. Diastereomer composition: α 5.8 %; β 19.3 %; γ 74.9 %
Incorporation of HBCDD into soil	Mixed in dry soil in batches per concentration
Test duration	56 days
Sampling	Day 0, 28 and 56

At day 28 it was observed that 2 worms could not be found in the lowest test concentration, they were therefore treated as dead.

A summary of the results are given inTable 3-158.

Table 3-158 Summary of the HBCDD effects on survival and reproduction of earthworm, and measurements of HBCDD in soil.

Parameter	**Results**
Survival day 28	Mg HBCDD/kg dry soil
EC_{10}	>4190
EC_{50}	>4190
NOEC	4190
Reproduction day 56	Mg HBCDD/kg dry soil
EC_{10}	21.6* (95 % confidence limits 0.000468-110)
EC_{50}	771* (95 % confidence limits 225-4900)
NOEC	128
Mean measured concentration of HBCDD in soil Day 28	<1.28 (control), 61..2, 145, 244, 578, 1150, 2180, and 4190 mg HBCDD/kg dry soil
Mean measured concentration of HBCDD in soil Day 56	<1.35 (control), 51.5, 128,235,543,1070,2020 and 3990 mg HBCDD/kg dry soil

*calculated using probit analysis.

Reproduction was a much more sensitive endpoint compared to survival. The average reproduction as well as max-min ranges, for each test concentration, is shown in Figure 3-14.

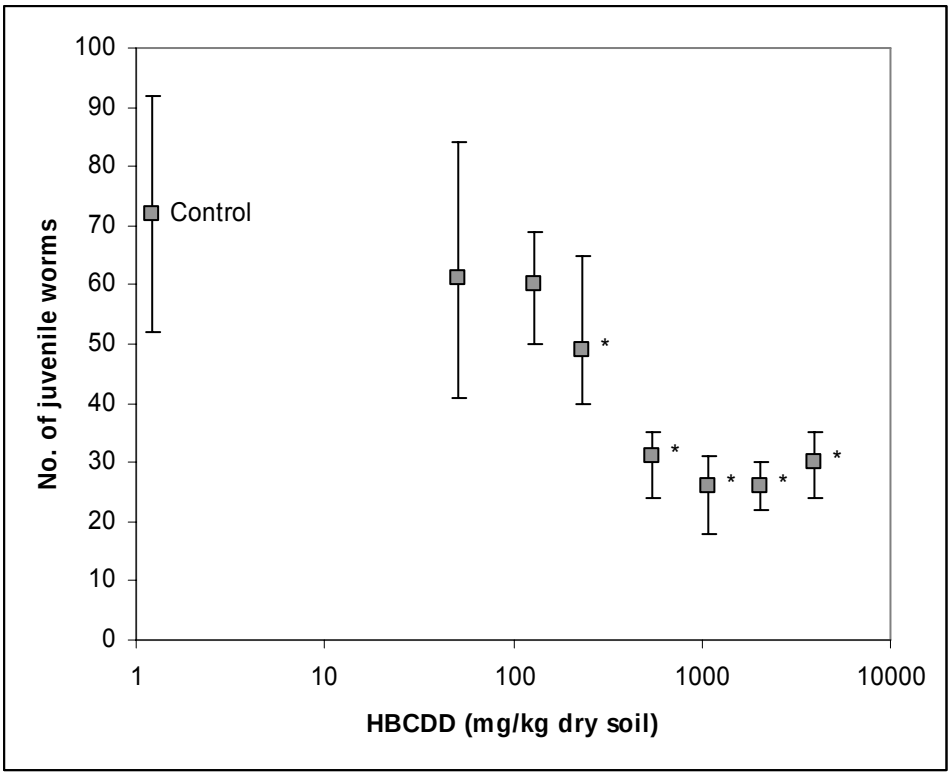


Figure 3-14: The results for the effects on reproduction, shown as averages and max-min ranges for each test concentration. The numbers of replicates were 8 for the control and 4 for the different test concentrations.

*Statistically significant ($p < 0.05$) reduction in the number of juvenile worms produced as compared to the control.

The study report give a NOEC of 128 mg HBCDD/kg dry soil for reproduction, while the estimated EC_{10} is 21.6 mg HBCDD/kg dry soil. The reason that the estimated EC_{10} is below the NOEC is due to high variability in the data at the lower test concentrations, which resulted in differences from the control that were not significant, also resulting in wide confidence limits for the EC_{10}. Based on the OECD document "Current Approaches in the Statistical Analysis of Ecotoxicity Data: A Guidance to Application (2005), finding no strong reasons not to assume monotonicity, the step-down approach using the Jonkheere-Terpstra method results in a NOEC of 128 mg HBCDD/kg dw. and a LOEC of 235 mg HBCDD/kg dw. (one-sided p-value=0.0026, exact permutation). For the purpose of this risk assessment it is therefore decided to use a NOEC of 128 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.

3.3.2.1.3 Micro-organisms

A study on the effects of HBCDD on micro-organisms in soil performed according to the OECD guideline No. 216 "Soil micro-organisms: Nitrogen Transformation Test" has been performed by ECT Oekotoxicologie sponsored by EBFRIP (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 were 104%, 83.1% and 75%, respectively.

No statistically significant differences in nitrate production between the controls and HBCDD treated soil samples were detected. (ANOVA, $p \leq 0.05$). The results are presented in Table 3-159.

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

Table 3-159 Mean concentration and standard deviation (SD) of nitrate in soil treated with HBCDD and in controls on day 0 and day 28 of the test.

Treatment Nominal concentration (mg HBCDD/kg soil dw)	Day 0 Nitrate concentration (mg/kg soil dw)	SD	Day 28 Nitrate concentration (mg/kg soil dw)	SD
0.0 (control)	4.57	0.15	56.18	5.57
0.0 (solvent control)	4.61	0.36	62.28	3.72
10..0	4.26	0.20	58.94	6.40
31.6	n.d.	n.a.	54.22	3.13
100.0	4.23	0.22	54.06	1.29
316.2	n.d.	n.a	60.11	3.52
1000.0	5.57	0.52	59.64	5.38

n.d- not determined, values on day 0 below or slightly below the detection limit of 5.0 mg NO_3 kg^{-1} dw.

n.a- not applicable

3.3.2.2 Predicted no effect concentration for terrestrial organisms ($PNEC_{soil}$)

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.

3.3.3 Atmosphere

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

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.

3.3.4 Non compartment specific effects

The assessment of the potential impact of substances on top predators is based on the accumulation of hydrophobic chemicals through the food chains. This accumulation may result in toxic concentrations in predatory birds or mammals ingesting biota containing the chemical. This effect is called secondary poisoning and should in principle be assessed by comparing the measured or estimated concentrations in the tissues and organs of the top predators with the no-effect concentrations for these predators expressed as the internal dose. In practice, data on internal concentrations in wildlife animals are hardly ever available and most no-effect levels are expressed in term of concentrations of the food that the organisms consume.

According to the revised TGD only toxicity studies reporting on dietary and oral exposure are relevant, as the pathway for secondary poisoning is referring exclusively to the uptake through the food chain. Secondary poisoning effects on bird and mammal populations rarely become manifest in short-term studies. Therefore, results from long-term studies are strongly preferred, such as NOECs for mortality, reproduction or growth.

3.3.4.1 Toxicity to mammalians

The results from the available studies are evaluated in the human health part (section **4.1.2.6**.). A NOAEL from a 90 days study would normally be preferred as the basis for derivation a PNEC for secondary poisoning, but the uncertainties introduced in the evaluation of the 90 days study by the dosing of HBCDD-particles to the animals, leads to the choice of a NOAEL form the recent 28-days study.
The most recent 28 days study in rats was performed using a benchmark model design and oral administration of dissolved HBCDD. The study shows effects on the liver, the thyroid, and the pituitary. Overall, a NOAEL/BMD-L of 22.9 mg/kg/day for liver weight increase is proposed for repeated dose toxicity (van der Ven *et al.*, 2006). A BMD-L of 22.9 mg/kg/day will most likely also cover for effects on the thyroid and pituitary system assuming that hepatic enzyme induction is one factor contributing to the effects on the thyroid. This NOAEL of 22.9 mg/kg bwt/day for rats will be used as mammalian toxicity data for secondary poisoning.

3.3.4.2 $PNEC_{oral}$ for non compartment specific effects

For the assessment of secondary poisoning, the results have to be expressed as the 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 (section **4.1.2.6**.) 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 obtain 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 characterisation 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.
The conversion factors and equations used for the calculation of $PNEC_{oral}$ are listed in Table 3-160.

Table 3-160 Equations, conversion factors and data used for calculation $PNEC_{oral}$.

Equations/conversion factor/data	Result/data	Reference/data source

Equations/conversion factor/data	Result/data	Reference/data source
$NOAEC_{diet}$	150 ppm dry weight	RAR, section 4.1.2.9
AF_{oral}	30	Table 23 in TGD
$PNEC_{oral} = \frac{NOAEC_{diet}}{AF_{oral}}$	5 mg/kg food	Eq 79 in TGD

Thus $PNEC_{oral}$ = 5 mg/kg food.

3.3.4.3 Toxicity to birds exposed as eggs (indicative evaluation)

There are no toxicity data for birds exposed to HBCDD via the feed, to base a $PNEC_{oral}$ on.

On the other hand, the fact that HBCDD has been found in the eggs of top predators, e.g. peregrine falcon (see section 3.1.7.2), is an important finding that should be considered in the assessment of long-term risks. However, there is no guidance in TGD on how to assess the risk from exposure to substances in birds' eggs. In agreement with the risk assessment report for bis(pentabromophenyl) ether, a provisional assessment to derive an indicative estimate of the effects on birds is performed: The measured levels in eggs are compared with the estimated 'internal dose' for developmental neurotoxicity in mice. Neurotoxicity is considered to be of special importance for predatory birds, since they are dependent on extremely well-developed senses (hunting behaviour).

In the neurotoxicity study by Eriksson *et al* (Eriksson *et al.*, 2002), evaluated in section **4.1.2.9**, neonatal mice were exposed on day 10 to HBCDD at 0.9 or 13.5 mg/kg body weight as a single oral dose by gavage. Spontaneous behaviour of the mice was significantly altered in both dose groups. Hence, the nominal dose 0.9 mg/kg body weight (wet weight) was set as an indicative LOAEL for behavioural disturbances in mice when they are exposed at a sensitive stage of brain development. In the human health evaluation there is a conclusion i) for this endpoint, i.e. the study should be repeated.

The internal dose is dependent on the absorption, which is assumed to be 100 % for HBCDD given by gavage, see section **4.1.2.1**. The estimated internal dose will then be 0.9 mg/kg wwt This internal dose will be used as a basis for a comparison (risk characterisation section **3.4.4.3**) with the levels found in birds' eggs.

3.3.5 Marine effects assessment

3.3.5.1 Toxicity results for marine aquatic organisms

Studies on the effects of HBCDD on marine organisms are scarce. For the aquatic compartment, only studies on marine algae exist. The lowest reliable EC_{50}, 52 µg/l of HBCDD, was observed for *Skeletonema costatum*, and the 72-hr NOEC for this algae was determined to 10µg/l <NOEC ≤40 µg/l, see section 3.3.1.1.3. There are no marine data available for other phyla than the phyla represented in the freshwater database.

3.3.5.2 Predicted No Effect Concentration for marine waters ($PNEC_{water, marine}$)

For the marine environment the PNEC is calculated with the use of same toxicity data as for the freshwater aquatic compartment

According to TGD (table 25) an assessment factor of 100 should be applied when PNEC is based on the lowest long-term NOEC from three freshwater or saltwater species, representing three trophic levels.

Hence, based on the lowest NOEC of 3.1 µg/l for *Daphnia magna* and an assessment factor of 100 the $PNEC_{water, marine}$ becomes 3.1/100 = 0.03 µg HBCDD/l.

There is no guidance in the TGD how to calculate a PNEC for intermittent releases to the marine environment. In fact, EUSES by default uses the long-term PNEC also for intermittent releases to the marine environment. However, it is likely that an intermittent release is rapidly diluted also in the marine recipient, which would justify the use of a PNEC based on short-term toxicity tests. For the calculation of a $PNEC_{intermittent}$ for the freshwater environment an assessment factor of 100 was applied on the lowest $L(EC)_{50}$ of three short-term tests. To take into account the greater diversity of species in the marine environment, and in line with the calculation of the long-term PNEC, it is proposed that an AF of 1000 is applied on the lowest EC_{50} from the freshwater database for calculation of $PNEC_{intermittent}$ for the marine environment.

The lowest EC_{50} for *Skeletonema costatum* in the algae growth inhibition test is 52 µg/l. Thus the PNEC for intermittent releases in marine water is 52/1000 = 0.05 µg/l.

3.3.5.3 Toxicity results for marine sediment organisms

There are no toxicity data available for effects of HBCDD on marine sediment organisms. In such cases fresh water data may be applied. According to TGD, only long-term sub-lethal endpoints are considered applicable to marine risk assessment because of the long-term exposure of benthic organisms to sediment-bound substances that occur under field conditions.

There are long-term freshwater NOECs available for *Hyalella azteca, Lumbriculus variegatus* and *Chironomus riparius* (see section 3.3.1.6). The lowest NOEC, 3.1 mg/kg dwt was obtained for *L. variegatus*. The organic carbon content in the sediment in this test was 1.8 %. This value corresponds well with the assumed organic carbon content in marine sediment (<2 % in whole-sediment according to TGD, section 4.3.2.2). However, since the organic carbon content in the standard sediment used for the PEC calculation is 5 % (EUSES 2.0 background report) test sediment with other organic carbon content has to be normalized according to equation 243:

$$NOECsed_{standard,i} = NOECsed_i \cdot \frac{Foc_{sed}}{Foc_{sed,exp}}$$

The organic carbon content in the test was 1.8 % and therefore the normalized NOEC will be 8.6 mg/kg dwt.

3.3.5.4 Predicted No Effect Concentration for marine sediment organisms ($PNEC_{sediment, marine}$)

For the marine environment the PNEC is calculated with the use of same toxicity data as for the freshwater aquatic compartment.

According to TGD (table 27) an assessment factor of 50 should be applied when PNEC is based on the lowest long-term NOEC of three species representing different living and feeding conditions.

Hence, based on the lowest NOEC of 8.6 mg/kg dwt for *Lumbriculus variegatus* and an assessment factor of 50 the $PNEC_{sediment, marine}$ becomes 8.6/50 = 0.17 mg HBCDD/kg dwt.

3.3.6 Summary of environmental effects data

The data on environmental effects from studies that are considered valid are summarised in Table 3-161

Table 3-161 Summary of data on the environmental effects of HBCDD from valid studies.

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, 1997b)**
Chronic toxicity			
Rainbow trout (*Oncorhynchus mykiss*)	Flow-through OECD 210 and OPPTS 850.1400	NOEC µg/l Hatching success ≥3.7 Swim-up ≥3.7 Larvae and fry survival ≥3.7 Growth ≥3.7	**(Drottar *et al.*, 2001)**
INVERTEBRATES			
Acute toxicity			
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, 1997a)**
Chronic toxicity			
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 mg/l	Study 2 **(Roberts and Swigert, 1997)**

Compartment/Species	Method	Results#	Remark and reference
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	Study 3 **(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	NOEC <40.6 µg/l EC_{50} >40.6	Study 4 **(Desjardins *et al.*, 2004)**
Skeletonema costatum	OECD 201	NOEC >10 µg/l **EC_{50} 52 µg/l**	Study 5 **(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 estimated. **(Schaefer and Siddiqui, 2003)**
SEDIMENT COMPARTMENT			
INVERTEBRATES			
Hyalella azteca (Amphipod)	Sediment toxicity test 28-day exposure period under flow-through conditions.	LOEC >1000 mg/kg dwt of sediment NOEC 1000 mg/kg dwt of sediment.	**(Thomas *et al.*, 2003b)**
Lumbriculus variegatus (Worm)	28d- sediment bioassay	LOEC = 28.7 mg/kg dwt **NOEC = 3.1 mg/kg dwt** **Normalized:** **NOEC = 8.61 mg/kg dwt**	**(Oetken *et al.*, 2001)**
Chironomus riparius (Mosquito)	28d- sediment bioassay Egg production of F generation	LOEC = 159 mg/kg dwt NOEC = 13.6 mg/kg dwt Normalized: NOEC = 37.8 mg/kg dwt	**(Oetken *et al.*, 2001)**
TERRESTRIAL COMPARTMENT			
Soil microorganisms	Nitrogen transformation test OECD 216	NOEC > 750 mg/kg dry soil	**(Förster, 2007)**
PLANTS			
Plants: corn (*Zea mays*), cucumber (*Cucumis sativa*), onion (*Allium cepa*), ryegrass, (*Lolium perenne*), soybean	Seedling emergence, suvival, height 21 days OECD 308 (proposal for	NOEC >5000 mg/kg dry soil	**(Porch *et al.*, 2002)**

Compartment/Species	Method	Results#	Remark and reference
(*Glycine max*), and tomato (*Lycopersicon esculentum)*	revision), 850.4100 and 850.4225 (public drafts)		
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)**

#Values in **bold** are used to calculate PNEC.

3.4 RISK CHARACTERISATION

General introduction

The quantitative estimates of the substance flow of HBCDD, relevant to exposure of the environment, are covered in the risk assessment by nine scenarios. Estimations of releases from point sources have been possible to quantify from site-specific data and default data from Emission Scenario Document. The amount and the coverage of site-specific data vary between the scenarios.

Issues that are not (fully) covered by the risk assessment

Industrial uses of HBCDD in (extruded, expanded and high impact) polystyrene as well as in textile coatings were included in the report. According to literature sources, HBCDD can be used also in *coatings, adhesives, polypropylene, SAN resins, and in ABS*. These applications have not been confirmed by industry to be applicable in the EU. Therefore, these uses are not included in the assessment at this stage, although, it cannot be exclude that they exist.

Distribution and transport of HBCDD, and the releases thereby, were not included in the risk assessment due to lack of data. Distribution and transport pertains to a variety of materials such as powder in bags, granules, beads, liquid dispersion in tanks on the road or by railway, backcoated textile and extruded, expanded and high impact polystyrene as well as preconsumer and postconsumer waste.

Diffuse releases from end products during service life. The releases of HBCDD from end-products during use, as well as from waste left in the environment, and from landfills, are dependent on the stock pile of the end-products, on the migration of HBCDD in the matrix enclosing the substance and, on the resistance of the matrix to degradation. Emissions from end-products due to migration of HBCDD is likely to occur since HBCDD is not chemically bound to the matrix. The service life time of articles containing HBCDD varies from short to very long, *i.e.* from less than one year (e.g. packaging material) to 50 or more (e.g. insulation on road banks). It is therefore probable that releases from end-products will show an increasing trend with constant HBCDD consumption since the amount of HBCDD stored in

the technosphere is likely to increase. It has not been possible to estimate with accuracy the accumulation of HBCDD in the technosphere and the subsequent emissions to the environment. Hence, the estimations made for regional releases from end-products are based on simplified assumptions on migration and technical lifetime of end-products. The estimations serve to indicate an order of magnitude of end-product releases, which could be compared to point source releases.

Waste and waste management. At present it seems that most of the HBCDD built into the construction sector since beginning of the commercialisation of the substance, is still in use. The stockpile is being built up. In some decades this material will begin to be left as waste in the environment, landfilled, recycled or incinerated. It seems, as a likely scenario for the future, that the amounts of HBCDD disposed off will increase markedly compared to amounts disposed off today. According to EU regulations, organic waste should not be disposed off in landfills and indeed industry's preferred solution for treating demolition waste is incineration. However, industry is aware that incineration capacities might be limited for a while in some EU states. During this transition period, demolition waste should be disposed off following the EU Directive and advice on landfills.

Disintegration of the polymer matrix takes place on the landfill and in the environment where fabrics, XPS and EPS have been left after use. HBCDD-containing polymer waste put on landfill could leak out when the polymer is degraded. Decomposition of PS under landfill conditions will occur. It is still an open question what happens to the HBCDD remaining in the PS matrix*Demolition of buildings.* When dismantling insulation boards containing HBCDD, or when demolishing buildings at the end of their lifetime, there is a risk that small particles are spread around.

Recovery/recycling of HBCDD-containing end-products, such as building material, are activities which will increase in importance in the future. If flame retarded material is not kept apart from other material in the recycling processes, there is a risk that the good intention to recycle results in a occurrence of HBCDD in end-products which do not need to be flame retarded. This potential flow of HBCDD has not been quantified in the risk assessment.

PNECs used in the risk characterisation

The calculated predicted no effect concentrations in different environmental compartments that will be used in the risk assessment of HBCDD are given in the Table 3-162 below. A $PNEC_{intermittent,\ marine}$ has been calculated and used for intermittent releases to the marine environment. There is no guidance in the TGD on how to calculate a $PNEC_{intermittent,\ marine}$. By default, EUSES uses the long-term marine PNEC. However, it is likely that an intermittent release is rapidly diluted also in the marine recipient, which would justify the use of a PNEC based on short-term toxicity tests. All PNECs in the water phase are below the maximum water solubility of HBCDD.

Table 3-162 PNECs used in the risk characterization.

Compartment	Assessment factor	PNEC
Aquatic compartment	10	0.31µg HBCDD/l

Compartment	Assessment factor	PNEC
Intermittent release, aquatic environment	100	0.5 µg HBCDD/l
Marine environment	100	0.03 µg HBCDD/l
Intermittent release, marine environment	1000	0.05 µg HBCDD/l
Sediment	10	0.86 mg HBCDD/kg dwt
Sediment, marine environment	50	0.17 mg HBCDD/kg dwt
Micro-organisms in STP	100	0.15 mg HBCDD/l
Atmospheric compartment	-	-
Terrestrial compartment	10	5.9 mg HBCDD/kg dwt
Non compartment specific effects	30t	5.0 mg HBCDD/kg food

The use of alternative biodegradation scenarios

The DT50s for biodegradation, that have been used in the calculations of the PECs, are 191 days (12^{o}C) for aerobic and 125 days (12^{o}C) for anaerobic sediment, whereas no degradation is assumed for soil (section 3.1.3.1.2., simulation study 2). These values are taken from a study where fairly high concentrations of HBCDD were used, and it has been disputed whether they are relevant for the environment. EUSES modelling has therefore also been performed with DT50s from a study (simulation study 1) where lower concentrations of HBCDD was used for comparison. The DT50s were 21 days for aerobic sediment, 2.8 days for anaerobic sediment, and 119 days for soil. In general the resulting PEC/PNEC-ratios are only slightly affected by the alternative DT50-values with exception of the soil compartment.For the sites/scenarios where the conclusion would change (PEC/PNEC-ratios change from >1 to <1) by the use of alternative DT50s, both values are included in the relevant PEC/PNEC-tables below, (*i.e.* for the soil compartment and terrestrial predators).

3.4.1 Aquatic compartment

This section includes PEC/PNEC for aquatic organisms (freshwater and marine), sediment dwelling organisms, and the sewage treatment process. The respective PNECs are shown in Table 3-162. For information on the calculation of the PEC values, see chapters 3.1.4-3.1.7 and chapter 3.2.3.

When there is information, for a local site, that the emissions are only directed to the marine environment, only marine risk characterisation has been performed. For sites known to be located far away from the sea, only fresh water assessment has been performed. In the cases where there are no information on the location, or when the emissions are known to be directed to a freshwater body close to the sea, risk characterisation has been carried out for both the freshwater and the marine environment.

For several sites information is missing regarding whether the site is connected to a STP or not. In such cases (and for the generic scenarios) risk characterisation is carried out for two scenarios, with and without STP.

According to the distribution pattern of HBCDD (low water solubility and high Kow) the majority of the HBCDD released can be expected to be rather rapidly adsorbed to particles and distributed to sediment.

The relevance of the PECs in sediment for sites/scenarios with intermittent release calculated by EUSES is questionable. The reason for this is that EUSES calculates a ´snapshot´ PEC, representative only for the intermittent emission period (1 or 2 days). EUSES does not take into consideration that the concentration of HBCDD in the sediment will be diluted by sedimentation of less contaminated particulate matter over time. The PEC will therefore be an overestimation of the exposure relevant for comparison to a long term PNEC. The PNEC for sediment is derived from studies where the organisms have been exposed for 28 days. A more relevant PEC could be a time weighted average over e.g. 30 days. This is presently not possible to do with EUSES 2.03. Therefore, a pragmatic approach is taken and the $PEC_{sediment}$ derived by EUSES for sites having intermittent releases to water is divided by 30 (c.f 3.1.4.1.). This is thought to give a more realistic PEC than just using the "snapshot" $PEC_{sediment}$ derived by EUSES.

3.4.1.1 Risk characterisation ratios for the aquatic compartment

3.4.1.1.1 PEC/PNEC ratios for production and micronising

PEC/PNEC-ratios for production, and micronising, are shown in Table 3-163. For micronising there are no releases to water, and therefore no site-specific risk characterisation is performed as it is covered by the regional risk assessment.

Table 3-163 PEC/PNEC ratios for STP, surface water and sediment. Production and micronising of HBCDD.

Site	Connected to municipal STP (Yes/No)	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
ProdA*	Yes	**3.8**	**170**	**280**
ProdB	Yes	0.0014	0.091	0.15
Micronising	Yes/No**	-	-	-

*Production site A closed down in December 2003 and is included for illustrative purposes only.

**STP connection not known, however no emissions to wastewater.

For production site A all PEC/PNEC ratios >1. However, since this site has closed the RCRs are kept for illustrative purposes only. The UK authorities have a monitoring programme to follow up the environmental concentrations (sediment and biota) in the vicinity of the plant.

Production site B is situated at a river mouth entering into the Western Scheldt. It is not known to the rapporteur whether the waste water is released to the river or the estuary. It is therefore, not clear if the aquatic compartment is of relevance for this site. However, if it is, there is based on present data no concern, and there are no concern for production site B for any of the compartments. Also when comparing PNEC with measured sediment concentrations close to site B the PEC/PNEC ratios are below 1. Micronising has no emissions to water and, hence there are no local concerns.

Conclusion (ii) is drawn for production site B and for micronising for the STP, aquatic, and freshwater sediment compartment.

3.4.1.1.2 PEC/PNEC ratios for formulation of compound for EPS and HIPS

PEC/PNEC-ratios for the formulation of EPS compound, including where relevant productionof HIPS, are shown in Table 3-164.

Table 3-164 PEC/PNEC ratios for STP, surface water and sediment. Formulation of compound for EPS and HIPS.

Site	Connected to municipal STP (Yes/No)	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
Site A + M	No	-	0.11	0.17

Site	Connected to municipal STP (Yes/No)	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
Site F + N	No	-	0.35	0.58
Site I + O	No	-	0.13	0.21
Site B	Yes	0.00028	0.00048	0.00079
Site C	No	-	0.16	0.26
Site D	No	-	0.14	0.23
Site E	No	-	023	0.37
Site G	Yes	0.0060	0.27	0.45
Site H	No	-	0.37	0.60
Site J	No	-	**4.7**	**7.7**
Site K	Yes	0.047	**2.2**	**3.6**
Site L	Yes	0.024	**1.1**	**1.7**
Site P*	No	-	**27**	**75 (2.5)***
GEN_EPS_FORM	Yes	0.091	**2.52**	**4**

* Intermittent releases. Figure within parenthesis represents PEC/PNEC ratio based on PEC sediment calculated by EUSES averaged over 30 days.

STP

No concern is indicated for the STPs for any of the sites.

Conclusion (ii) is drawn for all sites and for the generic scenario.

Freshwater

Conclusion (ii) is drawn for sites A+M, F+N, I+O, B, C, D, E, G and H.

Conclusion (iii) is drawn for sites, J, K, L and P and also for the generic scenario.

Freshwater sediment

Conclusion (ii) is drawn for sites A+M, I+O, F+N, B, C, D, E, G and H.

Conclusion (iii) is drawn for sites, J, K, L and P and for the generic scenario. For site P the relevance of the RCR is questionable due to intermittent release (c.f. 3.4.1). However, also when the PEC is divided by a factor of 30 to make it comparable to the long term PNEC for sediment the PEC/PNEC ratio is >1. Conclusion iii) is therefore considered relevant to draw for this site.

3.4.1.1.3 PEC/PNEC ratios for formulation of XPS compound

PEC/PNEC-ratios, for formulation of XPS compound for the manufacture of XPS, are shown in Table 3-165.

Table 3-165 PEC/PNEC ratios for STP, surface water and sediment. Formulation of XPS compound.

Site	Connected to municipal STP (Yes/No)	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
MasterbG	Yes	0.00028	0.10	0.17
MasterbH	Yes	0.00062	0.12	0.19
MasterbI	Yes	0.085	**3.9**	**6.4**
GEN_XPS_FORM	Yes	0.060	**2.8**	**4.6**

STP

No concern is indicated for the STPs for any of the sites including the generic scenario.

Conclusion (ii) is drawn for formulation of XPS compound.

Freshwater

Conclusion (ii) is drawn for sites MasterbG and MasterbH.

Conclusion (iii) is drawn for MasterbI and for the generic scenario.

Freshwater sediment

Conclusion (ii) is drawn for sites MasterbG and MasterbH

Conclusion (iii) is drawn for MasterbI and for the generic scenario.

3.4.1.1.4 PEC/PNEC ratios for formulation of polymer dispersions for textiles

PEC/PNEC-ratios for formulation of polymer dispersions of textiles are shown in Table 3-166.

Table 3-166 PEC/PNEC ratios for STP, surface water and sediment. Formulation of polymer dispersions for textiles.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
TexForm1	Yes	0.00023	0.10	0.16
	No	-	0.14	0.23

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
TexForm3	Yes/No*	-	0.090	0.15
TexForm4	Yes	0.00010	0.095	0.16
	No	-	0.11	0.19
TexForm5	Yes/No*	-	0.090	0.15
TexformA	Yes/No*	-	0.090	0.15
TexFormB	Yes	0.019	0.97	**1.6**
	No	-	**4.4**	**7.1**
GEN_TEX_FORM	Yes	0.22	**10**	**16**

#In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with and without STP.
* No emissions to waste water

STP

No concern is indicated for the STPs for any of the sites.

Conclusion (ii) is drawn for formulation of polymer dispersions for textiles

Freshwater

Conclusion (ii) is drawn for all local sites. It shall be noted however that the PEC/PNEC ratio for site TexFormB is >1 if it is assumed that it is not connected to a STP. However, the TGD default assumption is that sewage treatment is standard unless it can be assumed that direct discharges to water are widely practised. For this use area no such assumption can be made. .

Conclusion (iii) is drawn for for the generic scenario.

Freshwater sediment

Conclusion (ii) is drawn for sites TexForm1, TexForm3, TexForm4, TexForm5, and TexForm A.

Conclusion (iii) is drawn for site TexForm B and for the generic scenario.

3.4.1.1.5 PEC/PNECratios for industrial use of EPS compound

PEC/PNEC-ratios for industrial use of EPS compound are shown in Table 3-167.

Table 3-167 PEC/PNEC ratios for STP, surface water and sediment and marine. Industrial use of EPS compound

Site	Connected to municipal STP (Yes/No)	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
GEN_EPS_IndUse	Yes	0.0012	0.14	0.23

Site	Connected to municipal STP (Yes/No)	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
	No	-	0.35	0.57

Conclusion (ii) is drawn for the generic scenario for the STP, aquatic and freshwater sediment compartment.

3.4.1.1.6 PEC/PNECratios for industrial use of HIPS compound

PEC/PNEC-ratios for industrial use of HIPS compound are shown in Table 3-168.

Table 3-168 PEC/PNEC ratios for STP, surface water and sediment. Industrial use of EPS compound

Site	Connected to municipal STP (Yes/No)	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
GEN_HIPS_IndUse	Yes	0.0036	0.25	0.42
	No		0.88	**1.4**

STP

Conclusion (ii) is drawn for the STP.

Freshwater

Conclusion (ii) is drawn for surfacewater.

Freshwater sediment

Conclusion (ii) is drawn for sediment. It shall be noted however that the PEC/PNEC ratio becomes >1 if is assumed that there is no sewage treatment. However, the TGD default assumption is that sewage treatment is standard unless it can be assumed that direct discharges to water are widely practised. Since there is no information whatever for this use category, no such assumption is made.

3.4.1.1.7 PEC/PNEC ratios for industrial use of XPS compound

PEC/PNEC-ratios for industrial use of compound for manufacture of XPS are shown in Table 3-169.

Table 3-169 PEC/PNEC ratios for STP, surface water and sediment. Industrial use of XPS compound.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
XPS 1	Yes	0.10	0.14	0.22
	No	-	0.32	0.52
XPS 2*	Yes	0	0.090	0.15
XPS 3*	Yes	**4.2**	**2.2**	**6.1** (0.20)*
	No	-	**11**	**29** (0.83)*
XPS 11*	Yes	**7.0**	**39**	**110 (3.6)***
GEN_XPS_IndUse*	Yes	**26**	**150**	**400 (13)***

#In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with and without STP.

* Intermittent releases. Figure within parenthesis represents PEC/PNEC ratio based on PEC sediment calculated by EUSES averaged over 30 days.

STP

Conclusion (ii) is drawn for sites XPS 1 and XPS 2.

Conclusion (iii) is drawn for the STP for sites XPS 3 and XPS 11 and for the generic scenario.

Freshwater

Conclusion (ii) is drawn for sites XPS 1 and XPS 2.

Conclusion (iii) is drawn for sites XPS 3, XPS 11 and for the generic scenario.

Freshwater sediment

Conclusion (ii) is drawn for sites XPS 1, XPS 2 and XPS 3. Site XPS 3 has PEC/PNEC ratios > 1 if the PEC calculated by EUSES is used. However, when the PEC is divided by a factor of 30 to make it comparable to the long term PNEC for sediment the PEC/PNEC ratio is <1. Conclusion ii) is therefore considered relevant to draw for this site. In addition, possible emission reductions due to the risks for surface water will also reduce the exposure of the sediment.

Conclusion (iii) is drawn for site XPS 11 and for the generic scenario. Also when the PEC is divided by a factor of 30 to make it comparable to the long term PNEC for sediment the PEC/PNEC ratio is >1. Conclusion iii) is therefore considered relevant to draw for this site and the generic local site.

3.4.1.1.8 PEC/PNEC ratios for industrial use of HBCDD powder for flame retarded XPS.

PEC/PNEC-ratios for industrial use of HBCDD powder for the manufacture of XPS are shown in Table 3-170. No generic risk characterisation has been performed since data has been submitted for all of the sites.

Table 3-170 PEC/PNEC ratios for STP, surface water and sediment. Industrial use of HBCDD powder for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
XPS 4**, ***	Yes	**7.3**	**-**	**-**
XPS 5**	Yes	**1.9**	0.16	0.44
	No	-	0.57	**1.6** (0.052)**
XPS 6**	Yes	0.18	0.064	0.18
	No	-	0.10	0.28
XPS 7**	Yes	**12**	0.74	**2** (0.68)**
	No	-	**3.4**	**9.3** (0.31)**
XPS 8**	Yes	0.0040	0.076	0.21
	No	-	0.16	0.44
XPS 9	Yes/No*	-	0.090	0.15
XPS 10**	Yes	**20**	**80**	**220 (7)****
XPS 13**	Yes	0.0097	0.11	0.30
	No	-	0.32	0.86
XPS 14**, ***	Yes	0.0032	-	-
XPS 16	Yes/No*	-	0.090	0.15
XPS 17	No*	-	0.090	0.15
XPS 18	No*	-	0.090	0.15
XPS 20**	Yes	0.18	0.065	0.18
XPS 21**	Yes	**25**	**6.7**	**19** (0.62)**
	No	**-**	**33**	**89 (3.0)****
XPS 23**	Yes	0.00013	0.055	0.15
XPS 24**	Yes	0.00012	0.055	0.15
XPS 26**	Yes	0.071	045	**1.3** (0.041)**
XPS 27**	Yes	**8.4**	**47**	**130 (4.3)****

#In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with or without STP.

*No emissions to wastewater.

** Intermittent releases. Figure within parenthesis represents PEC/PNEC ratio based on PEC sediment calculated by EUSES averaged over 30 days.

***XPS4 and XPS14 are known to have releases to the sea.

STP

Conclusion (ii) is drawn for sites XPS 6, XPS 8, XPS 9, XPS 13, XPS 14, XPS 20, XPS 23, XPS 24 and XPS 26.

Conclusion (iii) is drawn for the STP for sites XPS 4, XPS 5, XPS 7, XPS 10, XPS 21 and XPS 27.

Freshwater

Conclusion (ii) is drawn for sites XPS 5, XPS 6, XPS 7, XPS 8, XPS 9, XPS 13, XPS 16, XPS 17, XPS 18 XPS 20, XPS 23, XPS 24 and XPS 26. It shall be noted that the PEC/PNEC ratio becomes >1 for site XPS 7 if it is assumed that there is no sewage treatment. However, the TGD default assumption is that sewage treatment is standard unless it can be assumed that direct discharges to water are widely practised. For this use area such an assumption cannot be made.

Conclusion (iii) is drawn for sites XPS 10, XPS 21 and XPS 27.

Freshwater sediment

Conclusion (ii) is drawn for sites XPS 5, XPS 6, XPS 7, XPS 8, XPS 9, XPS 13, XPS 16, XPS 17, XPS 18; XPS 20, XPS 21, XPS 23, XPS 24 and XPS26. For sites XPS 7, XPS 21 and XPS 26 the PEC/PNEC ratios are > 1 when the PEC calculated by EUSES is used. However, when the PEC is divided by a factor of 30 to make it comparable to the long term PNEC for sediment the PEC/PNEC ratio becomes <1. Conclusion ii) is therefore considered relevant to draw for these sites. For site XPS 21, the PEC/PNEC ratio is > 1 (also with the 30 day average PEC) if it is assumed that it lacks sewage treatment. However, as said earlier, the TGD default assumption is that sewage treatment is standard. In addition, possible emission reductions due to the risk for surface water will reduce the exposure of the sediment at this site.

Conclusion (iii) is drawn for sites XPS 10 and XPS 27 Even when the PEC calculated by EUSES is averaged over 30 days the PEC/PNEC ratio remains >1. It therefore, seems appropriate to draw conclusion iii).

3.4.1.1.9 PEC/PNEC ratios for industrial use of textile back-coating agent

PEC/PNEC-ratios for the life-cycle step industrial use of textile back-coating agent are shown in Table 3-171.

Table 3-171 PEC/PNEC ratios for STP, surface water and sediment. Industrial use of textile back-coating agent.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC STP	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
Backcoat.1	Yes	0.022	**1.1**	**1.7**
Backcoat.2	Yes	0.021	**1.1**	**1.7**
Backcoat.3	Yes	**3.7**	**170**	**270**
Backcoat.4	Yes	0.000043	0.092	0.15
	No	-	0.10	0.16
BackcoatC	Yes	0.0069	0.40	0.66
	No	-	**1.6**	**2.6**
GEN_TEX_IndUse	Yes	**3.7**	**170**	**270**
Monitoring data from textile industry area	River Viskan, SE			**1.9***
	River Calder, UK			**2.4***
	River Roch, UK			**3.1***

#In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with and without STP.

*River Viskan (1591 µg HBCDD/kg dwt)/ 860 µg HBCDD/kg dwt); River Calder (2037 µg HBCDD/kg dwt)/ 860 µg HBCDD/kg dwt); River Roch (2657 µg HBCDD/kg dwt)/ 860 µg HBCDD/kg dwt).

STP

Conclusion (ii) is drawn for sites Backcoat 1, Backcoat 2, Backcoat 4, and BackcoatC.

Conclusion (iii) is drawn for site Backcoat 3 and for the generic scenario.

Freshwater

Conclusion (ii) is drawn for site Backcoat 4.

Conclusion (iii) is drawn for sites Backcoat 1, Backcoat 2, Backcoat 3 and BackcoatC and for the generic scenario. It shall be noted that the PEC/PNEC ratio <1 for site Backcoat C if it is assumed that it is connected to a municipal sewage treatment plant. The TGD default assumption is that sewage treatment is standard unless it can be assumed that direct discharges to water are widely practised. For this use area it is considered appropriate to assume that direct discharges to water are (or has been) common based on the fact that measured HBCDD concentrations sediments associated to textile industry gives PEC/PNEC ratios > 1. It is therefore considered relevant to draw conclusion iii) also for site Backcoat C.

Freshwater sediment

Conclusion (ii) is drawn for site Backcoat 4.

Conclusion (iii) is drawn for sites Backcoat 1, Backcoat 2, Backcoat 3 and BackcoatC and for the generic scenario. In addition, PEC/PNEC ratios >1 are derived when using concentrations measured in freshwater sediment sampled in textile industry regions (River Viskan, SE, River Calder and River Roch, UK). This shows that this use area may give environmental concentrations resulting in concern for sediment dwelling organisms.

3.4.1.1.10 Regional PEC/PNEC ratios for surface water and sediment, both freshwater and marine

PEC/PNEC-ratios for the regional level are shown in Table 3-172.

Table 3-172 Regional PEC/PNEC ratios for surface water and sediment.

Scenario	PEC/PNEC aquatic	PEC/PNEC freshwater sediment
Regional	0.090	0.095

There is no concern at the regional level for freshwater and sediment as the regional PEC/PNEC ratios are well below 1.

Conclusion (ii) is drawn for the regional scenario for the aquatic and freshwater sediment compartment.

3.4.1.2 Conclusions to the risk assessment for the STP

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 (ii) applies to production, micronising, EPS formulation, XPS formulation, Textile formulation, Industrial use of EPS, industrial use of HIPS, most sites involved in industrial use of XPS compound, sites involved in industrial use of HBCCD powder for XPS and individual sites involved in textile backcoating.

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

Conclusion (iii) applies to some sites with industrial use of XPS having intermittent releases to waste water and for 1 textile backcoating site including the generic textile backcoating scenario.

3.4.1.3 Conclusions to the risk assessment for surface water

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 (ii) applies to production and micronising, industrial use of EPS and HIPS and for most sites in the other use areas.

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

Conclusion (iii) applies to some sites involved in EPS formulation including the generic scenario, one site involved in formulation of XPS compound and the generic scenario, the generic local scenario for formulation of polymer dispersions for textiles, individual sites involved in industrial use of XPS compound and XPS powder including the generic local scenario for industrial use of XPS compound and finally, sites involved in textile backcoating including the generic scenario.

3.4.1.4 Conclusions to the risk assessment for freshwater sediments

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 (ii) applies to production, micronising and industrial use of EPS, industrial use of HIPS, most sites for industrial use of HBCDD powder for XPS and for individual sites in the other use areas.

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

Conclusion (iii) applies to some sites involved in EPS formulation including the generic scenario, one site involved in XPS formulation including the generic scenario, one site involved in formulation of polymer dispersions for textiles including the generic scenario, individual sites involved in industrial use of XPS compound and HBCDD powder including the generic local scenario for industrial use of XPS compound and sites involved in textile backcoating including the generic scenario. A general conclusion (iii) is drawn for textile backcoating. based on measured concentrations in sediment downstream three different locations giving RCRs >1.

3.4.2 Terrestrial compartment

Calculations carried out according to the TGD show that sludge deposition on agricultural soil is the main contribution to the levels of HBCDD in soil

The PEC used is for agricultural soils, 30 days. The PNEC used for soil organisms is 5.9 mg/kg dwt.

EUSES calculates local PECs for agricultural soil by applying sewage sludge on regional natural soil which has a much lower HBCDD concentration than regional agricultural soil. The consequence of this is that many local $PEC_{agicultural\ soils}$ are lower than the regional PEC for agricultural soil.

In the calculations of PEC_{soil} no degradation is assumed as a worst case assumption based on results from simulation study 2. In addition the DT_{50}-value from simulation study 1 (119 days at 12 °C) is used for comparison (c.f. section **3.1.3.1.3**). The PECs and consequently the RCRs using the two DT_{50}-values differ approximately 10 times. The RCR derived from modelling with the lower half-life is only shown if the RCR changes from >1 to <1.

In addition, the relevance of the RCRs in soils for sites/scenarios with intermittent release is questionable. The reason for this is that EUSES calculates a ´snapshot´ concentration in sewage sludge, representative only for the intermittent emission period (1 or 2 days). EUSES does not take into consideration that the concentration of HBCDD in the sludge will be diluted by less contaminated sludge over time. The PEC will therefore be an overestimation of the exposure relevant for comparison to a long term PNEC. The HBCDD concentration in sewage sludge calculated by EUSES is therefore divided by a factor of 9.2 to take account of the retention time in the treatment plant (c.f 3.1.5).

3.4.2.1 Risk characterisation ratios for the terrestrial compartment

3.4.2.1.1 PEC/PNEC ratios for production and micronising of HBCDD

Sludge from the onsite STPs at the production sites is incinerated or put on landfill. Both site are however also connected to municipal STPs and the sludge from these STPs is assumed to be put on agricultural soil.

PEC/PNEC-ratios for the production and micronising, are shown in Table 3-173.

Table 3-173 PEC/PNEC ratios in soil for production and micronising of HBCDD.

Site	PEC/PNEC local $_{soil}$	Conclusion
ProdA*	**16**	
ProdB	0.0059	(ii)
Micronising**	0.00011	(ii)

*Production site A closed down in December 2003 and is included for illustrative purposes only.

**No emissions to wastewater.

Production at site A has recently closed down (December 2003) and is kept in the risk characterisation for illustrative puposes only. For site B as well as for micronising there is no concern.

Conclusion (ii) is drawn for production site B and micronising.

3.4.2.1.2 PEC/PNEC ratios for formulation of compound for EPS and HIPS

PEC/PNEC-ratios for formulation of EPS compound including, where relevant, production of HIPS are shown in Table 3-174. PEC/PNEC ratios are not shown for sites A, F, I, D, E, H, J and P. These sites are not connected to municipal STPs and therefore no HBCDD contaminated sewage sludge is spread on to soil locally at these sites. They all have PEC/PNEC ratios << 1.

Table 3-174 PEC/PNEC ratios for HBCDD in soil. Formulation of compound for EPS and HIPS.

Site	PEC/PNEC local $_{soil}$
Site B	0.0013
Site C	0.0078
Site G	0.022
Site K	0.19
Site L	0.097
GEN_EPS_FORM	0.37

Conclusion (ii) is drawn for all sites involved in formulation of compound for EPS and HIPS for which site specific information is available and also for the generic scenario.

3.4.2.1.3 PEC/PNEC for formulation of XPS compound for the manufacture of XPS

PEC/PNEC-ratios for formulation of XPS compound for the manufacture of XPS are shown in Table 3-175.

Table 3-175 PEC/PNEC ratios for HBCDD in soil. Formulation XPS compound

Site	PEC/PNEC local $_{soil}$	Conclusion
MasterbG	0.0013	(ii)
MasterbH	0.0027	(ii)
MasterbI	0.35	(ii)
GEN_XPS_FORM	0.24	(ii)

Conclusion (ii) is drawn for sites MasterbG, MasterbH, MasterbI and for the generic scenario.

3.4.2.1.4 PEC/PNEC ratios for formulation of polymer dispersions for textiles

PEC/PNEC-ratios for formulation of polymer dispersions of textiles are shown in Table 3-176.

Table 3-176 PEC/PNEC ratios for HBCDD in soil. Formulation of polymer dispersions for textiles.

Site	PEC/PNEC local $_{soil}$
TexForm1	0.0011
TexForm3	0.00011
TexForm4	0.00054
TexForm5	0.00012
TexFormA	0.00011
TexFormB	0.080
GEN_TEX_FORM	0.90

*

Conclusion (ii) is drawn for all sites involved in formulation of polymer dispersions for textiles for which site specific information is available and also for the generic scenario.

3.4.2.1.5 PEC/PNEC ratios for industrial use of EPS compound

PEC/PNEC-ratio for industrial use of EPS compound for the manufacture of EPS are shown in Table 3-177.

Table 3-177 PEC/PNEC ratios in soil for industrial use of EPS compound

Site	PEC/PNEC local $_{soil}$
GEN_EPS_IndUse	0.0049

There is no concern for this scenario.

Conclusion (ii) is drawn for industrial use of EPS compound.

3.4.2.1.6 PEC/PNEC ratios for industrial use of HIPS compound

PEC/PNEC-ratio for industrial use of HIPS compound for the manufacture of EPS are shown in Table 3-178.

Table 3-178 PEC/PNEC ratios in soil for industrial use of HIPS compound

Site	PEC/PNEC local $_{soil}$
GEN_HIPS_IndUse	0.015

There is no concern for this scenario.

Conclusion (ii) is drawn for industrial use of HIPS compound.

3.4.2.1.7 PEC/PNEC ratios for industrial use of XPS compound for flame retarded XPS

PEC/PNEC-ratios for industrial use of compound for manufacture of XPS are shown in Table 3-179.

Table 3-179 PEC/PNEC ratios for HBCDD in soil. Industrial use of XPS compound for flame retarded XPS.

Site	PEC/PNEC local $_{soil}$ *
XPS 1	0.42
XPS 2**	0.00076
XPS 3**	0.39
XPS 11**	0.65
GEN_XPS_IndUse**	**2.4** (0.25)

*Figures within parenthesis represent PEC/PNEC-ratios calculated with DT50s from simulation study 1 (119 d at 12 °C).

**Intermittent releases.

Conclusion (ii) is drawn for sites XPS 1, XPS 2, XPS 3 and XPS 11.

Conclusion (iii) is drawn for the generic scenario. TGD gives no guidance on how to handle the PEC estimation for sites having intermittent releases to waste water. In this risk assessment the HBCDD concentration in sewage sludge has been divided by a factor of 9.2 to take into account the retention time in the sewage treatment plant. However, the relevance of this PEC is still uncertain. It shall be noted that if the DT50 from simulation study 1 is used in the derivation of PEC_{soil} the PEC/PNEC ratio becomes <1.

3.4.2.1.8 PEC/PNEC ratios in soil. Industrial use of HBCDD powder for flame retarded XPS

PEC/PNEC-ratios for industrial use of HBCDD powder for the manufacture of XPS are shown in Table 3-180. No generic risk characterisation is performed since data has been submitted for all of the sites. PEC/PNEC ratios are not shown for sites XPS 9, XPS 16, XPS 17 and XPS 18 as these sites have no emissions to waste water. Therefore, no HBCDD contaminated sewage sludge is spread on to soil locally. These sites all have PEC/PNEC ratios << 1.

Table 3-180 PEC/PNEC ratios for HBCDD in soil. Industrial use of HBCDD powder for flame retarded XPS.

Site	PEC/PNEC local $_{soil}$*
XPS 4**	0.67
XPS 5**	0.18
XPS 6**	0.17
XPS 7**	**1.1** (0.12)
XPS 8**	0.00052
XPS 10**	**1.8** (0.19)
XPS 13**	0.0010
XPS 14**	0.00043
XPS 20**	0.017
XPS 21**	**2.3** (0.24)
XPS 23**	0.00013
XPS 24**	0.00013
XPS 26**	0.0067
XPS 27**	0.77

*Figures within parenthesis represent PEC/PNEC-ratios calculated with DT50s from simulation study 1(119 d at 12 °C).

**Intermittent releases

Conclusion (ii) is drawn for sites XPS 4, XPS 5, XPS 6, XPS 8, XPS 9, XPS 13, XPS 14, XPS 16, XPS 17, XPS 18, XPS 20, XPS 23, XPS 24, XPS 26 and XPS 27.

Conclusion (iii) is drawn for site XPS 7, XPS 10 and XPS 21. TGD gives no guidance on how to handle the PEC estimation for sites having intermittent releases to waste water. In this risk assessment the HBCDD concentration in sewage sludge has been divided by a factor of 9.2 to take into account the retention time in the sewage treatment plant. However, the relevance of this PEC is still uncertain. It shall be noted that if the DT50 from simulation study 1 is used in the derivation of PEC_{soil} the PEC/PNEC ratio becomes <1 for all three sites.

3.4.2.1.9 PEC/PNEC ratios for HBCDD in soil for industrial use of textile back-coating agent

PEC/PNEC-ratios in soil for industrial use of textile back-coating agent are shown in Table 3-181.

Table 3-181 PEC/PNEC ratios for HBCDD in soil. Industrial use of textile back-coating agent.

Site	PEC/PNEC local $_{soil}$*
Backcoat.1	0.088
Backcoat.2	0.088
Backcoat.3	**15 (1.6)**
Backcoat.4	0.00029
BackcoatC	0.028
GEN_TEX_IndUse	**15 (1.6)**

* Figures within parenthesis represent PEC/PNEC-ratios calculated with DT50s from simulation study 1 (119 d at 12 °C).

Conclusion (ii) is drawn for sites Backcoat1, Backcoat2, Backcoat4 and BackcoatC

Conclusion (iii) is drawn for the site Backcoat3 and for the generic scenario.

3.4.2.1.10 Regional PEC/PNEC ratios for HBCDD for agricultural soil

PEC/PNEC-ratios agricultural soil for the regional level are shown in Table 3-182.

Table 3-182 PEC/PNEC regional and continental for HBCDD for soil.

Scenario	PEC/PNEC regional$_{soil}$
Regional	0.039

There is no concern for the regional scenario.

3.4.2.2 Conclusion to the risk assessment for the terrestrial compartment

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 (ii) applies to production, micronising, EPS formulation, XPS formulation, Textile formulation, Industrial use of EPS, industrial use of HIPS, all sites involved in industrial use of XPS compound for which site specific information was available, most of the sites involved in industrial use of HBCCD powder for XPS and individual sites involved in textile backcoating.

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

Conclusion (iii) applies to the generic scenario for industrial use of XPS compound, three sites using HBCDD powder in the production of XPS and one site involved in textile backcoating including the generic scenario.

3.4.3 Atmosphere

No PNEC can be derived for the atmosphere. Therefore only a qualitative assessment can be made for this compartment.

Very low concentrations (<25 ng/m^3) of HBCDD are predicted for all scenarios (except for the production site A, no longer in use). Due to its physico-chemical properties (very low vapour pressure, high K_{ow}, low abiotic degradation etc) the major part of HBCDD in the air, is in particulate form, and is considered to present a negligible risk of adding to global warming, acidification or ozone depletion in the stratosphere.

Modelling of the potential of HBCDD to be transported long range indicates that the substance has a low potential to reach remote areas, and is dependent on behaviour of the atmospheric particulate matter to which they sorb. HBCDD has been found in samples of mosses from Norway, showing a south-north gradient, with the highest concentrations in the south. This may provide an indication that some transport via the atmosphere may occur for HBCDD, by the mechanism outlined above. The finding of detectable concentrations of HBCDD in birds-eggs and mammals in the Arctic, is suggestive that atmospheric long-range transport may be occurring.

3.4.3.1 Conclusions to the risk assessment for the atmosphere

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 (ii) applies to all use areas.

3.4.4 Non compartment specific effects relevant for the food chain (secondary poisoning)

Aquatic

When assessing secondary poisoning via the aquatic food chain the predicted concentration of HBCDD in fish are calculated as described below:

$PEC_{oral,\ predator} = (PEClocal_{freshwater} + PECregional_{freshwater}) \times 0.5 \times BCF_{fish} \times BMF_1$

BCF_{fish} : 18100

BMF_1: 10

Generally EUSES overpredicts $PEC_{sec\ poisoning}$ for the aquatic compartment (see 3.1.7.3).

As a consequence of this the $PECregional_{freshwater}$ is set to the concentration resulting in the regional part of $PEC_{oral,\ predator}$ becoming 20 µg HBCDD/kg wwt which is the median value of measured concentrations in fish representative for the region (see Table 3-111).

In addition the $PEC_{local\ surface\ water}$ is set to the water solubility limit (66 µg HBCDD/l) in those cases where EUSES calculates a higher PEC_{local} assuming that only the dissolved part of HBCDD is considered relevant when assessing secondary poisoning. The PECs are presented in appendix 1.

Despite these modifications there is still a large uncertainty in the PEC-values calculated with EUSES. Possible factors contributing to the uncertainty include chemical/physical- and fate-properties, emission data (e.g. dilution in the recipient) and the removal rate in the STP. Also the magnitude of the BCF- and BMF-factors may play a role but are considered to have less influence. The highest measured concentrations in fish are in the range of 5-10 mg/kg wwt, which stems from highly contaminated recipients (production, textile backcoating). EUSES calculates $PEC_{sec.\ poisoning}$ up to 6000 mg/kg wwt for the corresponding life cycle stages (scenario GenTex Ind use). The largest RCR based on monitoring data is approximately 0.6. This RCR is based on a measured concentration of 9.4 mg/kg wwt in eels captured in the vicinity of the former production site in Aycliffe,

Terrestrial

The $PEC_{local\ porewater}$ is set to the water solubility limit in those cases where EUSES calculates a higher $PEC_{local\ porewater}$ assuming that only the dissolved part of HBCDD is considered relevant when assessing secondary poisoning.

In the calculations of PEC_{soil} no degradation is assumed as a worst case assumption based on results from simulation study 2. In addition the DT_{50}-value from simulation study 1 (119 days at 12 °C) is used for comparison (c.f. section 3.1.3.1.3). The PECs and consequently the RCRs using the two DT_{50}-values differ approximately 15-20 times. The RCR derived from modelling with the lower half-life is only shown if the RCR changes from >1 to <1.

There is no valid experimental earthworm BCF study. However, the available data indicates that EUSES may overestimate the HBCDD concentration in earthworm by a factor of approx. 100 (c.f. section 3.1.3.2.4).

In order to be able to perform a more accurate risk assessment for the terrestrial predators, a valid earthworm BCF study is required.

Marine

When assessing secondary poisoning via the marine food chain for marine predators the predicted concentration of HBCDD are calculated as described below:

$PEC_{oral,\ predator} = (PEClocal_{seawater} + PECregional_{seawater}) \times 0.5 \times BCF_{fish} \times BMF_1$

BCF_{fish} : 18100

BMF_1: 10

Generally EUSES overpredicts $PEC_{sec\ poisoning}$ for the marine compartment (see 3.1.7.3).

As a consequence of this the $PECregional_{marine\ water}$ is set to the concentration resulting in the regional part of $PEC_{oral,\ marinepredator}$ becoming 1.8 µg HBCDD/kg wwt which is the median value of measured concentrations in marine fish in the Western Scheldt which is considered as being representative for the region (see 3.1.3.2.5). The PECs are presented in appendix 1.

When assessing secondary poisoning via the marine food chain for marine top-predators the predicted concentration of HBCDD is calculated as described below:

$PEC_{oral,\ toppredator} = (0.1 \times PEClocal_{seawater,ann} + 0.9 \times PECregional_{seawater}) \times BCF_{fish} \times BMF_1 \times BMF_2$

BCF_{fish} : 18,100

BMF_1: 10

BMF_2: 10

Generally EUSES overpredicts $PEC_{sec\ poisoning}$ for the marine compartment (see 3.1.7.3).

As a consequence of this the $PECregional_{marine\ water}$ is set to the concentration resulting in the regional part of $PEC_{oral,\ top\ predator}$ becoming 336 µg HBCDD/kg wwt which is the median value of measured concentrations in marine mammals in the Western Scheldt which is considered as being representative for the region (see 3.1.3.2.5). The PECs are presented in appendix 1.

Despite these modifications there is still a large uncertainty in the PEC-values calculated with EUSES. Possible factors contributing to the uncertainty include chemical/physical- and fate-properties, emission data (e.g. dilution in the recipient) and the removal rate in the STP. Also the magnitude of the BCF- and BMF-factors may play a role but are considered to have less influence. The highest measured concentrations in marine fish is 49 µg/kg wwt, which stems from the vicinity of production site B in the Netherlands whereas EUSES calculates a $PEC_{marine\ sec.\ poisoning}$ of 540 µg/kg wwt for this site. The highest measured concentration in marine mammals is 6400 µg/kg wwt which can be compared with the $PEC_{marine\ top\ predator}$ calculated with EUSES ranging from 18 to 3400000 µg/kg wwt. In addition to the uncertainties in the PEC derivation, the PNEC derived for secondary poisoning is also considered to be very uncertain (c.f. 3.3.4.2)

3.4.4.1 Risk characterisation ratios for secondary poisoning

The PEC/PNEC ratios for secondary poisoning are presented in Table 3-183 - Table 3-191.

3.4.4.1.1 PEC/PNEC ratios for production and micronising of HBCDD

PEC/PNEC-ratios for production and micronising are shown in Table 3-183.

Table 3-183 PEC/PNEC ratios in food of predators in the aquatic, terrestrial and marine food chains. Production and micronising of HBCDD.

Site	Connected to municipal STP (Yes/No)	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators	PEC/PNEC marine predators	PEC/PNEC marine top predators
ProdA*	Yes	**957**	**47**	**-**	**-**
ProdB	Yes	0.0075	0.14	0.0039	0.074
Micronising	Yes/No**	0.0040	0.12	0.00036	0.067

*Production site A closed down in December 2003 and is included for illustrative purposes only.

**Not known if connected to STP. However, no emissions to waste water.

Production site A has closed and the UK authorities has a monitoring programme to follow up the environmental concentrations (sediment and biota) in the vicinity of the plant.

Production site B is situated at a river mouth entering into the Western Scheldt. It is not known to the rapporteur whether the waste water is released to the river or the estuary. It is therefore, not clear if the aquatic food chain is of relevance for this site.

3.4.4.1.2 PEC/PNEC ratios for HBCDD formulation of compound for EPS and HIPS

PEC/PNEC-ratios for formulation of EPS beads including, where relevant, production of HIPS are shown in Table 3-184.

Table 3-184 PEC/PNEC ratios for HBCDD in food of predators in the aquatic, terrestrial and marine food chains. Formulation of compound for EPS and HIPS.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators	PEC/PNEC marine predators	PEC/PNEC marine top predators
Site A + M	No	0.087	-	**-**	**-**
Site F + N	No	0.71	-	**-**	**-**
Site I + O	No	0.12	-	**-**	**-**
Site B	Yes	0.0046	0.12	**-**	**-**
Site C	No	0.33	-	0.33	0.72
Site D	No	0.26	-	**-**	**-**
Site E	No	0.13	-	**-**	**-**

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators	PEC/PNEC marine predators	PEC/PNEC marine top predators
Site G	Yes	**1.3**	0.19	0.13	0.34
Site H	No	0.40	-	**-**	**-**
Site J	No	**21**	-	**2.1**	**4.3**
Site K	Yes	**9.8**	0.70	0.98	**2.0**
Site L	Yes	**4.9**	0.41	0.49	**1.1**
Site P*	No	0.71	-	0.071	0.21
GEN_EPS_FORM	Yes	**11**	0.76	**1.1**	**2.3**
	No	**53**	**-**	**5.3**	**11**

*Intermittent release.

For the generic scenario and in the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with or without STP.

3.4.4.1.3 PEC/PNEC ratios for HBCDD for formulation of XPS compound

PEC/PNEC-ratios for formulation of XPS compound for the manufacture of XPS are shown in Table 3-185.

Table 3-185 PEC/PNEC ratios for HBCDD in food of predators in the aquatic, terrestrial and marine food chains. Formulation of XPS compound for the manufacture of XPS.

Site	Connected to municipal STP (Yes/No) #	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators*	PEC/PNEC marine predators	PEC/PNEC marine top predators
MasterbG	Yes	0.061	0.12	0.0061	0.079
MasterbH	Yes	0.13	0.131	-	-
MasterbI	Yes	**18**	**1.2** (0.085)	**1.8**	**3.6**
GEN_XPS_FORM	Yes	**12**	0.85	**1.2**1	**2.6**4
	No	**60**	**-**	**6.0**	**12**

For the generic scenario and in the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with or without STP.

*Figures within parenthesis represent PEC/PNEC-ratios calculated with DT50s from the simulation study 1 (119 d at 12 °C).

3.4.4.1.4 PEC/PNEC ratios for HBCDD for formulation of polymer dispersions for textiles

PEC/PNEC-ratios for formulation of polymer dispersions of textiles are shown in Table 3-186.

Table 3-186 PEC/PNEC ratios for HBCDD in food of predators in the aquatic, terrestrial and marine food chains. Formulation of polymer dispersions for textiles.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators**	PEC/PNEC marine predators	PEC/PNEC marine top predators
TexForm1	Yes	0.051	0.12	0.0051	0.077
	No	0.23	-	0.023	0.11
TexForm3	Yes/No*	0.0040	0.12	0.00036	0.067
TexForm4	Yes	0.026	0.12	0.0025	0.072
	No	0.11	-	0.011	0.082
TexFormA	Yes/No*	0.0040	0.12	0.0036	0.067
TexFormB	Yes	**4.1**	0.36	0.41	0.88
	No	**20**	**-**	**2.0**	**4**
GEN_TEX_FORM	Yes	**46**	**2.8** (0.19)	**4.6**	**9.2**

For the generic scenario and in the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with or without STP.

*Not known if connected to STP. However, no emissions to waste water.

**Figures within parenthesis represent PEC/PNEC-ratios calculated with DT50s from the simulation study 1 (119 d at 12 °C).

3.4.4.1.5 PEC/PNEC ratios for HBCDD for industrial use of EPS compound

PEC/PNEC-ratios for industrial use of EPS compound for the manufacture of EPS are shown in Table 3-187.

Table 3-187 PEC/PNEC ratios for HBCDD in food of predators in the aquatic, terrestrial and marine food chains. Industrial use EPS compound

Site	Connected to municipal STP (Yes/No)	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators	PEC/PNEC marine predators	PEC/PNEC marine top predators
GEN_EPS_	Yes	0.25	0.13	0.025	0.12

Site	Connected to municipal STP (Yes/No)	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators	PEC/PNEC marine predators	PEC/PNEC marine top predators
IndUse	No	**1.2**	**-**	0.12	0.30

3.4.4.1.6 PEC/PNEC ratios for HBCDD for industrial use of HIPS compound

PEC/PNEC-ratios for industrial use of HIPS compound for the manufacture of HIPS are shown in Table 3-188.

Table 3-188 PEC/PNEC ratios for HBCDD in food of predators in the aquatic, terrestrial and marine food chains. Industrial use HIPS compound

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators	PEC/PNEC marine predators	PEC/PNEC marine top predators
GEN_HIPS_IndUse	Yes	0.079	0.16	0.0079	0.082
	No	0.37	-	0.037	0.14

3.4.4.1.7 PEC/PNEC ratios for HBCDD for industrial use of XPS compound

PEC/PNEC-ratios for industrial use of XPS compound for manufacture of XPS are shown in Table 3-189.

Table 3-189 PEC/PNEC ratios for HBCDD in food of predators in the aquatic, terrestrial and marine food chains. Industrial use of XPS compound.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators*	PEC/PNEC marine predators	PEC/PNEC marine top predators
XPS 1	Yes	0.015	**1.4** (0.090)	0.011	0.089
	No	0.056	**-**	0.052	0.17
XPS 2	Yes/No***	0.0040	0.12	0.0036	0.067
XPS 3**	Yes	0.060	**1.3** (0.083)	0.0056	0.18
	No	0.28	-	0.27	0.61
XPS 11**	Yes	2.0	**2.1** (0.14)	0.20	0.47
	No	**9.8**	-	0.98	**2.0**
GEN_XPS_IndUse**	Yes	**3.8**	**7.4** (0.52)	0.38	0.82
	No	**18**	**-**	**1.8**	**3.7**

#In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with or without STP.

*Figures within parenthesis represent PEC/PNEC-ratios calculated with DT50s from the simulation study 1 (119 d at 12 °C).

**Intermittent release.

***Not known if connected to STP. However, no emissions to waste water.

3.4.4.1.8 PEC/PNEC ratios for industrial use of HBCDD powder for flame retarded XPS

PEC/PNEC-ratios for industrial use of HBCDD powder for the manufacture of XPS are shown in Table 3-190. No generic risk characterisation is performed since data has been submitted for all of the sites.

Table 3-190 PEC/PNEC ratios for HBCDD in food of predators in the aquatic, terrestrial and marine food chains. Industrial use of HBCDD powder for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators *	PEC/PNEC marine predators	PEC/PNEC marine top predators
XPS 4**	Yes	**-**	**2.1** (0.14)	0.21	0.49
	No	**-**	-	**1.0**	**2.1**
XPS 5***	Yes	0.0095	0.65	-	-
	No	0.031	-	-	-
XPS 6	Yes	0.0042	0.56	-	-
	No	0.0053	-	-	-
XPS 7	Yes	0.022	**3.5** (0.24)	-	-
	No	0.09	-	-	-
XPS 8***	Yes	0.0051	0.12	0.00047	0.068
	No	0.0096	-	0.00092	0.0680.022
XPS 9	Yes/No****	0.040	0.12	0.0036	-
XPS 10	Yes	**2.1**	**5.7** (0.39)	-	-
	No	**10.0**	-	-	-
XPS 13***	Yes	0.0054	0.12	-	-
	No	0.011	-	-	-
XPS 14**	Yes	-	0.12	0.00045	0.068
XPS 16	Yes/No****	0.0040	0.12	0.00036	0.067
XPS 17	No	0.0040	0.12	0.0036	0.067
XPS 18	No	0.0040	0.12	0.0036	0.067
XPS 20***	Yes	0.0046	0.017	-	-
XPS 21***	Yes	0.35	**7.1** (0.49)	-	-

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators *	PEC/PNEC marine predators	PEC/PNEC marine top predators
	No	**1.7**	-	**-**	**-**
XPS 23***	Yes	0.004	0.12	0.00036	0.067
XPS 24***	Yes	0.0042	0.12	0.00038	0.067
XPS 26***	Yes	0.014	0.14	-	-
XPS 27***	Yes	**1.2**	**2.4** (0.16)	0.12	0.31
	No	**5.8**	-	0.58	**1.2**

#In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with and without STP.

*Figures within parenthesis represent PEC/PNEC-ratios calculated with DT50s from the simulation study 1 (119 d at 12 °C)

**XPS4 and XPS14 are known to have releases to the sea.

***Intermittent release

****Not known if connected to STP. However, no emissions to waste water.

3.4.4.1.9 PEC/PNEC for HBCDD for industrial use of textile back-coating agent

PEC/PNEC-ratios for industrial use of textile back-coating agent are shown in Table 3-191.

Table 3-191 PEC/PNEC ratios for HBCDD in food of predators in the aquatic, terrestrial and marine food chains. Industrial use of textile back-coating agent.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators*	PEC/PNEC marine predators*	PEC/PNEC marine top predators
Backcoat.1	Yes	**3.4**	0.38	0.34	0.74
	No	**16**	-	**1.6**	**3.3**
Backcoat.2	Yes	0.81	0.38	0.081	0.23
	No	**3.9**	-	0.39	0.85
Backcoat.3	Yes	**215**	**46 (3.2))**	**21.5**	**43**
Backcoat.4	Yes	0.0094	0.12	0.00090	0.068
	No	0.030	-	0.0030	0.073
BackcoatC	Yes	0.21	0.20	0.0210.0069	0.11
	No	**1.0**	-	0.10	0.27
GEN_TEX_ IndUse	Yes	**643**	**45 (3.2)**	**64**	**129**

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC aquatic predators	PEC/PNEC terrestrial predators*	PEC/PNEC marine predators*	PEC/PNEC marine top predators
Monitoring data from textile industry area Scheldt Oudenarde, River Scheldt basin, BE		**1.1*** **			

#In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with and without STP.

*Figures within parenthesis represent PEC/PNEC-ratios calculated with DT50s from simulation study 1 (119 d at 12 °C).

**Info representative for type of treatment of waste water available

***River Scheldt Basin: (5500 µg HBCDD/kg wwt)/ 5000 µg HBCDD/kg wwt)

3.4.4.2 Conclusions to the risk assessment for secondary poisoning

In the light of HBCDD being a PBT substance (c.f. 3.4.6) and considering the large uncertainties both in the derivation of PECs and in the derivation of PNEC it is not considered appropriate to draw conclusions for the individual sites. Since for PBT-substances the major concern is that accumulation of such substances in the foodchain may result in unpredictable effects in the long term it is appropriate to draw an overall conclusion (iii) for secondary poisoning.

3.4.4.3 Indicative risk assessment of effects on birds, exposed as eggs

HBCDD has been found in eggs of several bird species (Table 3-110). There are no toxicity data for birds exposed as eggs, and no guidance in the TGD on how to perform a risk assessment on measured concentrations in eggs. Therefore, as a provisional assessment of the effects on birds exposed during fetal development, and in line with the risk assessment report for bis(pentabromophenyl) ether (UK Environmental Agency, 2003), the measured levels in eggs are compared with the estimated 'internal dose' of 900 µg/kg ww derived from the indicative LOAEL for developmental neurotoxicity in mice assuming 100% absorption (section 3.3.4.3).

Most data on HBCDD levels in birds' eggs, are based on lipid weight. However, for the calculations below only data based on wet weight can be used. The ratios obtained when measured concentrations in eggs are compared to the estimated 'internal dose' are listed in Table 3-192 below.

This comparison results in ratios up to 0.7 for the highest individual HBCDD concentration found in eggs, common tern eggs sampled close to Terneuzen. If the 'internal dose' (LOAEL) used in this comparison had been used as the basis for PNEC derivation first a factor of 3 should have been used for the conversion to a NOAEL. Furthermore, in the calculation of a PNEC a minimum assessment factor of 30 should have been applied (i.e. the factor used with chronic data, according to TGD). This factor should cover uncertainties related to interspecies

variation, acute/subchronic to chronic extrapolation and laboratory data to field impact extrapolation. These are uncertainties that would apply also for the birds egg extrapolation case. Applying these factors would lead to ratios >1 for all species (and most locations), using the highest reported values. This would indicate that the highest levels of HBCDD, found in eggs from the environment is a possible reason for concern for several bird species.

Table 3-192 Indicative risk characterisation for birds exposed to HBCDD as eggs (without and with an assessment factor of 30).

Species	Level of HBCDD µg/kg wwt	Ratio measured level of HBCDD/indicative LOAEL*	Ratio when applying an assessment factor of 90 (3x30)**
Atlantic puffin (*Fratercula arctica*), **Norway**, two locations, 2003 **(Knudsen *et al.*, 2005)**	3.5-13 (min-max, n = 10)	0.004-0.014	0.3-1.3
Common tern (Sterna hirundo) **Netherlands**, Terneuzen, **(de Boer *et al.*, 2002a)**	35-640 (min-max, n = 10)	0.04-0.7	3.5-64
Glaucous gull (*Larus hyperboreus*) **Norway**, Bjørnøya, {Verreault *et al.*, 2004}}	2-70 (min-max, n = 10)	0.002-0.08	0.2-7
Norway, Bjørnøya, 2002 **(Knudsen *et al.*, 2005)**	5.2-23 (min-max, n = 4)	0.006-0.08	0.5-2.3
Guillemot (*Uria aalge*) **Sweden**, Baltic Sea, St. Karlsö, 2001 **(Sellström *et al.*, 2003)**	17 (aritm. mean of n = 10)	0.02	1.7
Herring gull (*Larus argentatus*) **Norway**, two locations, 2003 **(Knudsen *et al.*, 2005)**	5.7-18 (min-max, n = 10)	0.02	0.6-1.8
Kittiwake (*Rissa tridactyla*) **Norway**, two locations, 2003 **(Knudsen *et al.*, 2005)**	7.9-27 (min-max, n = 10)	0.009-0.03	0.8-2.7
Peregrine falcon *(Falco peregrinus)* **United Kingdom**, 2001-2001 **(Leslie *et al.*, 2004)**	3.6-30 (min-max, n = 7)	0.004-0.03	0.4-3.0
Sweden, 1998-1999 **(Lindberg *et al.*, 2004)**	2.19-160 (min-max, n = 15)	0.002-0.2	0.2-16
Denmark,Greenland, 2000-2003 **(Lindberg *et al.*, 2004)**	0.002-1.1 (mix-max, n = 8)	$2*10^{-6}$-0.001	0.0002-0.1

*Indicative LOAEL 900 µg/kg wwt, section 3.3.4.3.

** A factor 3 is used for converting the LOAEL to NOAEL and an assessment factor of 30 is for converting the NOAEL to a PNEC for secondary poisoning.

3.4.5 Marine Environment

3.4.5.1 Risk characterisation ratios for the marine environment

This section includes PEC/PNEC for aquatic organisms and sediment dwelling organisms. The respective PNECs are shown in Table 3-162. For information on the calculation of the PEC values, see Chapter 3.2.

When there is information, for a local site, that the emissions are only directed to the marine environment, only marine risk characterisation has been performed. For sites known to be located far away from the sea, only fresh water assessment has been performed. In the cases where there are no information on the location, or when the emissions are known to be directed to a freshwater body close to the sea, risk characterisation has been carried out for both the freshwater and the marine environment.

For several sites information is missing regarding whether the site is connected to a STP or not. In such cases (and for the generic scenarios) risk characterisation is carried out for two scenarios, with and without STP.

According to the distribution pattern of HBCDD (low water solubility and high Kow) the major part of the HBCDD released can be expected to be rather rapidly adsorbed to particles and distributed to sediment.

The relevance of the RCRs in sediment for sites/scenarios with intermittent release is questionable. The reason for this is that EUSES calculates a ´snapshot´ PEC, representative only for the intermittent emission period (1 or 2 days). EUSES does not take into consideration that the concentration of HBCDD in the sediment will be diluted by sedimentation of less contaminated particulate matter over time. The PEC will therefore be an overestimation of the exposure relevant for comparison to a long term PNEC. A more relevant PEC could be a time weighted average over e.g. 30 days. This is presently not possible to do with EUSES 2.03. Therefore, a pragmatic approach is taken and the $PEC_{sediment}$ derived by EUSES for sites having intermittent releases to water is divided by 30 (c.f 3.1.4.1.). This is thought to give a more realistic PEC than just using the "snapshot" $PEC_{sediment}$ derived by EUSES.

3.4.5.1.1 PEC/PNEC ratios for production and micronising

PEC/PNEC-ratios for production, and micronising, are shown in Table 3-193. For micronising there are no releases to water, and therefore no site-specific risk characterisation is performed as it is covered by the regional risk assessment.

Table 3-193 PEC/PNEC ratios for marine water and sediment. Production and micronising of HBCDD.

Site	Connected to municipal STP (Yes/No)	PEC/PNEC marine	PEC/PNEC marine sediment	Conclusion
ProdB	Yes	0.095	0.078	(ii) M, MS
Micronising	Yes/No*	*	*	(ii) M, MS

*STP connection not known, however no emissions to wastewater.

Production site B is situated at a river mouth entering into the Western Scheldt. It is not known to the rapporteur whether the waste water is released to the river or the estuary. It is therefore, not clear if the aquatic compartment is of relevance for this site. However, if it is, there is based on present data no concern, and there are no concern for production site B for any of the compartments. Also when comparing PNEC with measured sediment concentrations close to site B the PEC/PNEC ratios are below 1. Micronising has no emissions to water and, hence there are no local concerns.

Conclusion (ii) is drawn for production site B and micronising for the marine and the marine sediment compartment.

3.4.5.1.2 PEC/PNEC ratios for formulation of compound for EPS and HIPS

PEC/PNEC-ratios for the formulation of EPS compound, including where relevant productionof HIPS, are shown in Table 3-194.

Table 3-194 PEC/PNEC ratios for marine water and sediment. Formulation of compound for EPS and HIPS.

Site	Connected to municipal STP (Yes/No)	PEC/PNEC marine	PEC/PNEC marine sediment
Site C	No	0.80	0.65
Site G	Yes	0.36	0.30
Site J	No	**4.7**	**3.8**
Site K	Yes	**2.2**	**1.8**
Site L	Yes	**1.2**	0.94
Site P*	No	**29**	**46 (1.2)***
GEN_EPS_FORM	Yes	**2.5**	**2.0**
Monitoring data from producer of EPS beads, Åsnefjord, No			**47****

** Intermittent releases. Figure within parenthesis represents PEC/PNEC ratio based on PEC sediment calculated by EUSES averaged over 30 days.

**Åsnefjord: (8024 µg HBCDD/kg dwt)/ 170 µg HBCDD/kg ww

Marine water compartment

Conclusion (ii) is drawn for sites C and G.

Conclusion (iii) is drawn for sites J, K, L, P and the generic scenario.

Marine sediment

Conclusion (ii) is drawn for sites C, G and L.

Conclusion (iii) is drawn for sites J, K, P and the generic scenario. For site P the relevance of the RCR is questionable due to intermittent release (c.f. 3.4.1). However, also when the PEC is divided by a factor of 30 to make it comparable to the long-term PNEC for sediment the PEC/PNEC ratio is >1. Conclusion iii) is therefore considered relevant to draw for this site. In addition, a PEC/PNEC ratio of 47 is derived when using sediment concentrations measured in a fjord where a producer of EPS beads is situated (Åsnefjord, NO). This shows that this use area may result in environmental concentrations resulting in concern for marine sediment.

3.4.5.1.3 PEC/PNEC ratios for formulation of XPS compound

PEC/PNEC-ratios, for formulation of XPS compound for the manufacture of XPS, are shown in Table **3-195**.

Table 3-195 PEC/PNEC ratios for marine water and sediment. Formulation of XPS compound.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC marine	PEC/PNEC marine sediment
MasterbG	Yes	0.10	0.083
MasterbI	Yes	**3.9**	**3.2**
GEN_XPS_FORM	Yes	**2.8**	**2.3**

For the generic scenario and in the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with or without STP.

Marine water compartment

Conclusion (ii) is drawn for site MasterbG for the marine water compartment

Conclusion (iii) is drawn for site MasterbI, as well as for the generic scenario.

Marine sediment

Conclusion (ii) is drawn for site MasterbG.

Conclusion (iii) is drawn for site MasterbI, and for the generic scenario.

3.4.5.1.4 PEC/PNEC ratios for formulation of polymer dispersions for textiles

PEC/PNEC-ratios for formulation of polymer dispersions of textiles are shown in Table 3-196.

Table 3-196 PEC/PNEC ratios for marine water and sediment. Formulation of polymer dispersions for textiles.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC marine	PEC/PNEC marine sediment
TexForm1	Yes	0.099	0.081
	No	0.14	0.11
TexForm3	Yes/No*	0.089	0.073
TexForm4	Yes	0.094	0.076
	No	0.11	0.091
TexForm5	Yes/No*	0.089	0.073
TexFormA	Yes/No*	0.089	0.073
TexFormB	Yes	0.97	0.79
	No	**4.4**	**3.6**
GEN_TEX_FORM	Yes	**10**	**8.2**

In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with and without STP.
*No emissions to waste water

Marine water compartment

Conclusion (ii) is drawn for sites TexForm1, TexForm 3,TexForm4, TexForm 5 and TexForm A.

Conclusion (iii) is drawn for site TexFormB and for the generic scenario. It shall be noted that the PEC/PNEC ratio for TexForm B falls below 1 if it is assumed that the site is connected to a municipal STP. However, for marine risk assessment the default assumption of the TGD is direct discharge to water.

Marine sediment

Conclusion (ii) is drawn for sites TexForm1, TexForm 3, TexForm4, TexForm 5 and TexForm A.

Conclusion (i) is drawn for site TexFormB and for the generic scenario.

3.4.5.1.5 PEC/PNECratios for industrial use of EPS compound

PEC/PNEC-ratios for industrial use of EPS compound are shown in Table 3-197.

Table 3-197 PEC/PNEC ratios for marine water and sediment. Industrial use of EPS compound

Site	Connected to municipal STP (Yes/No)	PEC/PNEC marine	PEC/PNEC marine sediment
GEN_EPS_IndUse	Yes	0.14	0.12
	No	0.35	0.28

Conclusion (ii) is drawn for industrial use of EPS compound for the marine and the marine sediment compartment.

3.4.5.1.6 PEC/PNECratios for industrial use of HIPS compound

PEC/PNEC-ratios for industrial use of HIPS compound are shown inTable 3-198.

Table 3-198 PEC/PNEC ratios for marine water and sediment. Industrial use of EPS compound

Site	Connected to municipal STP (Yes/No)	PEC/PNEC marine	PEC/PNEC marine sediment
GEN_HIPS_IndUse	Yes	0.25	0.21
	No	0.88	0.72

Conclusion (ii) is drawn for industrial use of HIPS compound for the marine and marine sediment compartment.

3.4.5.1.7 PEC/PNEC ratios for industrial use of XPS compound

PEC/PNEC-ratios for industrial use of compound for manufacture of XPS are shown in Table 3-199.

Table 3-199 PEC/PNEC ratios for STP marine water and sediment. Industrial use of XPS compound.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC marine	PEC/PNEC marine sediment
XPS 1	Yes	0.55	0.45
	No	**2.3**	**1.9**
XPS 2**	Yes/No*	0.089	0.073
XPS 3**	Yes	**23**	**30 (1)****

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC marine	PEC/PNEC marine sediment
XPS 11**	Yes	**41**	**54 (1.8)****
GEN_XPS_IndUse**	Yes	**150**	**200 (6.8)****

#In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with and without STP.

*No emissions to wastewater.

** Intermittent releases. Figure within parenthesis represents PEC/PNEC ratio based on PEC sediment calculated by EUSES averaged over 30 days.

Marine water compartment

Conclusion (ii) is drawn for site XPS 2 for marine surface water.

Conclusion (iii) is drawn for sites XPS 1, XPS 3 and XPS 11 and for the generic scenario. It shall be noted that the PEC/PNEC ratio for site XPS 1 falls below 1 if it is assumed that the site is connected to a municipal STP. However, for marine risk assessment the default assumption of the TGD is direct discharge to water.

Marine sediment

Conclusion (ii) is drawn for site XPS 2 for marine sediment.

Conclusion (iii) is drawn for sites, i.e. XPS 1, XPS 3, and XPS 11, and for the generic scenario. The relevance of the RCRs is questionable due to intermittent release (c.f. 3.4.1). However, also when the PEC is divided by a factor of 30 to make it comparable to the long-term PNEC for sediment the PEC/PNEC ratio is >1. Conclusion iii) is therefore considered relevant to draw for these sites.

3.4.5.1.8 PEC/PNEC ratios for industrial use of HBCDD powder for flame retarded XPS.

PEC/PNEC-ratios for industrial use of HBCDD powder for the manufacture of XPS are shown in Table 3-200. No generic risk characterisation has been performed since data has been submitted for all of the sites.

Table 3-200 PEC/PNEC ratios for STP marine water and sediment. Industrial use of HBCDD powder for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC marine	PEC/PNEC marine sediment
XPS 4**, ***	Yes	**42**	**56 (1.9)***
XPS 8**	Yes	0.078	0.10
	No	0.17	0.22
XPS 14**, ***	Yes	0.056	0.074
XPS 16**	Yes/No*	0.089	0.073
XPS 17**	No	0.089	0.073
XPS 18**	No	0.089	0.073
XPS 23**	Yes	0.056	0.074
XPS 24**	Yes	0.056	0.074
XPS 27**	Yes	**78**	**130 (2.2)****

#In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with or without STP.

*No emissions to wastewater.

** Intermittent releases. Figure within parenthesis represents PEC/PNEC ratio based on PEC sediment calculated by EUSES averaged over 30 days.

***XPS4 and XPS14 are known to have releases to the sea.

Marine water compartment

Conclusion (ii) is drawn for sites XPS 8, XPS 14, XPS 16, XPS 17, XPS18, XPS 23 and XPS 24.

Conclusion (iii) is drawn for sites XPS 4, and XPS 27.

Marine sediment

Conclusion (ii) is drawn for sites XPS 8, XPS 14, XPS 16, XPS 17, XPS18, XPS 23 and XPS 24.

Conclusion (i) is drawn for sites XPS 4, and XPS 27. The relevance of the RCRs for sites with intermittent release is questionable (c.f. 3.4.1). However, also when the PEC is divided by a factor of 30 to make it comparable to the long-term PNEC for sediment the PEC/PNEC ratio is >1. Conclusion iii) is therefore considered relevant to draw for these sites.

3.4.5.1.9 PEC/PNEC ratios for industrial use of textile back-coating agent

PEC/PNEC-ratios for the life-cycle step industrial use of textile back-coating agent are shown in Table 3-201.

Table 3-201 PEC/PNEC ratios for marine water and sediment. Industrial use of textile back-coating agent.

Site	Connected to municipal STP (Yes/No)#	PEC/PNEC marine	PEC/PNEC marine sediment
Backcoat.1	Yes	**1.1**	0.87
	No	**4.8**	**3.9**
Backcoat.2	Yes	**1.1**	0.86
	No	**4.8**	**3.9**
Backcoat.3	Yes	**170**	**140**
Backcoat.4	Yes	0.091	0.074
	No	0.098	0.080
BackcoatC	Yes	0.40	0.33
	No	**1.6**	**1.3**
GEN_TEX_IndUse	Yes	**800**	**660**

In the cases where information is missing regarding wheter the site is connected to a STP or not, risk characterisation is carried out for two scenarios, with and without STP.

**Info representative for type of treatment of waste water available

Marine water compartment

Conclusion (ii) is drawn for site Backcoat 4.

Conclusion (iii) is drawn for sites Backcoat 1, Backcoat 2, Backcoat 3, BackcoatC and for the generic scenario. It shall be noted that the PEC/PNEC ratio for site Backcoat C falls below 1 if it is assumed that the site is connected to a municipal STP. However, for marine risk assessment the default assumption of the TGD is direct discharge to water.

Marine sediment

Conclusion (ii) is drawn for site Backcoat 4.

Conclusion (iii) is drawn for sites Backcoat 1, Backcoat 2, Backcoat C and for the generic scenario. It shall be noted that the PEC/PNEC ratios for sites Backcoat 1, Backcoat 2 and Backcoat C falls below 1 if it is assumed that sthe site is connected to a municipal STP. However, for marine risk assessment the default assumption of the TGD is direct discharge to water.

3.4.5.1.10 Regional PEC/PNEC ratios for marine water and sediment

PEC/PNEC-ratios for the regional level are shown in Table 3-202.

Table 3-202 Regional PEC/PNEC ratios for marine water and sediment.

Scenario	PEC/PNEC marine	PEC/PNEC marine sediment
Regional	0.089	0.020

There is no concern at the regional level.

Conclusion (ii) is drawn for the regional scenario for the marine and the marine sediment compartment.

3.4.5.2 Conclusions to the risk assessment for the marine environment

Surface water

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 (ii) applies to production, micronising and industrial use of EPS and HIPS and for individual sites in the other use areas.

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

Conclusion (iii) applies to some sites involved in EPS formulation including the generic scenario, and one site and the generic scenario for XPS formulation, one site involved in formulation of polymer dispersions for textiles including the generic scenario, individual sites involved in industrial use of HBCDD powder in XPS and use of XPS compound including the generic local scenario for industrial use of XPS compound, and some sites involved in textile backcoating including the generic scenario.

Marine Sediment

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 (ii) applies to production, micronising and industrial use of EPS and HIPS and for individual sites in the other use areas.

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

Conclusion (iii) applies to some sites involved in EPS formulation including the generic scenario, one site and the generic scenario for XPS formulation, one site involved in formulation of polymer dispersions for textiles including the generic scenario, individual sites involved in industrial use of HBCDD powder in XPS and use of XPS compound including the generic local scenario for industrial use of XPS compound, and some sites involved in textile backcoating including the generic scenario. In addition, measurements in marine sediment associated to a producer of EPS beads (EPS formulation) gives a RCR >1which indicates that there are concerns for this site and that there may be a general concern for this use area.

3.4.6 PBT-assessment

The marine risk assessment procedure requires a screening of the properties of a substance to see if it is considered as a persistent (P), bioaccumulative (B) and toxic (T) (or very persistent and very bioaccumulative (vPvB)) substance. The criteria are given in Table 3-203 below. The basis for these criteria is that an ordinary risk assessment using the PEC/PNEC approach is considered too uncertain for substances fulfilling these criteria.

Table 3-203 PBT- and vPvB-criteria according to the TGD

Criterion	PBT-criteria	vPvB-criteria
P	Half-life > 60 d in marine water or > 40 d in freshwater or half-life > 180 d in marine sediment or > 120 d in freshwater sediment	Half-life > 60 d in marine- or freshwater sediment or half-life > 180 d in marine or freshwater sediment
B	BCF > 2000	BCF > 5000
T	Chronic NOEC < 0.01 mg/l or CMR or endocrine disrupting effects	Not applicable

However, it should be noted that the TGD also says "certain flexibility is required in the application of the criteria for instance in cases where one criterion is marginally not fulfilled but the others are exceeded considerably. This may include for example substances that do not fulfil the persistence criteria but bioaccumulate significantly and are measured in marine biota distant from anthropogenic sources".

In summary, these two last sentences very precisely describe the situation for HBCDD. Thus, based on the present experimental data (including studies on the main degradation product CDT), HBCDD does not unequivocally fulfil the P-criterion , it very much exceeds the bioaccumulation criteria, it fulfils the toxicity criteria, and it is present in wildlife distant from areas of its use.

3.4.6.1 Persistence

Screening tests

HBCDD is not ready biodegradable according to the results from a Closed Bottle Test where no biodegradation was observed during 28-days at a test concentration of 7.7 mg HBCDD/l.

Simulation tests, soil

In the first aerobic soil-dissipation study **(Davis *et al.*, 2003a)** γ-HBCDD disappeared with a half-life of ca 4 months (119 days) at 12°C from a sandy loam soil amended with 5 mg/kg sewage sludge. The nominal test concentration was 25 µg technical HBCDD/kg 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. Questions can be raised about the efficiency of the extraction method and thus, the half-lives derived from this study may not solely represent biodegradation.

In aerobic soil in simulation study 2 **(Davis *et al.*, 2004)** there were no indications of any transformation of HBCDD during 112 days of incubation at 20±2°C. The nominal test concentration was 3.0 mg technical HBCDD/kg d.w. Even if metabolites could 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 adds to the suspicion that simulation study 1 may overestimate the degradability of HBCDD in soil.

Simulation tests, sediment

Two simulation tests studying the transformation of HBCDD in sediment have been performed.

In simulation study 1 (**Davis et al., 2003a**) only the transformation of the γ-diastereomer was studied 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 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 different systems, respectively. The 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 are involved, unless the steam sterilisation as such decrease the extractability of HBCDD (e.g., by affecting the structure of the sediment matrix (organic matter)). 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, mineralisation of HBCDD was not studied and no mass balance could be established. It was noted that the recovery varied a lot (33-125 %), even in un-aged samples, indicating problems with the extraction method. Therfore, it is not certain that the disappearance in this study only reflects biodegradation. The half-life values that can be calculated from this study may overestimate the degradability of γ-HBCDD.

In the second 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 simulation study 1 (4.7 mg/kg dw in aerobic sediment, 4.3 mg/kg 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. The half-life for the individual diastereomers under **aerobic** conditions recalculated to 12°C was approx. 210 days for α-, 130 days for β and 190 days for γ-HBCDD based on the disappearance of the individual diastereomers. The half-lives under **anaerobi**c conditions were approx. 210 days for α- 80 days for β- and 125 days for γ-HBCDD indicating that the α-diastereomer was not sensitive to reductive dehalogenation under anaerobic conditions. The study 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. There were no indications of further transformation of 1,5,9-cyclododecatriene as no CO_2 was formed during the course of the study.

The degradability of 1,5,9-cyclododecatriene (CDT) has been studied in a modified ready biodegradation test **(Davis, 2006b)**. ^{14}C-labelled 1,5,9-cyclododecatriene (CDT) was dispersed on silica gel and incubated at room temperature for up to 77 days. Approx. 50% $^{14}CO_2$ was produced during 63 days of incubation at a test concentration 0.2 mg HBCDD/l. At

1 mg/l the degradation seemed to be slower. After 63 days of incubation ca. 30% $^{14}CO_2$ had been formed whereas after 77 days of incubation 68% of the initial radioactivity had been identified as CO_2. This has been interpreted by the TCNES subgroup for PBT assessment as evidence that CDT clearly is not ready biodegradable, but does not fulfil the P criterion of the TGD.

It is difficult to judge the persistency of HBCDD because of the different results in the two studies in sediment mentioned above (**Davis 2003 and Davis 2004**).

Davis et al, 2006 argues that the difference in half lives between the two degradation studies in sediment can be attributed to the observation that biodegradation kinetics are significantly impacted by the concentration of the test chemical with kinetics often changing from first order to zero order at higher concentrations and that, at higher concentrations, for poorly soluble substances such as HBCDD, biodegradation rates are more dependent on mass transfer limitations than on true biodegradation kinetics. It might be the case that the differences are caused by the different concentrations. It must however, be recognised that the persistency of the three diastereomers differs, and that there is not one single half-life for HBCDD (the environmental occurrence also show large differences between the diastereomers).The three diastereomers have different chemical physical properties. α-HBCDD has an approx. 20 times higher water solubility than γ-HBCDD and a lower log Kow (5.1 compared to 5.5) which means that they also have different partitioning behaviour between pore water and the solid phase and thus it can, for example, be assumed that they are not equally affected by mass transfer limitations if tested at the same concentration. It is therefore not considered appropriate to draw conclusions on the biodegradability of α-HBCDD based on studies performed on γ-HBCDD.

Concentrations in four dated sediment cores have been reported in the literature. In a study by Kohler et al. (2006) a sediment sample was taken in Lake Griefensee at a depth of 31 m, sectioned and analysed. The HBCDD concentration was 1.3µg/kg dw in 1989, 1.8 µg/kg dw in 1995 and 2.5 µg/kg dw in 2001 i.e. the concentration of HBCDD in the 12 years old sediment layer was 50% of that found in the top layer. Moreover, Rembergeret al. (2004) detected concentrations of HBCDD in sediment layers 30 (±7) and 40 (±7) years old in two different sediment cores from the Stockholm archipelago. The concentrations were 25-33% of those found in the top layer.

These environmental measurements indicates that the dissipation rates obtained in the simulation degradation rates may not be relevant for the environment. It is not possible to determine a quantitative half-life of HBCDD in sediment based on these studies. However, it is clear that the DT50-values for γ-HBCDD derived in Davis et al 2003 of 21 and 62 days (12^{o}C), under aerobic conditions or 2-3 days (12^{o}C) under anaerobic conditions are not relevant in these cases as HBCDD is still present in sediment after 15-40 years.

In addition, sediment from some of the locations close to the former production site at Aycliffe, UK sampled in March 2002 were re-sampled 2 years after the close down of the factory in the autumn of 2005. In 2002 total HBCDD was analysed with GC. In 2005 an analysis method allowing for diastereomer specific analysis was used.

The HBCDD concentrations were higher in the 2005 survey than in 2002 at two sampling sites (BFR5 and BFR7). One site (BFR9) had a slightly lower concentration in 2005 than in 2002 whereas at the fourth site (BFR8) the concentration was below the detection limit both years (see Table 3-204).

Table 3-204 HBCDD concentrations in sediment (µg/kg dw) at Aycliffe, 2002 and 2005

Sampling site	2002	2005
BFR5	<50	2250
BFR7	11310	70085 ± 75391
BFR8	<50	<300
BFR9	441	112[a] 387[b]

a) only α– HBCDD (112 µg/kg dw) was above the detection limit

b) Estimated total HBCDD assuming not detected = detection limit/2. Detection limit for α–, β- and γ-HBCDD was 50, 50 and 500 µg/kg dw, respectively.

It is hard to draw any conclusions other than that the concentration of HBCDD in sediment in the vicinity of the former production site does not seem to have decreased two years after closure of the site.

In summary, HBCDD seems to be fairly persistent in aerobic soil.

No firm conclusion can be drawn 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.

α-HBCDD seems to be the least degradable with an aerobic DT_{50} in sediment at 12°C of approx 210 days, which is above the P-criterion of 120 days.

For γ-HBCDD the available data indicate very different half-lives depending on test concentration. The aerobic DT_{50}-value was below the P-criterion in sediment when tested at a concentration similar to the 90th percentile of measured concentrations in freshwater sediment. When tested at a concentration similar to what is measured close to polluted point the DT_{50} was 190 days (12°C).

The few data available from dated sediment cores does not support rapid degradation of HBCDD in sediment. Neither does the study on sediment concentrations at the former HBCDD production site at Aycliffe, UK.

3.4.6.2 Bioaccumulation

HBCDD has a log K_{ow} of 5.6. One study on fathead minnow gave a steady sate BCF of 18100 after 32 days of exposure. In another study rainbow trout were exposed for 35 days at two different concentrations of HBCDD with the diastereomer composition: α 6.4 %; β 4.5 %; γ 79.1 %. In the low concentration group (mean measured conc. 0.18 µg/l) apparent steady-state was not achieved and a BCF of 13085 was calculated using the average tissue concentration on uptake day 35. In the high concentration group (mean measured conc. 1.8 µg/l) HBCDD concentrations in edible tissues appeared to reach steady-state at day 14 and BCF was calculated to 8,974. Kinetic modelling (BIOFAC) gave BCF-values of 21940 and 16450 for the two groups, respectively. The diastereomer specific bioconcentration factors are not known, but it can be assumed that they differ. This is supported by the fact that it is the α-diastereomer that is found in the highest concentrations in biota despite having a much lower

concentration in technical HBCDD and often a lower concentration in e.g. sediment. Furthermore, bioaccumulation studies in eartwhworm indicated that α-HBCDD had a 15-60 times higher bioaccumulation factor than γ-HBCDD. Based on these data it can be concluded that HBCDD meets the vB criterion.

3.4.6.3 Toxicity

Two long term tests are available for HBCDD. The first study, a reproduction test on *Daphnia magna*, reports a NOEC of 3.1 µg/l. In the other study, a fish early life stage test, no effects were seen at the highest tested concentration, which was 3.7 µg/l. In addition, a marine algae test with Skeletonema Skeletonema where the all three diastereomers were tested together at their respective limit of solubility using a generator column when preparing the test solution gave an EC50 of 52 µg/l. Based on these data it can be concluded that HBCDD meets the T-criterion.

3.4.6.4 Occurrence in the environment

HBCDD has been detected in almost all environmental compartments in rural as well as in remote areas. The levels detected are higher close to local sources, e.g. production and use, and decrease with increasing distance from source(s).

The diastereomer pattern in abiotic samples e.g. sediment resembles in most cases that of the technical product with the γ-diastereomer having the highest concentrations. In biota (e.g. fish, birds and mammals) on the other hand, the α-diastereomer (<10 % of the technical product) is detected at the highest levels. The reason for this difference is not known.

Measured HBCDD levels in air range from a few pg/m^3 in remote areas of Sweden and Finland to 280 ng/m^3 in outdoor air at production facilities.

In freshwater sediment HBCDD levels range from <0.1µg/kg dwt in unpolluted sediments to above 2mg/kg dwt close to industry facilities (production of HBCDD, textile industries). Concentrations as high as 70 mg/kg dw have been measured close to a production facility. The median concentration for a total of 144 samples is 1.5 µg/kg dwt and the 90th percentile is 197 µg/kg dw. Measured levels in estuarine/marine sediments range from <0.5 µg/kg dwt to about 8.8 mg/kg dwt in the polluted Norwegian Åsnefjord. The median concentration for a total of 53 samples is 4.2 µg/kg dwt and the 90th percentile is 122 µg/kg dw.

Biota

HBCDD levels in European **marine fish** range from 0.001 to 49 µg/kg wwt (0.1-1110 based on lipid weigth).The higher levels are associated with industrial point sources. The median wet weight concentration of HBCDD in marine fish is 0.38 µg/kg wwt (n = 85). When based on lipid weight the median HBCDD concentration is 12 µg/kg (n = 77).

HBCDD levels in European **marine mammals** (seal, porpoise, dolphin) range from 0.5 to 6400 µg/kg wwt (7.5-21300 µ/kg based on lipid weigth). The median wet weight

concentration of HBCDD in marine mammals is 108 µg/kg wwt (n = 227). When based on lipid weight the median HBCDD concentration is 439 µg/kg (n = 205).

The levels of HBCDD in **marine birds eggs** range from a few µg/kg wwt at remote islands in northern Norway to around 100 µg/kg wwt close to production facilities.

HBCDD has also been detected in e.g. plankton, invertebrates, freshwater fish and terrestrial birds.

Biomagnification

Fish-marine mammals

The biomagnification potential of HBCDD (i.e. α-HBCDD) has been assessed by comparing measured levels of α-HBCDD in prey and predators using the monitoring data available to the rapporteur. Generally the highest levels of HBCDD are found in marine mammals such as seals and porpoises which are predominantly exposed to HBCDD via their food.

Overall, the median concentration ratios between marine mammals and fish on a wet weight basis and a lipid weight basis are 272 and 28, respectively. These figures are based on all data on HBCDD levels in marine fish and mammals from Europe known to the rapporteur.

As a way of reducing the influence of potential local sources, location specific comparisons have been made for the Baltic Sea and the Western Scheldt. The median concentration ratios between marine mammals and fish on a wet weight basis were 61 and 187, for the Baltic Sea and the Western Scheldt, respectively. The corresponding ratios on a lipid weight basis were 5.8 and 11, for the Baltic Sea and the Western Scheldt, respectively. The HBCDD concentration ratios between harbour porpoises and its prey from the U.K, based on an approximate whole wet weight basis for both the harbour porpoise and its fish diet, was 254.

Fish-Marine birds

Lundstedt-Enkel et al. (2005) used a statistical resampling method to calculate biomagnifications factors for a number of substances, including HBCDD, from Herring muscle and Guillemot eggs collected from the Baltic Proper. The biomagnification factor for HBCDD for the step between herring and guillemot was 9.1 calculated for lipid weight. For comparison, in the same study, the BMF for PBDEsum was 5.5, for PCBsum 24.6 and for DDTsum 36.

Freshwater foodchain

The biomagnification of α- and γ-HBCDD congeners in a Lake Ontario food web (invertebrates: plankton, *Mysis* and *Diporeia*; forage fish: alewife, sculpin and smelt; top predator: trout) has been studied (Tomy et al., 2003, Tomy et al., 2004).

Whole body concentrations of α- and γ-HBCDD were highest in the top predator lake trout samples; 0.4-3.8 µg/kg (wet wt) for the α-diastereomer and 0.1-0.8 µg/kg for the γ-diastereomer. There was linear relationship between the total HBCDD concentrations (wet weight) and trophic level based on $\delta^{15}N$ suggesting that HBCDD biomagnifies in the Lake Ontario fod web. The trophic magnification factor (TMF) was 6.3, derived from the slope of total HBCDD to trophic level relationship.

This TMF was higher than for p,p'-DDE, 6.1, and for sum of PCBs, 5.7. Lipid corrected biomagnification factors (BMF) for predator/prey, were variable between feeding relationships and highest for foragefish/zooplankton where it ranged from 3.5 (Sculpin/*Diporeia*) to 10.8 (Smelt/*Mysis*) for α-HBCDD.

In conclusion, all available data from monitoring studies shows that HBCDD (α-HBCDD) biomagnifies in the marine and aquatic food webs. The by far highest α-HBCDD concentrations are measured in air breeding marine predators such as seals and porpoises.

Time trends:

Several studies report increasing concentrations of HBCDD in biota. In a recent report Law and co-workers (2006, in press) presented concentrations of HBCDD in blubber of 85 harbour porpoises stranded or dying due to physical trauma in the U.K. during the period 1994-2003. α-HBCDD dominated over the other diastereomers and was detected in all samples at concentrations ranging from 11 to 21342 µg/kg lipid. The study shows increasing concentrations which was not confounded by age (length), sex, nutritional status, or location. The median concentration in the blubber increased from below 100 µg/kg lwt in the mid nineties to 9400 µg/kg lwt in year 2003.

Results from Roos (Swedish museum of natural history, personal communication, 2006) also indicate an increase of HBCDD over time in seals from the Baltic Sea. Median levels in the 1980-ies ranged between 16 and 35 µg/kg lwt with a median concentration of 28 µg/kg lwt (n = 7). In the 1990-ies the levels ranged between 34 and 177 g/kg lwt with a median of 73 µg/kg lwt. (n = 12). From 2000 and onwards data from only one seal is available having a HBCDD concentration of 64 µg/kg lwt. However, in another study Lundstedt-Enkel (2006) analysed blubber from 30 Grey seals during the period 2000-2002. the HBCDD concentration ranged from 31-554 µg/kg lwt with a mean of 101±98 µg/kg lwt (mean ±SD). The median concentration is not available but the results indicate that the levels have not decreased.

Sellström et al. (2003) presented results of measurements of HBCDD in eggs from Guillemot from St. Karlsö in the Baltic Sea from 1969 to 2001. The concentration of HBCDD approximately doubled during the study period from 8 µg/kg wwt in the early seventies to approx. 16 µg/kg wwt in the late nineties. The increase appears according to the authors to have levelled out since the mid-1990s.

Knudsen et al. (2005) analysed eggs from Atlantic puffins, Herring gull, and Kittywake from northern Norway (Hornøya and Røst) from 1983, 1993, and 2003. The HBCDD levels have risen from 1.1-2.9 µg/kg wwt in 1983 to 6.1-17.3 µg/kg wwt in 2003.

Increasing concentrations of HBCDD in biota are reported also from other parts of the world. The temporal trend in HBCDD content of three marine mammals in the Asia-Pacific was monitored in a recent Japanese study (Kajiwara et al, 2006).
In female northern fur seals (Callorhinus ursinus), collected during ten years between 1972-1998 off Sanriku, Japan in the Pacific Ocean, the average HBCDD concentration ranged from <0.1 ng/g lipid weight in 1972 to 33 ng/g lipid weight in 1997 (n=35, age=14-23 yrs).
In male melon-headed whales (Peponocephala electra), stranded on the Pacific coast of Japan in 1982 and 2001, the average HBCDD concentration was 7.0 ng/g lipid weight (n=5; range 2.7-9.7 ng/g) in 1982, and in 2001 the concentration had increased to 390 ng/g lipid weight (n=5; range 330-460 ng/g). In male finless porpoises (Neophocena phocaenoides), caught at the South China Sea coast, the HBCDD levels increased from 18 ng/g lipid weight in

porpoises collected in 1990 (n=7; range: 4.7-37 ng/g) to 35 ng/g lipid weight in 2000/2001 (n=5; range: 21-55 ng/g).

The most recent measurement of HBCDD in biota used in the time trend section is from 2003, which is the same year the production of HBCDD in production facility A ceased.

Long range transport

HBCDD has been detected in very remote areas, such as in air in northern Sweden and Finland, far from potential sources (Sternbeck et al., 2001; de Wit et al., 2002). HBCDD has also been found in fish from Swiss mountain lakes (Schmid et al., 2004), in mussels from Lofoten and Varanger and liver from Atlantic cod from northern Norway (Fjeld et al., 2004), in Polar cod and ringed seal from Svalbard in the arctic region (Jensen et al., 2004), in marine bird and bird eggs from northern Norway (Knudsen et al., 2005; Verreault et al., 2004; Gabrielsen et al., 2005), and in polar bears from Greenland and Svalbard in the Artic Ocean (Gabrielsen et al., 2004; Muir et al., 2004). These findings suggest that HBCDD undergoes long-range atmospheric transport, even though its importance relative to other sources and transport routes remains to be established. However, the potential use of HBCDD-containing articles by the small human populations living in these areas are not expected to explain the levels detected

3.4.6.5 Summary of PBT assessment

The PBT-criteria according to TGD and the corresponding data for HBCDD for each endpoint are shown in Table 3-205.

Table 3-205 Data for hexabromocyclododecane and PBT- and vPvB-criteria according to the revised TGD.

Criterion	PBT-criteria	vPvB-criteria	Hexabromocyclododecane
P	Half-life > 120 d in freshwater sediment	Half-life > 180 d in marine or freshwater sediment	Not readily biodegradable Sediment simulation tests: 1) Dissipation half-life at 12°C for γ-diastereomer in aerobic sediment 20-60 days. Half-lives may not only represent biodegradation, α- and β-diastereomers were not studied. 2) In aerobic sediment approximately half of the added HBCDD was transformed into the three dehalogenated metabolites within 4 months at 20°C. The half-lives of the individual diastereomers were approx. 210, 130 and 200 days for α-, β-and γ-HBCDD, respectively when temperature-corrected to 12°C.
B	BCF > 2000	BCF > 5000	BCF 18100
T	Chronic NOEC < 0.01 mg/l or CMR or endocrine disrupting effects	Not applicable	Chronic NOEC *Daphnia* survival, reproduction, growth 3.1 µg/l

Criterion	PBT-criteria	vPvB-criteria	Hexabromocyclododecane
			Chronic LOEC *Daphnia* reduced length 5.6 µg/l

HBCDD does not unequivocally fulfil the specific P-criterion, with some reliable studies indicating that biodegradation can occur. However in the light of findings of HBCDD at µg/kg levels in sediment cores dating 20 years back it can be questioned if the degradation rates obtained in these studies are relevant for the environment. In addition monitoring data indicate a significant degree of environmental transport and overall stability. The BCF of HBCDD is 18 100 and thus the vB criterion is fulfilled. Also the T-criterion is fulfilled according to available data. HBCDD is ubiquitous in the environment, being also found in remote areas far away from point sources (e.g. in polar bears in the arctic). The highest concentrations of HBCDD are detected in marine top-predators such as porpoise and seals showing that HBCDD bioaccumulates up the food chain. The trend also shows an increase in tissue concentrations of HBCDD in biota over time; this is especially so for marine mammals where for porpoise in the North Sea, the concentrations have increased rapidly in recent years. To conclude, although HBCDD does not unequivocally fulfil all individual criteria it is concluded that the substance **overall** fulfils the PBT-criteria of the TGD.

Conclusion

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

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 General introduction

See also chapter 2 for use pattern of HBCDD.

Due to the use of HBCDD in products in the society, humans may be exposed from different sources:

- at the workplace at the production of HBCDD,
- at the industrial use (formulation and industrial use of HBCDD as an additive and at the industrial uses of articles containing HBCDD ;
- from use of consumer products; and,
- indirectly via the environment via food, soil, water and air.

All known uses of HBCDD are as flame retarding additive in polymers. HBCDD is used foremost in extruded polystyrene (XPS) and expanded polystyrene (EPS) used mainly in building insulation, high impact polystyrene (HIPS), and in back-coating for textile.

HBCDD is a solid substance at room temperature (melting point >170 °C) and is most often handled as a solid powder or a compacted (pelletised) powder in the industry. Sizes of powder and granules are given in Table 1-5. The finest powder is mainly used at the formulation of textile backcoating. The vapour pressure is 6.3×10^{-5} Pa at 21 °C (Stenzel and Nixon, 1997). The density of solid HBCDD is 2.38 g/cm^3 at 20 °C. See also section 1 for the physico-chemical properties of HBCDD.

During formulation the HBCDD is encapsulated into a polymer matrix and is physically bound within the polymer matrix, however it is not chemically bound and may therefore migrate. The presence of HBCDD in dust collected from vacuum cleaners used in different buildings may support emissions from products, although emissions of HBCDD from the vacuum cleaner itself can not be completely ruled out (Leonards *et al.*, 2001).

The human population can be exposed to HBCDD by inhalation of vapour and airborne dust, ingestion and by dermal contact. In addition there is a risk that babies can be exposed during pregnancy and due to breast-feeding.

Based on information in chapter 2 the following exposure routes by exposed populations are considered to be relevant for this assessment:

- Inhalation of vapour and airborne dust and via dermal contact at the production of HBCDD, the industrial uses and the industrial end-uses
- Via inhalation of HBCDD emitted from articles as vapour and adsorbed to domestic dust and dermal exposure during private end-use (consumers)

- Exposure via the environment via inhalation of vapour and particles and via oral routes when exposed by food and water.

In this report, the identified sources of human exposure to HBCDD are summarised, but there may be other sources not yet identified. However, the most important sources are identified. Other populations/subpopulations than identified here may be exposed. These groups may represent more sensitive groups e.g. pregnant woman, breast-fed babies and children.

For the estimation of human exposure to HBCDD, similar activities can be clustered e.g. workers adding the substance to different processes and consumers use of similar but different articles containing HBCDD.

Humans may be exposed to a chemical for different periods during lifetime including single short-term exposure and persistent exposure for a lifetime. For HBCDD, humans could be exposed during their lifetime to HBCDD persistently present in the environment and from the residential environment. The cumulative exposure for a lifetime will depend on the magnitude of the persistent exposure.

4.1.1.2 Occupational exposure

4.1.1.2.1 General introduction

The following data were used for occupational exposure assessments for HBCDD:

- measured data of HBCDD
- physico-chemical data of HBCDD as the physical state, powder dimension and the vapour pressure at different temperatures
- qualitative and quantitative data regarding methods and use pattern of the product, temperature at which production processes take place; and the amount of HBCDD used in the different products.

The occupational inhalation exposure to HBCDD is dominated by exposure to airborne dust because of the low vapour pressure, the high melting temperature, the process temperature at the manufacture industrial uses and the particle size of the product.

Workers in the industry who may potentially be exposed are primarily those workers who come into direct contact with the pure substance. This includes workers working close to processes emitting HBCDD, workers packing the substance and workers transferring the substance to other systems in the chemical industries.

For all activities the exposure is strongly influenced by plant conditions and worker habits. Bad hygiene in a plant could lead to high background concentrations. Examples of this are broken bags, dusty pallets and dusty rooms. The presence of effective control measures can also have a great influence on the exposure.

Information on the product particle size distribution of HBCDD has been received from the manufacturers. The particle size of HBCDD differs between technical products, but can be grouped in either:

- fine powder (mean size 2-19 μm),

- standard grade powder (mean size 20-150 μm), and
- granules (mean size 560-2,400 μm).

For more information on the particle size of HBCDD see chapter **1.3.8**. Granulometry.

The assumptions used in the exposure assessment are described below.

The particle size is important for the risk characterisation, determining;

- site of deposition in the airways, i.e., the fraction inhalable/respirable (since 100 % absorption is assumed for both oral and inhalation exposure, 100 % absorption is assumed for all particles having a mean size <100 μm),
- degree of dusting (the handling of powder (100 % <100 μm) is assumed to give more respirable and inhalable airborne dust than handling of the granule grade qualities (10 % <100 μm). Thus, for granules only the fraction smaller than 100 μm is considered available), and
- degree of dermal absorption (a dermal absorption of 4 % is assumed for the powder, whereas an absorption of 2 % is assumed for the granules).

Some industrial users buy and use the HBCDD as a masterbatch. Exposure data from one plant producing masterbatch (17 samples on air concentrations), and three plants using masterbatch (40 air samples) was received in 2005 (Searl and Robertson, 2005). The concentration of HBCDD in the masterbatch was 50-70 %. Sub-scenarios for production of masterbatch and use of masterbatch in production of XPS have been included.

For the use of HBCDD in textile coatings, there is need to produce extra fine HBCDD powder by milling HBCDD (i.e., micronising HBCDD). Data on exposure in one plant (4 samples on air concentrations) specialised in micronising HBCDD has been received (Searl and Robertson, 2005), and a sub-scenario for the micronisation has therefore been included in the section on industrial use of HBCDD.

Information on the particle size of airborne HBCDD-dust at workplaces has been received, although the information is sparse for granules. In the absence of data, it is assumed that the particle size of the airborne dust probably is less than the particle size in the bulk material. For the standard grade powder on the market (from the three producers), 50, 75 and 100 % of the particles, respectively, seem to have a size less than 100 μm. The share of the airborne dust, which is inhalable, is therefore assumed to be 100 % as a reasonable worst case. For the granule grades, 1, 5 or 10 % of the particles in the bulk is assumed to have a size less than 100 μm. We chose 10 % as a reasonable worst case. Consequently, the concentration of inhalable dust in the air, when handling granules, will be 10 % of the concentration when handling powder.

Dermal exposure to HBCDD may occur in direct handling of HBCDD, either indirectly by contamination of surfaces or directly by dermal deposition of airborne dust.

Assuming that oral exposure is prevented by personal hygienic measures, ingestion of HBCDD does not seem to be a relevant route of occupational exposure, except to the extent that this follows initial inhalation exposure.

There are several industries in which HBCDD is produced or used. In some cases the processes and activities may lead to emission of HBCDD in the workplace and exposure of

the workers. This exposure may be similar in different industries. Therefore the industries have been clustered in similar exposure scenarios based upon the type of process and activity and the possibilities for exposure that relate to that process and activity. An example of clustered activities is opening, and emptying bags containing HBCDD.

There are three industry sectors where occupational exposure to HBCDD may occur;

- Manufacture of HBCDD
- Industrial use of HBCDD as an additive (formulation and processing in the polymer- and textile industry)
- Industrial use of semi-finished or end-products containing HBCDD

The exposure is assessed without taking account of the possible influence of personal protective equipment (PPE). Information of the effectiveness of PPE to reduce exposure to HBCDD in practical situations is limited. The use of PPE normally reduces the level of exposure. PPE are usually intended for use during work operations entailing risk for increased exposure such as repair work, service and maintenance.

The exposure may be reduced by PPE, but incorrect or careless use may lead to unforeseen and unexpected exposure. One example is when using protective gloves coming in contact with e.g. the face.

In this chapter on occupational exposure, inhalation and dermal exposure from the EASE-model (Estimation and Assessment of Substance Exposure) are presented. EASE is a general-purpose predictive model for workplace exposure assessments. The model is in widespread use across the European Union for the occupational exposure assessments of new and existing substances. All models are based upon assumptions. Their outputs are approximates. EASE is only intended to give generalised exposure data and works best in exposure assessments when the relevance of the modelled data can be compared with and evaluated against, measured data.

There is at present no validated method for measuring exposure to HBCDD. In the recent study by Searl and Robertson (Searl and Robertson, 2005), HBCDD in air has been sampled using IOM samplers and HBCDD has been analysed by HPLC. Limited data on the realistic total number of exposed employees in the EU have been submitted by the industry, but one recent estimate is that there are 50-70 plants using HBCDD in Western Europe (Searl and Robertson, 2005). No information on the sex and age of the exposed workers in the EU is available. In the USA, approximately 12,000 employees at 420 facilities are exposed to HBCDD (US,EPA., 1996)

The following exposure scenarios are considered:

- the filling of bags at the manufacture of HBCDD
- charging HBCDD to processes producing end-products or semi-products containing HBCDD (micronisation of HBCDD, production of XPS and EPS, and textile coating)
- sewing

At workplaces where the substance is handled as the pure substance, the premises and equipment are expected to be more or less contaminated with the substance. This seems especially true for the processes of micronisation and textile coating (Searl and Robertson,

2005). This causes exposure during cleaning and during the everyday presence and activity at the workplace. This exposure is assumed to be mainly via the dermal route and less than the more direct exposure at filling/adding operations. Similar exposure scenarios can occur at workplaces where materials containing HBCDD is used, but this is assumed to be low.

Measured data are given for sites producing HBCDD, for sites using HBCDD as fine powder, standard grade, granules, and masterbatch. The use of HBCDD as such in fine powder takes place at, at least 16 sites and, in standard grade in at least 30 sites. In the measured exposure data no specific information on the particle size of the used HBCDD is given. In the older data, no information on the size distribution of the powder in the bulk material is given, but in the new study by Searl and Robertson (Searl and Robertson, 2005) both respirable and inhalable HBCDD in the air are measured. The given measured data from the production sites and sites using HBCDD in XPS and EPS production are assumed to be more representative than the measured data given for the sites using HBCDD for micronisation and textile coating.

Not enough data for estimation of the exposure at industrial end-uses of semi- and end-products containing HBCDD are available. Scenarios with possible exposure might be all kind of handling of material containing HBCDD, e.g. work with building insulation material, insulation boards for roads and railways, and backcoated textile. The exposure depends on how the material is handled (dust generating cutting, heating e.g. with a hot wire for cutting boards), the circumstances in the work environment (ventilation, the shaping of the workplace) and the frequency and duration of the work task. However, these scenarios lead probably to lower exposure levels than the handling of the pure substance.

As a general approach, RWC values have been set using measured data and EASE modelling, with most weight on the measured data. Typical inhalation exposure has been set at half the RWC-value, which is a general approach supported by the measured data (median approximately half RWC). For dermal exposure, there is no measured data and EASE has therefore been used. Typical dermal exposure has not been specified in the RAR. Based on the relation between RWC and typical inhalation data for HBCDD, one may estimate that typical dermal data also is half of RWC data. However, because the EASE- estimate is likely a conservative value and there is likely a bigger variation in dermal exposure (depending on varying personal habits/work routines) than in inhalation exposure, the difference between typical and RWC could be bigger than a factor of 2.

4.1.1.2.2 Occupational exposure to HBCDD during its manufacture

Background

Technical grade HBCDD is generally produced from *cis trans, trans*-1,5,9-cyclododecatriene (CDT), one of four CDT isomers, (CAS No. 27070-59-3). The reaction, *trans*-addition of bromine to the double bounds of CDT, results in the three diastereomers α-, β- and γ-HBCDD. It is a batch wise process and each batch takes 12-15 hours, as described by one of the producers. The manufacture of HBCDD is described in Chap. 2.1.

HBCDD is currently produced in two plants in the EU. We assume that five persons per process is a representative staffing requested to run one manufacture process. This implies

less than 50 persons working at the HBCDD-manufacture sites in the EU if the work is performed in three shifts.

The process temperature during the manufacture varies between 20 ºC and 70 ºC. The manufacture of HBCDD, up to the storage step, takes place in closed systems. However, exposure may occur through system leaks, during sampling from the manufacturing process, in the compactation to granules, during filling of bags (packing), when cleaning the process rooms, during service and maintenance and at incidental releases of HBCDD.

Filling of bags is assumed to be the working task during normal operation at manufacture of HBCDD with the highest exposure. A normal situation at the production of HBCDD is that two individuals work in the packaging area. Exposure may be significant when changing dust filters in the ventilation, during maintenance and repair work of the manufacture units including the ventilation and during cleaning of the process rooms. It is envisaged that both inhalation or airborne dust and dermal exposure may occur during the manufacture of HBCDD.

Scenario FILLING. Filling of bags at the manufacture of HBCDD

The exposure to HBCDD at the filling of bags is assumed to last for 8 h every working day as a reasonable worst case, even though it is likely that workers some days work with other tasks.

Inhalation exposure data

The highest exposure to HBCDD at the manufacturing site is assumed to be during the packing of HBCDD into 25 kg bags. The product is a crystalline powder (20-150 μm). There may also be production of granules via a compactation process, and filling of granules into 500 or 1000 kg bags. The earlier process-steps, e.g., bromination, crystallisation, centrifugation and drying, are closed and the release of HBCDD to the working environment is probably low during normal conditions. Grinding (micronisation) of HBCDD to 3-4 μm particles for some applications may take place at the manufacture of HBCDD, but no manufacturer in the EU perform micronisation. However, productions of fine grade HBCDD by micronisation do occur in the EU at a few plants. Some exposure data has been received from one plant specialised in producing fine grade HBCDD by micronisation (not a production plant), and there is a specific sub-scenario for production of fine grade HBCDD by micronisation.

Only a few data on occupational exposure by inhalation at the production of HBCDD is available, representing 2 or 3 plants, although only one active in 2005. The background information, with regard to where the measurements were taken or the number of measurements, varies. However, the measured values can be used with support from modelled values.At one manufacturer (Broomchemie B.V., Terneuzen) inhalable dust and HBCDD in the air at fixed locations at workplaces were measured; see Table 4.1.(e-mail from M. Karp to KemI 2000-02-01). The samples were taken at a reactor were HBCDD were produced in a batch process. The plant was working during the time of measurements. At the big bag filling station five and three bags respectively were filled during the sampling period. The whole process is controlled from a control-room. The point of the air-measurement at the reactor is not a workplace. Sometimes the staffs have to do sampling at this place. The big bag filling

station is a workplace. The activities of the employees are: controlling function, blow up the bags with compressed air, filling the bags, sampling after each filling, preparation of the bags and transportation. There is a big difference between weighted total dust and the analyses of HBCDD. No information on the composition of the airborne dust, except for HBCDD, is given. No other substances seem to be handled at these places. No information on the product powder size is given.

At another manufacturer, samples taken during operating conditions (bagging off process) revealed concentrations of total inhalable dust of 4.0 - 4.5 mg/m^3 (Biesemeier, 1996). No information if the samples were personal or stationary is given.

Ten samples (from 2-3 shifts in one of the plants mentioned above) have been measured in 2005 (Searl and Robertson, 2005). The plant produces standard grade powder. The samples represent packing, compactation, process operation, and working in the warehouse. Most HBCDD was found in the inhalable fraction and, with the exception of the warehouse, the air concentrations of HBCDD were fairly similar between the different tasks. The highest measured concentration of total HBCDD was approximately 3 mg/m^3, although the 90th percentile total HBCDD concentration for four all tasks was lower (1.9 mg/m^3). No respiratory protective equipment was used at the plant according to the report.

Measurements of air concentrations of HBCDD were performed in one plant producing fine grade HBCDD by micronisation of HBCDD (Searl and Robertson, 2005). This plant is by far the most important microniser in the EU. In all four samples, most HBCDD was found in the inhalable fraction (>90 %). The total mean concentration was 23 mg/m^3, and the 90th percentile 35 mg/m^3. The work area was reported to be very dusty, and even though the workers used PPE, HBCDD was reported to permeate both gloves and the suit (around the collar and zip). The report states that there is one operator working per shift, and that the workers rotate between different tasks. Since no worker has this task every day, a more reasonable long-term RWC exposure value of 10 mg/m^3 will be used in the RC. The typical exposure is assumed to be half this value.
The workplace air-measurements of inhalable dust and HBCDD at manufacturers of HBCDD are summarised in Table 4-1.

Table 4-1 Workplace air-measurements of inhalable dust and HBCDD at manufacturers of HBCDD.

Measuring point Place position	Substance	Number of samples (time/ sample, min)	Concentration (mg/m^3)	Reference
Ambient air	Inhalable dust HBCDD	(120) (120)	0.04 0.00028	(Waindzioch, 2000)
Big bags filling station for HBCDD	Inhalable dust HBCDD	(88) (88)	2.5 0.0094	
- " -	Inhalable dust HBCDD	(48) (48)	2.2 0.097	
Reactor	Inhalable dust HBCDD	(90) (90)	0.1 0.0234	
- " -	Inhalable dust HBCDD	(51) (51)	0.2 0.0285	
Operating conditions	Inhalable dust	No data	4.0 - 4.5	(Biesemeier, 1996)

Measuring point Place position	Substance	Number of samples (time/ sample, min)	Concentration (mg/m³)	Reference
(bagging off process)				
Production of standard grade HBCDD,	Inhalable HBCDD	10 (480)	Mean: 1.2 90 %-ile: 1.9	(Searl and Robertson, 2005)
Production of fine grade HBCDD,	Total HBCDD	4 (480)	Mean: 23 90 %-ile: 35	(Searl and Robertson, 2005)

Inhalation exposure is assessed by EASE. The reasonable worst-case inhalatory dust exposure during the manufacture of standard grade HBCDD is 2-5 mg/m^3 (See Appendix 2, EASE-modelling). For granules, 10 % of the mass is represented by particles having a size <100 µm, and inhalation exposure to 0.2-0.5 mg/m^3 is assumed for granules. The modelling is not considered to apply for micronisation, which according to the measured data is an extremely dusty operation. Considering the availability of measured data from micronisation, and the extreme process, a modelling of inhalation exposure is not considered to improve the exposure assessment, and modelling is therefore not performed for micronisation.

Dermal exposure data

Dermal exposure is assessed by EASE. It is assumed that exposure at the manufacture of HBCDD occurs principally during packing and compactation of the product. The reasonable worst-case dermal exposure level (potential exposure) during the manufacture of standard grade HBCDD is estimated to be 1 mg/cm^2/day because little direct handling is expected, but contamination of surfaces due to emissions to air is foreseen. It is assumed that both hands are exposed. This corresponds to an exposed area of 840 cm^2, which leads to an exposure of: 840 mg/day (see Appendix 2, EASE-modelling). For granules, the exposure is thought to be 10 % of that with powder, because of less dusting, i.e., 84 mg/day.

A separate modelling is performed for the micronisation, where a dispersive use is assumed. The reasonable worst-case dermal exposure level (potential exposure) during the use of HBCDD is estimated to be 1-5 mg/cm^2/day, (see Appendix 2, Ease-modelling). It is assumed that both hands are exposed. This corresponds to an exposed area of 840 cm^2, which leads to an exposure of 4200 mg/day, when no PPE is used. Even when PPE is used, it is assumed to be a considerable exposure.

Protective suits and gloves are reported to be used in these production processes (Searl and Robertson, 2005).

Conclusions: Manufacture of HBCDD

Inhalatory exposure

For product packing and compactation at the manufacture of HBCDD a reasonable worst-case exposure level to be used for risk characterisation is considered to be 1.9 and 10 mg/m^3 for the production of standard and fine grade HBCDD, respectively. At the packaging of granules the exposure is assumed to be 10 % of the above value for standard grade powder, i.e. 0.19 mg/m^3.

Exposure during the manufacture (packing, filling of sacks) of HBCDD may be full shift, but short-term exposure (15 minutes) may be higher. It is assumed that this may be twice as high as the reasonable worst case, but for micronisation there are measured values up to 40 mg/m^3.

At a typical working day, the worker is assumed not to work in direct contact with the substance for 8 hour. Therefore, a typical level of exposure via inhalation can be about 0.95 mg/m^3 (TWA), representing 4 h contact with the standard grade substance. There are uncertainties in the measured values, e.g., with respect to few samples, but the EASE estimations support them.

Dermal exposure

Estimation of the dermal exposure has been derived with EASE. The reasonable worst-case dermal exposure level (potential exposure) during the manufacture of standard grade powdered HBCDD is estimated to be 1 mg/cm^2/day (see Appendix 2, EASE-modelling). It is assumed that both hands are exposed. This corresponds to an exposed area of 840 cm^2, which leads to an exposure of: 840 mg/day. For granules, the exposure is thought to be 10 % of that with powder, because of less dusting, i.e., 84 mg/day. Modelling of dermal exposure during micronisation (fine grade HBCDD) leads to an exposure to 1-5 mg/cm^2/day, this is equivalent to 4200 mg/day if assuming exposure of two hands.
At a typical working day, the worker is assumed not to work in direct contact with the substance for 8 hour and the extent of dermal contact with the substance can be limited because of careful handling of sacks, etcetera.

4.1.1.2.3 Occupational exposure during industrial use of HBCDD as an additive (formulation and processing in the polymer industry)

Background

Workers in the polymer industry using HBCDD are potentially exposed especially those workers who are working in direct contact with the pure substance. Activities leading to direct contact concerns workers handling the pure substance or products containing the substance and workers transferring the substance or products to other systems in the chemical industries.

Exposure may occur when adding (charging) HBCDD in the processes, during mixing the agent and moulding the agent into shapes - (extruding, expanding, coating), during taking samples from the process, during service and maintenance, during cleaning the rooms and at system leaks. The exposure depends on the particle size of the HBCDD used, i.e., 2-19 μm for fine powder, 20-150 μm for powder of standard grade and 560-2,400 μm for granules. The powder used in the textile formulation are smaller (2-19 μm) than the powder and granules used for polystyrene (20-150 μm and 562-2,400 μm, respectively), see Chapter 1.3.8 "Granulometry".

Manual charging HBCDD to the process is assumed to be the working task during normal operation of processes with the highest exposure. In this assessment the exposure when adding HBCDD is assumed to be the same at all processes irrespective of the kind of processes, provided the same HBCDD grade and particle size is used.

Scenario ADDING. Industrial use of HBCDD as an additive

Inhalation exposure data

Due to information on the particle size distribution of HBCDD from one manufacturer, the airborne HBCDD-dust is assumed to be mainly inhalable with a fraction of respirable dust. This is also supported by recent measurements (Searl and Robertson, 2005).

Data on air concentrations of HBCDD was submitted for the **production of XPS-masterbatch**, i.e., HBCDD-polystyrene granules (Searl and Robertson, 2005). The highest concentrations were measured during mixing and weighing (7.5 mg/m^3 for 2 hours), with a 90th percentile concentration of 5.4 mg/m^3 based on 10 samples representing durations of 19-295 minutes. The size distribution seemed highly variable during the weighing and mixing, as indicated by the observation that the inhalable fraction was 3-35 fold higher than the respirable fraction. Recalculated into 8 hours personal exposure concentrations, the mean and 90th percentile concentrations of total HBCDD for mixing and weighing were 0.88 and 1.36 mg/m^3, respectively. During these tasks, the workers were reported to use PPE (including a half mask disposable respirator), but to intermittently take off and replace the gloves.

For the extruder operators (4 samples), the 90th percentile was 0.16 mg/m^3 during approximately 5 hours and no PPE was used.

Three samples (no information on sampling) from another plant for master batch production with automised handling of HBCDD reported negligible air concentrations of HBCDD (Searl and Robertson, 2005).

One single measured value on occupational inhalation exposure from one user (**suspension polymerisation**) was submitted (Hüls, 1996). The dust concentrations during handling of HBCDD were amounted to an average of 1.6 mg/m^3. The background information was not well documented with regard to how the measurements were taken or the number of measurements.

Measured exposure data from the **manufacturing of extruded polystyrene (XPS)** foam have been submitted from eight surveys (Abbot, 2001). The surveys were carried out on the same process at seven different sites in various European countries. The number of people potentially exposed was assumed to be 10 per plant. Free flowing HBCDD, granulated or powder of HBCDD were delivered in 850 kg multiple use boxes (with the trademark “TNT”) with inliner. In one of the plants HBCDD powder was used at one occasion and at another plant HBCDD powder was used during the time of the survey. At all other locations granulated HBCDD was used.
The TNT-boxes are emptied into hoppers by a vacuum system. The solid feed stream is metered to the process using a closed system. During the processing fines of unknown particle size are generated by attrition. Leaks at the metering system can occur. This requires manual cleaning. The work tasks assumed to be relevant for exposure sampling were emptying of TNT boxes and cleaning of feed deck. Feed deck cleaning is performed about once a week and takes about one hour. Emptying TNT boxes using the vacuum system takes about 15 minutes and is performed once per day. Personal Protective Equipment is used during the work tasks. The samplings were performed on cellulose ester membrane filters. The filters were analysed specifically for bromine using an X-ray fluorescence technique.

The studies, designed as worst case monitoring, consisted mainly of stationary 4 to <14 hour TWA measurements at the plants´ feed deck. At one plant personal monitoring was carried out specifically during cleaning of the feed deck.

During use of HBCDD powder in a plant having no effective engineering controls or LEV in place the measured exposure was between 0.09 and 1.6 mg/m^3.

In total 43 samples of inhalable dust were collected across the XPS foam manufacturing industry. Most of the samples were taken at fixed locations representing typical operator positions. Percentile calculations revealed that 95 % of the data were below 0.47 mg/m^3 and 90 % did not exceed 0.43 mg/m^3. 51 % of all results were at the limit of detection, which ranged from 0.004 to 0.02 mg/m^3. The results of these measurements are summarised in .Table 4-2

Table 4-2 Summary of the results of the measurements in seven plants producing XPS.

PLANT	Number of samples	FORM OF USE:Powder	FORM OF USE:Powder	FORM OF USE:Granules	Reference
		Total inhalable HBCDD(mg/m3)	Respirable HBCDD(mg/m3)	Total inhalable HBCDD(mg/m3)	
Plant 1 Stationary monitoring Sampling time: 4: 40-8.00 h: min		n.d. (<0.026) (10 samples)	n.d. (<0.026) (8 samples)	n.d.(<0.026)-0.013 (6 samples, 1 n.d.)	(Abbott, 2001)
Plant 2 Personal Exposure monitoring Sampling time: 180-440 min	9			n.d.(<0.006)-0.015 (7 samples n.d.)	(Abbott, 2001)
Plant 3 Personal Exposure monitoring Sampling time: 60 min	6			n.d.(<0.08) (6 samples n.d.)	(Abbott, 2001)
Plant 4 Stationary monitoring Sampling time: 480 min	10			n.d.(<0.02)-0.43 (6 samples n.d.) Mean: 0.3[a] 90 % -ile: 0.42[a]	(Abbott, 2001)
Plant 5 Stationary monitoring Sampling time: 480 min	6	Range: 0.09-1.6 Mean: 0.66 90 %-ile: 1.45			(Abbott, 2001)
Plant 6 Stationary monitoring Sampling time: 262-480 min	6			Range: n.d. (<0.02)-0.40 (2 samples n.d..) Mean: 0.21[a] 90 %-ile: 0.36[a]	(Abbott, 2001)
Plant 7 Stationary monitoring Sampling time: 315-1435 min	5			Range: 0.22 – 0.88 Mean: 0.6 90 %-ile: 0.95	(Abbott, 2001)

[a] n.d. samples are not included in the calculations

XPS-production using standard grade HBCDD is rare. Data has been received from 2 plants (table 4.2), showing maximal concentrations of inhalable HBCDD of 1.6 mg/m^3. Based on these limited data, and when comparing with EPS-production using standard grade

HBCDD where more data has been generated (with a RWC of 2.5 mg/m^3), a RWC concentration of 2.5 mg/m^3 is used in the RC.

Data from one plant, using **HBCDD-granules for the production of XPS**, was received in 2005 (Searl and Robertson, 2005). The plant used 1000 kg sacks of HBCDD-granules. HBCDD was detected in 4 out of 12 samples, representing logistics, extruding, and laboratory work. The calculated 90th percentile 8 hours exposure concentration was 0.04 mg/m^3 (when setting the concentration in samples where no HBCDD was detected to half the detection limit). The workers used PPE (gloves, overall, eye protection) but not respiratory protection. Based on the description of the plant, where the addition of HBCDD was conducted in an isolated room and the rest of the process was fully automated, it is not clear how representative this plant is for other XPS-producers. Data received in 2001(Table 4-2) indicate air concentrations of up to 0.4 in two plants and up to 0.88 mg/m^3 in one plant. Based on all the data and putting more emphasis on personal sampling, a RWC concentration of 0.22 mg/m^3 will be used in the RC for XPS-production using granules.

The air concentrations of respirable and inhalable HBCDD have been measured in three plants using **master batch for the production of XPS** (Searl and Robertson, 2005). Based on altogether 42 samples, secondary processing of boards and reclamation seemed to be the processes leading to the highest exposure, even though HBCDD was only detected in 3 out of 14 samples. The 90th percentile total air concentrations of HBCDD for these processes were 0.22 mg/m^3. The workers were reported to use PPE, sometimes also including disposable respirators. A RWC air concentration of 0.22 mg/m^3 will be used in the RC for XPS production using masterbatch.

The HBCDD exposure of workers at an industrial plant in Norway producing expandable polystyrene (XPS), flame retarded with HBCDD, has been analysed by measuring HBCDD concentrations in the workplace air and in the workers' serum (Thomsen *et al.*, 2007). A control group was constituted from subjects expected to have a background exposure to HBCDD.
Ten workers were either working in the so called "reactor" or "mixer" area. Ten persons from the general Norwegian population were also included in the study in order to compare the serum concentrations of HBCDD in the workers at the plant with serum concentrations found in persons expected to have a background exposure to HBCDD.
30 samples were obtained. Personal sampling equipment for total dust was used, sampling near the breathing zone of the workers throughout the eight hour shift.
Blood samples from the ten workers were collected twice; the first sampling was done between the fourth and ninth day of the production period when air particulate was sampled, and the second blood sampling was performed about six weeks after the end of the production period.
The HBCDD concentration varied from 0.24 µg/m^3 to 150 µg/m^3, with a mean of 12 µg/m^3 and a median of 2.1 µg/m^3. The concentrations found in the samples from the mixer area (mean 0.5 µg/m^3, median 0.5 µg/m^3) were significantly lower than those measured in the reactor area (mean 15 µg/m^3, median 2.7 µg/m^3), and the variability was much lower.
The mean serum concentrations were 162 ng/g lipids and 218 ng/g lipids, respectively, the medians were 92 ng/g lipids and 117 ng/g lipids. HBCDD was not detected above the limit of detection (LOD = 0.5 ng/g lipid) in any of the serum samples from the subjects in the control group. A large variation in the HBCDD serum concentrations in the workers was observed, ranging from 6 ng/g lipids to 856 ng/g lipids, with a tendency to increasing concentration with age (R2=0.39).

There was no correlation of serum HBCDD levels with average HBCDD concentrations in the corresponding particulate air samples, except that considerably lower levels were found in the serum from the two subjects working in the low contaminated mixer area, but they were also the only ones that had used personal protection equipment.

An occupational exposure study was carried out in September 1996 at one plant **producing EPS beads**. The study followed five operators, equipped with personal monitoring devices, involved in the dosing operations at the reactors including supervising HBCDD addition. The maximum TWA (8h) figures found were 0.5 mg/m^3 for respirable dust (0.5-5 μm) and 2.0 mg/m^3 for total inhalable dust. Workplace exposure measurements of organic dusts were carried out at another EPS plant in Europe: all 22 samples were less than 6 mg/m^3 (Ransbotyn, 1999).

Air concentrations of respirable and inhalable HBCDD were measured in 4 plants (totally 12 samples) using 25 kg bags of HBCDD standard grade powder for the production of EPS beads (Searl and Robertson, 2005). Measurements were only done during the addition of HBCDD to the reactor. The generation of HBCDD dust during this task was highly variable depending on how it was done. The ratio of inhalable to respirable HBCDD dust varied from 3 to 30-fold more inhalable than respirable HBCDD. The total concentrations measured during this task varied from 2.9 to 21.5 mg/m^3 (during 15-60 minutes); with mean and 90th percentile total concentrations of 7 and 10.5 mg/m^3, respectively. When these short-term measurements were recalculated into 8 hours concentrations, mean and 90th percentile total concentrations of 1 and 1.3 mg/m^3, respectively, were obtained. However, full shift measurements were also conducted, giving mean and 90th percentile total concentrations of 1.2 and 1.1 mg/m^3 for adding HBCDD to the reactor. The high mean value (in relation to the 90th percentile) is caused by one individual being exposed to a mean 8 hours concentration of 14.7 mg/m^3. Full shift measurements were also done during weighing out the HBCDD to be added to the reactor. Four samples gave mean and 90th percentile total concentrations of 7.2 and 10.5 mg/m^3 for the task of weighing. Although individuals did spend up to the whole working day doing this task, the workers are reported to do this perhaps 10-15 % of their long-term working time, because of rotation between tasks. *Most* workers were reported to use gloves, overall, eye protection, and disposable respirator. Based on the 90th percentile 8 hours concentrations of 1.3 (recalculated from short term measurements) and 1 mg/m^3, and that occasional weighing will contribute to the long-term exposure, a RWC concentration of 2.5 mg/m^3 is used in the RC for EPS-production using standard grade HBCDD. The typical exposure is assumed to be half this value.

Measured data for the use of **fine powder HBCDD in textile backcoating** has been produced for one plant, claimed to be representative for the textile backcoaters (Searl and Robertson, 2005). Fourteen samples were analysed, with 6 samples representing laboratory work, 4 backcoating when HBCDD was actively used, and 4 backcoating when HBCDD was not used in the process. Total (respirable and inhalable) HBCDD 90th percentile concentrations in the laboratory and during backcoating when no HBCDD was used, were 0.5 and 1 mg/m^3, respectively. The latter value indicates a general contamination of the facility, and thus a rather poor hygienic standard. During backcoating with an active use of HBCDD, the total mean and 90th percentile air concentration were 1.4 and 3.1 mg/m^3, respectively (range of 4 samples 0.4-4.3 mg/m^3). Workers were reported to use respiratory protection while handling powder, but not otherwise. Likewise, gloves and eye protection were not used, and several workers were reported to be smeared with HBCDD both in face and hair. The value of 3.1

mg/m^3 will be used in the RC as a RWC concentration. The typical exposure is assumed to be half this value.

The data from workplace air-measurements of airborne dust at industrial uses of HBCDD as an additive is summarised in Table 4-3.

Table 4-3 Workplace air-measurements of airborne dust at industrial uses of HBCDD as an additive.

Measuring point **Place position**	**Substance**	**Number of samples** **(time/ sample, min)**	**Concentration** **(mg/m^3)**	**Reference**
Handling at suspension polymerisation	Airborne dust		Mean: 1.6	(Padberg and Schaaf, 1996)
Production of XPS-masterbatch	Total HBCDD (inhalable)	10 (480) (240)	Mean: 0.88 90 %-ile: 1.4 90 %-ile: 5.4	(Searl and Robertson, 2005)
XPS manufacturing	Inhalable dust	16 (480 min) 43 (60 - 1435 min)	Powder Range: 0.24 - 1.6[a] Mean: 0.66[a] 90 %-ile: 1.45[a] (10 samples n.d.) Granules Range: 0.005-0.9[a] Mean: 0.24[a] 90 % -ile: 0.47[a] (16 samplings n.d.)	(Abbot, 2001).
	Total HBCDD	8 (480)	Granules; or powder Mean: 0.03 90 % -ile: 0.04	(Searl and Robertson, 2005)
	Total HBCDD	8 (480)	Masterbatch Mean: 0,08 90 % -ile: 0,22	(Searl and Robertson, 2005)
EPS beads manufacturing	Respirable dust Inhalable dust		<0.5 2.0	(Ransbotyn, 1999)
	Organic dust	22	n.d. <6	(Ransbotyn, 1999)

Measuring point Place position	Substance	Number of samples (time/ sample, min)	Concentration (mg/m³)	Reference
	Total HBCDD	18 (480) 4 (480)	Adding (standard grade); Mean: 1.2 90 % -ile: 1.1 Weighing Mean: 7.2 90 % -ile: 10.6	(Searl and Robertson, 2005)
Textile backcoating	Total HBCDD	4 (480)	Powder: Mean: 1.35 90 %-ile: 3.1	(Searl and Robertson, 2005)

a: n.d. samples are not included in the calculation

The information on the handling and the hygiene at the sites using HBCDD as an additive is limited. It is assumed that the levels of exposure at the user sites may be higher than at the producer sites.

Inhalation exposure is assessed by EASE. The reasonable worst-case inhalatory dust exposure during the use of HBCDD is estimated to be 2-5 mg/m^3 for the loading of fine and standard grades HBCDD (see Appendix 2, EASE-modelling). At loading of granules, the exposure is estimated to be 0.2-0.5 mg/m^3, because of 10 % of the particles being <100µm.

Dermal exposure data

There are most likely differences between dermal exposure in the different sub-scenarios, as based on the descriptions by Searl and Robertson (Searl and Robertson, 2005) on degree of dusting and workers habits. Dermal exposure is assessed by EASE. One modelling is done for use of fine and standard grades HBCDD, thus applying both for XPS/EPS-production and textile backcoating. It is assumed that exposure at the use of HBCDD when making products containing HBCDD occurs principally during manual adding of the substance to the process. The reasonable worst-case dermal exposure level (potential exposure) during the use of fine and standard grade HBCDD is estimated to be 0-0.1 mg/cm^2/day (see Appendix 2, Ease-modelling). It is assumed that both hands are exposed in XPS/EPS-production, whereas hands and face are assumed to be exposed in textile coating. This corresponds to exposed areas of 840 and 1200 cm^2, respectively. This leads to an exposure to 0-84 mg/day in XPS/EPS-production and 0-120 mg/day in textile coating. For granules, the exposure is thought to be 10 % of that with powder, because of less dusting, i.e., 8.4 mg/day.

Conclusions - Industrial use of HBCDD as an additive

Inhalatory exposure

For HBCDD charging to a process, reasonable worst-case exposure levels for fine grade and standard grade HBCDD according to the EASE modelling is 2-5 mg/m^3, and the measured data support this range. Although the recent survey on air concentrations of HBCDD by Searl

and Robertson (Searl and Robertson, 2005) is based on rather few samples, the data seems reliable and will therefore be used in the RC to the extent possible. The sub scenarios XPS-production, EPS-production, and textile back-coating will be used, and the values to be used for the risk characterisation are 2.5 (standard grade), and 3.1 (fine grade) mg/m^3 (TWA), respectively. At loading of granules or use of masterbatch (XPS and EPS production), the exposure is estimated to be 0.22 mg/m^3.
At a typical working day, the worker is assumed not to work in direct contact with the substance for 8 hour. However, in the data by Searl and Robertson (Searl and Robertson, 2005), this is considered by the recalculation of measured data into 8 hours mean air concentrations. On the other hand, workers are reported to rotate and perform different tasks on different days, some most likely leading to lower exposure, and the typical level of long-term exposure via inhalation can therefore be assumed to be about half of the values reported above.
Exposure during the industrial use of HBCDD may be full shift, which the values above represent, but short term exposure may be higher. It is assumed, and supported by the measured data, that this may be twice as high as the reasonable worst case values above and in some cases even four-fold higher than the RWC.

Dermal exposure

There are no measured dermal exposure data, and EASE-modelling has been performed. The reasonable worst-case dermal exposure level (potential exposure) during use of fine and standard grade HBCDD in XPS/EPS-production and textile coating is 0.1 mg/cm^2/day. For XPS/EPS-production, it is assumed that both hands are exposed. For textile coating, exposure of hands and the face is assumed. There is no default area for the face so it is assumed that the area for the front of the face is slightly less than the total skin area of one hand. This corresponds to exposed areas of 840 and 1200 cm^2, which leads to an exposure to 84 and 120 mg/day for XPS/EPS-production and textile coating, respectively. For granules, the exposure is thought to be 10 % of that with powder, because of less dusting, i.e., 8.4 mg/day.
At a typical working day (or working week), the worker is assumed not to work in direct contact with the substance all the time and the extent of dermal contact with the substance can be limited because of careful handling of sacks etcetera.

4.1.1.2.4 Occupational exposure during industrial end-use of semi- and end-products containing HBCDD

Background
Workers in the industry using HBCDD-containing semi- or end-products are potentially exposed to HBCDD.
Examples of industrial uses of semi- and end-products containing HBCDD are processing of polystyrene boards (XPS and EPS), e.g. cutting, gluing and heating and the manufacturing of upholstered furniture and sewing of textiles

The release of HBCDD from products containing HBCDD is depending on

- the concentration of HBCDD in the material.
- the mobility of HBCDD in the matrix.

- the relative surface area of the product. The relative surface area depends on the conformation of the matrix and the use of the product.
- physical conditions of the surrounding media.

Dermal exposure to HBCDD may occur during e.g. sewing textiles with latex back-coating containing HBCDD. Due to the low vapour pressure the release to air from products is assumed to be relatively low at room temperature, but the release may be larger because of a temporary rise in temperature or because of weathering and abrasion.
Release from a product to surrounding water is a possible route of dermal exposure e.g. during washing products containing HBCDD.

The exposure at workplaces handling products and semi-products are probably much lower than the exposure at the handling of the pure substance.

At workplaces using compound or masterbatch of e.g. polystyrene with relative high concentrations of HBCDD (40 %), the workers may be exposed to HBCDD. The exposure is probably lower than the exposure at workplaces handling the pure substance. The release of dust and vapour containing HBCDD is probably relatively small. Polystyrene flame-retarded with HBCDD (EPS-F, XPS-F and HIPS-F) contains HBCDD in concentrations of 0.5-5 %. The exposure to HBCDD during dust generating handling of these materials will therefore be low.

One scenario for occupational exposure at industrial end-use of HBCDD is chosen in this assessment. It is the sewing of flame-retarded textiles containing HBCDD. This is assumed to be a reasonable worst-case scenario.

Scenario SEWING (Occupational). Industrial end-use; Textile worker

No measured data on inhalation exposure or dermal exposure during the industrial end use of products containing HBCDD has been provided. A possible scenario for a professional textile worker sewing flame-retarded textiles for e.g. furniture for public rooms can be designed as follows;

> A textile worker is sewing flame retarded textiles with backcoating containing 25 vol.-% HBCDD, 8 hours/day. The body weight of the exposed individual is 60 kg and the ventilation rate 10 m^3/day. Many textile workers are women and therefore 60 kg is chosen as a realistic body weight.

Inhalation exposure

A concentration of 5 mg/m^3 airborne textile dust is assumed to be realistic at this work environment. If the concentration of HBCDD in the material and in the airborne dust is 10 % (most airborne dust probably comes from the textile fraction of the material and less from the backcoating, resulting in a lower content of HBCDD in the airborne dust than in the back-coating), the air concentration will be 0.5 mg/m^3. HBCDD in vapour form is assumed to be negligible.

Dermal exposure

A dermal exposure to 1 mg/cm^2/day on 840 cm^2 (both hands) of textile dust is assumed to be a possible estimate at this work environment. The dust probably comes from the textile

fraction of the material and less from the backcoating, and a concentration of HBCDD in the dust of 10 % is therefore assumed. The total dust exposure is 840 mg/day, resulting in a total exposure to 84 mg HBCDD/day.

4.1.1.2.5 Summary; Occupational exposure

Table 4-4 The identified reasonable worst-case exposures for occupational exposure to HBCDD, representing three scenarios (typical exposures within brackets).

Scenario	Product grade	Inhalation		Dermal[c]			Multiple routes exposure
		mg/m^3	mg/kg/ day	mg/day	dermal abs (%)	mg/kg/ day	mg/kg/day
FILLING. Filling of bags at the production of HBCDD[a]	Fine powder (via micronisation)	10 (5)	1.42	4200	4	2.4	3.82
	Powder	1.9 (0.95)	0.27	840 (	4	0.49	0.76
	Granules	0.19 (0.1)	0.03	84	2	0.02	0.05
ADDING. Industrial use of HBCDD as an additive[a]	Formulation of textile. Fine powder	3.1 (1.55)	0.44	120	4	0.07	0.51
	Formulation of polystyrene (EPS, XPS and HIPS), standard grade powder	2.5 (1.25)	0.36	84	4	0.05	0.41
	Formulation of polystyrene (EPS, XPS and HIPS), granules or masterbatch	0.22 (0.11)	0.031	8.4	2	0.002	0.033
SEWING (Occupational). Industrial end-use[b]		0.5 (0.25)	0.08	84	4	0.06	0.14

[a]In calculating the internal exposure, a body weight of 70 kg and an inhalation volume of 10 m3/working day is assumed.

[b]A body weight of 60 kg and an inhalation volume of 10 m^3/working day is assumed.

[c]An exposed area of 840 cm^2 is assumed (both hands), except for textile backcoating where an area of 1200 cm^2 is used based on descriptions of exposure also of the face.

Other occupational exposure scenarios may be at building sites handling insulation boards containing HBCDD or other materials containing HBCDD. These scenarios probably result in much lower exposure levels than the scenarios arising during direct handling of the pure substance. Common for all scenarios, is that they may be significantly higher during shorter periods. The exposed populations of workers may differ between the scenarios, as the distribution between men and women can be different for workers in the plants producing HBCDD and at sites sewing textiles.

4.1.1.3 Consumer exposure

HBCDD is used in several products, some of which are available to consumers.
Some examples of end-products containing HBCDD are:

- flat and pile upholstered furniture (residential and commercial furniture)
- upholstery seatings in transportation, draperies, and wall coverings
- bed mattress ticking
- interior textiles e.g. roller blinds
- automobile interior textiles
- car cushions
- 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)
- parking decks
- insulation boards against frost heaves of road and railway embankments
- packaging material
- electrical and electronic equipment e.g. distribution boxes for electrical lines
- video cassette housings
- polyvinyl chloride wire, cable and textile coating
- protecting paints for military purposes

Other exposure can occur due to misuse of products containing HBCDD e.g. when children play with articles containing HBCDD not intended to play with.
Any foreseeable misuses of HBCDD have not been identified.

There are some useful data on the potential exposure to HBCDD from consumer products.

There is no known direct private use of HBCDD as the substance as such. However articles containing HBCDD are sold to consumers. The general uses of end-products containing HBCDD can be found in Table 2-2. End-products used by consumers are e.g. packing material, insulation boards, electric housing, and textile backcoating in e.g. home furniture and automobile interior and children car seats and videocassettes. The concentrations of HBCDD in various products are given in Chapter 2.

The flame retardant is physically bound within the polymer matrix; however it is not chemically bound and may therefore migrate out of the matrix. Therefore, release of HBCDD from the surface of the product and to atmosphere from plastic products may be a potential way of exposure.

Measured data for the assessment of consumers' exposure of HBCDD from backcoated fabric were submitted by the industry.

There is no obvious way of separating articles containing HBCDD from similar products without HBCDD. Therefore, there may be a risk for misuse of articles containing HBCDD in a way that HBCDD comes in contact with food. However, this risk is assumed to be relatively small.

The release of HBCDD from products containing HBCDD is depending on (1) the concentration of HBCDD in the product (2) the mobility of HBCDD in the matrix, (3) the relative surface area of the product and (4) the physical conditions of the surrounding media. The mobility of HBCDD in latex in e.g. textile coatings is assumed to be greater than the mobility in XPS and EPS and the mobility of HBCDD in HIPS is assumed to be greater than in XPS and EPS. The relative surface area depends on the conformation of the matrix and the use of the product. The concentration of HBCDD in textile latex coating is assumed to be about 25 % (however, lower when expressed as % of weight of the whole textile), in HIPS 3 % and in XPS and EPS about 1 %. We assume that latex coating on textile results in greatest relative release of HBCDD from products. Due to the low vapour pressure the release to air from products is assumed to be relatively low.
Direct dermal contact with products containing HBCDD may give dermal exposure.

A consumer exposure assessment of HBCDD was made in Toxicological Risks of Selected Flame-Retardant Chemicals (National Research Council, 2000). It encompassed

(1) dermal exposure assuming exposure from furniture upholstery backcoated with HBCDD, estimated to be 1.3×10^{-6} mg/kg/day,
(2) inhalation exposure in a room caused by wear of and evaporation of HBCDD from fabric upholstery treated with HBCDD, estimated to give a total air concentration of 3.9 μg/m^3 HBCDD,
(3) oral exposure of children to HBCDD by sucking a fabric (50 cm^2) backcoated with HBCDD daily for 2 years, 1 hr/day, estimated to be 26 μg/kg/day HBCDD.

Three scenarios for consumer's exposure to HBCDD are presented here;

- **TEXTILE IN FURNITURE (AND CURTAINS)**
  Subscenario. Oral exposure to dust
  Subscenario. Inhalation exposure
  Subscenario. Oral exposure by mouthing of textile
- **INDOOR AIR**. Exposure from XPS construction boards
- **MATTRESS TICKING**. Lying down in a bed on a mattress with flame-retarded ticking.

These scenarios may give an exposure. The information on the release of HBCDD from the materials and on the actual use of the materials giving support for calculations on the exposure is very limited.

Scenario **TEXTILE IN FURNITURE (AND CURTAINS)**

The Polymer Research Centre at the University of Surrey and the Bolton Institute undertook studies to determine the release of flame-retardants from backcoated textiles (Thomas and Stevens, 2006). The effects of ageing and wear on the potential release were investigated. Where possible, accepted standard methodologies were used for testing. Ageing was simulated by exposure to higher temperatures, alone or in combination with increased humidity, exposure to UV-A light or by a physical process, the latter being a modified standard method for testing abrasion resistance of fabrics. The wear was simulated by a "Martindale abrasion test" according to ISO 12947-1:1999; this test is commonly used in the textile industry to simulate abrasion of textiles over their product life. The release was measured both as formation of debris (and size of the particles/fibres in the debris) and as

volatile release. The ageing and wear simulations were designed to simulate the entire lifetime of the textile. The extraction of HBCDD from fabric by simulating oral suckling and aqueous phase dermal exposure was also investigated.
The tests were performed on one single cotton fabric with 7.7 wt-% HBCDD, but the textile is not further described in the report. The coating was applied at a rate of 271 g/m^2, giving an area density of HBCDD in the final coating of around 1.98 mg/cm^2. It is assumed to be a representative cotton fabric, but the lack of information on the textile makes it difficult to evaluate the representativity of both the textile and the results, as abrasion depends a lot on the structure/construction of the textile. Fabric only based on cotton is commonly used in furniture fabric, but blends of cotton and e.g. wool and synthetic polymers are also common. According to the study director, the choice of cotton would possibly be a worst-case choice, as other materials wear at slower rates.

It is an extensive study, which has generated a huge amount of data. However, the methods used in the study are poorly described and the calculations are often difficult to follow. Therefore, it is difficult to evaluate the results and how representative they are for real world conditions.
In the study, the samples were subject to various standard accelerated ageing protocols as follows:

e) control conditions (20°C and 65% relative humidity for either 18 or 36 weeks),
f) environmental ageing (30°C at 65% relative humidity for 20 weeks or 60°C at 75% relative humidity for 2, 8, 14 or 20 weeks),
g) thermal ageing (60°C, 70°C[5] or 90°C all at ambient relative humidity for up to 10 weeks (60 and 70°C) or 4 weeks (90°C) and
h) UV-ageing (exposed to 340 nm at room temperature for 5, 10, 20 or 30 days).

Following ageing under the various conditions, the samples were subject to various wear and leaching tests. Volatile emissions to the head space were also determined during the thermal ageing tests. No HBCDD could be detected in these samples indicating minimal loss by volatilisation during the ageing process.
To determine particulate and volatile release during wear of the textile, an adaptation of the Martindale abrasion test using an enclosed system was used. The test equipment was modified to allow settled debris and particulates to be collected and the concentrations of volatiles and airborne particulates to be determined. Using this equipment, the mass balance was found to be over 99.5% over a period of up 30,000 wear cycles (this was taken to be representative of a full service-life wear history).

The tested fabric exhibit a low wear rate for all ageing conditions with the exception of UV aged (wavelength 340 nm) materials that wear with increased ageing. The UV exposure is thought to be representative for the indoor environment as UV-A goes through glass windows, and it is well known that cotton and wool fibres are weakened by sunlight.

The debris produced in the fabric wear test contains a large number of short and long cotton fibres from the fabric. In addition, particulates are present in the debris with the largest quantity (both number and weight of particles) in the 10-90 μm size ranges with a low quantity by weight of smaller particles. Some are aggregates from the backcoating material and these consist of submicron particles in many cases.

[5] This temperature appears as 75°C in places in the report.

Flame-retardant compounds were found to be heterogeneously distributed throughout_the debris which is consistent with the fact that the deposition of the backcoating on the fabric is non-continuous and the distribution of flame-retardant compounds within the fabric is non-uniform.
UV-aged fabric produced greater amounts of debris during wear testing than the non-aged fabric, which seems reasonable. No HBCDD were detected as volatiles. After the maximum UV-aging and number of wear cycles, the report says that debris contained 0.47 % HBCDD by debris weight, which would represent a maximum of 44 or 84 mg of HBCDD per m^2 of fabric surface area released through wear of the unaged or UV-aged samples. However, the calculation of the concentration of HBCDD in the debris in the report is not easy to follow, and there are different data on the amount of debris being produced. Breakthrough of flame-retardant from the backcoating to the front face of fabrics occurs and appears to be related to particle transport from the backcoating to the front face.
Airborne particle analysis from the fabric wear tests confirm that particle emission in the size range 30 nm to 6.5 µm occurs with a particle size distribution and concentration which varies with ageing time and cycle number. This size is within the respirable range. It is notable that particles of nano-size is being generated, and that there are some uncertainty as to if they exert the same type of toxicity as larger particles. No chemical analyses of the particles were made. The UV aged materials produced the lowest concentration of airborne particles, but more debris, suggesting that more extensive wearing produces larger particles in contrast to the finer particles produced from new textiles.
Volatile emissions during the thermal ageing of fabrics up to 90 ºC and from the collection of volatiles from fabric wear testing shows that no HBCDD could be detected above that of the analytical detection limit. Still, minute and unquantifiable emissions of volatiles may occur.
Extraction of the unaged fabrics, to determine the total amount of HBCDD potentially available for recovery, showed a recovery of 93±5 % of the load level. The corresponding values after thermal or UV-aging was 72.6 % or 74.6 %, respectively, indicating that 20 % of the HBCDD could not be recovered after aging. Thus, thermal or UV-aging resulted in similar reductions in total extractability. These reductions could be due to an actual reduction in the fabric content of HBCDD or a reduction in the extraction efficiency due to an increase in the binding of the flame-retardant to the fabric or other processes. The reductions could also be related to, e.g., debromination of the flame retardant in the textile, and that the modified (debrominated) substance is not identified in the chemical analyses as mentioned. A very small follow up study by Industry indicates that when measuring the bromine content of the textile before and after aging, the reduction was less than reported in this study (personal comm. BSEF), supporting that release of HBCDD is not the only reason to the observed reduced amount of HBCDD in the textile after aging.
The test for simulating skin contact, where a moist filter paper was pressed to the textile, showed no detectable HBCDD. The method developed by the US Consumer Products Safety Commission for testing upholstered furniture was used (Ref: upholstered furniture; 1994; 59, 30735-30738). However, this is not to be expected because HBCDD has low water solubility. A similar test with a filter-paper covered with some lipid layer, corresponding to the dermal fat, might have given other results. In contrast, in the test to simulate oral sucking of fabric, up to 2.75 % HBCDD was extracted from either the nonaged, thermally aged or UV-aged fabric. This test has been used to assess release of phthalates from toys, but it is difficult to assess the relevance of this test for textiles.

Overall, the most important information from this study is that debris that contains HBCDD is being formed, and that the concentration of HBCDD in the debris is at least 0.47 %. The amount of dust generated under conditions said to mimic life-time wear (UV-light and

mechanical wearing) was 0.86 % of the weight of the textile (2.3 g dust/m^2). A concentration of 0.47 % will be used in the RC for a scenario where children are exposed orally to the dust. Particles of different sizes (from nano-size and upwards) are being generated, but no volatile HBCDD. After a simulated sucking on the textile, up to 2.75 % of the HBCDD content was lost. The amount of dust generated under conditions said to mimic life-time wear (UV-light and mechanical wearing) was 0.86 % of the weight of the textile (2.3 g dust/m^2).

SUBSCENARIO. ORAL EXPOSURE TO DUST

If assuming a 4 m^2 textile area (2 sofas in a room), 9.2 g dust would be generated during a life-time assumed to be 10 years (3650 days). If assuming a continuous emission, the daily amount of dust available for oral exposure (pica behaviour) would be 2.5 mg/day. The report says that the content of HBCDD in the dust was 0.47 %, leading to an oral exposure to 12 µg HBCDD/day. If a 10 kg child is eating all dust generated from the sofas, the daily exposure would become 1.2 µg/kg/day, which is considered insignificant and therefore not brought forward to the risk characterisation.

Subscenario. Inhalation exposure

No HBCDD was found in the gas phase, but dust containing HBCDD was released. A small amount of the dust that was being generated was found to have a size in the respirable size region, and some particles were even of nano-size. Particle release rates from 3×10^4 to 5×10^6 particles/cm^3/cycle were observed. However, there is no information on the total weight of these particles or on their content of HBCDD. As there is no information that can be used as such, there is a need for simplifications and assumptions when estimating the exposure to HBCDD via inhalation of dust generated from textiles.

A scenario has been constructed where all dust released from the textiles are assumed to be airborne and inhalable. The study reports on emissions of 84 mg HBCDD/m^2 textile in dust from the wear of the textiles. The value is thought to represent emissions during a lifetime of 10 years. As more HBCDD can be assumed to be present in particulates (mainly coming from the backcoating) than in fibres (from the cotton), and 84 mg HBCDD/m^2 represent the HBCDD concentration in debris mainly made up of fibres, it is possible that the concentration of 84 mg HBCDD/m^2 underestimates the concentration in inhalable dust. It should be noted that rather high particle release rates were observed, but the HBCDD content was not measured in the particles. It has been assumed that a room with a volume of 60 m^3 contains 4 m^2 of textiles, e.g., two sofas or one set of curtains. The resulting release (per hour) will be as calculated below.

$$R = \frac{84 \times 4}{10 \times 365 \times 24} = 3.8 \cdot 10^{-3}\, mg/h$$

The concentration of HBCDD in airborne dust in a room with a volume of V = 60 m^3 can be calculated. The ventilation (air changes) rate is a = 0.35/h×24 h air, whereas setting and removal is assumed to be insignificant.

$$C_{indoors} = \frac{R}{V \cdot (a)} = \frac{3.8 \cdot 10^{-3} \times 24}{60 \cdot 0.35} = 4.4 \cdot 10^{-3}\, mg/m^3$$

The exposure of an adult being in the room 24 hours with an inhalation rate of 20 m^3/day will be 88 µg/day. If the body weight is 60 kg and the absorption is 100 %, the internal exposure will be 1.5 µg/kg bwt/day. Considering the low exposure value and the rather unrealistic construction of this scenario, with assumingly only a part of this dust amount being in the respirable size, this scenario is not brought forward to the risk characterisation.

ORAL EXPOSURE BY MOUTHING OF TEXTILE

Emission of HBCDD from textiles has been measured using extraction with surrogate biological aqueous media, using the same set-up as used for studying leaching of phthalates from toys. According to the report, 0.2-2.75 % of original HBCDD content could be released by, e.g., artificial saliva and citric acid (mean of 3 samples). However, the variation was big, both between samples and in between the different media used. The technique involved immersion of the whole textile (both sides) for 1.5 hours in the media, and subsequent analyses of HBCDD in the media.

The mouthing time is based on a thorough study by the US CPSC (Kiss, 2001) where trained personnel studied the mouthing behaviour of 169 children (age 3-36 months) for a total time of 4 hours/child, during two different days. The average total mouthing time was 70 minutes/day for children <1 year, and the mouthing time decreased slowly with age. The mouthing time was divided between different items roughly as follows; 30 % of the time mouthing on pacifiers, 25 % on the child's body, 20 % on plastic toys, and 25 % on other items (representing hard plastic tableware, furniture, clothing and miscellaneous items). Thus, in average, mouthing on items in this last category (including furniture) lasted for 17 minutes per day. Although no 95th percentile values are reported, a RWC mouthing time of 30 minutes per day is assumed for this assessment.

Considering that the extraction time of the textile was 90 minutes, and assuming a linear leakage with time, the extracted amount (2.75 % per 90 minutes) is divided with 3 to represent the potential extraction during 30 minutes mouthing (0.9 %). It is also acknowledged that a daily 30 minutes mouthing on furniture is not that likely, and for the sake of this assessment, one mouthing every three days will be assumed.

Another aspect relating to the extraction time chosen, is that it is likely that leaching will decrease with time, both during the experiment above (the surface content will be released much faster than substance being deeper inside the back-coating), and, if assuming that a child mouth the same area of the textile many times, in between mouthing episodes. Therefore, a shorter extraction period could possibly have given a higher release rate, as the value reported is the mean release during 1.5 hours. On the other hand, if a child mouths the same piece (area) of fabric each time, the value above would most likely overestimate the true exposure.
With regard to the immersion of *both* sides of the textile (thus, including the back-coating), the set-up will only mimic situations where both sides of the fabric are available for mouthing.

Whether the release also includes particles has not been studied, but if it does, the absorption of HBCDD from those particles could be expected to be lower than from dissolved HBCDD. As it is likely that some release could be related to particles, and we assume 100 % absorption of ingested HBCDD, this assumption will lead to some overestimation of the exposure. On the other hand, if simultaneous chewing would occur, the release could possibly be higher than during a passive extraction.

Considering the factors discussed above, and acknowledging that daily mouthing is a conservative assumption, especially when assuming mouthing to different areas of the textile every day, a release value of 0.9 % has been chosen from this study for the calculation where both sides of the textile is available for mouthing (e.g., textiles used as curtains). However, it is acknowledged that when textiles are used in, e.g., furniture, they are not likely to be mouthed on the back-coated side. There is no information on release when only the textile side is available for mouthing, but the study has shown that HBCDD can migrate from the back-coating to the textile part. It is, however, arbitrarily assumed that it may be a factor 10 lower than when both sides are available for the mouthing. Based on these assumptions, calculations are performed for when mouthing of both sides are possible and for situations where only the textile side is available for mouthing.

If assuming daily mouthing on a 50 cm^2 fabric back-coated with HBCDD (2 mg HBCDD/cm^2 = 100 mg/50 cm^2), 0.9 % release during 0.5 hours mouthing represents 0.9 mg HBCDD released per event (day). The oral absorption is assumed to be 100 %. For a child weighing 10 kg (assumed to represent a 1 year old child), the daily exposure is 90 µg/kg bwt/day HBCDD. However, considering one mouthing every three days, the average daily mouthing will be 30 µg/kg bwt/day. If the back-coated side is not available, the exposure would become 3 µg/kg/day.

Scenario **INDOOR AIR**. Exposure from XPS construction boards

The release of HBCDD from construction boards can be calculated. A not very realistic worst-case estimate is outlined below. If walls are covered with construction boards with an area of A≈50 m^2 and the boards are 7 cm thick (l), have a density of 40 kg/m^3 (ρ) and 3 weight-% HBCDD (C), the release (R) can be calculated. Two different emissions factors are used, one default from the TGD and one measured by Industry. An emission factor (E) of 0.05 % release during lifetime of the total amount HBCDD in the article is obtained from the TGD. The lifetime can be assumed to be 20 years giving a release factor of 2.5×10^{-5} per year, if the release rate is constant during the lifetime (see Chapter 2.2.3.3.). The measured emission factor is 2.4×10^{-7} per year (Mills, 2002). The resulting exposure is calculated below, using the default factor as example in the formulas.

$$R = \frac{A \cdot l \cdot \rho \cdot C \cdot E}{365} = \frac{50 \cdot 0.07 \cdot 40 \cdot 0.03 \cdot 0.000025}{365} = 0.29 mg / 24h$$

The corresponding R using the measured emission factor is 0.0028 mg/24 h.

The vapour concentration of HBCDD in a room with a volume of V = 60 m^3 with walls covered with construction boards with an area of A≈50 m^2 = 500,000 cm^2 can be calculated. The ventilation (air changes) rate is a = 0.35/h×24 h air, whereas setting and removal is assumed to be insignificant.

$$C_{indoors} = \frac{R}{V \cdot (a)} = \frac{0.29}{60 \cdot 0.35 \cdot 24} = 0.58 \mu g / m^3$$

The $C_{indoors}$ based on the measured emission factor is 5.6 ng/m^3.

This concentration is less than the calculated saturated vapour concentration at 20 °C which can be calculated to 4.2 10^{-3} mg/m^3. HBCDD can condense to aerosols or on dust particles and therefore the concentration of airborne HBCDD may be higher than the calculated saturated concentration.

The exposure of an adult being in the room 24 hours with an inhalation rate of 20 m^3/day will be 11.6 μg or 0.11 μg based on the default or the measured emission factor, respectively. If the body weight is 60 kg and the uptake is 100 %, the internal exposure will be 0.19 or 0.002 μg/kg bwt/day, based on the default (0.05 % release during lifetime) or the measured emission factor, respectively. Considering the rather unrealistic construction of this scenario (with the construction boards not covered by anything), the exposure is considered insignificant and therefore not brought forward to the risk characterisation.

Scenario; MATTRESS TICKING. Lying in a bed on a mattress with flame-retarded ticking.

The textile on mattress ticking might be flame-retarded with HBCDD. There is no information on how common this use could be. Individuals might lie on such mattress with bedding substantial time per day because of handicap or disorders of different kind. The temperature and the humidity in the bed and in the mattress ticking can be increased compared with the room, increasing the release of HBCDD from the back-coating.
A person is lying down in a bed with a mattress with a flame retarded textile cover with back-coating containing 25 weight-% HBCDD. The textile area is assumed to be = 2 m^2. The time lying in the bed is 8 hours per day, 365 days a year.
The weight of backcoating layer per m^2 (W) is 350 g/m^2. An emission factor (E) of 0.1 % release during lifetime of the total amount HBCDD in the article is used. The lifetime can be assumed to be 10 years giving a release factor of 1×10^{-4} per year, if the release rate is constant during the lifetime (see Chapter 2.2.3.3.).
The release of HBCDD

$$R = \frac{A \cdot W \cdot C \cdot E \cdot \frac{8}{24}}{365} = \frac{2 \cdot 350 \cdot 0.25 \cdot 0.0001 \cdot \frac{8}{24}}{365} = 1.6E-5 g / day$$

For the purpose of the assessment, the uptake is assumed to be 4 % of the emitted HBCDD and the body weight of the exposed individual 60 kg. This exposure is assumed to be via dermal route, with an uptake of 4 % of the external exposure. The exposure will be;
$1.6 \times 10^{-5} \times 0.04/60 = 0.01$ μg/kg bwt/day.
This exposure level is considered insignificant and therefore not brought forward to the risk characterisation.

4.1.1.3.1 Summary – Consumer exposure

For the three textile backcoating scenarios, the oral exposure of dust and the inhalation of airborne dust are considered insignificant are not brought forward to the risk characterisation.
For the scenario with a child mouthing textiles the daily exposure is assumed to be 30 µg/kg bwt/day HBCDD if the back-coated side is available. If only the textile side is available, the exposure would become 3 µg/kg/day.
For the two other consumer scenarios, indoor air and mattress ticking, the calculated exposure levels are 0.002 and 0.01 µg/kg/day, respectively, which is considered insignificant and therefore not brought forward to the risk characterisation.

These scenarios must be regarded as examples of possible consumer exposure scenarios. Other examples of scenarios might be children sitting in car chairs for children, persons handling insulation boards at home (sawing and cutting), or sewing flame-retarded textiles. These scenarios are assumed to typically result in lower exposures than the two scenarios summarised above. Common for all scenarios, is that they may be significantly higher during shorter periods. The exposed populations of consumers may differ between the scenarios, e.g., the distribution among men and women and the age of the exposed persons can be different for consumers handling building insulation at home and persons sewing textiles

4.1.1.4 Indirect exposure via the environment

HBCDD may be released to the environment through wastewater and air effluents from manufacture, formulation, industrial use, use and disposal of HBCDD containing products. These releases are described in section 3.1.1. Since HBCDD is a rather persistent and bioaccumulating substance emitted from both point sources and diffuse sources, it could be expected that the exposure to man via food is an important route of exposure.

Multiplying the concentrations in the intake media by the daily intake rates of each medium and summing the contribution of each medium will estimate the total daily intake.

Table 4-5 Daily human intake of drinking water, different foodstuff and daily inhalation rate (TGD).

Parameter	Value Adult	Unit
Daily intake of drinking water	0.002	m^3/day
Daily intake of fish	0.115	kg_{wwt}/day
Daily intake of leaf crops (incl. fruit and cereals)	1.20	kg_{wwt}/day
Daily intake of root crops	0.384	kg_{wwt}/day
Daily intake of meat	0.301	kg_{wwt}/day
Daily intake of dairy products	1.333	kg_{wwt}/day
Daily inhalation rate	20	m^3/day
Body weight	70	kg

There is considerable uncertainty in the approach the TGD is taking in estimating the concentrations of substances with high *log* K_{ow} values in the food chain.

Predicted daily intake from local emission sites are presented in the tables below.

Tabell 4-6 Predicted total daily intake via the environment resulting from production and micronising of HBCDD and the fraction of the total intake that is due to the intake of fish or crop, respectively.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/d)	Fraction of intake	
			Fish (%)	Root crops (%)
ProdB	Yes	0.00085	1.1	78
Micronising	Yes/No*	0.000043	7.6	30

*Connection to STP not known. However, no emissions to waste water.

Table 4-7 Predicted total daily intake via the environment resulting from formulation of compound for EPS and HIPS and the fraction of the total intake that is due to the intake of fish or crop, respectively.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/d)	Fraction of intake	
			Fish (%)	Root crops (%)
Site A+M	No	0.00019	73.7	6.7
Site F+N	No	0.0013	86	1.0
Site I+O	No	0.00039	48	3.4
Site B	Yes	0.00024	1.8	59
Site C	No	0.00073	74	1.8
Site D	No	0.00056	74	2.4
Site E	No	0.00025	85	5.0
Site G	Yes	0.0050	41	54
Site H	No	0.00070	93	1.8
Site J	No	0.035	99.7	0.037
Site K	Yes	0.038	42	57
Site L	Yes	0.019	41	57
Site P	No	0.0012	98.4	1.1
GEN_EPS_FORM	Yes	0.042	42	57
	No	0.087	99.7	0.015

Table 4-8 Predicted total daily intake via the environment resulting from formulation of XPS compound and the fraction of the total intake that is due to the intake of fish or crop, respectively.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/d)	Fraction of intake	
			Fish (%)	Root crops (%)
Masterb G	Yes	0.00047	21	30
Masterb H	Yes	0.00062	34.6	48.1
Masterb I	Yes	0.069	42.4	57
GEN_XPS_FORM	Yes	0.048	42	57
	No	0.10	99.7	0.014

Table 4-9 Predicted total daily intake via the environment resulting from formulation of polymer dispersion for textiles and the fraction of the total intake that is due to the intake of fish or crop, respectively.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/d)	Fraction of intake	
			Fish (%)	Root crops (%)
TexForm 1	Yes	0.00033	25	36
	No	0.00052	73	2.5
TexForm 3	Yes/No*	0.000022	15	58
TexForm 4	Yes	0.00016	24	38
	No	0.00025	71	5.2
TexForm 5	Yes/No*	0.00011	2.9	12
TexForm A	Yes/No*	0.00002	16	64
TexForm B	Yes	0.016	43	57
	No	0.033	99.9	0.040
GEN_TEX_FORM	Yes	0.18	42.8	56.9
	No	0.37	99.9	0.0036

*Connection to STP not known. However, no emissions to waste water.

Tabell 4-10 Predicted total daily intake via the environment resulting from industrial use of EPS compound at the manufacture of flame retarded EPS and the fraction of the total intake that is due to the intake of fish or crop, respectively.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/d)	Fraction of intake	
			Fish (%)	Root crops (%)
GEN_EPS_IndUse	Yes	0.001	40	55
	No	0.002	97	0.64

Tabell 4-11 Predicted total daily intake via the environment resulting from industrial use of HIPS compound at the manufacture of flame retarded HIPS and the fraction of the total intake that is due to the intake of fish or crop, respectively.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/d)	Fraction of intake	
			Fish (%)	Root crops (%)
GEN_HIPS_IndUse	Yes	0.0018	7	92
	No	0.00063	95	2

Tabell 4-12 Predicted total daily intake via the environment resulting from industrial use of EPS compound for flame retarded XPS and the fraction of the total intake that is due to the intake of fish or crop, respectively.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/d)	Fraction of intake	
			Fish (%)	Root crops (%)
XPS 1	Yes	0.048	0.04	99.6
	No	0.00013	68	9.8

XPS 2	Yes	0.0007	0.46	12.2
XPS 3	Yes	0.045	0.21	98.4
	No	0.0017	27	1.1
XPS 11	Yes	0.076	4.3	94.9
	No	0.017	95.1	0.095
GEN_XPS_IndUse	Yes	0.28	2.2	97
	No	0.031	95	0.061

Table 4-13 Predicted total daily intake via the environment resulting from industrial use of HBCDD powder for flame retarded XPS and the fraction of the total intake that is due to the intake of fish or crop, respectively.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/d)	Fraction of intake	
			Fish (%)	Root crops (%)
XPS 4	Yes	0.079	4.3	95
	No	0.017	99.1	0.078
XPS 5	Yes	0.020	0.062	99.3
	No	0.00019	25.2	7.0
XPS 6	Yes	0.018	0.021	98.9
	No	0.00034	1.6	4.2
XPS 7	Yes	0.13	0.025	99.6
	No	0.00029	50	4.5
XPS 8	Yes	0.00010	5.0	56.8
	No	0.00012	10	10.5
XPS 9	Yes/No*	0.000045	7.3	35
XPS 10	Yes	0.21	1.6	98
	No	0.016	99.6	0.078
XPS 13	Yes	0.00015	3.8	79
	No	0.000090	15.8	14.3
XPS 14	Yes	0.000059	8.1	81
XPS 16	Yes/No*	0.000032	10.2	44
XPS 17	No	0.00017	1.9	7.7
XPS 18	No	0.00018	1.8	7.5
XPS 20	Yes	0.0020	0.21	97
XPS 21	Yes	0.26	0.21	99
	No	0.0029	95	0.46
XPS 23	Yes	0.000071	46	20
XPS 24	Yes	0.000099	36	14.4
XPS 26	Yes	0.0011	1.8	68
XPS 27	Yes	0.089	2.2	97
	No	0.0096	99.6	0.13

*STP connection not known. However, no emissions to waste water.

Tabell 4-14 Predicted total daily intake via the environment resulting from industrial use of HBCDD as textile back-coating agent and the fraction of the total intake that is due to the intake of fish or crop, respectively.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/d)	Fraction of intake	
			Fish (%)	Root crops (%)
Backcoat 1	Yes	0.016	36	64
	No	0.027	99.9	0.047
Backcoat 2	Yes	0.011	12	88
	No	0.0064	99.7	0.18
Backcoat 3	Yes	2.1	17.2	82.5
	No	1.7	99.9	0.0000074
Backcoat 4	Yes	0.000049	25	67
	No	0.000063	73	20
Backcoat C	Yes	0.0036	9.6	90
	No	0.0017	99	0.76
GEN_TEX_IndUse	Yes	2.75	38.5	61.3
	No	2.0	100	0.00065

Tabell 4-15 Regional predicted total daily intake via the environment and fraction of the total intake that is due to the intake of fish or crop, respectively.

Site	Total daily intake (mg HBCDD/kg/d)	Fraction of intake	
		Fish (%)	Root crops (%)
Regional	0.0044	0.0007	99.4

According to the EUSES calculation, the largest amount of HBCDD ingested by man is estimated to come from fish and root crops. The calculation methods are simple methods for predicting indirect exposure. Owing the considerable uncertainties accompanying the methodology, they serve primarily as screening methods.

It should be kept in mind that only point source releases to the environment and diffuse source releases, i.e., from end-products, are estimated quantitatively. Waste left in the environment and disposals are described in qualitative terms. This means that the contribution to human exposure to HBCDD from some diffuse sources is not included in the estimated values. In view of the potential ability of HBCDD to be transported long range, releases from diffusive sources are likely to influence areas remote from point sources and by that exposure to humans.

4.1.1.5 Measured levels in biota and food

The first study in the EU involving HBCDD was measurements in Sweden in pike downstream of possible point sources (textile industries) (Sellström *et al.*, 1998). At two of the locations, HBCDD was found at concentrations of between 4 and 8 µg/g lipid weight (n = 5). These figures equal approximately 0.02 and 0.06 mg/kg muscle, on a wet weight basis, and would result in a daily intake in humans of up to 0.115 µg/kg bwt. HBCDD has later been measured in fish at different locations in Europe and in different kinds of water (brackish, marine and fresh). The results show a great variation, with the highest values found in eel from the Skerne-Tees River system UK (39000 µg/kg fat basis or 9.4 mg/kg wet weight). The highest concentrations were found in the samples caught closest to the Newton Aycliffe plant and the concentrations fell with distance downstream from this location. Production of HBCDD in the UK by Great Lakes ceased in December 2003, so with no further inputs from this source it is reasonable to expect that the concentration of HBCDD in fish from the Skerne-Tees River system will fall. Based on this value (as a worst case estimation), a daily intake of 0.115 kg fish and a body weight of 70 kg, this measured data indicates a daily maximum intake of 21 µg HBCDD/kg/day from fish.
Twenty four species of fresh wild fish, seven of farmed fish, seven of fresh shellfish and ten of canned or processed fish and shellfish commonly available on the UK market were sampled between 2002 and 2004 (UK, 2006). The samples that were analysed comprised composites consisting of 30 or 60 individual samples. No individual fish samples were tested in the current survey. The samples were analysed for brominated dioxins, polybrominated biphenyls and brominated flame retardants. The highest concentrations for HBCDD were found in dogfish, eel and sprats and the α-diastereomer was the most frequently occurring diastereomer. Table 4-16 contains a representative selection of measured values that are also presented in chapter 3.

Table 4-16 Measured levels of HBCDD in fish

Species	Location	Water	Measured Level µg/kg lwt , dwt or wwt	Recalculated Level to mg/kg wwt[a,b]	Reference
Mussels	The Netherlands	brackish	125-177 µg/kg dwt	37.5×10^{-3}-53.1×10^{-3}	(Bouma *et al.*, 2000)
Yellow eel	The Netherlands	brackish	<1.7-3300 µg/kg lwt	<0.34-657×10^{-3}	(de Boer *et al.*, 2002a)
Sprat	The Netherlands	brackish	65.5 µg/kg dwt	19.7×10^{-3}	(Bouma *et al.*, 2000)
Sprat	United Kingdom	unknown	3.96 µg/kg wwt	3.96×10^{-3}	(UK, 2006)
Herring	Baltic Proper, Sweden	brackish	10×10^{-3} µg/kg lwt	0.53×10^{-3}	(Nylund *et al.*, 2001)
Herring	Southern Baltic Proper, Sweden	marine	32×10^{-3} µg/kg lwt	3.94×10^{-3}	(Nylund *et al.*, 2001)
Herring	United Kingdom	unknown	2.34 µg/kg wwt	2.34×10^{-3}	(UK, 2006)
Cod, liver	Southern Norway	marine	0.3×10^{-3} -9.9×10^{-3} µg/kg wwt	0.3×10^{-6} -9.9×10^{-6}	(Schlabach *et al.*, 2002)
Yellow eel	The Netherlands	fresh	2.3-110 µg/kg wwt	2.3×10^{-3}-110×10^{-3}	(de Boer *et al.*, 2002a)
Eel	Sweden	fresh	65-1808 µg/kg lwt	12.9×10^{-3}-360×10^{-3}	(Sternbeck *et al.*, 2001)

Species	Location	Water	Measured Level µg/kg lwt , dwt or wwt	Recalculated Level to mg/kg wwt[a,b]	Reference
Eel	Skerne-Tees River System, UK	fresh	204-39×10^3 µg/kg lwt (range)	0.04-9.4[3]	(Anonymous, 2004)
Eel	United Kingdom	unknown	5.29 µg/kg wwt	5.29×10^{-3}	(UK, 2006)
Salmon	Sweden	fresh	7-490 µg/kg lwt	0.67×10^{-3}-46.8×10^{-3}	(Peltola, 2002)
Trout	Skerne-Tees River System, UK	fresh	309-161×10^3 µg/kg lwt (range)	0.003-7[c]	(Anonymous, 2004)
Dogfish	United Kingdom	unknown	2.63 µg/kg wwt	2.63×10^{-3}	(UK, 2006)
Sea bass (farmed)	United Kingdom	unknown	2.59 µg/kg wwt	2.59×10^{-3}	(UK, 2006)

a: lwt recalculated to wwt according to **Table 3-105**.

b: the dwt is assumed to be 30 % of the wwt.

c: not recalculated figures. The data are presented in the report.

Based on all EU data, a regional average concentration of HBCDD in fresh water fish has been calculated in the environmental part of this risk assessment (chapter 3) to 20 µg/kg wet weight. Based on this value a daily intake of HBCDD (0.115 kg/day and a body weight of 70 kg) from fish is approximately 33 ng/kg bwt/day, see calculation below.

$$\frac{0.115 \text{ kg/day} \times 20 \mu\text{g/kg}}{70 \text{ kg}} = 32.8 \text{ ng/kg b.w day}$$

Based on a screening study on a very limited number of food-samples bought in food-stores in Sweden, representing fish, meat, chicken, milk, and egg (Sternbeck *et al.*, 2001), and the amount of these types of food normally consumed (Table 4-5), a maximum intake of 22 ng HBCDD/kg/day was calculated. The medium value was 10-fold lower (Lind *et al.*, 2002).
The mean dietary intake of a number of brominated flame retardants by the Dutch population was estimated using analytical data of the Netherlands Institute for Fisheries Research (RIVO) and the consumption data from the third Dutch National Food Consumption Survey (DNFCS). The concentration of HBCDD was determined in 91 samples from the food categories dairy, meat, animal fat, eggs, fish and vegetable oil (De Winter-Sorkina *et al.*, 2003). The processed samples were analysed by gas chromatography with mass spectrometry.
DNFCS describes the consumption pattern of the Dutch population and includes information on the daily consumption over two consecutive days and a record of age, sex and body weight of 6250 individuals. The average dietary intake of brominated flame retardants by the Dutch population were calculated based on mean data from the database, i.e., by multiplication of the average compound concentration with the average consumption per food group. Two scenarios were calculated. In scenario 1, samples with a BFR level lower than the limit of detection (LOD), a value of 0.5×LOD was assumed. When a compound could not be detected at all in a food group, the food group was omitted for that compound. In scenario 2, the concentrations of non-detects are set to zero. The reason to perform two calculations was that for some samples RIVO reported very high detection limits.
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 food groups with the highest

contributions to the total intake of HBCDD were beef, poultry and vegetable oils and fats. The total average dietary intake of HBCDD by the Dutch population, according to scenario 1 were 2.9 ng/kg bwt/day and according to scenario 2; 1.5 ng/kg bwt/day. The difference between the results of the two scenarios indicates, not surprisingly, that the choice of detection limits affects the results.
The average concentration in EU fish could indicate en exposure to 33 ng/kg bwt/day for frequent fresh water fish consumers. The limited Swedish food basket study indicates a maximum intake of 22 ng HBCDD/kg/day, and the Dutch study indicates for a typical diet, an average exposure level of 3 ng/kg bwt/day. A typical exposure level of 3, a maximum level of 22, and a RWC level of 20 ng HBCDD/kg/day will be considered in the risk characterisation.

Table 4-17 Measured levels in food.

Sample type	HBCDD (mg/kg lwt/mg/kg wwt)	Number of samples	Reference
Mixed fish and salmon	6.7×10^{-3} - 0.051/1×10^{-3} - 4.9×10^{-3}	3 samples	(Sternbeck *et al.*, 2001), (Remberger *et al.*, 2004)
Lamb (fat)	1.4×10^{-3}/ 1.3×10^{-3}	1 sample	
Pork (fat)	1.0×10^{-3}/ 0.8×10^{-3}	1 sample	
Beef (fat)	$<1\times10^{-3}$ -4.1×10^{-3}/$<0.8\times10^{-3}$ -3.4×10^{-3}	2 samples	
Chicken (fat)	6.5×10^{-3}/ 4.1×10^{-3}	1 sample	
Milk	1.8×10^{-3}/ 0.07×10^{-3}	1 sample	
Egg yolk	9.4×10^{-3}/ 2.4×10^{-3}	1 sample	
Mothers milk	mean: 0.45 ng/g fat, max: 2.4 ng/g fat (samples below DL set to ½ DL)	33 samples	(Aune and et al., 2001)
	mean:0.45 ng/g fat, max:1.5 ng/g fat, (samples below DL set to ½ DL)	30 samples	(Lignell and et al., 2003)
potatoes	0.1µg/kg wwt	?	(Anonymous, 2006)

Based on monitoring, a regional concentration of 20 µg HBCDD/kg/day in fish has been calculated, and used as in input parameter in the EUSES modelling.
The EUSES model predicts a *regional* intake of approximately 5 µg/kg/day, with the highest contributions coming from root crop (99 %). EUSES predicts a regional concentration of 840 µg/kg in root crop, resulting in the intake of 4.6 µg/kg/day. There is few measured data to confirm the modelling in crops. In potatoes from the UK, HBCDD has been measured at a concentration of 0.1 µg/kg wwt, most likely representing a regional contamination with HBCDD. This measured concentration is three orders of magnitude lower than the regional root crop concentration estimated by EUSES. However, potatoes has a fat concentration which is 10 times lower than the fat concentration generally assumed for root crops by EUSES, making it possible that other more 'fatty' root crops, such as carrots, could have higher concentrations of HBCDD. Uptake of another lipophilic substance (a medium chain chlorinated paraffin) into carrots was recently measured, and it was concluded that EUSES overpredicts the root crop uptake of that substance also for carrots (EU RAR, MCCP). Thus, the root crop concentration of HBCDD is most likely an overestimation. If assuming a lower water solubility of HBCDD (e.g., 2 µg/l), the EUSES-predicted root crop concentration would still be two orders of magnitude higher than the measured concentration in UK potatoes. The

presence of HBCDD in vegetable oil and fats in the Dutch DNFCS-study does, however, indicate uptake into plants. In the risk characterisation, the food basket studies will be used rather than the EUSES-modelling, thus resulting in a regional intake of 20 ng/kg/day, representing both fish and vegetable oils and fats.

For the *local* scenarios, comparisons can be made between predicted and monitored concentrations in fish, involving the production sites and some textile industries. These comparisons indicate that EUSES overpredicts the fish concentrations considerably, e.g., the highest local modelled fish concentration is 700 mg/kg fish (textile backcoating) whereas the highest measured fish concentration is 9.5 mg/kg (outside a production plant). For root crops, there is no basis to validate the local EUSES-predictions, but as stated above, the root crop estimation is likely an overestimation based on comparison of regional predictions and 'regional' concentrations in potatoes. If comparing the EUSES-predicted local root crop concentrations with the 'regional' concentrations in potatoes, the local EUSES-predictions are between 26 and 10^6-fold higher than the measured potato-concentrations. For the risk characterisation, EUSES-data has to be used for the local scenarios, with some exceptions, , considering that the exposure is overestimated by at least a factor of 10. These differences in estimated versus measured concentrations in fish and root crops will be considered in the risk characterisation.
The local exposure values can be summarised as follows;

Exposure is <0.1	mg/kg/day in	Production and Formulation/industrial use of EPS, XPS, and HIPS
Exposure is 0.1-1	mg/kg/day in	Formulation in textile backcoating
Exposure is >1	mg/kg/day in	Industrial use of backcoating

4.1.1.5.1 Blood and breast milk

The National Food Administration in Sweden has analysed HBCDD and other substances in mother's milk from Swedish women in two different studies. The first study (Aune and et al., 2001), detected HBCDD in 12 of 33 samples. The samples were taken from primiparous, age 19-40, living in Uppsala County, Sweden, two weeks after delivery. The limit of detection was <15 pg HBCDD/g mothers milk (fresh weight). On a lipid weight basis, the mean and max concentrations were 0.45 and 2.4 ng/g fat, respectively.

In the second study (Lignell and et al., 2003), breast milk was sampled from 30 primiparous mothers who delivered at Uppsala University Hospital from March 2002 to February 2003. The milk was sampled during the third week after delivery (day 14-21 post partum). The limit of detection was 0.006 ng/g milk, which corresponds to 0.20-0.37 ng/g/milk fat. On a lipid basis, the mean and max concentrations were 0.42 and 1.5 ng/g fat.

In 1986, 1993 and 2001, Norwegian breast milk samples from 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). Samples collected in 1993 and 2001 in Tromsø, Hamar and Skien/Porsgrunn were pooled. From the 1986 study, only two individual samples from Tromsø were available. HBCDD was found in all samples, but at very varying levels, range 0.25-2 ng/g lipids. (Thomsen *et al.*, 2003)

A recently performed 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. Milk was collected from healthy native Swedish mothers. Equal amounts of milk from individual mothers were pooled, from years 1980, 1984/85, 1988-2002, 2003 and 2004, with each pool representing milk from 116, 102, 20, 15 and 20 mothers respectively. The average age of the mothers was 27-28 years in 1980 and 1984/85, and between 29-31 years in 1988-2004. Fourteen milk samples were taken out from 1980 to 2004 for analysis.
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. The analysis technique is not described.
(Fängström *et al.*, 2005)The study has also been presented as an extended abstract, with more information (Fängström *et al.*, 2006).

Blood and mothers milk samples from Mexico and Sweden was screened for both PBDEs and HBCDD. The Mexican samples were taken from women living in an urban environment and from indigenous rural women. Blood was donated by 5 women from San Luis Potosi City, and milk from 7 women from La Huastecha Potosina, located 300 km east of San Luis Potosi City. Swedish milk (5 individuals) was bought from the mothers' milk central at a hospital pharmacy in Stockholm. The HBCDD from the blood plasma was extracted and analysed by GC/MS. The study shows the presence of HBCDD in blood and in mothers' milk, both from Mexican and Swedish women. The mean concentration in Mexican plasma was 1.2 ng/g lipid weight (range 0.7-2.5), in Mexican milk 2.1 ng/g (range 0.8-5.4) and in Swedish milk 1.1 ng/g (range 0.3-3.2). The sample levels varied but possibly indicate somewhat higher levels in the indigenous Mexican women. The study shows that HBCDD will be transferred via the mothers' milk to the nursing child (López *et al.*, 2004).
HBCDD and PBDE levels in serum from mothers and infants from a Dutch cohort were investigated in a study, which also had the aim to establish a clean up method for HBCDD analysis in human serum. A total of 90 human serum samples were analyzed. Serum samples were obtained from the Dutch-Groningen-PCB-Infant-Cohort, and contained 8 samples from mothers at the 20^{th} week of pregnancy, 70 samples from mothers at the 35^{th} week of pregnancy and 12 cord blood samples.
HBCDD was detected in almost all samples, with concentrations up to 6.9 ng/g lwt. The HBCDD concentrations were similar to serum concentrations in Mexican and Swedish women (López *et al.*, 2004). HBCDD concentrations, on a lipid weight basis, were similar in maternal and cord blood (infant level). But if the relative fat content is considered, i.e. 0.23 % lipids in cord blood and 0.77 % in maternal serum, the total exposure to HBCDD is lower for an infant than for the mother. The mean concentration of HBCDD in cord blood was 2.4 ng/g lwt (median 0.32 and range 0.16-4.2) and in mothers serum 1.1 ng/g (median 0.72 and range 0.16-6.9) at pregnancy week 20 and 35. Concentrations of HBCDD were within the same range as PBDE congeners in both cord and maternal serum (Weiss *et al.*, 2004).

Table 4-18 Measured levels in breast milk

Country	Year	Number of samples	Concentration of HBCDD in breast milk(ng/g fat)
Sweden		12	0.45-2.4
Sweden	2003	30	0.42-1.5

Norway	1993-2001	10	0.25-2
Sweden	1980-2004	14 [a]	0.084-0.93
Sweden	2004	5	0.3-3.2
Mexico	2004	7	0.8-5.4

[a]Equal amounts of milk from individual mothers were pooled from years 1980, 1984/85, 1988-2002, 2003 and 2004, with each pool representing milk from 116, 102, 20, 15 and 20 mothers respectively

HBCDD levels in plasma from 10 pregnant women living in Bodø, Norway and from 10 women living in Taimyr, Russia were analysed by LC-MS. The samples were collected in august- December 2002. The women's ages were 20-35 and they had all giving birth to one child before. None of the locations had any known local HBCDD source. 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). (Odland *et al.*, 2005)

Blood samples were taken from 47 members of the European Parliament, representing 17 European countries, in Brussels in December 2003. The samples were weighed and dried by mixing with sodium sulphate and then extracted by Soxhlet extraction with hexane:acetone as solvent. HBCDD in blood extracts were measured by gas chromatography/mass spectrometry (GC/MS). HBCDD was detected in one individual. The concentration, 0.063 ng/g blood was too low to allow identification of the separate HBCDD diastereomers. (Brandsma *et al.*, 2004)

However, Weiss et al, 2006, have made a stereoisomeric analysis of HBCDD in human serum using LC/MS-MS. Two serum pools were analysed, each based on serum from 25 individuals (elderly women being married to fishermen). The study shows that (-) α- HBCDD was the dominating diastereomer, with only a few percents contribution from γ-HBCDD (Weiss *et al.*, 2006).

Calculation of breast-feeding intake levels

It is assumed that an infant breastfeeds for 1 year, and that this year of life is subdivided into two periods (0 to 3 months and 3 to 12 months), reflecting the changing feeding demands of the infant. It is assumed that over the first 3 months the infant has an average weight of 6 kg, that the infant ingests 0.8 kg of milk per day, that 100 % of the ingested HBCDD is absorbed and that the breast milk has a fat content of 3.5 %. From 3 to 12 months it is assumed that the infant has an average weight of 10 kg, that the infant ingests 0.5 kg of milk per day, that 100 % of the ingested HBCDD is absorbed and that the breast milk has a fat content of 3.5 % (WHO, 1998). It is also assumed that the content of HBCDD remains constant during the breast-feeding period.

Using the following equation and the assumptions, as detailed above, the average daily uptake (U_{milk}) of the breast-feeding infant is estimated for both the 0-3 month and 3-12 month periods of infant life. The resultant uptakes are then summed to generate an average uptake for the infant in mg/kg/day.

$$U_{milk} = \frac{C_{milk} \times \mathrm{B}_{\mathrm{ing}} \times IR_{milk} \times F_{milk}}{\mathrm{BW}_{\mathrm{infant}}}$$

where:
$C_{milk,}$ represents the concentration of HBCDD in mg per kg milk fat
B_{ing}, represents the bio-availability of the ingested HBCDD (100/100 = 1)
IR_{milk}, represents the ingestion rate of milk by the infant (kg/day)
$BW_{infant,}$ represents the average infant body weight over the exposure period (kg)
F_{milk} represents the percentage of fat in breast milk

HBCDD in human breast milk have been identified in four Scandinavian studies. The highest concentration measured (3.2 ng/g fat) is used for calculation of reasonable worst case.

0-3 months: $U_{milk} = \frac{3.2 \times 1 \times 0.8 \times 0.035}{6} = 0.015\ \mu g/kg/day$

3-12 months: $U_{milk} = \frac{3.2 \times\ 1 \times 0.5 \times 0.035}{10} = 0.0056\ \ \mu g/kg/day$

4.1.1.6 Combined exposure

Due to the use of HBCDD in the society and the diffuse emissions from products, humans may be exposed from different sources mentioned in Chapter 4.1.1.1. The total exposure (body burden) is the summary of all the specific exposures. The most important sources of human exposure to HBCDD are probably identified. Additions of individual scenarios are not considered to change any of the conclusions, and no calculation on combined exposure has therefore been performed.

4.1.1.7 Summary

Human exposure of different populations and subpopulations by multiple exposure routes is possible. These are workers, consumers, and humans exposed to HBCDD via the environment (via food, drinking water and air). Worker and consumer exposure are mainly via the dermal and inhalation routes, whereas exposure via the environment occurs via the oral route.
Combined exposure of different populations and subpopulations is also possible and may occur, but is not estimated.

Few measured data on the exposure to HBCDD are available and these are not considered to be entirely representative for the populations and hence are not used in this risk characterisation exclusively. Instead modelled realistic worst-case scenario information has been generated when considered relevant.

Although the most important exposure situations probably have been covered, it is recognised that not all exposed consumers may have been identified since it was not possible to obtain information on all the possible exposure situations for HBCDD in the European Union.

4.1.1.7.1 Assumptions applied for the calculation of internal exposure

The absorption for the oral and inhalation route is set to 100 %. An inhalation rate of 10 m^3/day has been used for all worker populations. Recent experimental data on the dermal absorption rate is available. The dermal uptake is assumed to be 4 % if the mean particle size <100μm. When the particle size >100 μm, the dermal absorption is set to 2 %.

4.1.2 Effects assessment - Hazards identification and Dose (concentration) - Response (effect) assessment

The studies quoted below seem to have been conducted with commercially available qualities of HBCDD.

In some of the studies,where the administration is by oral route, the animals received HBCDD suspensions in oil, i.e., with HBCDD-particles of a mean size of 142 μm (10 %< 3.6 μm, 10 % >280 μm). At this particle size only a smaller fraction of HBCDD will be dissolved. Consequently, with increasing concentrations of HBCDD the relative fraction being dissolved and absorbed is getting lower. Furthermore, there was a strong binding of HBCDD to glassware, which likely lead to contamination problems (e.g. of the control animals in the 90-day rat oral toxicity study). In case the same washing procedures was used in other studies, the same contamination problems might have occurred in those studies. These issues, particles of HBCDD in oil and absorption to glass, cause uncertainties when comparing doses and effects.

4.1.2.1 Toxicokinetics, metabolism and distribution

Information on the toxicokinetics of HBCDD is limited, and no data are available on absorption via inhalation.

Oral administration

The absorption, distribution, excretion, and metabolism of γ-HBCDD were investigated in Sprague-Dawley rats after a single oral dose of ^{14}C-γ-HBCDD diluted 1:10 with technical HBCDD (7-9 mg total HBCDD/kg in acetone:olive oil 2.1:4.1) (Yu and Atallah, 1980). As the HBCDD was first dissolved in acetone, and the resulting solution later was mixed with olive oil, one can assume that the relatively low dose of HBCDD was fully dissolved in this particular study.

The rats, 8 females and 2 males, weight 220-275 g, were grouped into five subgroups, fed *ad libitum* and sacrificed 8, 24, 48, and 72 hours (females) and 48 hours (males) after dosing. One female rat, fed olive oil only, served as a control. Urine and faeces were collected daily, blood samples during the first 24 hours from 4 animals only, and tissue samples were collected at the time of sacrifice. Unchanged HBCDD and its metabolites were analysed by liquid scintillation technique and thin-layer chromatography (TLC). The estimated absorption

half-life was 2 hours (k_a = 0.35h^{-1}) from the gastrointestinal tract and peak radioactivity in blood was reached 4 hours after administration. After 8 hours the highest activity was found in adipose tissue and muscle followed by liver, with much lower activities present in lung, kidney, blood and brain. In the gonads, 0.2 % of the administered dose was found. After 8 hours 43 % of the total administered dose was recovered in tissues, with about 20 % in fat, 14 % in muscle and 7 % in the liver. At 24 hours, 0.8 % of the administrated dose was found in the liver. At 48 hours, the radioactivity in fat and muscle was about 14 and 3 %, respectively and 0.5 % in the liver. At 72 hours the percentage of the administered dose was still about 14 % in fat but only about 2 % in muscle and 0.28 % in the liver. The kinetics of elimination followed a two-compartment open model system, and was faster in males than in females (94 % vs. 54 % of the administered dose was eliminated in faeces within 48 h, respectively). Disappearance of radioactivity was obviously slower from body fat (k = 0.03 hr^{-1}), than from the central compartment representing blood, muscle, liver and kidney (k = 0.17 hr^{-1}). For the initial period of 3 days, elimination of HBCDD and its metabolites mainly occurred in faeces (77 %) with a minor part excreted in urine (16 %). In faeces about 28 % of the radioactivity was in the form of metabolites, while the remaining 72 % was not extractable by organic solvents (ethyl acetate, not even after acid or base hydrolyses) and thus possibly constituted covalently bound metabolites. In urine, 64 % of the radioactivity was metabolites while the remaining 36 % of urinary ^{14}C was not further identified as it was not extractable from the urine. The three metabolites resolved by TLC were not identified. It should be noted that these data are associated with some uncertainty in view of that the variation between the limited number of animals used is not presented (2 animals/data point). Additionally, 93 % of the dose is reported to have been excreted within 3 days, as metabolised HBCDD, whereas the tissues are reported to contain 17 % of the given dose at the same time. However, keeping in mind that the results are only valid for γ-HBCDD, the major (but least bioaccumulating; see below) diastereomer in technical HBCDD, the results are useful, indicating a high degree of metabolism/absorption. As 93 % of the dose was excreted as 'transformed substance' (metabolites or non-extractable radioactivity), an oral absorption close to 100 % is indicated, assuming that the possible contribution of gut microflora metabolism in the generation of 'transformed substance' is low.

The concentration of HBCDD as individual α-, β- and γ- diastereomers in rodent fat tissue was determined in a 90-day oral (gavage) toxicity study (Chengelis, 2001). The rats, Crl:CD(SD)IGS BR, were divided in two satellite groups, 20 animals/sex. They were treated in an identical manner, receiving a suspension of HBCDD at dose levels of 0 and 1000mg HBCDD/kg/day for up to 90 days. The dosage volume was 5 ml/kg. The control animals received the vehicle, corn oil, only. Two animals/sex/group were euthanized on study days 2, 6, 9, 13, 20, 27 55, 89, 104 and 118 and blood and body fat (mesenteric and/or omental) were collected. The body fat was analysed for concentration of HBCDD as individual α-, β- and γ-diastereomers. The analytical methodology for the analysis was high performance liquid chromatography with mass spectrometry. The possibilities to draw quantitative conclusions from this study is somewhat hampered by the use of only two animals/sex/time point, especially during the elimination phase (see Figure 4-1 below).

The stereoisomer composition of the test article was γ >>α >β, but the concentration in adipose tissue were at all time points α >>γ >β (Table 4-19). Although the study report states that a steady state appeared to have been reached by day 27, the actual data (Figure 4-1) indicate that steady state levels roughly seemed to be achieved by study day 55 in males and perhaps by 89 days in the females. However, in both sexes the highest concentration of HBCDD was achieved in fat tissue on day 89. On day 89, the α concentration was 8-12 times higher than the concentration of β, and 6-8 times higher than the concentration of the γ stereoisomer, thus indicating a 100-fold higher relative bioaccumulation of the α-diastereomer

than of the major γ- diastereomer. The concentration of α-diastereomer was higher in females than in males at all time points (roughly 15-100 %), with mean levels of 3101 µg/g fat for males and 4342 µg/g fat for females at day 89. Although difficult to draw any conclusions on elimination based on so few time points, the data may perhaps suggest elimination half-life in the order of weeks to months for the three diastereomers, with the longest half-life for the α-diastereomer.

Figure 4-1 Mean concentration of total HBCDD in fat during an oral 90 days toxicity study, followed by a 30 days depuration period.

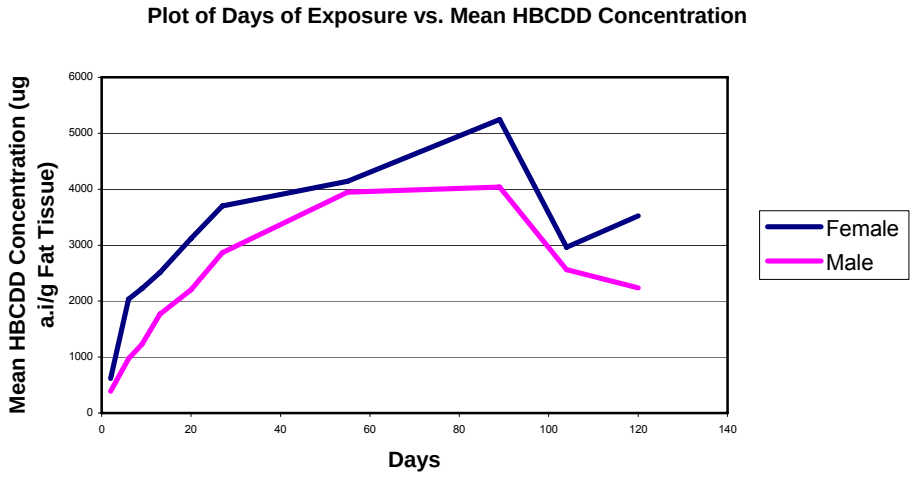


Table 4-19 Relative bioaccumulation factors (BAF) for the three HBCDD diastereomers in an oral 90 days toxicity study (a total dose of 1000 mg 'technical' HBCDD/kg/day by gavage)

	α-HBCDD	β-HBCDD	γ-HBCDD
Composition of administered dose (%)	6.4	4.5	79.1
Administered dose (mg/kg/day)	64	45	791
Concentration in females day 89 (µg/g fat)	4340	357	544
Apparent bioaccumulation factor [a]	68	7.9	0.69
Relative BAF (γ-HBCDD set to1)	99	11	1

[a] The administered dose is normally expressed as a concentration in the diet (in the same unit as the concentration in the adipose tissue is expressed), and the BAF is calculated as concentration in fat divided by the concentration in the diet. Since the dose is given by gavage in the study above, only an apparent relative bioaccumulation factor can be calculated.

HBCDD was also found in the satellite control animals, with the highest level on day 55 (a mean value of 15 µg/g fat for the 4 control animals). The most likely explanation to this contamination was poor washing procedures for the glassware and reuse of the dosing suspension containers in between the groups, as proposed after a GLP study audit.
Because HBCDD was administrated as a suspension of particles (mean size 0.14 mm) in oil, and not properly dissolved, the oral absorption is probably low. The basis for this statement is the general knowledge that surface area will affect the solubility, and a paper by Scholz *et al.*

(Scholz *et al.*, 2002)showing that particle size do mater for oral absorption. Scholz et al studied a substance (Felodipine) that is similar to HBCDD with regard to its poor water solubility. Felodipine was chosen as a typical example of a poorly water-soluble drug (1 mg/l at 37 °C). It does not ionise over the pH range in the GI tract and the variations in pH are not likely to influence its solubility. Furthermore, the high lipophilicity of felodipine (log 4.5) indicates that permeability would not likely be a limitation to its oral bioavailability. Two particle sizes, besides fully dissolved substance, were used: micronised powder with a median particle size of 8 μm; and coarse grade powder with a median particle size of 125 μm.
Three male Labradors, age 2-3 years and weighing between 30 and 35 kg were used for this study. All three animals had a chronic nipple valve fistula in the mid-jejunum.
A combination of infusion and oral administration of either normal saline or a 5 % glucose solution was used to maintain, "fasted" and establish "fed" state motility patterns of the GI tract, respectively.

The orally administrated felodipine formulations were as follows:
Solution:
Ten milligrams of felodipine fully dissolved in 2 ml ethanol 99 % (v/v). This was followed by the addition of 20 ml polysorbate 80 1.5 % (w/v) and adjustment of the volume to 198 ml using either 0.9 % saline or 5% glucose solution containing 0.8 % PEG 4000.
Suspension-micronised powder:
Ten milligrams of micronised felodipine and 20 ml of a 1.5% hydroxypropylmethylcellulose (HPMC) solution containing 0.85 PEG 4000 was added. The suspension was sonicated for approximately 1 min. This suspension was administrated promptly with either 178ml of 0.9 % saline solution or 5 % glucose solution containing 0.8 % PEG 4000.
Suspension-coarse grade powder:
After wetting the oro-gastric tube with approximately 40 ml of the 198 ml of either 0.9 % saline or 5 % glucose solution containing 0.8 % PEG 4000, the coarse grade felodipine was administrated via a funnel into the oro-gastric tube and subsequently rinsed through with the reminder of the co-administrated solution.

In each case, a total of ten milligrams of felodipine and 198 ml of accompanying fluid were administrated to feed dogs, resulting in a dose of approximately 0.3-0.35 mg/kg.

Blood samples were taken from either foreleg or neck surface veins. Samples were collected at 0, 0.25, 0.5, 1, 1.5, 2, 3, 5, 7 and 24 hours after administration. Mean C_{max} values were highest for the solution: 24.4 and 20.3 μg/l for the "fasted" and the "fed" states, respectively. Maximum concentrations after administering the micronised suspension reached 11.2 and 12.5 μg/l in the "fasted vs. fed state, while after the coarse suspension C_{max} values were much lower, around 0.5 and 1.2 μg/l, respectively. Time to peak (tmax) values commensurate with the C_{max} behaviour. Times to peak were shortest for the solution (0.5 hours fasted vs. 0.4 hours fed), somewhat longer for the micronised suspension (1.3 vs. 0.8 hours) and much longer for the suspension containing the coarse grade felodipine (3.7 vs. 4.0 hours). No significant difference in time to peak between the two dosing conditions was observed for any of the formulations. The solution was more completely absorbed than either suspension, with AUC essentially the same under both fasted and fed conditions (33.9 vs. 35.1 μg/hr/l). The relative bioavailability of the micronised suspension was about 75 % and independent of dosing conditions (24.5 vs. 25 μg/hr/l). Absorption of the coarse grade suspension was much poorer and varied with dosing conditions, about 5 % (1.7 μg/hr/l) in the fasted and just over 10 % (3.7 μg/hr/l) in the fed state.

The mean particle size in the 90 days study by (Chengelis, 2001) was 142 μm. This is comparable to the coarse grade powder with a median particle size of 125 μm used in Scholtz study. Thus, the results from the Scholtz study, indicates that the oral absorption in Chengelis study might be incomplete. The physico-chemical similarities between HBCDD and Felodipine are not sufficient to allow a strict read-across between these chemicals, but the Felodipine study indicate that the absorption of the HBCDD particles are probably low. Therefore, the LOAEL of the Chengelis study will be adjusted to a corrected LOAEL based on an expected 10-20 % oral absorption in that study.

Four male Wistar strain rats, at a body-weight of 260-300 g, were used in an oral study on the toxicodynamics of HBCDD (Arita *et al.*, 1983). The animals were kept separated in glass cages, and urine and faeces were collected daily. A fine suspension of 500 mg/kg HBCDD (Pyroguard SR-103; Daiichi Kogyo Seiyaku K.K.) in olive oil (purity and particle size of the test substance is unknown) was administered for 5 days consecutively by oral intubation. Autopsy was performed 24 hours after the final administration, and samples from spleen, pancreas, kidneys, liver, heart, and adipose tissues were collected for HBCDD analyses. The limit of detection was given as 5 μg/ml in urine and about 20 μg/ml in homogenates of the biological samples.

In this study, no urinary excretion of unchanged HBCDD was reported. The average daily rate of faecal excretion of HBCDD was 29-37 % of the administered amount in the four rats, and the cumulative faecal excretion of HBCDD was roughly 32-35 % with respect to the total administered amount. Considering the high dose (500 mg/kg) and the use of a particle suspension, the degree of absorption of HBCDD in the gastrointestinal tract can be questioned. Unchanged HBCDD was detected only in body fat with no recovery of the compound from other tissues, like liver and kidney. The study is poorly documented, and due to the fact that HBCDD may decompose under typical gas chromatographic conditions (temperatures up to 300 °C were used), the reliability of the results can be questioned. Gas chromatography can be used for analyses of HBCDD if relevant controls are used to check/compensate for possible decomposition. It is not clear whether such controls have been used in this study.

Zegers at al (2004) investigated the total-HBCDD levels in blubber of female harbour porpoises (*Phocoena phocoena*) and common dolphins (*Delphinus delphis*) stranded on Western European coasts between Scotland (UK) and Galicia in Spain by GC/MS. The isomeric composition of HBCDD in blubber was investigated by LC/MS in a selection of 19 samples, representing all sampling areas. The possible influence of cytochrome P450 mediated biotransformation on the changes in the isomer composition (α-, β- and γ-HBCD) in the residues in blubber with respect to the commercial mixture was investigated by in vitro assays with hepatic microsomes of rat and harbour seal (*Phoca vitulina*). All the 19 samples (10 harbour porpoises and 9 common dolphins) contained exclusively the α-diastereomer, despite the dominance of the γ-diastereomer in commercial mixtures. In the four replicate preparations of (phenobarbital induced) rat microsomes and an artificial 1:1:1 mixture of the three diastereomers, the peaks of the β- and γ-diastereomers of HBCDD showed a highly significant decrease, but the peak of the α-diastereomer had not become significantly smaller even after 90 minutes incubation at 37°C. In the 90-minutes incubation with harbour seal microsomes, the β- and γ-diastereomer seemed to decrease, whereas no significant disappearance of α-HBCDD was observed. For β-HBCD, three bromine-containing metabolites could be observed, while for γ-HBCDD two such metabolites were observed. Hydroxy-metabolites of both the β-diastereomer and γ-diastereomer were found.

Dermal administration

An in vitro study on human skin has been performed according to OECD Test Guideline 428 in order to assess the rate and extent of dermal absorption of HBCDD (Roper, 2005). Samples of full- thickness breast human skin were obtained from seven patients, 19-68 years old. The samples were stored in -20°C until required. After thawing, the samples were cut into smaller pieces and split-thickness membranes (200-400 µm depth) were prepared. An automated flow-through diffusion cell apparatus was used. The surface area of exposed skin within the cells (stratum corneum uppermost) was 0.64 cm^2. The receptor chamber volume was 0.25 ml, and the peristaltic pumps were adjusted to maintain a flow-rate of ca 1.5 ml/h. A physiological receptor fluid containing bovine serum albumin (ca 5 %, w/v), glucose (ca 1 %, w/v), streptomycin (ca 0.1 mg/ml) and penicillin (ca 100 units/ml) was used.

^{14}C-HBCDD was mixed in acetone by inversion until the test item had dissolved. Analysis of seven aliquots of the test item by liquid scintillation counting resulted in a coefficient of variation of 3 %. Six µl of the test preparation was applied over the stratum corneum surface of the exposed skin, and once the acetone had evaporated from the skin surface the application was repeated 4 more times until a total of 30 µl (corresponding to 640 µg ^{14}C-HBCDD) had been applied to the skin. The dosing procedure took 15 minutes to complete. Receptor fluid was collected in hourly fractions from 0-8 hrs post dose and then in 2 hourly fractions from 8-24 hrs post dose. All receptor fluid samples were mixed with scintillation fluid and analysed by liquid scintillation counting. At 8 h post dose, the exposed skin surface was washed with soap solution. The skin wash was mixed with acetone and analysed by liquid scintillation counting. The cell and skin surface was dried with tissue paper swabs. These swabs were pooled and analysed by combustion/liquid scintillation counting. The radioactivity in the aliquots was added (summed together) to determine the total radioactivity in the skin wash. At 24 h post dose, each diffusion cell was disconnected from the receptor fluid pump lines. The underside of the skin was washed (receptor rinse) with receptor fluid, which was analysed by scintillation counting. The donor and receptor chambers were also taken for analysis by liquid scintillation counting. The stratum corneum was removed with 20 successive tape strips. The first 5 tapes were pooled and analysed together. This was repeated for tapes 6-10, 11-15 and 16-20. The skin under the cell (unexposed skin) was cut away from the exposed skin with scissors. These samples were analysed by combustion/liquid scintillation counting. Since the results showed that the scintillation vials contained 85.24 % of the radioactivity of the combustion samples a conversion factor of 85.24 % was applied to the combustion samples, and the corrected total mass of ^{14}C-HBCDD applied to the skin was calculated to be 606.84 µg.

The results are based on 9 samples of skin obtained from 6 different donors (Table 4-20). It was suggested by the authors that 2 cells should be rejected: C 2 because it had a poor mass balance, 86 % (should normally be within the range 90-110% but may be considered a borderline case) and C 9 because it just failed the tritiated water barrier integrity assessment (the value was borderline to their rejection criteria) and had an amount of HBCDD in the exposed skin being more than two S.D. higher than the mean value. It is noted that other contract laboratories use slightly different rejection criteria, and based on them this sample would not have been rejected. It should also be noted that C 2 and C 9 show a higher absorption than the other samples, and that they are samples from the same individual, thereby to some extent supporting each other. After evaluating all data, both cells are considered as borderline cases. If they were included in the calculations the absorption would

increase, e.g., the amount in the exposed skin would increase with roughly 1 % from what is calculated in the study (1.34 %).

Table 4-20. Distribution of radioactivity (% applied dose) at 24 hours post dose following topical application of HBCDD to human Split-Thickness skin. C 2-C 14 are different cells, and SC denote Stratum Corneum

	C 2	C 4	C 5	C 8	C 9	C 11	C 12	C 13	C 14	Mean*	SD*
Subject	0105	0082	0067	0086	0105	0082	0067	0070	0109		
Dislodgeable dose	58.1	65.5	63.5	72.3	56.6	75.6	41.1	71.2	54.4	63.4	12.1
SC 1-5	18.1	33.4	23.5	20.8	25.7	15.1	42.1	18.6	26.5	25.7	9.31
SC 6-11	2.65	1.60	1.71	3.04	2.04	1.77	5.43	3.54	4.74	3.12	1.54
SC 12-15	1.53	1.05	0.91	1.38	4.56	1.21	1.16	1.16	3.90	1.54	1.05
SC 16-20	0.97	0.59	0.83	1.59	1.47	0.72	1.13	1.09	1.94	1.13	0.49
unexposed	0.14	0.10	0.51	0.64	0.57	0.73	0.28	0.59	0.31	0.45	0.23
Exposed skin	4.11	0.38	1.49	1.54	7.57	1.53	1.37	1.96	1.14	1.34	0.49
Receptor fluid	0.01	0.00	0.00	0.01	0.01	0.00	0.01	0.01	0.01	0.01	0.00
Receptor rinse	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Mass balance	85.6	103	92.4	101	98.5	96.6	92.6	98.1	92.9	96.7	4.25

*excluding cell C 2 and C9

Following topical application of ^{14}C-HBCDD to human split-thickness skin in vitro the cumulative absorption in the receptor fluid increased over the full 24-hrs study period. There are indications from the cumulative absorption that the substance has a considerable lag-time (4 hours). This would imply that the absorption could continue for a long time. However, due to limited "life-time" of the skin in the *in vitro* assay, it has not been possible to measure for longer time than 24 hours.

There were two steady-state fluxes observed in the study; the first from 0-6 hours and the second from 6-24 hours. Since steady state was reached, additional amount applied would not increase the absorption. The low flux rate is an argument against a high absorption figure. However, it is difficult to relate the flux rate to the proposed absorption figure, considering that the amount in the receptor fluid was less than one percent of the amount found in the exposed skin.

The solubility of HBCDD in water is low (2.1, 14.7 and 48.8 µg/l for γ-, β- and α- HBCDD, respectively). Addition of bovine serum albumin to the receptor fluid is expected to increase the solubility. The amount ^{14}C-HBCDD found in the receptor fluid is close to or below the solubility of γ-HBCDD in water and it is unclear whether solubility is a problem (limiting the transport to the receptor fluid) in this study.

A limitation of the study is that there is only one dose tested (the guideline says two), even though this dose is considered to be relevant when compared to the dermal occupational exposure values used in the RAR. It is likely that a higher dermal absorption (when expressed as percentage) would have been obtained with a lower dose.

One problem when evaluating this study is that the stratum corneum contained 31.5 % of the applied dose. The bulk of this (25.7 %) was recovered in the first 5 tape strips. Since the bulk of the stratum corneum associated material was found in the first 5 tapes strips, this indicates that the ^{14}C-HBCDD was on the surface of the skin and that there is a slow transport over the

stratum corneum. Usually, there is a case-by-case decision as to whether the amount in the stratum corneum should be included or not in the calculation of the absorption. The amount on the surface (the 5 outermost tape strips) is not relevant to include in this case. However, based on the slow transport and long lag-phase and on the fact that there is a turn-over time of the cells of the stratum corneum of perhaps one month, it is not evident how many of the tape strips (1-15) that should be included. Considering the uncertainties in the study, an expected low solubility of the substance in the aqueous receptor fluid (in spite of the addition of albumin), the fact that there is only one dose level studied (a higher percentage could be expected with a lower load of substance), and indications from the cumulative absorption that the substance has a considerable lag-time, we find it appropriate to include strips 11-20 in the absorbed dose. The total dermal absorption is estimated to 4 % (representing 1.5+1.1+1.3+0.01 %). Although respecting that 2 samples were excluded based on the rejection criteria used in that laboratory, it is noted that they are truly borderline cases and that the high absorption noted in these samples (representing the same individual) is felt to support the proposed absorption value of 4 %.

In conclusion:
Three available studies demonstrate that HBCDD can be absorbed from the gastro-intestinal tract. The highest concentrations are subsequently reached in adipose tissue and muscles followed by liver, and with much lower activities present in lung, kidney, blood, brain, and gonads. At long-term exposure, higher concentrations are achieved in females than in males, but the substance is bioaccumulating in both sexes. Of the three diastereomers constituting HBCDD, the α-form is much more accumulating than the others (the relative bioaccumulation factor is 99:11:1 for α-, β- and γ- HBCDD, respectively). The time to reach steady-state seems to be in the order of months. Γ-HBCDD can be metabolised, and three polar metabolites as well as unextractable radioactivity were detected. However, the extent of the metabolism of technical HBCDD is not known. For an initial period of 3 days post dosing, elimination of HBCDD and its metabolites mainly occurred 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 diastereomers possibly being in the order of weeks to months. For the risk characterisation, the oral and inhalation absorption is set to 100 %, whereas a value of 2 % (granules) or 4 % (powder) is used for the dermal absorption, depending on the size of the particles occurring at the exposure situation.

4.1.2.2 Acute toxicity

Dermal: In an acute dermal toxicity study 2 male and 2 female New Zealand White rabbits were used (Wilson and Leong, 1977). The hair was removed from the back (20-30 % of the body surface). HBCDD (a white powder identified as ”Firemaster 100”) was applied once at a dosage level of 20 g/kg. The area of application was bandaged and occluded. The bandages were removed 24 hours later and the backs were washed. The animals were observed daily thereafter for a total of 14 days. None of the animals died. There is no information if any non-lethal signs of toxicity appeared. Minimal lethal dose by dermal route is thus greater than 20 g/kg. The study was not performed according to current guidelines.

In a similar study, 3 male and 3 female New Zealand White rabbits were given 8 g HBCDD/kg as a single dermal application under occluded patch (Lewis and Palanker, 1978a). The test material was identified as ”GLS-S6-41A”, without further details, physiological

saline was used as a wetting agent. The observation period was 14 days. None of the animals died. There is no information on whether any non-lethal signs of toxicity appeared. The study was not performed according to current guidelines.

Oral: In an acute oral toxicity study of HBCDD, 5 male and 5 female rats (Charles River CD strain) were used (Wilson and Leong, 1977). The test material (a white powder identified as ”Firemaster 100”) was suspended in corn oil (particle size unknown) and administered orally by gavage at a dosage level of 10 g/kg in a volume of 20 ml/kg. The rats were observed for mortality and toxic signs during the first four hours after dosing, at 24 hours and then daily for 14 days. The following non-lethal toxicity signs were observed during the 14-day observation time. Females: diarrhoea in 1 of 5, hypoactivity in 1 of 5. Males: hypoactivity in 3 of 5, corneal opacity in 3 of 5 and ptosis in 3 of 5. None of the animals died. Minimum lethal dose by oral dosing is then greater than 10 g/kg. The study was similar to the OECD guideline limit test.

In a similar study, 10 Wistar rats, 5 of each sex, were given a single oral (by gavage) dose of 10 g HBCDD/kg The test material was identified as ”GLS-S6-41A”, without further details, and it was administrated as a 25 % gravimetric suspension in corn oil (particle size unknown) (Lewis and Palanker, 1978b). The animals were observed for a period of up to 14 days. No toxic or gross pathological changes were observed, although one male rat died at day 6, having fibrous tissue encasing heart and lungs. The cause of death could not be identified. The study was comparable to current guidelines.

An acute toxicity test with HBCDD was performed in CRJ:SD-male SPF rats (Ogaswara *et al.*, 1983). The test substance, identified as Pyroguard SR-103, was suspended in a 1 % CMC aqueous solution (particle size unknown) and administered by gavage on day 0, 2, 4 and 6. The test was carried out with two dosage groups (10 and 20 g/kg) and one control group (solvent only), with 10 animals/dose. The rats were observed for 14 days from the first administration for assessment of toxicity. None of the animals died. A yellow excreta was observed in both exposed groups on the day after administration but it disappeared on the 8th day. A slight, but not statistically significant, inhibition of body weight gain was observed in the both exposed groups compared to the control group. No abnormalities were observed at autopsy in the exposed groups.

In an acute toxicity study on mice, HBCDD was administered by gavage as a 30 % aqueous tragacanth suspension (doses and particle size were not reported) (EPA, 1990c). The observation period was 7 days. Toxic signs included increasing apathy and trembling. The LD_{50} was >6400 mg/kg. At necropsy it was found that the product was not fully absorbed. There is no explanation regarding this statement but the rapporteur interprets it, as it is particles/crystals of the test substance left in the stomach at necropsy. The study was not performed according to current guidelines.

An acute toxicity test on HBCDD (manufactured by Daiichi Kogyo Seiyaku) was carried out by using B6C3F1/Slc mice (Tobe, 1984). The test substance was suspended in 5 % CMC or olive oil to obtain 30 % suspensions (particle size unknown). The administered doses were 10, 15, and 20 g HBCDD/kg in the CMC suspension and 20 g HBCDD/kg in olive oil. Control groups received 5 % CMC or olive oil in the same volumes as those used for the 20 g HBCDD/kg groups. There were 10 animals in each dosage group and the substance was administered once by gavage. After administration the animals were observed until sacrifice on the 14th day. Both males and females in each group exhibited depressed activity immediately after administration, but recovered after about 30 min. There was no mortality and no effect on body weight during the 14 days observation period and no significant findings at autopsy. The study was comparable to current guidelines.

In an acute toxicity test HBCDD was administered to male ICR-SLC mice (number not stated) by gavage (Ishizu *et al.*). The test substance (supplied by Daiichi Kogyo Yakuhin K.K.) was suspended in olive oil for administration (particle size unknown). The doses were 30, 35, and 40 g/kg. No deaths were observed. Slight diarrhoea and body weight reduction were reported. However, no further information is available. Livers appeared to be swollen, and slight necrotic foci were sporadically found. No other changes were reported. The study is poorly described and not performed according to current guidelines.

Inhalation: Five Charles River CD rats of each sex were used in an acute inhalation toxicity study (Wilson and Leong, 1977). The animals were whole body exposed to a dust of HBCDD (a white powder identified as ”Firemaster 100”, unknown particle size)) at a concentration of 202 mg/l for 4 hours. The authors noted, “the chamber atmosphere was extremely dusty”. The immediate (first 10 minutes) response to the dusty atmosphere was an increase of activity in preening. 90 minutes after initiation of exposure to the end of the 4 hours exposure period the rats exhibited slight dyspnea. During the 14 days observation period the animals appeared normal. No deaths occurred. The study was not performed according to current guidelines.

In another inhalation study, five Wistar rats of each sex were whole body exposed to a dust of HBCDD (GLS-S6-41A) at a concentration of 200 mg HBCDD/l for one hour and thereafter observed for the two following weeks (Lewis and Palanker, 1978a. None of the animals died and no gross changes were observed. The study was not performed according to current guidelines.

The particle size distribution of the material used in these two studies was not stated, and it is therefore not clear to which extent the aerosol was at all inhalable. Without this information, no general conclusions can be drawn with respect to the acute toxicity by inhalation. However, it is clear that the tested technical preparations showed low toxicity.

In conclusion: The substance has a very low acute toxicity by the oral and dermal routes of administration, and it has not been possible to determine a LD_{50} value. The minimum oral lethal dose is higher than 20 g/kg in rats, and exceeds 40 g/kg in mice. By the dermal route, LD_{50} is higher than 20 g/kg in rabbits. The acute toxicity by inhalation has not been adequately investigated, but seems low in some technical preparations.

4.1.2.3 Irritation

4.1.2.3.1 Eye irritation

Three male and 3 female New Zealand White rabbits were used for an eye irritation study (Wilson and Leong, 1977). The eyes of the rabbits (weight 2003 to 2220 g) were examined with ultraviolet light following instillation of a sodium fluorescein solution. The rabbits received 100 mg of HBCDD (”Firemaster 100”) placed into the cupped conjunctival sac of the right eye, then the eyelids were gently held together for one second. The left eye served as the untreated control for each rabbit. The eyes were examined 24, 48, 72 hours and 7 days following instillation. The scores for iris and cornea were zero so the results refer to the effects on conjunctiva. The group average scores 0.6, 1.0, 1.0, and 0 respectively at the different time points. The total maximum score possible was 110. Based on the scores

obtained it was concluded that the test material is not a primary eye irritant and according to EU criteria it does not classify as an eye irritant.

In a similar test, 6 New Zealand rabbits, mixed sex, were given 0.1 g HBCDD (”GLS-S6-41A”) (Lewis and Palanker, 1978b). Their eyes were not washed for 24 hours. The group’s average scores according to Draize were 6.2 after 24 hours, 2.8 after 48 hours, 0.3 after 72 hours, and 0 on days 4-7. For two of the animals no irritation at all was observed. For one animal signs of irritation was observed in cornea, iris and conjunctivae, the latter grade two otherwise grade 1. For the three remaining animals signs of irritation (grade 1) were observed in the conjunctivae and for one of the three also in iris (grade 1). The scores were provided by adding up the scores from each animal and then divided the sum with the number of animals. It was concluded by the author that the test substance is a mild ocular irritant in rabbits under the conditions of the test. HBCDD does not classify as an eye irritant according to EU criteria.

In an acute eye irritation test, six rabbits were given 100 mg HBCDD (white powder) in one eye (Crown *et al.*, 1984). The animals were examined after 1, 24 and 48 hours. Slight conjunctival redness was observed in five rabbits (grade 1 according to Draize) and mild redness in one rabbit (grade 2) after 1 hour. Slight conjunctival chemosis (grade 1) was observed in four rabbits and mild redness in two rabbits (grade 2) after 1 hour. After 24 hours two rabbits showed slight redness and one animal showed chemosis. Opacity (grade 1, according to Draize) over an area up to a quarter of the cornea was observed on one rabbit 24 and 48 hours after instillation. After 3 days no irritation was observed.

In a similar study, 50 mg HBCDD (granular powder) was applied to rabbit eye (Zeller, 1962). No irritation was reported 1 hour, 24 hours or 8 days after application.

4.1.2.3.2 Skin irritation

In a primary skin irritation study, six rabbits were given 0.5 g HBCDD, moistened with aqua dest. as an 80 % suspension (firm paste), as a single semi-occluded application (EPA, 1990b). The test site area was clipped the day before dosing. The application time was 4 hours. Assessment of skin irritation responses was made 1, 24, 48, and 72 hours after removal of the dressing. No signs of irritation were observed at any test site, except for a slight erythema (barely perceptible) on one rabbit. The study was performed according to OECD guideline 404.

In a similar study, HBCDD (80 % suspension in water) was applied on the dorsal skin of rabbits. The exposure period was 20 hours. After 24 hours a slight erythema was reported and after 8 days a slight scaling (EPA, 1990d).

In an irritation test with cloth containing HBCDD (amount not given), squares of 10×10 cm were placed on the shaved back of 3 New Zealand White rabbits (Pharmatox, 1990). The piece of cloth was moistened with warm water. The study report does not clarify if HBCDD was impregnated or applied to the cloth. After 4 hours the cloth was removed and the skin scored for irritation. Observation time was 7 days. No irritative effects were noted. The study was performed according to OECD guideline 404.

In a primary skin irritation study three male and three female New Zealand White rabbits were used (Wilson and Leong, 1977). The hair was removed from the back of each rabbit and the skin was abraded on three of the rabbits. 0.5g HBCDD (”Firemaster 100”) was applied to the back using physiological saline as a wetting agent. The application sites were covered with gauze bandaging and occluded. Four hours later the bandages were removed. The sites

were examined for skin irritation in accordance with the scale that is described in OECD guideline 404 at 4, 24, 48 and 72 hours. Only a very slight erythema, barely perceptible, was noted. The results indicate that the test substance is not a primary skin irritant and minimally irritating to the skin. The substance was applied to intact skin of 3/6 rabbits and 3 animals are sufficient according to the recently revised OECD guideline 404; the study is performed according to current guideline.

In a similar study six New Zealand rabbits were used. 0.5 g HBCDD (”GLS-

6-41A”) was administrated to clipped areas of intact and abraded skin under occlusion for 4 hours (Lewis and Palanker, 1978b). The sites were examined and scored separately at 24 and 72 hours. The test substance did not cause skin irritation, neither erythematic or edema. The study was not performed according to current guidelines since the sites were only examined at 24 and 72 hours.

A primary skin irritation test on female Hartley guinea pigs was performed according to the Draize method (Momma *et al.*, 1993). There were 10 animals in the exposed group and 10 animals in the control group. 0.5 g of the stock powder (HBCDD from Daiichi Kogyo Seiyaku) was wetted in 0.4 ml distilled water and 3 drops of ethanol before use. The hair on the back of the animals was cut and thereafter shaved with an electric shaver the day before the drug application. The left side was set as an intact skin section and the right side was set as an abraded skin section by scratching it with an injection needle. A lint fabric patch 2×3 cm was coated uniformly with moistened HBCDD (0.5 g), placed in aluminium foil chamber, and fixed on the back of the animals, and occluded with elastic bandage. The bandages were removed after 24 hours, and the sites were examined for irritation after 24 and 48 hours.

No primary skin irritation was observed on intact or abraded skin sites of guinea pigs.

4.1.2.3.3 Respiratory tract irritation

In an acute inhalation study, five male and five female Charles River CD rats were whole body exposed to a dust of HBCDD (a white powder identified as ”Firemaster 100”, unknown particle size) at a concentration of 202 mg/l for 4 hours (Wilson and Leong, 1977). The authors noted, “the chamber atmosphere was extremely dusty”. Observations for ocular and nasal irritation, respiratory distress and mortality were made during and immediately following the 4 hours exposure and daily thereafter for 14 days. The immediate (first 10 minutes) response to the dusty atmosphere was an increase of activity in preening. 90 minutes after initiation of exposure to the end of the 4 hours exposure period the rats exhibited slight dyspnea. During the 14 days observation period the animals appeared normal. There was not an autopsy performed so it is difficult to draw any conclusions but there were no clinical signs of irritation in the respiratory tract. No deaths occurred. The study was not performed according to current guidelines.

In conclusion: HBCDD is mildly irritating for the eye, but does not qualify as an eye irritant according to EU criteria. The substance is not irritating to skin in skin irritation studies or to the respiratory system according to clinical symptoms in acute toxicity studies by the inhalation route.

4.1.2.4 Corrosivity

A group of 6 New Zealand White rabbits (sex not stated) were used in a dermal corrosion test (Lewis and Palanker, 1978b). Applications of 0.5 g of HBCDD (described as "GLS-S6-41A") were made under occlusion to clipped areas of intact skin. The wrapping and test material were removed 4 hours following application. The sites were examined for erythema and oedema at 4 and 48 hours. It was concluded that the test material is not corrosive. A second study gave similar results (Crown *et al.*, 1984).

In conclusion: HBCDD is not corrosive to skin.

4.1.2.5 Sensitisation

4.1.2.5.1 Human data

McDonnell (McDonnell, 1972) reported no reactions on the arms of 10 men or on the arms or legs of 10 women who wore 1-inch squares of Tyvek T-12 fabric treated with 10 % HBCDD (purity not stated) and held in place with Dermicel tape for 6 D. After a two-week rest period, new patches were applied for 48 hours as a challenge test for skin sensitization. Skin under the patches was examined at 2 and 6 days after the first application on removal of the challenge patch. No skin reactions were observed on any subject at any examination.
According to information from three HBCDD manufacturing plants no cases of skin sensitisation have been observed. However, the number of exposed workers or the level of exposure is not reported and therefore this information cannot be used to judge the sensitising potential of HBCDD.

4.1.2.5.2 Animal data

A composite of the EU-marketed HBCDD was administered to male Dunkin Hartley albino guinea pigs in a dermal sensitisation test according to the Magnusson-Kligman protocol (Magnusson and Kligman, 1969; Wenk, 1996). The animals were divided into a control group of 10 animals (Group 1) and a test group of 20 animals (Group 2). Three pairs of intra-dermal injections (induction dosing) were given to each animal at three clipped sites. Each injection had a volume of 0.1 ml. The first pair of injections consisted of 50:50 solutions of corn oil and Freund's complete adjuvant. The second pair consisted of corn oil (Group 1) or HBCDD at a concentration of 5 % in corn oil (Group 2) and the third pair of injections consisted of corn oil (Group 1) or HBCDD at a concentration of 5 % in Freund's adjuvant (Group 2). On day 7 the interscapular area was shaved again and treated with sodium lauryl sulphate in petroleum jelly in order to create a local irritation. On day 8, a topical application of HBCDD (0.5 g moistened with corn oil) was applied on the clipped interscapular area of each Group 2 animals using a chamber. The chambers were left in place for 48 hours. After a two weeks rest period all animals were given a challenge dose (24 hours exposure). An empty chamber was applied to the right flank and a chamber loaded with 0.5 g HBCDD (moistened with corn oil) was applied to the left flank on clipped skin. The chambers were removed after 24 hours and approximately 21 hours thereafter the animals were scored according to Draize. Dermal lesions were scored up to 120 hours after the challenge dose, and after that the animals were

sacrificed. HBCDD produced no erythema or oedema in any animal and the substance was considered a non-sensitiser.

In another guinea pig sensitisation test on HBCDD produced in Japan, groups of 10 female Hartley albino guinea pigs were used in a study performed according to the Magnusson-Kligman protocol (Magnusson and Kligman, 1969; Nakamura *et al.*, 1994; Wenk, 1996). For the induction 0, 5000 or 50,000 ppm HBCDD in olive oil was used for the intra-dermal injection and 250,000 ppm HBCDD in petrolatum for the topical application on shaved skin. For the challenge, 21 days after the intra-dermal injection, 0, 500, 5000 or 50,000 ppm HBCDD in acetone was used in an open patch test on shaved skin. At the highest concentration of induction and challenge 9/10 animals were sensitised and there was a clear dose-effect relationship.

Similar results on Japanese HBCDD were reported in a study by Momma et al. (Momma *et al.*, 1993). Groups of 10 female Hartley albino guinea pigs were used. The study was performed according to the Magnusson-Kligman protocol (Magnusson and Kligman, 1969). For the induction 0.05 ml of 0, 0.05, 0.5 and 5 % HBCDD in olive oil was used. Seven days later, filter paper coated with 25 % HBCDD in vaseline (0.2 g) was applied for the topical application on shaved skin. 21 days after the intra-dermal injection, 0.02 ml of 0.005, 0.05, 0.5 and 5 % HBCDD in acetone was used for the challenge in an open patch test on shaved skin. The results showed that an induction dose of 0.5 % or more and a challenge dose of 0.05 % elicited a positive response. At the highest concentration of induction and challenge 9/10 animals were sensitised and there was a clear dose-effect relationship.

The contact sensitisation potential of HBCDD has also been investigated using the Local Lymph Node Assay (LLNA) on young CBA/J female mice (Wolhiser and Anderson, 2003). The methodology used is in compliance with OECD Guideline 429 and GLP standards. The animals were housed in plastic “shoebox” cages with filter lids in rooms designed to maintain adequate conditions, and acclimatized to the laboratory for approximately one week prior to the start of the study. The animals were 8 weeks of age at the start of the primary irritancy study and 9 weeks of age at the start of the LLNA.
The HBCDD test material, a mixture of three manufacturers HBCDD commercial product (Wildlife International Ltd), was comprised of three diastereoisomers: 5.8 % α-, 19.3 % β-, and 74.9 % γ- HBCDD.
The study was divided in two parts: a primary irritancy study (ear swelling response) and LLNA. The HBCDD concentrations that were used for the LLNA evaluation were selected following the primary irritancy study. The primary irritancy study was performed on 12 mice, 2 in each group. Prior to the administration and on day three, 24 hours after the final exposure, the thickness of each ear was measured with a digital micrometer. 25 µl/ear was administrated on the dorsal surface of each ear for two consecutive days using an adjustable pipette. Each mouse received one concentration of either HBCDD in the vehicle (N,N-dimethylformamide, DMF), or DMF alone. The preferred vehicle for a LLNA test is 4:1 aceton:olive oil but HBCDD was not adequately soluble in that media. According to OECD guideline 429, DMF is another alternative to use. The highest concentration of HBCDD (50 % w/v) was based upon maximal solubility of HBCDD in DMF; lower concentrations were selected to provide a range of doses, 25 %, 10 %, 5 %, or 1 %, to evaluate ear swelling potential of HBCDD. Mean (2 ears) percent ear swelling was 1.0, 2.0, 6.2, 19.8, 26.5, 19.8 in the vehicle, 1 %, 5 %, 10 %, 25 %, and 50 % HBCDD groups respectively. Erythema evaluations of the ear surfaces were not assessed because the ears of the CBA/J mice are brown.

In the LLNA, the administration of the materials (25µl/ear) was made on the dorsal surface of both ears using an adjustable pipettor fitted with a disposable tip. 30 mice, 6 in each group received one of three concentrations of HBCDD; 2 %, 20 %, or 50 %, or DMF on days 1-3. These concentrations are in agreement with OECD Guideline 429. α-hexyl cinnamaldehyd (HCA) at 30 % v/v was run concurrently as a positive dermal sensitisation control. On day 6, all mice received an intravenous injection, via the lateral tail vein, of 250 µl of phosphate buffered saline (PBS) containing 20 µCi of ^{3}H-thymidine. Five hours later, the mice were sacrificed and the draining auricular lymph nodes excised and pooled for each mouse. A single cell suspension of lymph node cells was prepared. The cells were washed two times in PBS and were precipitated in 5 % trichloroacetic acid. ^{3}H-thymidine incorporation was measured on a β-scintillation counter as disintegration per minute (dpm) per mouse, and a mean dpm value ± SD (standard deviation) was calculated for each experimental group. In addition, a Stimulation Index (SI) was calculated for each mouse using the absolute dpm value for each mouse as the numerator, and the mean dpm value from the vehicle control mice as the denominator. The mean SI ± SD was calculated for each experimental group (Table 4-21).
Chemicals that elicit a SI of $\geq$3 in the LLNA are considered positive for dermal sensitisation potential.

The initial and terminal body weights were obtained and recorded. Once each day clinical examination was conducted and the following parameters were evaluated: skin, fur, mucous membranes, respiration, nervous system function, animal behaviour, moribundity, mortality, and the availability of feed and water. There were no toxic effects on these parameters.
The mean lymphocyte proliferation in vehicle-exposed (DMF) mice was 2015±749 (Table 4-21). This was higher than historical values (237-739 dpm) from the same laboratory using 4:1 aceton:olive oil as the vehicle, but according to the report consistent with values reported elsewhere in the literature for this vehicle. One mouse in particular demonstrated 3481 dpm in its draining lymph nodes and was confirmed to be a statistical outlier. Exclusion of this outlier still resulted in a control value of 1722 dpm and did not alter the conclusion.

Table 4-21 Summary of Lymph Node Proliferation data

Treatment group	DPM (mean ± SD)	SI (mean ± SD)
Vehicle (DMF)	2015 ± 749	1.0 ± 0.4
HCA (30 % v/v)	5970 ± 1909*	3.0 ± 0.9
2 % HBCDD	1744 ± 181	0.9 ± 0.1
20 % HBCDD	1670 ± 155	0.8 ± 01
50 % HBCDD	1946 ± 664	1.0 ± 0.3

*= represents statistical difference from control mean at p <0.05 using Dunnet`s T-test.

HCA administration elicited proliferation that was 3-fold greater than that of vehicle controls. While this SI value for HCA was slightly lower than reported values when diluted in acetone and olive oil, HCA was still positive according to guideline criteria. Exclusion of the statistical outlier in the vehicle group increased the HCA SI value to above 3.5.

HBCDD did not demonstrate results that were consistent with dermal sensitisation. SI values were consistently around 1.0 at all doses tested (Table 4-19). The conclusion from this study is that (this composite of EU-marketed) HBCDD is not a skin sensitizer.

The results from the studies on the EU-marketed HBCDD (conducted in the U.S.) and the two valid Japanese investigations on dermal sensitisation are obviously difficult to reconcile. A difference in composition of the test material used (additives, impurities) could be one explanation for the divergent outcomes, see Table 4-22. The use of acetone as a vehicle for HBCDD that will promote its penetration on shaved skin in the challenge phase of the Japanese studies, vs. the use of the suspended substance in corn oil on clipped skin, may also have contributed to the divergent outcome of these investigations. The use of open chambers could hardly be cited as a reason for obtaining a *positive* result. However, as the LLNA study also involved HBCDD properly dissolved in DMF, the most likely explanation to the different results is the presence of different impurities in the EU and Japanese HBCDD. As the two studies on the EU-marketed HBCDD were negative, we conclude that the HBCDD available in the EU is not sensitising.

Table 4-22 Comparison of the methodology used in 3 guinea pig skin sensitization studies conducted on HBCD.

	Wenk- 1996	Momma-1993	Nakamura-1994
INDUCTION - ID			
VOLUME	0.1 ml	0.05 ml	?, Assume 0.05 ml
CONCENTRATION	5 %	0.05, 0.5, 5 %	0.5, 5 %
DOSE	0.005 mg	0.000025, 0.00025, 0.0025 mg	0.00025, 0.0025 mg
VEHICLE	Corn oil	Olive oil	Olive oil
INDUCTION –TOPICAL			
AMOUNT	500 mg	200 mg	?, Assume 200 mg
CONCENTRATION	100 %	25 %	25 %
DOSE	250 mg	50 mg	50 mg
VEHICLE	Corn oil*	Vaseline	Petrolatum
CHALLENGE			
VOLUME/AMOUNT	500 mg	0.02 ml	0.1 ml
CONCENTRATION	100 %	0.005, 0.05, 5 %	0.05, 0.5, 5 %
DOSE	250 mg	0.000001, 0.00001, 0.0001, 0.001 mg	0.00005, 0.0005, 0.005 mg
VEHICLE	Corn oil*	Acetone	Acetone

*Moistened with corn oil.

The phototoxicity as well as the photosensitisation potential of HBCDD was investigated by the same laboratory in the guinea pig using 8-methoxypsoralene (Momma et al., 1993) as a positive control for the phototoxicity study, and 3,5,4-tribromosalicylanilide (TBS) as a positive control for photosensitisation using an adjuvant-strip procedure. Since HBCDD has no appreciable light absorption in the UVA region, and a very small absorption around 300 nm, irradiation was performed with a mixed UVA/UVB source. No reactions were reported to be induced by the irradiation in presence of HBCDD. However, crucial data such as the characteristics of the light source as well as dosimetric data, including the minimal erythemic dose, were not given (Nilsson et al., 1993). The strong reactions elicited by the positive controls indicate, on the other hand, that a sufficient radiation dose had probably been applied. In view of the fact that HBCDD – that was found to be a sensitizer in the same laboratory upon conventional testing – did not elicit any reaction in the photosensitization test using the same animal species, seems to indicate that the protocol used would only detect strong photosensitizers.

In conclusion: Two Magnusson-Kligmann studies performed with HBCDD of unknown specification have given positive results. However, studies of HBCDD of known specification by the major producers of HBCDD, has shown negative results both in a Magnusson-Kligmann test and in an LLN assay. Overall, HBCDD is therefore not regarded as a skin sensitizer.

No data are available to assess the potential for respiratory sensitisation.

4.1.2.6 Repeated dose toxicity

Two of the following repeated dose studies presented below were carried out in the same laboratory (Chengelis 1997, Chengelis 2001). HBCDD contamination of control rats was reported in the 90-day study. It should therefore be kept in mind that there might also be a question of contamination in the second study carried out in the same laboratory.

In a 28-day study on Sprague-Dawley Crl:CDBR rats, according to OECD Guideline 407 and in compliance with US EPA GLP, HBCDD was administered as a suspension (mean particle size 142 μm) in corn oil by gavage at 0 (control), 125 (low), 350 (mid) or 1000 (high) mg/kg daily for 28 days (Chengelis, 1997). The HBCDD mixture consisted of; HBCDD α-diastereomer (6.3 %), HBCDD β-diastereomer (9.1 %), HBCDD γ-diastereomer 76.9 %, Isobutanol (0.2 %), Tetrabromocyclododecane (1.0 %), and other unknowns (6.5 %). The control group comprised of 12 males and 12 females, while the test groups consisted of 6 males and 6 females in the low and mid dose groups, and 12 males and 12 females in the high dose groups. At the end of the dosing period 6 animals/sex/group were killed and necropsied. The remaining 6 animals/sex in the control and high dose groups remained untreated for a 14-day recovery period and then killed and necropsied.

All animals survived to the scheduled necropsy. Clinical signs during the study were non-specific and not considered to be related to the test substance. Body weight gain and food consumption was not affected by treatment, and no significant histopathological lesions attributed to the treatment were reported. Functional observation battery and motor activity results were neither affected by the treatment. No significant histopathological or serum chemistry changes related to exposure to HBCDD were observed. With the exception for livers, there were no statistically significant differences in the absolute and relative organ weights relative to controls. The relative liver weight was significantly increased with respect to the control group at the day of sacrifice (day 28), in mid- (+17 %), and high dose (+29 %, $p < 0.01$) in males, and at low (+16%, $p < 0.05$), mid (+22 %, $p < 0.01$), and high dose (40 %, $p < 0.001$) in females. At the sacrifice after the recovery period, the relative body weight in males had normalised to control levels. The relative liver weight in females had decreased, but was still significantly elevated (+17 %) in the high dose group in comparison to control rats. Histopathological analysis of the thyroids revealed no significant lesions (such as follicular cell hypertrophy). There was a slightly increased colloid loss in the thyroids of male rats (high-dose) in comparison to controls that persisted in a less pronounced form to the recovery sacrifice. No such changes were observed in females. The thyroid gland weight and serum concentration of TSH, T3 and T4 were not measured. The effects on the liver, especially in the female rats (weight increase by 16 % at low dose), indicate a LOAEL of 125 mg/kg/day.

In a 28-day feeding study (not carried out in accordance with present standards), 40 Sprague-Dawley rats of each sex were divided into 4 groups, receiving 0 (control), 1.0, 2.5 or 5.0 % HBCDD (Hexabromid-S), respectively, in the feed (Zeller and Kirsch, 1969). This is approximately equivalent to about 940 (low dose group), 2400 (mid dose group) or 4700 mg/kg/day (high dose group), respectively. The rats were given powdered feed, which was mixed with 1 % olive oil to reduce dusting.

There were no overt signs of clinical toxicity at the lowest dose level, but the rats in the two higher dose groups were in poor condition after the first two weeks; they lost hair, and they had an uncertain gait. In the control group, the food consumption increased 56 % for males and 34 % for females during the 28 test days. This should be compared to 53 % (low), 62 %

(mid) and 4 % (high) for males and 29 % (low), 25 % (mid), 18 % (high) for females. A weight increase was also observed in both male and female rats in the mid and high dose groups. The weight of the animals in the control group increased with 209 % for males and 92 % for females. In the mid dose group the weight increase was 136 % (male) and 83 % (female), and in the high dose group it was 86 % (male) and 63 % (female). There were no significant differences with respect to serum chemistry data (serum enzyme levels were not analysed). In females, the relative liver weight increased with 33 %, 62 %, and 108 % in the low-, mid- and high dose groups. In males, the relative liver weight increased with 27 %, 60 % and 105 % in the low, mid and high dose groups. The significant relative liver weight increase was dose related but was not accompanied by any signs of fatty infiltration or by other pathological findings.

The thyroids of exposed animals showed microfollicular hyperplasia and increased activity of the epithelium. The changes were slight at the lowest exposure level, but became more evident in the higher dose groups. At the highest dose, the animals exhibited a marked hyperplastic thyroid tissue with adenomatose proliferation and epithelial hyperactivity. Serum concentrations of TSH, T3 and T4 were not measured.
The females in the high dose group showed signs of inhibited oogenesis in most of the follicles and ripening follicles in the ovaries were sparse. The testes and epididymides of the males in the high dose group showed normal differentiation of the inner sexual organs and undisturbed spermiohistogenesis, although the epididymides were small. This study does not comply with the present standard, a NOAEL/LOAEL can therefore not be determined based on these data. Nevertheless, the data supports that the liver and thyroid glands are targets for HBCDD toxicity in rats.

In summary, the two 28-day studies both demonstrate a rather low order of toxicity of HBCDD upon repeated administration. The increase in liver weight was not accompanied by any pathological findings. This scenario might reflect a reversible adaptive change characterised by induction of the microsomal enzymes and cell proliferation in the endoplasmic reticulum. The observed hypertrophy was reversible for short time exposure, but if the exposure continues for a longer period it may exceed the metabolic capacity of the liver, and cause necrosis (Newberne, 1982).

Seven weeks old Wistar rats were administered HBCDD by gavage for 28 days (van der Ven *et al.*, 2006). The HBCDD was properly dissolved in corn oil. This was achieved by first dissolving the HBCDD in acetone, then mixing the HBCDD-acetone solution in corn oil, and finally evaporating the acetone. HBCDD was supplied by Industry (BSEF), and was of the same composite of substance (from three producers), as used in many other studies reported in the RAR. The composition of diastereomers was 10.3 - 8.72 - 81.0 % of α, β, and γ, respectively. Five rats per sex were used per dose-group, but fewer animals were used for analyses of some parameters. The doses were 0, 0.3, 1, 3, 10, 30, 100, and 200 mg/kg bwt/day. The study was carried out according to the OECD 407 guideline, with a focus on endpoints sometimes altered by persistent organic pollutants, including effects on the thyroid hormone (TH) axis, haematology, and bone parameters. The study did not follow GLP. Only absolute organ weights were reported, as the authors considered it more accurate than relative weights since changes in body weights may introduce new uncertainties. Analysis of residue-levels of HBCDD-diastereomers was done by LC-MS/MS.

Dose-response analysis was done by fitting a model (a nested family of purely descriptive (exponential) models was used) to the data (based on external dosing). From the best curve fit

(significant at the 5 % level) a benchmark dose (in the paper referred to as a critical effect dose, CED) was defined as the dose at which a predetermined change in effect level was obtained. This predetermined change in effect level was determined by expert judgment for each parameter. In practice a 10 % change was defined as a default, considering that this would cover the hazard for most sensitive subjects in a population, while a 20 % change was used for liver weight and immune parameters. A 95 % confidence interval (two-sided) was calculated of the Benchmark dose, and the lower confidence bound was proposed as an alternative to NOAEL. Finally, a ratio of the Benchmark dose and the lower confidence bound was used as a measure of the statistical uncertainty in the data set, and hence the validity of the result of the dose response modelling. In case of a more than tenfold ratio the Benchmark model was considered uncertain by the authors.

A few animals died from erroneous administrations, but no clinical signs of effects were observed. The chemical analysis showed that nearly no β-HBCDD was present in the animals. The concentration of α-HBCDD and γ-HBCDD in liver fat increased dose-dependently, but there were signs of a plateau being reached at the highest dose level(s), and perhaps earlier for γ than for α. Accordingly, with increasing dose, more and more of the HBCDD-residues consisted of the α diastereomer. Females consistently accumulated more HCBDD in the liver compared to males, but as no absolute concentrations are presented, it is difficult to quantify the difference. At doses up to the levels where the residue levels seem to plateau (30-200 mg/kg/day), the accumulation of α seemed to be roughly twice as high as γ. At doses of 100 mg/kg/day and above, the relative concentration of α increased much further compared to γ. This may be explained by either reduced absorption or increased metabolism of γ at high doses. The latter is possibly explained by induction of CYP 2B; see below.
A significant dose-dependent liver weight increase (BMD-L 23 mg/kg/day) and an induction of the liver enzyme T4-UGT were observed in female rats (Table 4-23, Table 4-24). BMD-L for T4-conjugation (T4-UGT) was 4 mg/kg/day in females. A similar trend was noted in male rats ((Table 4-23, Table 4-24)). Histopathological examination showed a slight non-significant induction of hepatic endoplasmatic reticulum in female rats. No such effect was noted in male rats.
The thyroid weight was significantly increased in female rats, accompanied by a reduction of serum T4 (BMD-L 55 mg/kg/day) ((Table 4-23, Table 4-24)). The serum T3 was unaffected in female rats whereas it was decreased in males (non-significant). The thyroid weight and serum level of T4 were unaffected in male rats. Histopathology showed a dose-related increase of thyroid activation in both sexes. However, male thyroid samples had a higher background activation (moderately activated), which make changes to advanced activation less obvious. The most evident effects in female rats were decreased follicular cell height and nuclear size.
In females, the pituitary weight was significantly increased (Table 4-23), and immunohistochemical staining for TSH showed a low but significantly increased ratio between high and low intensity immunostained thyrotrophic cells. This finding was consistent with the increased pituitary weight observed in females. No effects on pituitary weight or TSH staining of thyrotrophic cells were observed in males.
The most evident observation in bone was increased mineral density of trabecular bone at femur and tibia metaphysic in female rats (Table 4-23). There were no changes of any of the bone parameters in males.
Analysis of plasma showed reduced alkaline phosphatase and glucose, and increased cholesterol and total protein/albumin in female rats (Table 4-24). In males, cholesterol and glucose were decreased, whereas total protein/albumin was increased.

Table 4-23 Significant HBCDD induced dose response effects on organ weights and bone density in a 28 oral study in rats (van der Ven *et al.*, 2006)

Females										
	Organ weights					Total bone[d] Femur		Trabecular bone[d] Femur		Tibia
HBCDD dose mg/kg bwt	n	Pituitary (mg)	Thyroid (mg)	Liver (g)	Thymus (g)	n	Area (mm^2)	Min. cont[c] (mg/mm)	Mineral density (mg/cm^3)	
0	5	5 ± 1	17 ± 2	9.7 ± 1	0.42 ± 0.06	3	16 ± 1.5	10.5 ± 0.5	228 ± 34	199 ± 45
0.3	4	12 ± 1	18 ± 1	8.9 ± 1.1	0.28 ± 0.10	3	15 ± 0.9	10.2 ± 1.1	262 ± 34	226 ± 53
1	5	11 ± 3	22 ±4	8.6 ± 1.3	0.36 ± 0.09	4	14 ± 0.6*	9.5 ± 0.6	231 ± 40	199 ± 24
3	5	13 ± 2	15 ± 4	9.5 ± 0.4	0.35 ± 0.07	5	14 ± 0.7	9.2 ± 0.7*	219 ± 20	186 ± 23
10	5	11 ± 3	18 ± 3	8.9 ± 0.6	0.44 ± 0.07	3	15 ± 0.9	9.5 ± 1.1	211 ± 45	201 ± 70
30	5	8 ± 2	35 ± 17	11 ± 1.0	0.43 ± 0.08	3	16 ± 0.4	9.9 ± 0.4	248 ± 10	181 ± 33
100	5	13 ± 1	27 ± 7	13 ± 0.5	0.42 ± 0.08	3	16 ± 1.0	10.4 ± 0.8	228 ± 22	195 ± 29
200	5	13 ± 2	26 ± 3	12 ± 0.6	0.37 ± 0.10	3	16 ± 0.7	11.1 ± 0.5	265 ± 20	251 ± 12
CED (mg/kg bwt)		50.6	3.4	29.9	-		>200	134.6	139.3	91.1
BMD-L mg/kg bwt)		29.9	1.6	22.9	NSE		129.8	86.7	69.9	49.3
CED/BMD-L		1.7	2.1	1.3			1.8	1.6	2.0	1.8
Males										
	Organ weights					Total bone[d] Femur		Trabecular bone[d] Femur		Tibia
HBCDD dose mg/kg bwt	n	Pituitary (mg)	Thyroid (mg)	Liver (g)	Thymus (g)	n	Area (mm^2)	Min. cont[c] (mg/mm)	Mineral density (mg/cm^3)	
0	5	10 ± 2.0	27 ± 4.8	13.9 ± 0.7	0.47 ± 0.08		20 ± 1.1	11.1 ±0.4	145 ± 14	143 ± 17
0.3	5	6 ± 2.0	23 ± 5.0	17.1 ± 3.4	0.45 ± 0.08		19 ± 0.9	10.9 ± 0.9	142 ± 15	126 ± 30
1	5	14 ± 2.0	26 ± 5.4	16.2 ± 3.0	0.52 ± 0.17		19 ± 1.9	10.3 ± 1.4	136 ± 28	131 ± 18
3	4	8 ± 2.0	28 ± 5.6	15.0 ± 1.6	0.47 ± 0.07		20 ± 2.7	11.3 ± 1.5	174 ± 16	158 ± 15
10	5	12 ± 4.0	27 ± 8.6	17.7 ± 2.3	0.50 ± 0.09		18 ± 0.5	10.7 ± 0.4	145 ± 9	136 ± 9
30	5	10 ± 2.00	27 ± 4.3	15.7 ± 0.5	0.37 ± 0.06		19 ± 1.6	10.8 ± 0.9	151 ± 28	143 ± 19
100	5	8 ± 4.0	24 ± 3.5	16.4 ± 2.3	0.42 ± 0.09		19 ± 1.1	11.3 ± 0.7	184 ± 18	168 ± 24
200	5	11 ± 3.0	25 ± 5.9	16.4 ± 3.2	0.38 ± 0.13		18 ± 1.5	10.3 ± 1.2	149 ± 10	135 ± 6
CED (mg/kg bwt)		-	-	-	176.6		-	-	-	-
BMD-L mg/kg bwt)		NSE	NSE	NSE	104.2		NSE	NSE	NSE	NSE
CED/BMD-L		-	-	-	1.7		-	-	-	-

Figures are average ± standard deviation of n replicates per dose group. No significant effects where observed (NSE), critical effect dose (CED), Benchmark dose (BMD-L). [a] % compared to control. [b] no plateau reached within applied dose range. [c] min.content., mineral content. [d] Measured at femur and tibia metaphysic.

Table 4-24 Significant HBCDD induced dose response effects in plasma chemistry and liver values in a 28 oral study in rats (van der Ven *et al.*, 2006).

Females										
		Plasma chemistry								Liver
HBCDD dose mg/kg bwt	n	ALP (U/l)	CHOL (mmol/l)	GLU (mmol/l)	Total protein (g/l)	Albumin	N	TT4 (mmol/l)	TT3 (mmol/l)	T4-UGT (pmol/min/ mg protein)[c]
0	5	4.66 ± 2.91	1.78 ± 0.18	9.76 ± 0.89	56.1 ± 2.0	42.0 ± 1.6	5	41.3 ± 2.6	0.91 ± 0.10	0.56 ± 0.23
0.3	3	3.10 ± 2.76	1.71 ± 0.10	9.56 ± 1.19	56.9 ± 2.2	43.2 ± 1.3	4-5	41.9 ± 3.1	0.84± 0.15	0.61 ± 0.15
1	3	4.74 ± 2.50	1.68 ± 0.28	9.84 ± 0.95	56.4 ± 1.8	42.6 ± 1.4	5	40.2 ± 7.3	0.88 ± 0.12	0.53 ± 0.17
3	3	3.72 ± 2.14	1.86 ± 0.27	10.3 ± 1.18	56.3 ± 0.7	42.3 ± 1.1	5	37.2 ± 4.7	0.81 ± 0.11	0.43 ± 0.09
10	3	2.30 ± 1.21	1.61 ± 0.20	9.40 ± 0.35	56.8 ± 2.4	42.1 ± 1.7	5	38.6 ± 1.7	0.80 ± 0.09	0.57 ± 0.14
30	4	2.36 ± 0.33	1.94 ± 0.31	9.76 ± 1.13	59.2 ± 3.1	42.6 ± 2.8	4-5	38.0 ± 6.1	0.74 ± 0.15	0.74 ± 0.22
100	4	2.73 ± 1.55	2.42 ± 0.22	8.73 ± 0.88	64.1 ± 1.3	45.6 ± 1.1	4-5	35.8 ± 5.2	0.92 ± 0.20	1.39 ± 0.27
200	3	2.42 ± 2.71	1.84 ± 0.22	8.14 ± 1.81	61.0 ± 3.6	44.2 ± 2.6	5	30.4 ± 5.9	0.82 ± 0.13	0.99 ± 0.43
CED (mg/kg bwt)		33.9	23.6	100.0	194.3	316.8		76.1	-	14.5
BMD-L mg/kg bwt)		18.9	7.4	70.8	142.7	197.5		55.5	NSE	4.1
CED/BMD-L		1.8	3.2	1.4	1.4	1.6		1.4	-	3.5
Males										
		Plasma chemistry								Liver
HBCDD dose mg/kg bwt	n	ALP (U/l)	CHOL (mmol/l)	GLU mmol/l)	Total protein (g/l)	Albumin	N	TT4 (mmol/l)	TT3 (mmol/l)	T4-UGT (pmol/min/ mg protein)[c]
0	5	7.34 ± 5.59	2.08 ± 0.20	10.6 ± 1.1	56.4 ± 2.1	39.2 ± 1.0	5	40.2 ± 3.6	0.81 ± 0.06	0.36 ± 0.05
0.3	3	5.30 ± 3.66	2.13 ± 0.31	10.4 ± 3.2	58.1 ± 2.9	39.1 ± 0.7	4-5	40.4 ± 5.0	0.84 ± 0.14	0.44 ± 0.15
1	3	3.68 ± 1.82	2.10 ± 0.23	10.3 ± 2.6	58.6 ± 2.2	39.7 ± 1.9	5	40.6 ± 5.3	0.85 ± 0.16	0.40 ± 0.14
3	3	7.43 ± 7.43	1.74 ± 0.30	10.9 ± 1.8	58.1 ± 1.5	40.1 ± 0.3	5	49.4 ± 7.2	0.89 ± 0.04	0.69 ± 0.37
10	3	4.88 ± 5.75	2.01 ± 0.23	10.9 ± 2.5	57.6 ± 2.0	38.8 ± 1.2	5	43.3 ± 1.3	0.97 ± 0.16	0.60 ± 0.24
30	4	5.10 ± 2.54	2.31 ± 0.41	10.5 ± 1.5	60.1 ± 1.2	39.2 ± 1.6	4-5	41.9 ± 4.6	0.90 ± 0.13	0.73 ± 0.26
100	4	3.74 ± 1.61	1.99 ± 0.39	9.7 ± 2.6	61.4 ± 1.7	40.8 ± 1.8	4-5	35.4 ± 4.2	0.82 ± 0.06	0.99 ± 0.41
200	3	3.48 ± 1.95	1.63 ± 0.19	8.4 ± 1.2	59.6 ± 3.6	40.6 ± 1.8	5	41.4 ± 3.5	0.89 ± 0.05	0.88 ± 0.45
CED (mg/kg bwt)		-	102.9	98.1	>200	>200		-	-	1.1
BMD-L mg/kg bwt)		NSE	65.9	57.0	>200	>200		NSE	NSE	0.1
CED/BMD-L		-	1.6	1.7	1.7	1.8		-	-	11.0

Figures are average ± standard deviation of n replicates per dose group. ALP, alkaline phosphattase; CHOL, cholesterol; GLU; glucose; T4-UGT; T4-uridine-diphosphate glucoronyl transferase. No significant effects where observed (NSE), critical effect dose (CED), Benchmark dose-low (BMD). [a] % compared to control. [b] no plateau reached within applied dose range. [c] a similar dose-response is observed when activities are expressed per min/g liver.

The BMD-L for a decreased weight of the male thymus was 104 mg/kg/day, whereas no effects were noted in females. Investigation of immunological spleen parameters was only performed in males. A few immunological parameters were affected (number of total spleen cells, T-helper cells, NK cells and NK cell activity), but the data are based on very small number of animals and have been generated in one sex only (Table 4-25). However, the results indicate a biological relevant dose response effect of HBCDD, even if the data are not statistically confirmed. However, other studies have not indicated effects on the immune system, and no effects were noted in the spleen by histopathology or weight. Therefore, the toxicological relevance of this finding is dubious.

The main results from the study are summarised in table (Table 4-26) below, in order of increasing BMD-L in the females. The experimental data are also presented in a more conventional manner (presented as means ±SD/dose group) in Table 4-23 and Table 4-24, as some of the effects were minimal and the variation was high.

Table 4-25 Effects of HBCDD on immune parameters in males of a 28 days oral study in rats (van der Ven *et al.*, 2006).

HBCDD dose (mg/kg/day)	No. of animals analysed	Total cells per spleen	CD4 (T-helper cells)	CD161a (Natural killer cells)	NK cell activity
0	4	48.7 ± 10.5	14.0 ± 4.7	4.8 ± 1.2	100.0 ± 29.4
0.3	1	49.6	15.2	3.8	99.9
1	2	47.1 ± 15.4	13.3 ± 4.8	3.6 ± 0.1	89.6 ± 1.2
3	1	44.4	11.4	4.6	102.6
10	2	39.4 ± 3.8	10.5 ± 0.9	4.1 ± 1.2	87.3 ±15.5
30	1	29.7	9.0	2.7	52.6 ±6
100	1	37.0	11.2	2.9	59.3
200	2	35.8 ± 1.1	10.0 ± 2.0	2.6 ± 0.1	104.3 ± 43.2
Dose response		+	+	+	a

Data represent mean ± standard deviation. [a] absent of effect based on mean values, dose response analysis was not performed. The immunological parameters were not assessed in female rats.

Table 4-26 Effects of HBCDD in a 28 days oral study in rats, where HBCDD (properly dissolved in corn oil) was administered by gavage (van der Ven *et al.*, 2006). The main results from the study are shown in order of increasing BMD-L in the females

Endpoints	BMD-L[a] (mg/kg/day) *(CED ± 95 % ci)*	
	Females	Males
Thyroid weight increase	1.6[b] (3.4 ± 1.8)	NSE[c]
T4-uridine-diphosphate glucoronyl transferase (T4-UGT) (pmol/min/mg protein)	4.1 (14.5 ± 10.4)	0.1 (1.1 ± 1)
Cholesterol (mmol/l)	7.4 (23.6 ± 16.2)	65.9 (102.9 ±37)

ALP (U/l)	18.9 (33.9 ± 15)	NSE
Liver weight increase	22.9 (29.9 ± 7)	NSE
Pituitary weight increase	29.9 (50.6 ±20.7)	NSE
Bone density		
Trabecular bone/tibia, mineral density	49.3 (91.1 ± 41.3)	
Trabecular bone/femur, mineral density	69.9 (139.3 ± 69.4)	NSE
Total bone; femur, mineral content	86.7 (134.6 ± 47.9)	
Total bone; femur area	129.8 (>200 ± 70.2)	
TT4 (nmol/l)	55.5 (76.1 ± 20.6)	NSE
TT3 (nmol/l)	NSE	NSE
Glucose (mmol/l)	70.8 (100 ± 29.2)	57 (98.1 ± 41.1)
Total Protein (g/l)	142.7 (194 ± 51.3)	>200
Albumin (g/l)	197.5 (316.8 ± 119.3)	>200
Thymus weight decrease	NSE	104.2 (176.6 ± 72.4)

[a] For each parameter, a critical effect dose (CED) was defined and calculated as the dose at which a predetermined critical effect size was obtained (10-20 % deviation from control values). A 95 % confidence interval (two-sided) was calculated for the CED, and the lowest value from the interval was defined as the BMDL, i.e., BenchMark Dose at the Lower confidence bound.

[b] Based on the numerical data presented, a NOAEL of 10 mg/kg/day seems more reasonable.

[c] NSE; no significant effects were observed.

[d] becasue of a high variation in the male data, this BMD-L is not considered valid by the authors, but there is a clear trend indicating a doubled enzyme activity at 3 mg/kg/day and an almost two-fold increase at 100-200 mg/kg/day.

Germer at al (2006) have studied hepatic cytochrome P-450 (CYP 450) levels and CYP 450 activity in HBCDD treated rats. Hepatic mRNA and microsomes were isolated from rats in the oral 28-day study cited above (van der Ven *et al.*, 2006). The main focus of the study was to evaluate the effect of HBCDD on the CYP 1A, 2B, and 3A enzyme families (enzymatic activity, mRNA and protein level). The enzyme activity was assessed with the following methods; 7-ethoxyresorufin *O*-deethylase assay (EROD), 7-pentoxyresorufin-*O*-dependentylase (PROD), and luceferin benzylether debenzylase (LBD) -assay, believed to represent 1A, 2B, and 3A4 enzymatic activity. mRNA levels (CYP 1A2, 2B1, 3A1/3). The amount of enzyme protein (CYP 1A1/2, 2B1/2, 3A1) were measured according to standard RT-PCR and Western blotting procedures.

The enzymatic activity in females and males are shown in Table 4-27. The only significantly induced enzymatic activities were detected for CYP 2B in males and CYP 3A4 in females from 10 mg/bwt/day. The induced CYP 2B enzymatic activity in males was confirmed on mRNA and protein level (5-fold and 3-fold induction at the highest dose, respectively), while the lack of CYP 2B activity induction in females were contrasted by high mRNA and protein levels (50-fold and 8-fold induction at the highest dose, respectively). However, the standard deviation at the highest dose on CYP 2B mRNA level was very high, which make the data difficult to interpret. The induced 3A4 activity in females was confirmed on mRNA and protein level (30-fold and 5 fold at the highest dose, respectively). The absence of effect on CYP 1A enzymatic activity induction in both genders was confirmed on mRNA and protein level.

In summary, HBCDD did not cause a general enzyme induction in rats. The detected HBCDD-induced effects where rather specific with induced CYP 2B and CYP 3A4 in males and females respectively.

Table 4-27 CYP activities in liver microsomes isolated from HBCD-treated rats

FEMALES			
HBCDD treatment (mg/kg bwt/ day)	EROD (CYP 1A) (pmol/mg protein/min)	PROD (CYP 2B) (pmol/mg protein/min)	LBD (CYP 3A4) (pmol/mg protein/min)
0 (DMSO)	28 ± 7	31 ± 11	1.0 ± 1.0
0.3	29 ± 7	30 ± 2	1.2 ± 0.8
1.0	30 ± 6	37 ± 7	1.8 ± 1.0
3.0	25 ± 5	28 ± 4	1.7 ± 1.1
10	26 ± 5	35 ± 7	3.8 ± 2.5*
30	27 ± 6	32 ± 4	5.0 ± 3.9*
100	26 ± 5	35 ± 7	4.1 ± 2.3
200	26 ± 4	26 ± 5	10.2 ± 5.2*
MALES			
HBCDD treatment (mg/kg bwt/day)	EROD (CYP 1A) (pmol/mg protein/min)	PROD (CYP 2B) (pmol/mg protein/min)	LBD (CYP 3A4) (pmol/mg protein/min)
0 (DMSO)	25 ± 7	30 ± 6	1.0 ± 0.4
0.3	27 ± 5	35 ± 10	1.3 ± 0.5
1.0	28 ± 3	39 ± 7	1.1 ± 0.3
3.0	24 ± 4	37 ± 5	0.4 ±0.4
10	23 ± 2	45 ± 6*	0.9 ± 0.3
30	25 ± 5	41 ± 6	0.6 ± 0.6
100	31 ± 7	51 ± 7*	1.7 ± 1.2
200	27 ± 6	35 ± 8	1.4 ±0.8

Data represent mean ± SD from three independent experiments. *Significantly different from controls ($p \leq 0.05$)

In concluding the 28 days study (van der Ven *et al.*, 2006), effects of HBCDD were observed on the thyroid, liver, pituitary, immune system, and on bone density. Study designs based on the benchmark model are rare, but they can be used according to the TGD. In setting the NOAEL/BMD-L for this study, a few issues have been considered.

For the increased thyroid weight, a BMD-L of 1.6 mg/kg/day has been proposed by the authors. However, when looking at the numerical data (mean value for control 17, means of thyroid weights for the exposed groups in order of increasing dose 18, 22, 15, 18, 35, 27, 26), a NOAEL of 1.6 mg/kg/day seems too conservative, considering that an undisputable increase only was observed at a dose of 30 mg/kg/day and above. The histopathology does not indicate effects at very low exposure levels either. 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). Taken together, we consider the thyroid effect being an adverse effect, particularly as human foetuses are particularly sensitive to small changes in thyroid hormone levels during the embryogenesis (Anselmo et al., 2004; Mead, 2004). Still, considering the uncertainty with regard to setting a NOAEL for this effect, we chose not to use this effect as basis for the NOAEL/BMD-L.
The BMD-L for liver weight increase was 22.9 mg/kg/day, using a 20 % weight increase as the critical effect level. Hepatic enzyme induction is probably one reason for this effect, but it should be noted that enzyme induction occurred also in male rats without any concomitant liver weight increase, indicating that other mechanisms also could be involved. Enzyme induction is well known to occur in humans, and this effect is therefore relevant for the risk characterisation. A BMD-L of 22.9 mg/kg/day will most likely also cover for effects on the thyroid (see above) and pituitary system (BMD-L 29.9 mg/kg/day).
Overall, this study supports the earlier RdT-studies, and it is proposed to use the NOAEL/BMD-L of 22.9 mg/kg/day from the 28 days study using dissolved HBCDD (van der Ven *et al.*, 2006) in the risk characterisation.

In a recent 90-days oral toxicity study, according to GLP and OECD guideline 408, 15 Sprague-Dawley rats of each sex were dosed daily by gavage for 90 days with 0, 100, 300, or 1000 mg HBCDD/kg/day (Chengelis, 2001). The test substance (90 % pure HBCDD, 0.7 % tetrabromocyclododecane, 0.1 % isobutanol, 9.2 % unknown, a mixture of three commercial lots) was administered as a suspension in corn oil. The mean particle size was 142 µm (10 % <3.6 µm, 10 % >280 µm). Ten animals of each sex were killed and necropsied at the end of the 90-day period, and five rats were killed after an additional recovery period of 28 days. Two satellite groups of 20 animals/sex/group were treated in an identical manner at dose levels of 0 or 1000 mg/kg/day, and 2 animals/sex/dose were killed at different time points during the study for fat-residue analyses. The results of the chemical analyses are described in chapter 4.1.2.1. However, it should be noted that the finding of HBCDD in control animals of the satellite group raises some questions regarding this study. Up to 15 µg HBCDD/g fat were found in the controls, as compared to approximately 4300 µg/g fat in the animals dosed with 1000 mg/kg/day. Study audits at the involved laboratories have not fully resolved the questions, although it is pointed out in the audit reports that poor washing procedures and re-use of the suspension containers in between the exposed groups likely have led to the contamination of control animals. Considering that the control values for the toxicity endpoints are within historical control values (Chengelis, 2002), and the rather flat dose-response relationships, it seems possible to use this study despite the uncertainties regarding the contamination of the control animals. However, the contamination issue will be further discussed in the risk characterisation.

The animals of the main study were examined for clinical signs, and effects on haematology, serum chemistry, thyroid hormones, oestrous cycle, sperm number and motility, and tissue histology. A few animals (n = 5) were also subjected to a Functional Observational Battery and Locomotor Activity evaluation.
In this study no effects were observed on body weight, haematology, or oestrous cycle of females or male reproductive system. The main effects were observed in the liver, thyroid, and prostate.

The relative liver weights were statistically increased in a dose-dependent manner in both sexes in all dose groups (18-44 % in males, and 24-48 % in females). Minimal hepatocellular vacuolisation was also observed in both sexes at all dose groups. In females (mid and high dos groups), some animals also exhibited mild or moderate hepatocellular vacuolisation, whereas minimal to mild hepatocellular hypertrophy was observed in females in the high dose group. Staining procedures indicated that the vacuoles contained lipid, but the amount was within the normal range. The effect on the relative liver weight (approximately +10 %) remained after the 28-day recovery period, albeit only statistically significant in the males. The relative weight of the thyroid was statistically increased by 33 % in the mid and high dose females, an effect that also remained after the recovery period.
There were also minimal thyroid follicular cell hypertrophy in the mid dose females, and mild hypertrophy in high dose animals of both sexes. In the high dose females, some histological effects remained after the recovery period. Serum concentrations of T4 and TSH were dose-dependently affected in all groups of both sexes; a reduction of T4 (21 % and 37 % in the high dose females and males, respectively) and an increase in TSH (approximately 10-fold). There was a dose-dependent increase in relative prostate weight, reaching statistical significance in the high dose group (+42 %), but no histological effects were observed.
The large weight increase of prostate may indicate a hormonal imbalance. Serum protein concentrations were dose-dependently increased in both sexes, with significantly increased albumin in males (7-14 %) and total protein in females (8-14 %).
These changes in the liver, thyroid hormone system and prostate could possibly be explained by enzyme induction in the liver as hepatic glucuronidation is known to be the rate limiting step in the biliary excretion of T4. This hypothesis remains to be proven as liver enzyme activities were not evaluated in the present study. The functional observation battery did not show any effects.

Although the study report concludes on a NOAEL of 1000 mg/kg/day, with the argument that the effects on the liver were not very serious and seemingly slowly reversible, a LOAEL of 100 mg/kg/day is deduced from this study based on increased relative liver weights (18 and 24 % in males and females, respectively). Changes in the thyroid hormone system were also observed at this dose (LOAEL of 100 mg/kg/day) (serum concentrations of T4 decreased 19 % in males and TSH increased several-fold in both sexes). Disturbances in the thyroid system could be a secondary effect to hepatic enzyme induction in the liver. Due to the expected low absorption of the HBCDD-suspension (see explanation in chapter 4.1.2.1 on toxicokinetics), the study LOAEL is transformed into an internal (systemic) corrected LOAEL of 10-20 mg/kg/day based on the assumed 10-20 % oral absorption.

In a 90-day study, rats were given HBCDD (Hexabromid-S, unknown purity), powered in food The HBCDD was mixed with 1 % olive oil to reduce dusting (Zeller and Kirsch, 1970). Five groups with 20 Sprague-Dawley rats of each sex per group were dosed with HBCDD. The concentration of HBCDD in food was 0 (control), 0.16, 0.32, 0.64 or 1.28 %, respectively, equivalent to about 0, 120, 240, 470, or 950 mg/kg, respectively based on actual body weights and food consumption After the exposure period, 10 extra rats per sex from the control group and the highest dose group were kept for 42 days on control food.

None of the animals showed any visible signs of toxicity after ingestion of HBCDD for 13 weeks. About one third of the animals, control inclusive, showed signs of respiratory infections. No information of the severity of the respiratory infections or when the infections first were noted is available. Male rats in the highest dose group weighed slightly more than rats in the other dose groups, but consistently less than the controls. No histopathological or

serum chemistry changes related to exposure to HBCDD were observed. The relative liver weights increased significantly in a dose-dependent manner in both sexes in all dose groups (8-22 % in males, and 5-27 % in females). Hepatic lipoid phanerosis was observed in many animals, varying from very slight adipose specks to disseminated droplets. The incidence of lipoid phanerosis and hepatic reticuloendothelial system showed a general tendency to rise with the level of HBCDD in the diet. The lesion on the hepatic reticuloendothelial system is not further described in the study report. Lipoid phanerosis in male rats increased from 20 % in the control group to 95 % in the highest dose group. Among female rats the incidence started at 50 % and raised to 95 % in the mid dose group, and decreased slightly to 80 % in the high dose group. On average the incidence of lipoid phanerosis in the liver increased with increased liver weight. There was no increase in the levels of liver specific glutamatic pyruvic transaminase (ALT). This study does not comply with present standard so a NOAEL/LOAEL cannot be based on these data, but the data supports the hypothesis that the liver is the target for HBCDD toxicity.

In a life time bioassay, mice (B6C3F1) were exposed via the feed to HBCDD for 18 months (see section 4.1.2.8 for more details) (Kurokawa *et al.*, 1984). The mice (n = 50/group/sex) were exposed to 0, 13, 130 or 1300 mg/kg/day. Numerous tissues were collected for histopathological examination.
The survival was high, and there were no differences in mortality between the groups. The body weight (males and females) was lower in the groups with HBCDD supplementation compared to the control group, but the difference was not statistically significant. There were also large variations in food consumption between the HBCDD supplemented mice and control groups (males and females), but no significantly differences were detected. The main effects of HBCDD in this study were liver lesions, such as hepatocytic swelling, degeneration, necrosis, vacuole formation and fatty infiltration (Table 4-29), although the dose-response relationships were not clear-cut. The study was apparently not performed according to current guidelines, and the data are only available as a study summary. The study supports that the liver is an HBCDD target organ, but a NOAEL cannot be deduced from this study.

Also in a two-generation reproductive toxicity study (Ema et al 2008), where animals were administered HBCDD particles mixed in ground food, effects were observed on the liver and on the thyroid system (a more detailed description is present in the fertility section 4.1.2.9). Because of dosing HBCDD-particles, the accuracy of the higher dose levels can be questioned. The concentrations in the diet was 0, 150, 1500, or 15,000 ppm, which numerically has been translated to 10-14, 101-141, or 1008-1363 mg/kg/day in low, mid, and high dose animals of the F0 and F1-generations, with the low end of the ranges representing males and the high females. Considering the dosing of particles, the top doses should be considered as $\leq$ stated above (i.e., $\leq$101-141 and $\leq$1008-1363 mg/kg/day).

The relative liver weight was increased at the top dose in both sexes of all generations. Relative liver weight increases were also observed at the mid dose in male F0-animals (magnitude not stated), male and female F1-weanlings (+10 %), and male F2-weanlings (+7 %). No “gross lesions or remarkable microscopic alterations” were observed in the livers by histopathology.

Histopathology revealed a statistically significant increased incidence of animals with decreased size of the thyroid follicles in the F0 and F1 mid and high dose groups. Thyroid weight was measured in adult F0 and F1 animals, and an increased relative weight was observed in high dose male F0 and F1 animals and in high dose female F1 animals. Serum

hormone T4 levels appeared decreased in all high dose animals, although statistically significantly decreased only in male and female F0 animals. Serum TSH was statistically increased att all dose levels in F0 females, and at the two highest doses in F1 females. In males, there appeared to be increases at least at the highest dose, but they were not statistically significant. There were no effects on T3 in any groups.

Consistent liver weight increases were observed at the top dose. There were also weight increases in some of the groups/sexes at the mid dose, most notably in F1 weanlings but not in adult F1. The mid dose (≤101-141 mg/kg/day) can thus be considered a LOAEL for effects on the liver, and the low dose (10-14 mg/kg/day) a very conservative NOAEL. For effects on the thyroid system, the mid dose (≤101-141 mg/kg/day) is a clear effect level (decreased thyroid follicle size and increased serum TSH). Serum TSH seems increased in females also at the low dose, although statistically significant only in F0 females. Still, the low dose is considered a NOAEL.

Mechanistic in vitro/in vivo studies

Studies have indicated that some chemicals may act as endocrine disruptors and modulate the thyroid metabolism. Disturbances in thyroid function may lead to altered energy metabolism and abnormal development (particularly neural and postembryonic development).

Yamada-Okabe et al studied HBCDD *in vitro* for its effect on the thyroid receptor (TR) (Yamada-Okabe *et al.*, 2005). Human liver cells (HeLaTR) were transfected with a plasmid construct containing a luciferase reporter gene and a thyroid hormone response element. HeLaTR cells were chosen as they over-express the human TR α1. Thus, transcription of the luciferase gene was dependent on TR activation. Transfected HeLaTR cells were treated for 2 days with HBCDD (3.12, 6.25, 12.5 and 25 μM), in the presence or absence of T3 (50 ng/ml). The cells were thereafter lysed and analysed for luciferase activity. All the tested concentrations of HBCDD increased TR-mediated luciferase activity significantly (1.6-1.8 fold) in the presence of T3, but the effects were modest in comparison with the effects of 4,4'-diidiodobiphenyl. The effects were not due to toxicity or proliferation, as HBCDD was tested in the MTS-cell growth assay prior to analysis, and concentrations that were cytotoxic were not further tested. The TR is 50 % homologous with the ERα. HBCDD was therefore also tested for ER-mediated gene activation in MCF7 cells, but no such effect was detected. In conclusion, HBCDD modulates TR-mediated gene expression in HeLaTR cells, but only in the presence of T3.

Recently, a similar T3 potentiating effect of HBCDD has been reported in an *in vitro* study using rat pituitary cells (Hamers *et al.*, 2006). In this study, 27 brominated flame retardants (BFRs) were tested for their potential effects as endocrine disruptors. All BFRs were tested for their potency to interfere via five nuclear receptor-mediated pathways; arylhydrocarbon receptor (AhR), androgene receptor (AR), progesterone receptor (PR), estrogen (ER), and thyroid receptors (TR).
HBCDD was evaluated for agonistic and antagonistic interaction with the AhR, AR, PR and ER in the CALUX assay. Basically, the cell lines used were stably transfected with the reporter gene luciferase and a response element for the tested receptor. Cells were treated for 24h with HBCDD (maximum concentration 10-12.5 μM) in the presence or absence of a reference agonist. Thereafter the cells were lysed and the luciferase activity was measured in a luminometer. HBCDD was also tested for its potency to act as a TR agonist or antagonist in the T-screen assay. Thyroid depleted rat pituitary tumour cells (GH3) were treated with

HBCDD (maximum tested concentration 1 μM) or reference compound in the presence or absence of T3 hormone. After 96 h, cell proliferation was determined by measuring the total metabolic activity of the cells by adding resazurin and measuring the fluorescent metabolite in a fluorometer. HBCDD was also tested for cytotoxicity in all cell lines used to ensure that the reported effects were not due to cell death.
Moderate AR and PR antagonistic activity were reported for HBCDD γ (IC_{50}; 3.7 ± 1 and 1.4 ± 0.0, respectively). Very low or no antagonistic activation was detected for the other two receptors, AhR and ER. No agonistic effects of HBCDD were observed for any of the studied nuclear receptors. HBCDD α and γ showed T_3-enhanced activity at 1 μM in the T-screen assay (38±3 and 40 % (n = 1) additional effect, respectively at T3 EC_{50}). This T_3 potentiating effect may lead to hyperthyroidal effects. HBCDD was also tested for inhibition of estradiol (E2) sulfotransferase, but no inhibition was observed. A-HBCDD and β-HBCDD did compete with T_4 for binding to transthyretin (TTR), but with a low potency (EC_{50} = 12-15 μM). In summary, HBCDD exhibited antiandrogenic, antiprogesteronic and T_3-potentiating properties *in vitro*, and a low binding to TTR.

As discussed above, HBCDD have T_3-potentiating properties in the in vitro assays; CALUX transfection assay and T-screen assay. However, none of these assays detect effects resulting from metabolic activation, or are validated according to OECD guidelines. Recently, the Xenopus metamorphosis assay (XEMA-assay) has been proposed in the OECD as an alternative method to address effects of thyroid hormone disruptors.
Thyroid hormone is an important regulator of metamorphosis in amphibian animals, including the transformation of a tadpole into a froglet and regression of the tail. This process was utilised in a bioassay to detect thyroid hormone disruptive effects by HBCDD (Schriks *et al.*, 2006c). Premetamorphic *Xenopus laevis* tadpoles were pre-treated with the goitrogen meithimazole to inhibit TH synthesis prior to isolation of tadpole tips (6-8 mm). The tadpole tips were cultured *ex vivo* in dishes for 24 h, and thereafter exposed to DMSO (solvent control, T_3 alone (20 nM or 100 nM), HBCDD alone (1000 nM or 10000nM) or T_3 (20 nM) in combination with HBCDD (10, 100, 1000, or 10000 nM). On day 6, exposure of tail tips to 1000 nM HBCDD in combination with 20 nM T_3, significantly ($p \leq 0.05$) potentiated tail tip regression with 35 ± 5% relative to the 20 nM T_3- control . All lower HBCDD exposures, including HBCDD alone (1000 nM), did not have any effects on tail tip regression. 10000 nM HBCDD alone or in combination with 20 nM T_3 resulted in a very fast regression of tail tips in the first two days of exposure. This was faster than tail tip regression in the 100 nM T_3-control, but after two days of exposure tail tip regression roughly stayed the same during the rest of the experiment period. According to the author this fast regression was due to cytotoxic activity at this concentration. The T_3-potentiating effect of HBCDD is consistent with previous results obtained from the T-screen assay in the same laboratory (Schriks *et al.*, 2006b).

In many of these *in vitro/ex vivo* assays it is difficult to elucidate if HBCDD act via the thyroid axis, or exert its toxic response through alternative routes. An alternative approach could be to measure cell proliferation in *Xenopus Laevis* tadpoles *in vivo*.
Ten one week old Xenopus laevis tadpoles were placed in petri dishes containing buffer (Marc's modified ringers solution), and exposed to T3 or T3 in combination HBCDD for 8 days. Increasing concentrations of T3 (0-100 nM) were used for dose-response curves, and 1 nM T3 in combination with 10, 100, 100, 1000 nM were used in the HBCDD experiments. 1000 nM HBCDD was also tested in the absence of T3 hormone. The tadpoles were not fed during the exposure period, and after the exposure the animals were fixed in a 4 % paraformaldehyde solution and prepared for immunocytochemistry. The rostal head region

and brain were immonocytochemical labelled with a fluorescent antibody against the phospho-histone H3 mitosis marker, and the proliferating cells were visualized in a fluorescence microscope. The rostal area was defined as the area in front of the brain that includes the majority of the proliferating cells in the head, including the lower jaw and olfactory epithelia.
HBCDD significantly enhanced the number of proliferating cells in the brain at the two highest doses 100 and 1000 nM (in combination with 1 nM T3) with 33.2 % and 24.5 %, respectively. The effect was not dose-related. T3-treatment alone resulted in a dose-related increase in proliferating cells in the brain and rostal head region, with a peak at 1 nM. The effective concentration of HBCDD relative to T3, was a 100-fold higher than T3 (100 nM to 1 nM) compared to 400-fold higher in the T-screen (100 nM to 0.25 nM) (Schriks et al, 2006, chapter 2). The high cell proliferation in the brain correlated very well with results previously obtained using the *in vitro* T-screen assay (T3-mediated cell proliferation) (Schriks et al, 2006, chapter 2). HBCDD did not affect cell proliferation in the absence of T3.

Evaluation of Mode of Action for the effects on the thyroid system

Introduction
Five repeated dose toxicity studies have been performed Table 4-28, each with more or less shortcomings. The only really consistent effect is a liver weight increase in female rats, although most studies also have found liver weight increases in male rats. The discrepancy may be related to the use of different doses and preparations (suspension/solution) in the different studies, but the result could possibly indicate that males are less sensitive than females to the liver weight increase.

It has been suggested that the liver weight increase is caused by hepatic enzyme induction, as indicated by histopathology (proliferation of SER; (Chengelis, 2001)and induced hepatic enzyme activities/mRNA/protein (van der Ven *et al.*, 2006); (Germer *et al.*, 2006). However, 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 (Chengelis, 2001), (van der Ven *et al.*, 2006)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, and will be discussed below basically using a structure that has been proposed by the IPCS when analysing the mode of action for carcinogens. The discussion below will solely be based on the data presented by (van der Ven *et al.*, 2006), as only this study has been designed to allow this analysis.

Table 4-28 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

Postulated mode of action

Hepatic enzyme induction (T4 conjugation) leads to increased excretion of T4, compensatory activation of the pituitary, increased serum TSH concentration and activation of the thyroid. Depending on the magnitude of the T4-decrease, the feed-back system may manage to produce sufficient amounts of T3, or if the reduction is severe, lead to a condition of hypo-thyroidism.

Key events

This MoA is based on the assumption that hepatic enzymes involved in the metabolism of T4/T3 are induced by HBCDD. There should be a reasonable relationship between induction of hepatic enzymes and thyroid effects in both sexes, acknowledging that females are somewhat more sensitive to thyroid effects than males. The van derVen study (van der Ven *et al.*, 2006) has indeed identified induction of T4-UGT transferase by HBCDD, and the induction occurred in both sexes.

Dose-response

The hypothesis postulates that the first effect (occurring at the lowest exposure level) is enzyme induction, followed by activation of the pituitary (resulting in TSH synthesis), activation of the thyroid (hyperactive cells/weight increase), and if the thyroid is incapable of producing sufficient amounts of T4/T3, effects in other tissues/systems regulated by T4/T3.
In females, the BMD-L for induction of T4-UGT is 4.1 mg/kg/day (and LBD/CYP3A4 is induced as from 10 mg/kg/day), the BMD-L for pituitary weight 29.9 mg/kg/day, the (uncertain) BMD-L for thyroid weight 1.6 mg/kg/day (the weight increase seems undisputable at 30 mg/kg/day), and there are histological signs of thyroid hyperactivity at 25, 47, and 177 mg/kg/day for nuclear size, cell height, and vacuolization, respectively. Considering the variation in all data, this sequence of events could support the theory with regard to females.

In males, enzyme induction occurs (the uncertain BMD-L for T4-UGT transferase is 0.1 mg/kg/day; PROD/CYP2B is induced as from 10 mg/kg/day), there are no effects of T4 or the pituitary, but there are histological signs of thyroid hyperactivity at 39, 90, and 199 mg/kg/day for nuclear size, vacuolization, and follicle size, respectively. There are no effects on the thyroid weight.

Strength, Consistency, and Specificity of association of response with key events

There is always some variation in biological data, making firm conclusions difficult. However, the key event (enzyme induction) seems to occur at low exposure in both males and females. In females, the chain of events could support the theory, although the data are not clear-cut. The effect on the pituitary would be expected to appear before effects in the thyroid are evident, but it is acknowledged that if only some pituitary cells are affected, the weight of the whole pituitary will be a rather insensitive parameter. Still, the occurrence of only few and mild effects in males are unexpected, even when considering that the male thyroid system is known to be less affected than the female by chemicals.

Other modes of action

Considering that *in vitro* data indicates an interaction of HBCDD with many different hormone systems, it is possible that HBCDD also could affect the thyroid system via other mechanisms than hepatic enzyme induction. In cell cultures, HBCDD was found to exert antagonistic effects at the progesterone receptor, androgen receptor, and estrogen receptor (IC_{50} 1-5 µM for γ-HBCDD). A low binding of α-HBCDD to the thyroxin-binding transport protein TTR was also indicated *in vitro* (IC_{50} 12 µM). α-HBCDD was a T3-agonist (21 % of maximal T3-effect at 1 µM α-HBCDD) and enhanced T3-dependent effects both in rat cells and *ex vivo* in tadpole tail tips (Hamers *et al.*, 2006), (Yamada-Okabe *et al.*, 2005), (Schriks, 2006a).
It is not likely that these *in vitro* effects can explain all *in vivo* effects on the thyroid system, but theoretically they could be factors contributing to effects on the thyroid hormone homeostasis.

For instance, the binding of HBCDD (or metabolites of HBCDD) to TTR could displace T4 from TTR, making T4 more susceptible to metabolism and excretion. Binding of chemicals to TTR is usually increased by hydroxy-groups (the endogenous ligand thyroxin is hydroxylated), and one may speculate that hydroxy-HBCDD could have a higher binding affinity to TTR than HBCDD itself. OH-HBCDD is formed from HBCDD by microsomes in vitro (Zegers *et al.*, 2004), but it is not known if there is any sex-dependent difference in formation of OH-HBCDD.
A T3-potentiating effect could possibly occur simultaneously as the concentration of T4 is decreased, masking some of the hypothyroidogenic symptoms.

Assessment of postulated MoA
The postulated mode of action is a plausible mechanism, but the lack of effects (other than some effects on thyroid histology) in males in spite of enzyme induction in males is surprising, even though females are known to be more sensitive than males. The big difference between males and females in sensitivity towards effects on the thyroid system could possibly be explained if HBCDD has other effects that contribute to the thyroid effects in females or antagonize the effects in males.

In conclusion: No repeated dose studies with inhalation or dermal exposure as route of administration are available. Five repeated dose studies with oral administration of HBCDD in rats have been conducted; three 28-day and two 90-day studies.
In the first four studies, the rats were dosed with HBCDD particles in suspension, and doses of ≥100 mg/kg/day resulted in a dose-dependent (but reversible) increase in liver weight not accompanied by any clear pathological signs. In addition, the prostate weight was statistically increased in a 90 days study (Chengelis, 2001) at exposure to 1000 mg/kg/day and in a 28-day study (Zeller and Kirsch, 1969) females showed signs of inhibited oogenesis in most of the follicles and sparse ripening follicles in the ovaries at exposure to 4700 mg/kg/day. The effect on the prostate and the oogenesis has not been confirmed in any other studies and the inhibited oogenesis occurred at a very high dose. In addition, the study design does not comply with today´s standards and therefore this result is not used in the risk assessment. All studies showed effects on the thyroid hormone system. A study LOAEL of 100 mg/kg/day is deduced form the recent 90 days study mainly based on relative liver weight increases (18-24 %) although disturbances in the thyroid hormone system (T4 and TSH) also occurred. The study LOAEL is transformed into an internal (systemic) corrected LOAEL of 10-20 mg/kg/day based on an assumed 10-20 % oral absorption of the HBCDD-particles. A 2-generation reproductive toxicity study has also shown the liver and thyroid system to be target organs. However, also in this study HBCDD particles were administered to the rats, although this time mixed into ground food. Because of dosing HBCDD-particles, with the absorption kinetics likely being dependent on particle size and amount of particles administered, the actual doses received at the top doses are uncertain.
The mid dose (≤101-141 mg/kg/day) can thus be considered a LOAEL for effects on the liver, but considering the big dose spacing, the low dose (10-14 mg/kg/day) is a very conservative NOAEL. For effects on the thyroid system, the mid dose (≤101-141 mg/kg/day) is a clear effect level, with decreased thyroid follicle size and increased serum TSH.
A 28 days study (van der Ven *et al.*, 2006), performed by using a benchmark model design and oral administration of dissolved HBCDD, showed effects on the liver, the thyroid, and the pituitary. Overall, a NOAEL/BMD-L of 22.9 mg/kg/day for liver weight increase is

proposed for repeated dose toxicity (van der Ven *et al.*, 2006). Enzyme induction is a likely cause to the liver weight increase, and enzyme induction is clearly relevant also to humans.
A NOAEL from a 90 days study would normally be preferred in the risk characterisation, but the uncertainties introduced in the evaluation of the 90 days study by the dosing of HBCDD-particles to the animals, leads to the choice of a NOAEL form the recent 28-days study.

4.1.2.7 Mutagenicity

In a mutagenicity study, five strains of *Salmonella typhimurium* (TA 1535, TA 1537, TA 1538, TA 98 and TA 100) were tested in the presence and in the absence of a metabolic activation system (from Aroclor induced rat liver). Doses used were 1, 10, 50, 100, 500, 1000 or 5000 µg per plate. HBCDD was neither mutagenic nor cytotoxic in this study (Simmon *et al.*, 1976). Other laboratories have obtained similar results using the same system (Baskin and Phillips, 1977; GSRI, 1979; Zeiger *et al.*, 1987; Ogaswara and Hanafusa, 1993) (Hossack *et al.*, 1978; EPA, 1990a).

HBCDD has been tested in the *in vitro* mammalian cytogenetic test (OECD Guideline 473), according to GLP, using human peripheral blood lymphocytes, from a healthy 40 year adult female, both in the absence and presence of metabolic activation (Gudi and Schadly, 1996). The assay was performed in two phases. The first phase, the initial chromosome aberration assay, was conducted to establish the dose range for testing and to evaluate the clastogenic potential of the test article. The second phase, the independent repeat chromosome aberration assay, was performed to confirm the test system response to the test article seen in the initial test.

In the initial phase, lymphocytes were exposed to nine concentrations of HBCDD ranging from 0.25 to 2500 µg/ml. The exposure period was 20 hours in the absence, and 4 hours (+16 hours recovery) in the presence of metabolic activation. Dose levels of 2500 µg/ml (non-activated), and 750 µg/ml and higher (activated), gave a complete mitotic inhibition. Toxicity (as measured by mitotic inhibition) was also observed at 750 µg/ml (non-activated) and 250 µg/ml (activated). Based on the initial phase the following concentrations were used in an independent repeat chromosome aberration assay: 10, 19, 38, 75, 150, 300 and 600 µg/ml. The lymphocytes were exposed to HBCDD continuously for 20 or 44 hours in the non-activated and for 4 hours in the activated system. Metaphase cells were collected and scored at 20 and 44 hours after initiation of treatment. Toxicity was more than 50 % at the two highest exposure levels. No statistically significant increases in structural chromosome aberrations were observed in either the non-activated or activated studies, regardless of dose level or harvest time. Nor were there any increases in numerical chromosome aberrations. The positive and negative controls fulfilled the requirements for a valid test.

HBCDD was tested *in vivo* for clastogenicity and for the ability induce spindle poison effects in NMRI mice using the micronucleus test method as specified in the OECD Guideline 474 and under GLP conditions (Engelhardt and Hoffman, 2000). For this purpose 90 % pure HBCDD, dissolved in DMSO, was administrated twice intraperitoneally, with a 24-hour interval between administrations, to male animals at dose levels of 500 mg/kg, 1,000 mg/kg and 2,000 mg/kg body weight in a volume of 4 ml/kg body weight in each case. There were 5 animals in each dose group, one vehicle control group and two positive control groups. The administration of HBCDD led to evident clinical signs of toxicity like squatting posture. The

control animals did not show any signs of toxic influence. The animals were sacrificed and the bone marrow of the two femora was prepared 24 hours after the second administration. After staining of the preparations, 2,000 polychromatic erythrocytes were evaluated per animal and investigated for micronuclei. The normochromatic erythrocytes with and without micronuclei occurring per 2,000 polychromatic erythrocytes were also registrated. There was no significant dose-related increase in the frequency of micronuclei in the treated animals as compared to the vehicle controls. The recorded frequencies were within the normal historical control range. The positive controls gave the expected increase in the incidence of micronuclei. Evidence of a slight inhibition of erythropoiesis as reflected by the ratio of polychromatic to normochromatic erythrocytes was recorded at the highest dose, 2,000 mg/kg. Thus, this test shows that HBCDD has no chromosome-damaging (clastogenic) effect nor does it lead to any impairment of chromosome distribution in the course of mitosis.

When investigating the effects of HBCDD on somatic recombination in a non-standard assay using two Chinese hamster cell lines containing duplication mutations in the *hprt* gene, a slight, but statistically significant increase of somatic recombinations was recorded (Helleday *et al.*, 1999). The Sp5 and SDP8 clones used in these assays exhibit a spontaneous partial duplication of the *hprt* gene, resulting in a non-functional HG-PRT protein. These duplication mutants revert spontaneously to a functional hprt gene phenotype by recombination with a frequency of approximately $1x10^{-5}$ reversions/cell generations. The Sp5 clone reverts spontaneously to a functional hprt gene via non-homologous recombination; whereas the SPD8 clone reverts spontaneously via a homologous recombination mechanism.
In the system employing Sp5 cells, HBCDD caused statistically significant (Student`s t-test) increase in the reversion frequency. Linear regression analysis indicated that HBCDD in the SPD8 assay and in the Sp5 assay gave statistically significant, dose-dependent increases in the reversion frequencies. 2.2-fold in the Sp5 assay and 1.9-fold in SPD8 cells. In total, 10 compounds were examined; four of these agents are well known environmental pollutants. In comparison with a clearly recombinogenic agent like Cr (VI), the effect was small, and its biological significance with respect to the *in vivo* situation is not clear.

In conclusion:
The preponderance of evidence from available studies indicates that HBCDD lacks significant genotoxic potential *in vitro* as well as *in vivo*.

4.1.2.8 Carcinogenicity

In a lifetime bioassay, 200 B6C3F1 mice of each sex, divided in four dose groups, were exposed orally to HBCDD for 18 months (Kurokawa *et al.*, 1984). There were three exposure levels: 100, 1000 or 10000 ppm in feed (this is equivalent to about: 13, 130, and 1300 or mg/kg, respectively) and a control group, with 50 males and 50 females at each level.
The HBCDD used in this study was described as a fine white powder, soluble in acetone and xylene, slightly soluble in benzene and olive oil and insoluble in water.

The general condition of the mice was observed every day during the period of administration and the body weight and the consumption of the diet were measured once every week during the first three months of the test, and then once every month. After the administration period the mice were sacrificed by exsanguination under ether anaesthesia. The following organs were collected for histopathological examination: brain, heart, lung, liver kidneys spleen, testicles, epididymis, seminal vesicles, uterus, ovaries, pituitary, thyroid, submandibular gland, thymus, adrenals, oesophagus, stomach, duodenum, jejunum, ileum, rectum, pancreas,

urinary bladder, mesenteric lymph nodes, sternum and femur. These organs were fixed with 10 % neutral formalin solution. Paraffin-embedded sections of the organs and tissues were prepared by the conventional method, and stained with H-E (hematoxylin-eosin) for histopathological examination. Similar treatments and examinations were performed on animals that died during the test period.

The survival was high, and there was no difference in mortality between the groups. The body weight was lower in the exposed groups but no apparent signs of overt toxicity were reported that could be related to the administration of HBCDD. 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 (which may be caused by a low absorption of the HBCDD particles especially at the highest concentrations of the HBCDD in the feed), 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, see Table 4-29 (Haseman *et al.*, 1984).

Table 4-29 Findings (number of animals responding) in the 18-month carcinogenicity study on mice.

Examination, Organ		Exposure concentration mg/kg bwt/day							
		Control group		13		130		1300	
	Sex	M	f	M	f	m	f	m	f
	No of killed	45	48	47	49	45	49	45	49
	No of died	5	2	3	1	5	1	5	1
Gross findings	Liver nodules	12	2	21	1	30	5	24	6
	Liver, small ponctate on surface	5	2	7	0	12	0	5	0
	Lung nodules	6	1	7	1	3	2	1	1
	Uterus swelling	-	31	-	27	-	24	-	3
	Ovary cyst	-	1	-	0	-	4	-	4
Histopat. findings, Liver	Hepatocellular carcinoma	12	0	17	0	25	1	13	5
	Hemangioma	2	1	2	0	2	0	0	1
	Altered foci	20	12	20	6	41	10	25	10
	Hepatocytic swelling	29	37	31	39	43	41	39	42
	Necrosis	14	12	19	6	27	10	15	8
	Vacuole formation	8	17	19	18	4	18	4	9
	Fatty infiltration	0	0	3	0	9	0	4	0
	Cyst	3	0	5	0	7	0	3	0
	Mixed vacuoli.+fatty change	8	17	9	18	31	20	20	28
Histopat. findings, Lung	Adenoma	5	1	9	5	8	3	5	2
	Lymph node hyperplasia	11	13	5	5	2	4	2	1
Histopat. findings, Kidneys	Cell infiltration	23	20	17	26	11	6	16	6
Histopat. findings, Uterus	Cystic change	-	31	-	27	-	21	-	22

Data from (Kurokawa *et al.*, 1984)

m = male

f = female

No of killed = the number of animals that survived the entire exposure period

No of died = the number of animals that died during the exposure period

In conclusion:
This study is not reported according to current guidelines, it is only available as a study summary lacking significant details. The study might have been performed according to current guidelines, but there is no such information. The summary does not say anything about the uptake, the test substance purity or the particle size. Further, the pathological examination is not described and reported in detail as it should be according to guideline 451. The conclusion is that no adequately performed cancer study has been reported for HBCDD. Still, based on the present brief report and the lack of mutagenicity, there is no reasons to explore this endpoint further.

4.1.2.9 Reproductive toxicity

Fertility

A two-generation reproductive toxicity study in rats has been conducted (Ema et al., 2008). The study was performed according to OECD guideline 416 and in accordance with the principles for good laboratory practice.
The test substance was a composite of HBCD commercial products from three leading producers and the preparation was a mixture of three enantiomers, HBCD-α, -β and –γ, and their respective proportions in the used batch were 8.5, 7.9 and 83.7%. The test substance was 99.7% pure.
One hundred and ninety-two Crl:CD(SD) rats were randomly assigned in four groups, 24/sex/group as F0 animals. Animals were housed individually, except during acclimation, mating and nursing periods. Rats were given dietary HBCDD at a concentration of 0, 150, 1500 or 15000 ppm, which numerically has been translated to 10-14, 101-141, or 1008-1363 mg/kg/day in low, mid, and high dose animals with the low end of the ranges representing males and the high females. The daily intake of food increased during lactation, leading to daily intake of 20-23, 179-240, and 1724-2200 mg/kg/day in dams of the low, mid, and high dose groups, respectively. Diet preparations were formulated by mixing HBCDD particles into an appropriate amount of a powered diet for each dietary concentration. Administration was continued for 10 weeks prior to the mating period, throughout the mating, gestation and lactation periods.
Twenty-four male and 24 female weanlings in each group were selected as F1 parents on PNDs 21-25. The day on which F1 parental animals were selected was designated as 0 week of dosing for the F1 generation. F1 selected rats were administered HBCDD as described for F0 rats. On PND 26, unselected F1 weanlings and all F2 weanlings were necropsied.
Each female was mated with a single male of the same dosage group until copulation occurred or the mating period (three weeks) had elapsed. During the mating period, vaginal smears were examined for the presence of sperm, which was considered as evidence of successful mating. Successful mating was designated as day 0 of pregnancy. For F1 matings, cohabitation of siblings was avoided.
All adult rats were observed twice a day for clinical signs of toxicity, bodyweights and food consumption were recorded weekly. For females exhibiting evidence of successful mating, body weight and food consumption were recorded on days 0, 7, 14, and 20 of pregnancy and days 0, 4, 7, 14, and 21 for lactation. Daily vaginal lavage samples of each F0 and F1 female were evaluated for estrous cyclicity. After weaning their pups, parental female rats were necropsied at the proestrous stage of the estrous cycle. For each female, the number of uterine implantation sites was recorded.

Once insemination was confirmed, female rats were checked at least three times daily on days 21-25 of pregnancy to determine the time of delivery. The females were allowed to deliver spontaneously and nurse their pups until PND 21. The day on which parturition was completed was designated as PND 0. Total litter size and the numbers of live and dead pups were recorded, and live pups were counted, sexed, examined grossly, and individually weighed on PNDs 0, 4, 7, 14, and 21. On PND 4, litters were randomly adjusted to 8 pups comprising of 4 males and 4 females. No adjustment was made for litters of fewer than 8 pups.
All F1 and F2 pups were observed for pinna unfolding on PND 3, incisor eruption on PND 11, and eye opening on PND 14. One male and one female F1 and F2 pup selected from each dam were evaluated for the surface righting reflex on PND 5, negative geotaxis refex on PND 8, and mid-air righting reflex on PND 18. All F1 offspring selected as F1 parents were observed daily for male preputial separation beginning on PND 35 or female vaginal opening beginning on PND 25. Body weight of the respective F1 rats was recorded on the day of preputial separation or vaginal opening. The anogenital distance (AGD) was measured using callipers on PND 4 in all F1 and F2 pups, and the normalized value of AGD to body weight, AGD per cube root of body weight ratio, was calculated.
Spontaneous locomotor activity was measured with a multi-channel activity monitoring system in 10 male and female F1 rats selected from each group at 4 weeks of age. Rats were placed individually in transparent polycarbonate cages, which were placed under an infrared sensor that detects thermal radiation from animals. Spontaneous motor activity was determined for 10 min intervals and for a total of 60 min. A test in a water-filled multiple T-maze was conducted in 10 male and 10 female F1 rats selected from each group at 6 weeks of age. The water temperature of the maze was kept 21-22°C. The elapsed time between entry into the water at the starting point and touching the goal ramp and number of errors were recorded.
Parental rats were necropsied: males after the parturition of paired females, females after weaning of their pups. A complete necropsy was performed on all rats found dead and those killed at the scheduled sacrifice. Weights of the brain, pituitary, thyroid, thymus, liver, kidney, spleen, adrenal, testis, epididymis, seminal vesicle, ventral prostate, uterus, and ovary were recorded. Weights of the thyroid and seminal vesicle were measured after fixation.
Histopathological evaluation of F0 and F1 adults was performed on the tissue specified below after fixation, paraffin embedding, and sectioning and staining with hematoxylin and eosin: the pituitary, liver, thymus, kidney, spleen, adrenal, bone marrow, mesenteric lymph node, Peyer´s patches, testis, epididymis, seminal vesicle, coagulating gland, ventral prostate, ovary, uterus, vagina and mammary gland of all male and females in the control and highest dose (15000 ppm) groups and of females with abnormal estrous cycles, males and females without evidence of copulation or insemination and females with abnormal delivery or totally dead pups in all groups. Any organs or tissues of F0 and F1 adults showing gross alterations were evaluated histopathologically. The thyroid in all rats in all groups was examined histopathologically. In ten F1 females of each group, the number of primordial follicles was counted. The right ovary was fixed in 10% neutral buffered formalin and dehydrated and embedded in paraffin in a longitudal orientation by routine procedures. Sections were cut serially at five µm and every 20th section was serially mounted on a slide and stained with hematoxylin and eosin. About 40 sections per ovary were used to determine the primordial follicles.
Following the adjustment of litter size on PND 4, culled pups were euthanized. No tissues from these pups were collected.
The weanlings not selected to become parents were euthanized and necropsied as described for the adults. Organ weights of one male and one female F1 and F2 weanling selected from

each dam were measured as described for adults. The weights of the pituitary, thyroid and seminal vesicle were not determined. All pups found dead before weaning were also necropsied. In all male and female F1 and F2 weanlings whose organs were collected, histopathological evaluations of the liver, in the control and 15000 ppm groups, and thyroid, in all groups, were performed. On the day of the scheduled sacrifice, blood samples were collected. Hematological examinations and blood chemical evaluations were performed for 10 males and 10 females of F0 and F1 rats randomly selected from each group. Eight males and eight proestrous females of F0 and F1 generations from each group were selected randomly for blood collection. Serum levels of several hormones were measured with a radioimmunoassay kit.
Sperm parameters were determined for all F0 and F1 male adults on the day of the scheduled sacrifice. The right testes were used to count testicular homogenization-resistant spermatid heads. The right cauda epididymis was weighed and used for sperm analysis. Sperm motility and the percentage of motile sperm and progressively motile sperm, and the swimming speed and pattern were determined. After recording sperm motion, the cauda epididymal fluid was diluted and the sperm were enumerated. Sperm count per gram of epididymal tissue was obtained, and the percentage of morphologically abnormal sperm was calculated.

General observations: Unscheduled deaths and euthanasia due to moribund condition were noted in a few animals, but these events were inconsistent across doses, sex and generations, and not thought to be attributed to the administration of HBCD. Body weight was significantly decreased (9-23%) compared to controls at 15,000 ppm in F1 male weanlings and F2 male and female weanlings, but the decrease was not seen in adult F1 animals.

Reproductive effects: In F0 females, HBCD produced no significant effects on the estrous cycle, although a few control and HBCD-exposed animals had an extended estrous or diestrous. Copulation was not observed in two male and two female F0 animals at 1500 ppm, and in two males and one female at 15,000 ppm. Two of the mated females at 150 ppm and four each at 1500 and 15,000 ppm did not become pregnant (Table 4-30.). There was thus a seemingly dose-dependent decrease in fertility index in both F0 and F1, although only statistically significant in F0. It should be noted that the fertility index includes effects both on copulation ability and impregnation ability. At 1500 ppm there was a significantly longer gestation length and lower sex ratio in F0 animals compared to controls. Total litter loss was observed in one F0 dam at 15,000 ppm by day 5 of lactation. In the sperm analyses, F0 males at 15,000 ppm had a higher mean amplitude of lateral head displacement compared to controls.

In F1 animals, all pairs in all dose groups copulated, but one female each at 0 and 150 ppm, and three each at 1500 and 15,000 ppm, were not impregnated (Table 4-30.). At 1500 ppm, one female did not deliver pups. Total litter loss was seen in one dam in the control group (by day 4 of lactation), and in one dam in the 150 ppm group (by day 2 of lactation). Eight F1 dams in the 15,000 ppm group experienced total litter loss at different days of lactation (up until day 18) without clear signs of toxicity. At 15,000 ppm, a significantly decreased viability index was seen in F2 pups on PND 4 and 21. Mean body weights were significantly lowered at 15,000 ppm in male F2 pups on PNDs 7, 14 and 21, and in female F2 pups on PNDs 4, 7, 14 and 21, compared to controls.

No significant effects between HBCD-treated animals and controls were seen in copulation index, gestation index, pre-coital interval, number of implantations, delivery index or number of pups delivered in either F0 or F1 animals.

Table 4-30. Fertility index (%) in male and female animals, no. of pregnant females and no. of litters in the F0 and F1 parental generation, respectively, in control and HBCD-exposed animals.

	Dose (ppm)			
	0	**150**	**1500**	**15,000**
F0 parent/F1 offspring				
Fertility index in male/female (%)	100/100	91.7/91.7	90.9/90.9	85.7/86.4
No. of pregnant females/total no. females	24/24	22/24[a]	20/24[a]	19/23[a]
No. of litters/total no. females	24/24	21/24	20/24	18/23
F1 parent/F2 offspring				
Fertility index (male/female) (%)	95.8/95.8	95.8/95.8	87.0/87.5	87.5/87.5
No. of pregnant females/total no. females	23/24	23/24	21/24	21/24
No. of litters/total no. females	23/24	23/24	20/24	21/24

a: The rapporteur has performed a statistical analysis of the fertility index. Using the Cohran-Armitage test on the number of pregnant females vs. the total number of females for the F0 parent generation results in an exact one-sided P-value of 0.02, indicating a significant trend.

*Necropsy and histopathology(F0, F1 and F2 animals):*In F0 and F1 adult animals, no compound-related gross lesions and microscopic alterations could be found in any organs except for the thyroid where a decreased size of follicles could be seen, see section 4.1.2.6. In F1 females at 1500 and 15,000 ppm, the number of primordial follicles in the ovary was significantly decreased compared to controls (Table 4-31). Although there is no clear dose-response, the decreases in the two top doses are of such a magnitude (-30 %) that the effect has to be considered. The primordial follicle pool is formed just after birth in rodents (Mc Gee & Hsueh, 2000; Skinner, 2005) and is, according to current knowledge, not renewable. Hence a decreased number of primordial follicles can lead to a decreased fertility, and the decrease in fertility index seen in F1 animals at 1500 ppm and above could be a consequence of the significant decrease in number of primordial follicles (only measured in F1 animals) at the same dose levels.

Table 4-31 Number of primordial follicles in the ovary in F1 females of the control and HBCD-treated groups, respectively.

Dose (ppm)	No of follicles
0	316.3 ± 119.5
150	294.2 ± 66.3
1500	197.9 ± 76.9*
15,000	203.4 ± 79.5*
Historical controls[1]	295.6 (189.5-353.4)

* = significantly different compared to controls ($p<0.05$)

[1] The historical control data is taken from studies performed in the laboratory that performed the current study and is based on data from a total of 100 female rats (10 studies with 10 animals each performed 2005-2006).

*Organ weights:*In F0 males at 1500 and 15,000 ppm, the relative weight of the seminal vesicle was decreased (magnitude not given), and in F0 females at 15,000 ppm significant increases

were found in the absolute weight of the adrenal. A significant increase in absolute and relative testis weight in F1 male weanlings at 150, 1500 and 15,000 ppm was observed, but this was not seen in F2 males or in F1 adults, and is considered a chance finding. In F2 male weanlings, significantly decreased absolute weights of the adrenal, epididymis and ventral prostate was seen at 15,000 ppm. In F2 female weanlings, there were significantly reduced absolute weights of the adrenal and uterus at 15,000 ppm, and increased relative weights of the ovary at 150 and 15,000 ppm. Absolute, but not relative, brain weight was decreased in the highest dose group in F1 and F2 male and female weanlings, and in F1 adults of both sexes. There were changes in weight in other organs, but these were not consistent, and there is no clear pattern in the weight changes in the reproductive organs either. Hence, we have not used these results in the determination of the NOAEL. Absolute and/or relative liver and thyroid weights were increased in all generations in both sexes, see section 4.1.2.6.

Developmental landmarks and behavioural effects: Some effects were seen on the completion of eye opening and the surface righting reflex response in F1 and F2 pups, but this was not consistent over generations or sexes and hence was not considered substance-related. A significantly lower completion rate of mid-air righting reflex was seen in F2 female pups at 15,000 ppm (76.9% vs. 100% in controls). There was no significant difference compared to controls in F1 and F2 pups in the AGD or AGD per cube root of body weight ratio, nor any difference in age at preputial separation in males or vaginal opening in females, or body weight at preputial separation or vaginal opening. In the behavioural tests, there was no significant difference between groups that could be associated with exposure to HBCD.

Serum hormone levels: There were significantly increased levels of TSH in F0 females at 150 ppm and higher (~35-100%), and in F1 females at 1500 ppm and higher (~80%). In F0 males and females, lower levels of T4 compared to controls were seen at 15,000 ppm. There were no effects on T3 in any groups. Serum FSH levels were significantly decreased in F0 males at 1500 ppm, and significantly increased in F0 females at 15,000 ppm. Levels of DHT were significantly higher in F1 males at 1500 ppm. There were no significant differences in serum testosterone, estradiol, progesterone and LH levels in HBCD-treated F0 and F1 animals compared to controls. The effect on TSH levels is consistent through dose groups and generations, and is considered an effect of HBCD-expsosure, while no clear pattern can be seen for the other changes in hormone levels.

Conclusion: 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. There were indications of effects on liver and thyroid weights (section 4.1.2.6). The effect of HBCD 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. In our assessment, we have chosen to deal with this endpoint as an indication of effects on fertility. 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 this study.
Considering that the dose-spacing in the study (nominally) was a factor of 10, the observed dose-responses are surprisingly flat. However, since the animals were dosed with HBCDD-

particles mixed in the feed, and that the particle size in general affects the absorption (rate), the actual systemic doses are uncertain. Consequently, the systemic doses in the mid and high dose groups are likely to be lower than the calculated nominal doses, which may be related to the observed rather flat dose-response relationships. A NOAEL of 10 mg/kg/day is suggested to be used in the RC for the fertility end-point.

In conclusion:

A NOAEL of 10 mg/kg/day has been deduced in a two-generation reproductive toxicity studie in rats. The NOAEL is based on dose-dependent decrease in fertility-index and a reduced number of primordial follicles.

Developmental toxicity

A developmental toxicity study was carried out by using 80 non-pregnant female Wistar rats, divided in four groups (Murai *et al.*, 1985).

In the main developmental study the 20 rats, of each dose group, were given 0, 0.01 %, 0.1 % and 1 % HBCDD in the diet during day 0-20 of gestation. Thus, this study also covered potential effects during the preimplantation stage. The doses are approximately equivalent to 0, 7.5, 75, and 750 mg/kg/day, respectively. These calculations are based on assumptions that the animals mean weight is 200g and their food consumption is 15 g/day. On day 20 of gestation 14 rats per group were killed by cervical dislocation, and served for abdominal surgery to visual observation of major organs. Organs were weighed. Numbers of corpora lutea, implants, resorptions, and live foetuses were measured. The incidence of abnormalities on external examination was examined, sex was identified and both body weight and placental weight were measured for each live foetus. 1/3 of the foetuses were examined for visceral anomalies and 2/3 of the foetuses were examined for skeletal abnormalities. The remaining 6 dams per group were delivered naturally, and the pups monitored through weaning. Number and body weight of delivered foetuses, number of live foetuses, and abnormalities resulting from external examination was recorded. From the third week, males and females were separated, and the new-borns growth and survival was observed until the 7th week. In the highest dose group the maternal food intake was slightly suppressed, and both the absolute and relative maternal liver weights were significantly increased by 13 %. The mothers showed no signs of toxicity, nor were body-weight gain affected. No significant changes in number of implants, number of resorbed, dead or live foetuses was reported, or external, visceral or skeletal anomalies of foetuses that could be attributed to exposure to HBCDD. No difference was seen in the number of live new-borns, or in the number of dead new-borns, and no abnormalities were observed based on external examination. No abnormality of newborns was observed during parturition, during the weaning period, and after the weaning period. Normal body weight changes of both male and females of each administration group were observed. No significant difference in either the weaning index or the survival index, obtained when the experiment was over, was observed between administration groups and the control group. Although a somewhat lower number of animals per dose level was used (14) in comparison with the 20 individuals called for in OECD Guideline 414, about 150 foetuses per dose level were examined without any adverse effects being detected, and it is highly unlikely that the inclusion of an additional 6 dams per dose group would have given a significantly different result. This study gives a foetal NOAEL of 750 mg/kg/day, the highest dose tested, and a maternal NOAEL of 75 mg/kg/day, with a liver weight increase by 13 % and the next higher dose.

The lack of fetotoxic and teratogenic potential found in the above mentioned study has been confirmed in a recently performed investigation that fully complies with OECD Guideline 414 as well as with U.S. EPA Health Effects Test Guidelines OPTS 870.3700, "Prenatal Developmental Toxicity Study "(Stump, 1999). The material used consisted of a composite of the commercial products from three different producers. As determined by NMR, the material consisted of 90 % HBCDD diastereomers (about 6.4 % α-, 4.5 % β-, and 79 % of the γ-diastereomer, plus 0.5-0.9 % tetrabromocyclododecane and about 9 % unknown constituents) that were found to be stable during the time of the study. Three groups of 25 female Charles River CD rats (Crl:CD(SD)IGS BR) were administered 0, 500, or 1000 mg HBCDD per kg and day orally in corn oil (suspension with a mean particle size of 142 µm) once daily from gestation days 6 through 19, whereby clinical observations, body weights and food consumption were recorded. All animals survived until sacrifice on gestation day 20. Body weight gain and food consumption were not adversely affected at any dose level, and no significant clinical signs were observed. However, histopathological analyses of selected organs of the dams were not performed. On day 20 all maternal animals were subjected to laparohysterectomy, and uteri and ovaries were examined as well as the number of foetuses, early and late resorptions, total implantations and number of corpora lutea. Mean gravid uterine weights and net body weight changes were assessed. The foetuses were weighed and examined for external soft tissue and skeletal malformations as well as variations. At necropsy, no treatment-related clinical signs were observed at any dose level. Intrauterine growth and survival were unaffected, and no treatment-related foetal malformations or developmental variations were observed in any of the treated groups. In the 500 mg/kg/day group, one foetus had a facial cleft as well as exencephaly and another foetus exencephaly. However, since no malformations were observed in the 1000 mg/kg/day group foetuses, these malformations were not considered treatment related. There were no soft tissue malformations in any of the examined foetuses, but soft developmental variations were observed in one foetus in each of the 500 and 1000 mg/kg/day groups. However, these single occurrences cannot be considered to have any significance. Skeletal variations occurred in all dose groups as well as in controls and consisted primarily of unossified sternebrae (2 foetuses), ossified cervical centrum (1) as well as rudimentary ribs (2). Based on this study a foetal and maternal NOAEL of 1000 mg/kg/day may be determined.

A two-generation study in rats was conducted by Ema et al (2008). The study is described in detail above in the fertility section (4.1.2.9). Rats were given dietary HBCDD at a concentration of 0, 150, 1500 or 15000 ppm, which numerically has been translated to 10-14, 101-141, or 1008-1363 mg/kg/day in low, mid, and high dose animals with the low end of the ranges representing males and the high females.

In F0 females at 1500 ppm, there was a significantly longer gestation length and lower sex ratio in F0 animals compared to controls. There was no significant difference compared to controls in F1 and F2 pups in the AGD or AGD per cube root of body weight ratio, nor any difference in age at preputial separation in males or vaginal opening in females, or body weight at preputial separation or vaginal opening. Total litter loss was observed in one F0 dam at 15,000 ppm by day 5 of lactation. Body weight was significantly decreased (9-23%) compared to controls at 15,000 ppm in F1 male weanlings and F2 male and female weanlings, but the decrease was not seen in adult F1 animals.

In F1 animals, one female from the 1500 ppm group did not deliver pups. Total litter loss was seen in one dam in the control group (by day 4 of lactation), and in one dam in the 150 ppm group (by day 2 of lactation). Eight F1 dams in the 15,000 ppm group experienced total litter loss at different days of lactation (up until day 18) without clear signs of toxicity. At 15,000 ppm, a significantly decreased viability index was seen in F2 pups on PND 4 and 21. Mean body weights were significantly lowered at 15,000 ppm in male F2 pups on PNDs 7, 14 and 21, and in female F2 pups on PNDs 4, 7, 14 and 21, compared to controls.

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, it indicates a NOAEL of 150 ppm (10 mg/kg/day) for pup mortality in this study.

In conclusion: Two ordinary developmental toxicity studies have failed to demonstrate any fetotoxicity, teratogenic potential or adverse effects from HBCDD on development of rats. However, an increased pup mortility during lactation was observed in a 2-generation study.

Developmental neurotoxicity

A neurotoxicity study investigated if HBCDD exposure during brain development affected spontaneous behaviour, learning or memory later in life (Eriksson *et al.*, 2006).

Neonatal male NMRI mice were exposed on day 10 to HBCDD [0.9 or 13.5 mg (1.4-21μmol)/kg body weight] as a single oral dose by gavage (8-10 animals/dose group). The substance was dissolved in a mixture of egg lecithin and peanut oil. The day of administration (postnatal day 10) corresponds to the peak in rodent brain growth spurt. At the age of three months, the mice were observed regarding spontaneous behaviour, learning and memory capability. Spontaneous behaviour was studied in a total of 10 animals from three different litters on a once-only occasion. The animals were placed in cages and locomotion (horizontal movement), rearing (vertical movement) and total activity (all types of vibrations within the test cage) were monitored for 3×20 minutes. Movements were measured by infrared beams that intersected the cages. Vibrations were measured by a sensory pickup. Learning and memory were observed in a Morris water maze (Morris, 1981) where a total of 12-17 mice, from three different litters, were observed trying to find a submerged platform for four consecutive days (5 trials/animal/day). On the fifth day, the platform was moved, and the re-learning ability of finding the platform was registered. It is unclear whether different animals from the same dose group were tested on different days in the water maze. Spontaneous behaviour of mice exposed to HBCDD (both dose groups) was significantly altered, manifested as reduced habituation with initial hypoactivity followed by hyperactivity in a novel environment, as compared to controls. In the high-dose group (13.5 mg/kg), all three parameters (locomotion, rearing and total activity) were significantly affected during the first and the last 20-minute-period, where hypo- and hyperactivity, respectively were demonstrated, as compared to controls. The results for the high-dose group are only presented in graphs. The locomotion and total activity during the first 20-minute-period are estimated to 50 % of control values, and rearing was even more affected with approximately 6 times less activity compared to controls. In contrast, total activity during the last 20-minute-period was twice as high in the high-dose animals, than in the control mice and differences were even greater for locomotion and rearing. In the low-dose group (0.9 mg/kg), locomotion and

rearing was significantly decreased by a factor of approximately 1.3, during the first 20-minute-period, but there were no effects in the later measurements or in total activity.

The ability to find the platform in the water maze improved in all mice (exposed and controls) over the four-day trial. However, the mean latencies in finding the platform on the fourth day were significantly longer in the high dose group compared to the lower dose group and to controls. The latency periods were estimated, from the presented graphs, to be 20-30, 15 and 10 seconds, respectively, in the high-dose, low-dose and control groups. Moreover, the re-learning ability on day five was impaired in high-dose animals with significantly longer latency periods (20-30s) compared to controls (10-20s). Mice exposed to 0.9 mg HBCDD/kg did not differ from controls in the water maze experiment.

The effects observed in this study were increased in a dose-related fashion although only two exposure groups were used, and the behavioural alterations were induced at doses that did not affect the weight gain or evoke any clinical signs of ill-health. The mice were exposed during the peak period of rapid brain growth, known as the "brain growth spurt" (BGS). During the BGS, the brain undergoes several fundamental phases, such as dendritic and axonal outgrowth and establishment of neural connections. Whereas this period is neonatal in rats, spanning the first 3-4 weeks of life, this period begins during the third trimester of pregnancy in humans and continues throughout the first two years of life.

Although the study does not follow the current guidelines, it is well performed. The laboratory has a good repeatability for control values and for relevant active substances tested several times. Previous studies with known neurotoxic agents, such as PBDEs and certain PCBs have shown that the experimental model is very sensitive. The study focus on effects on a population (infants/newborn) believed to be the most susceptible to this type of effects as well as the most exposed to highly lipophilic substances (e.g. via mother's milk). The LOAEL-value will be used as if representing repeated exposure. Overall, the study seems adequate for setting an indicative LOAEL. However, as discussed in the ESR-program for other substances tested in this model, the results need to be confirmed by other laboratories, and a conclusion (*i*) is therefore proposed for this endpoint. Thus, the results indicate that HBCDD can cause developmental neurotoxic effects at low exposure levels, with clear effects on all parameters at 13.5 mg/kg, and on some at 0.9 mg/kg, giving an indicative LOAEL of 0.9 mg/kg/day.

In a two-generation study in rats by Ema et al (2008), some developmental parameters were studied. The study is described in detail above in the fertility section (4.1.2.9). Rats were given dietary HBCDD at a concentration of 0, 150, 1500 or 15000 ppm, which numerically has been translated to 10-14, 101-141, or 1008-1363 mg/kg/day in low, mid, and high dose animals with the low end of the ranges representing males and the high females.

Some effects were seen on the completion of eye opening and the surface righting reflex response in F1 and F2 pups, but this was not consistent over generations or sexes and hence was not considered substance-related. A significantly lower completion rate of mid-air righting reflex was seen in F2 female pups at 15,000 ppm (76.9% vs. 100% in controls). In the behavioural tests, i.e., assessment of spontaneous locomotor activity and how the animals handle a water-filled multiple T-maze, there was no significant difference between groups that could be associated with exposure to HBCD.

A study according to OECD guideline 415, using a benchmark design, was conducted to investigate the neurotoxicity of HBCDD in rats. The study is part of the FIRE- project, but the study is still only reported as an extended conference abstract (Lilienthal *et al.*, 2006). Wistar rats (number not reported) received HBCDD in the diet, and the exposure started before conception and was continued throughout mating, gestation, lactation, and after weaning of the offspring. There were eight dose groups; vehicle, 0.1, 0.3, 1; 3, 10, 30, or 100 mg/kg bwt. No data on reproductive outcome was presented.
The toxicological database for HBCDD contains effects on circulating thyroid hormones, and studies indicate that an insufficient supply with thyroid hormones in the early postnatal period may affect the function of the apical part of the cochlea, and thus hearing in the lower frequency range. A test called brainstem auditory evoked potentials (BAEPs) was therefore selected to study this effect further.
In addition, effects on the dopamine system were studied as an *in vitro* study has indicated impairment of dopamine uptake into synaptosomes preparations (see below). Haloperidol is a substance that blocks a variety of receptors in the brain, particularly dopamine receptors. Therefore, haloperidol-induced catalepsy was selected for investigation of HBCDD effects on the dopamine uptake in the nervous system in vivo. The dopamine uptake was estimated by measuring cataleptic behaviour, as defined as bar, grid, and box.

Effects of HBCDD were observed in the lower frequency range as measured by the BAEP test. The effects were only observed in male offspring. No progressive delays in peak latencies were detected in later waves of the BAEP, indicating a cochlear origin of the hearing impairment. The lowest BMD-L values were 0.2 and 0.9 mg/kg bwt for thresholds at 0.5 kHz and for clicks, respectively. BMD-L values for wave II latency were approximately 30 mg/kg bwt for clicks and about 40 mg/kg bwt at 1 kHz. In contrast to earlier studies, no effects on circulating thyroid hormones were detected in littermates in the present study.
In the catalepsy test, HBCDD exposure resulted in decreased latencies to movement onset in all three situations used to measure cataleptic behaviour, namely, bar, grid, and box. The lowest BMD-L values were approximately 3 mg/kg for foreleg retraction in male offspring in the box. In females, these values were 0.6 mg/kg bwt for foreleg retraction in the box and about 4 mg/kg on the grid and the sum of latencies in all three situations. This outcome may according to the author be due to HBCD-related induction of hepatic xenobiotic metabolizing enzymes in the liver, resulting in enhanced metabolism of haloperidol, or/and to lower dopaminergic activity in the brain. Too little information is available from this study for an independent review of its relevance.

Mariussen and Fonnum (2002), studied the plasma membrane uptake of the neurotransmitters dopamine, glutamate and γ-amino-*n*-butyric acid (GABA) in rat brains. Male Wistar rats (150-200g) were killed by decapitation and the brains were quickly removed and kept on ice. The brains were homogenised in sucrose, centrifuged, and the supernatant was mixed with sucrose and centrifuged a second time to get a crude synaptosomal pellet without myelin. The pellet was pre-incubated at 25 ºC for 15 min in absence or presence of HBCDD in Tris-Krebs buffer. HBCDD was tested in four different concentrations (2-20 μM). The reaction was started by adding substrate containing either ^{3}H-glutamate, ^{3}H-GABA or ^{3}H-dopamine, and terminated by a bovine serum dilution, and rapid filtration into a glass-fibre filter mat. The filters were dissolved and counted for retained radioactivity in a liquid scintillation spectrophotometer. Blanks were treated similarly.
HBCDD inhibited neurotransmitter uptake into synaptosomes and dopamine uptake into synaptic vesicles.

The dopamine uptake was inhibited at low concentrations with an IC_{50} value of 4 ± 1μM. HBCDD also inhibited glutamate uptake at low concentrations with 26 ± 9 % inhibition at 1 μM, but it never achieved more than 40 ± 6 and 50 ± 4 % inhibition at 20 and 50 μM, respectively. The study does not report any effects concerning GABA inhibition. Glutamate uptake was inhibited to an equal extent in synaptosome fractions from cerebellum and forebrain. Kinetic studies of HBCDD indicated that HBCDD inhibit dopamine non-competitively.

HBCDD inhibited the high affinity uptake of neurotransmitters into synaptosomes at similar concentration levels as previously shown for polychlorinated biphenyls (PCBs) (Mariussen and Fonnum, 2003). The present study indicates that HBCDD might have a neurotoxicological potential but the results are not taken forward to the risk assessment.

In conclusion: A recent study has indicated 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 they can be used in the RC for the end-point developmental neurotoxicity. Thus, a conclusion (i) is proposed.

4.1.3 Risk characterisation

4.1.3.1 General aspects

4.1.3.1.1 Physical/chemical properties

HBCDD is a solid substance at room temperature and is mostly handled in industry as a solid powder (mean size of 2-5 μm or 20-150 μm), pelletised (560-2400 μm) or as a masterbatch. The vapour pressure is low and the density is high. HBCDD decomposes at temperatures above 230 ºC. The information available shows that HBCDD is not readily biodegradable, and that the substance may accumulate in biota.

4.1.3.1.2 Life-cycle stages

The sole known use of HBCDD is as a flame retardant. The main downstream uses of HBCDD are in the polymer (mainly polystyrene) and textile industries (in polymer dispersion). The flame retardant is not chemically reacted, but physically bound within the polymer matrix, and therefore has a potential for migration, and consequently also for diffuse emissions. The end-products are to a large extent built into the society, where they have a very long 'half-life'. After their use, they are disposed, mainly by incineration, recycling or by landfill.

4.1.3.1.3 Exposure

Although humans may be exposed to HBCDD at the workplace, from use of consumer products, and indirectly from the environment via food, soil, water and air, the highest exposures undoubtedly occur in the workplace environment. Still, there are no occupational exposure limits for HBCDD. Diffuse releases from sources such as use of end-products and from disposal are difficult to estimate, but could possibly be important in a longer time perspective. More information on the degradation of HBCDD under various environmental conditions has shown that whereas reductive debromination of HBCDD occurs in anaerobic compartments (e.g., anaerobic sediment) no biodegradation occurs in aerobic environments (e.g., soil), indicating that there is a big potential for indirect exposure to this substance.

The release of HBCDD, principally as particles, when dismantling insulation boards containing HBCDD or pulling down houses with this kind of insulation, has not been considered because of the substantial uncertainties in estimating how carefully and by whom (professionals or private citizens) these activities will be performed in the future. This might be a considerable source of release in the future when the technical lifetime of these products runs out, and thus affect the future exposure of man via the environment.

There are generally few measured HBCDD-levels in the occupational environment, but some reliable data has been received recently, although there are still serious data gaps for some scenarios. In the risk characterisation, both measured (inhalation) data and modelled (dermal) data are used. Information on exposure of consumers has not been located, and for indirect exposures data are also limited, although a few surveys recently have been performed.

Therefore, to identify potentially significant risks, while excluding uses of less concern, model calculations have been used to generate data. It should be realised that combining quantitative data obtained in this manner for several worst-case scenarios may give a gross overestimation of the most likely total exposure.

In a personal communication from Industry it is indicated that a large portion of the EU production (possibly in the order of one third) may be used in the Eastern Europe, with Poland as a large market. Nothing is known regarding to what extent the use in these countries contribute to the overall diffuse releases.

4.1.3.1.4 Toxicokinetics

Information on the toxicokinetics of HBCDD is limited. No data are available on absorption via inhalation. An in vitro dermal absorption study has been performed, and a dermal absorption value of 4 % has been selected. When the substance is *properly* dissolved in the vehicle, it 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. Although the exact extent of oral absorption is unknown, it is probably in the order of 50-100 %. 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, 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 (see Figure 4-1).

An oral absorption of 100 % is assumed for *dissolved* HBCDD. Data on absorption by inhalation exposure is lacking, and for the purpose of modelling, the efficiency of inhalation uptake can be considered equal to uptake by the oral route (100 %). When applicable, this should be based on the fraction respirable particles. However, as the inhalable fraction is cleared from the upper respiratory pathway and then swallowed with saliva, also the inhalable fraction will eventually be absorbed, although via the oral route but with the same expected absorption rate as the respirable fraction is absorbed through the lungs. Therefore, both the respirable and inhalable fraction has been considered for inhalation exposure, even though the actual absorption involves also the oral route. 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.

4.1.3.1.5 Effects

Most studies have been performed with HBCDD-particles suspended in oil. The mean particle size of the HBCDD used in many studies is reported to be 0.14 mm (10 % <3.6 μm, 10 % >280 μm). With the reservation that the absorption of these suspended particles may have been low in these studies (i.e., the solubility of the particles in the GI-tract is expected to decrease with the amount of particles administered), and that the internal dose is likely to have been lower than the administered dose, HBCDD has demonstrated a low acute toxicity.

Acute toxicity The minimum lethal dose is greater than 20 g/kg for both dermal and oral routes of administration, and greater than 200 mg/l from inhalation for 4 hours. Therefore, the acute toxicity is not considered further in the risk characterisation.

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 or corrosive to skin.

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. Sensitisation will not be considered further in the risk characterisation. No information is available on respiratory sensitisation.

Repeated dose toxicity No repeated dose studies with inhalation or dermal exposure as route of administration are available. A 90-days toxicity study with oral exposure to a suspension of HBCDD particles has shown effects on the liver, the thyroid and the prostate. As from doses of 100 mg/kg/day, a dose-dependent increase in liver weight that was not accompanied by any clear pathological signs was noted, as well as effects on the thyroid hormone system. The liver weight increase was slowly reversible upon cessation of exposure. All other repeated dose studies on HBCDD have also shown the liver to be the target organ. In addition, the prostate weight was statistically increased at exposure to 1000 mg/kg/day. A LOAEL of 100 mg/kg/day is deduced for repeated dose toxicity based on liver weight increases (18-24 %). In addition, a disturbed thyroid hormone system (T4↓ and TSH↑) was observed after 90 days

oral exposure to HBCDD, potentially being secondary to the liver effect. The use of a suspension of HBCDD particles in most toxicity studies has likely led to a low absorption rate. Therefore, based on an assumed conservative oral absorption of 10-20 % (chapter 4.1.2.1) for this suspension, the study LOAEL of 100 mg/kg/day is transformed into a corrected LOAEL of 10-20 mg/kg/day. A 2-generation reproductive toxicity study has also shown the liver and thyroid system to be target organs. However, also in this study HBCDD particles were administered to the rats, although this time mixed into ground food. Because of dosing HBCDD-particles, with the absorption kinetics likely being dependent on particle size and amount of particles administered, the actual doses received at the top doses are uncertain. The mid dose (≤101-141 mg/kg/day) can thus be considered a LOAEL for effects on the liver, but considering the big dose spacing, the low dose (10-14 mg/kg/day) is a very conservative NOAEL. For effects on the thyroid system, the mid dose (≤101-141 mg/kg/day) is a clear effect level, with decreased thyroid follicle size and increased serum TSH. The most recent 28 days study is performed using a benchmark model design and oral administration of dissolved HBCDD. The study mainly shows effects on the liver, the thyroid, and the pituitary, with a NOAEL/BMD-L of 22.9 mg/kg/day for liver weight increase.

Overall, it is proposed to base the NOAEL for repeated dose toxicity on the NOAEL/BMD-L of 22.9 mg/kg/day for liver weight increase observed in a 28 days oral study in rats (van der Ven *et al.*, 2006). Enzyme induction is a likely cause to the liver weight increase, and enzyme induction is clearly relevant also to humans.

A NOAEL from longer studies (e.g., a 90 days study) would normally be preferred in the risk characterisation, but the uncertainties introduced in the evaluation of the 90 days study by the dosing of HBCDD-particles to the animals, leads to the choice of not using the 90 days study. Concerning the 2-generation study, with a LOAEL of ≤101-141 mg/kg/day for liver weight increase, a very conservative NOAEL of 10-14 mg/kg/day could be deduced from that study. However, it is in this case preferable to use an overall NOAEL of 22.9 mg/kg/day for liver effects in the recent 28-days study as the wide dose spacing in the 2-generation study does only allow a less accurate estimation of the effect level than the benchmark dose 28d study.. It should also be pointed out that effects on the endpoint liver weight increase does not seem to worsen with time, e.g., the magnitude of the weight increase is similar in studies of different length, and weight increases observed in young animals did not remain in adults in spite of continous exposure.

Mutagenicity The preponderance of evidence from available studies indicates that HBCDD lacks significant genotoxic potential in vitro and in vivo.

Carcinogenicity Based on the only available lifetime bioassay, it is not possible to assess the carcinogenic potential of HBCDD. However, the available data (including mutagenicity) gives no reason for further exploration of this endpoint.

Reproductive toxicity

Fertility

A NOEAL of 10 mg/kg/day has been deduced in a two generation reproductive toxicity study in rats (Ema, 2008). The NOAEL is based on a dose-dependent decrease in fertility index observed in both generations (8-14 % in the mid and high dose groups)(with a statistically significant trend in F0). A 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.

Developmental toxicity

Two ordinary *developmental toxicity* studies have failed to demonstrate any fetotoxicity, teratogenic potential, or adverse effects from HBCDD on development postpartum. However, an increased pup mortility during lactation was observed in a 2-generation study, with a NOAEL of 10 mg/kg/day. No risk characterisation for developmental toxicity is currently performed, as it anyway would result in identical results as presented for fertility in this report. In addition, because of the conclusion (i) on hold for developmental neurotoxicity, the end-point developmental toxicity will be re-visited at a later stage.

A study on *developmental neurotoxicity* in adult mice exposed to HBCDD as pups at day 10 postpartum was recently conducted. It indicated that HBCDD may cause statistically significant changes in spontaneous behaviour, learning and memory defects. An indicative LOAEL of 0.9 mg/kg/day can be deduced from this latter study. The study is published, but would benefit from being confirmed by other laboratories. We propose **conclusion (i)** with regard to developmental neurotoxicity. However, awaiting the confirmation of a similar study on decaBDE, we propose to put this conclusion **on hold**.

Table 4-32 Summary of effects brought forward to the risk characterisation.

Endpoint	Study NOAEL (mg/kg/day)	Effects observed
Repeated dose toxicity	22.9	Liver weight increase, thyroid weight increase (and decreased serum T4 levels), and increased pituitary weight
Reproductive toxicity/fertility	10	Dose-dependent decrease in fertility index and a reduced number of primordial follicles

4.1.3.1.6 Issues

Uptake

A high degree of accumulation of HBCDD in body fat of rats has been observed, with a time to reach steady state levels in the order of months suggested by the data. The apparent bioaccumulation is much higher of the minor α-diastereomer relative to the γ-diastereomer, which is the main constituent of the tested technical product. The accumulation indicates a potential for bioaccumulation, which is also supported by bioconcentration data in fish. Excretion of HBCDD via breast milk to infants is possible because of its physico-chemical properties (e.g. a log K_{ow} of 5.6). Indeed, HBCDD has been found in Swedish breast milk samples, with an indication of increasing concentrations during the eighties and nineties. The composition of HBCDD in milk and in food-stuff is not known, but there seems to be a

preponderance of the α-diastereomer in most biotic samples (Table 3-64-Table 3-65). Thus, the PBT-properties of HBCDD has to be considered.

Toxicological 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. The contamination of control animals observed in one recent performed 90-days oral study is likely to have been caused by insufficient washing of the glassware used for the substance. Since the same laboratory has performed two other recent studies cited in this report, there is a possibility (that cannot be checked) that the controls of those studies also were contaminated with HBCDD. However, it should be noted that HBCDD also was found in the female (but not male) control animals in the 'FIRE' 28 days study. The concentrations were much lower than in the 90 days study, and the authors speculated that the commercial feed was the source of HBCDD.

4.1.3.2 Risk characterisation for workers

Occupational exposure to HBCDD occurs primarily by dermal and respiratory routes of exposure. However, ingestion is not considered for workers in this risk assessment. Exposure by multiple exposure routes is considered to more accurately represent total exposure levels for each population, and is estimated as the sum of the highest exposures from each route during a working day.

Dermal exposure levels used for manufacture and industrial use have been derived from EASE, whereas most inhalation exposure levels are based on measured data. In the case of sewing (industrial use), the exposure level was estimated by the rapporteur. The mean particle sizes for the different applications vary considerably between the producers. In general, the size of the fine powder used by the textile industry is in the range of 2-19 μm, the powder 20-150 μm, and the granules 560-2400 μm. The extent of use of personal protective equipment (PPE) seems to vary between the occupational settings using HBCDD. However, in setting the reasonable worst case exposure levels, it is assumed that no PPE is used. Risk characterisation is performed for the endpoints repeated dose toxicity and reproductive toxicity /fertility (see also above).

Three scenarios have been identified for which there will be a risk characterisation:

- manufacture of HBCDD, where exposure mainly occur during filling operations with HBCDD powder or granules
- industrial use of HBCDD, i.e., production of fire-proofed products (e.g., polystyrene), where exposure mainly occur during adding of HBCDD powder or granules to the formulation. HBCDD may also be added as a masterbatch.

- end use of HBCDD during sewing textiles treated with HBCDD. Other end-uses, such as handling of polystyrene-boards are assumed to result in very low exposure levels, and, therefore, there is no scenario for this use.

4.1.3.2.1 Calculation of minimal MOS for workers

The following assessment factors are applied in setting the minimal MOS for **repeated dose toxicity**;

- a factor of 5 for intraspecies differences; this will cover the expected variation in sensitivity in the worker population.
- a factor of 4 for interspecies differences; a factor of 4 represents the difference in caloric demand between rats and humans. If speculating that enzyme induction explains the liver and thyroid weight increases, then humans are not expected to be more sensitive than rats to the enzyme induction/liver weight increase, and no factor for differences in sensitivity is thus needed.
- a factor 1 for differences between the experimental sub-chronic (28 days) and chronic exposure; Although a steady-state has not been reached in female rats in the 28 days study, and that extrapolation from 28 days to chronic exposure situation normally require a higher assessment factor, a factor of 1 is proposed as 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.

The resulting minimal MOS for comparison with the obtained MOS-values is 20.

The following assessment factors are applied in setting the minimal MOS for **reproductive toxicity/fertility;**

- a factor of 5 for intraspecies differences; this will cover the expected variation in sensitivity in the worker population.
- A total factor of 10 for interspecies; 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).

The resulting minimal MOS for comparison with the obtained MOS-values is 50.

4.1.3.2.2 Production of HBCDD

There is one manufacturing plant in the EU. It is assumed that five persons are required to run each production process. It is also assumed that within the industry producing HBCDD in EU, there are probably less than 100 individuals exposed to this compound.

The highest exposure to HBCDD at the manufacturing site is assumed to exist during filling of HBCDD into various kinds of containers. Earlier process steps; bromination, crystallisation, centrifugation and drying, are all of the closed system type, resulting in a low level of release HBCDD during normal operating conditions.

Scenario FILLING. Filling of bags at the manufacture of HBCDD

Inhalation exposure to HBCDD
For filling operations, the reasonable worst-case exposure to be used for risk characterisation is 10 and 1.9 mg/m^3 for fine (micronisation) and standard grade powder, respectively. Assuming a pulmonary ventilation rate of 10 m^3 per 8 hr (light work), inhalation exposure to 10 and 1.9 mg/m^3 (8 hour TWA) and 100 % uptake gives a total intake of about 1.42 and 0.27 mg/kg/day for a 70 kg individual, respectively. The corresponding value for granules is 0.03 mg/kg/day.

Repeated dose toxicity
The exposure level should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 16, 85, and 763 for fine powder, standard grade powder, and granules, respectively. When compared with a minimal MOS of 20 (see above) there is concern for repeated dose toxicity after inhalation exposure to fine powder. For standard powder and granules, there is no concern.

Reproductive toxicity
The exposure level should be compared with a NOAEL of 10 mg/kg/day for reproduction toxicity/fertility, giving a MOS of 7, 37, and 333 for fine powder, standard grade powder, and granules, respectively. When compared with a minimal MOS of 50 (see above) there is concern for reproductive toxicity after inhalation exposure to fine and standard powder. For granules, there is no concern.

Conclusion:

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

Dermal exposure to HBCDD
The derived reasonable worst-case dermal exposure during filling of fine powder HBCDD is 5 mg/cm^2/day and with standard powder 1 mg/cm^2/day. It is assumed that both hands are exposed, which corresponds to an exposed area of 840 cm^2. A 4 % uptake is assumed, which gives an exposure of 168 and 34 mg/day, resulting in an estimated uptake of 2.4 and 0.49 mg/kg /day HBCDD, for fine and standard grade HBCDD, respectively. The corresponding value for granules is 0.02 mg/kg/day.

Repeated dose toxicity
The exposure level should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 10 and 47 for fine and standard grade HBCDD, respectively, and 1145 for granules. When compared with a minimal MOS of 20 (see above) there is concern for repeated dose toxicity after dermal exposure to fine powder, but not after exposure to standard powder and granules.

Reproductive toxicity
The exposure level should be compared with a NOAEL of 10 mg/kg/day for repoductive toxicity/fertility, giving a MOS of 4 and 20 for fine and standard grade HBCDD, respectively, and 500 for granules. When compared with a minimal MOS of 50 (see above) there is concern for reproductive toxicity after dermal exposure to fine and standard powder, but not after exposure to granules.

Conclusion:

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

Multiple routes
Occupational exposure to HBCDD mainly occurs by inhalation and dermal contact. The extent of exposure by multiple exposure routes can be calculated as the sum of the highest exposure from each route during a working day, giving a total intake during filling operations of about 3.82, 0.76, and 0.05 mg/kg/day for fine powder, standard powder, and granules, respectively.

Repeated dose toxicity
The exposure level should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 6 and 30 for fine and standard powder and 458 for granules. When compared with a minimal MOS of 20 (see above) there is concern for repeated dose toxicity after multiple exposures to fine powder. Considering the substantial exposure also expected at typical exposure conditions, the concern also applies for typical exposure.

Reproductive toxicity
The exposure level should be compared with a NOAEL of 10 mg/kg/day for reproductive toxicity/fertility, giving a MOS of 3 and 13 for fine and standard powder and 200 for granules. When compared with a minimal MOS of 50 (see above) there is concern for reproductive toxicity after multiple exposures to fine and standard powder. Considering the substantial exposure also expected at typical exposure conditions, the concern also applies for typical exposure.

Conclusion:

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

4.1.3.2.3 Industrial use of HBCDD

The exposures at sites where HBCDD are added as powder to a formulation are assumed to be similar, independent of which formulation scenario. The situation at these sites when the exposure is assumed to be highest is when emptying bags or other containers with HBCDD in powder form to the formulation equipment.

It is assumed that exposure HBCDD from manufacturing fireproofed material occur principally during manual adding of the substance during the manufacturing process.

Scenario: ADDING- Industrial use of HBCDD as an additive

The information on number of exposed individuals and the frequency and duration of the exposures is limited.

Inhalation exposure
For operations involving manual addition of HBCDD, the reasonable worst-case exposure to be used for risk characterisation is, based on measured data, estimated to be about the same as for manual filling of containers, i.e., 3.1 for fine powder (textile backcoating), 2.5 for standard powder (XPS and EPS production), and 0.22 mg/m^3 for granules and masterbatch (XPS and EPS production). Assuming a pulmonary ventilation rate of 10 m^3 per 8 hr, inhalation exposure to 3.1, 2.5, and 0.22 mg/m^3 (8 hour TWA) and 100 % uptake gives a total intake of about 0.44 and 0.36 mg/kg /day for a 70 kg individual, for fine and standard powder, respectively. The corresponding value for granules/masterbatch is 0.031 mg/kg/day.

Exposure during adding of HBCDD may be full shift. It is assumed that short-term exposure may be twice as high as the reasonable worst case, e.g. up to 10 mg/m^3, but will not significantly alter the total intake.

*Repeated dose toxicity*The exposure level should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 52-64 for powder and 739 for granules/masterbatch. When compared with a minimal MOS of 20 (see above) there is no concern for repeated dose toxicity after inhalation exposure.

Conclusion:

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

Reproductive toxicity
The exposure level should be compared with a NOAEL of 10 mg/kg/day for repeated dose toxicity/fertility, giving a MOS of 23-28 for powder and 323 for granules/masterbatch. When compared with a minimal MOS of 50 (see above) there is concern for reproduction toxicity when using fine and standard powder after inhalation exposure, but not when using granules.

Conclusion:

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

Dermal exposure
More thorough descriptions of the conditions at the different occupational settings have led to separate dermal exposure calculations for textile backcoating on the one hand and XPS/EPS-production on the other. The derived reasonable worst-case dermal exposure during filling of fine and standard powder HBCDD is 0.1 mg/cm^2/day. It is assumed that both hands are exposed in XPS/EPS-production, whereas hands and face are exposed in textile backcoating, which corresponds to exposed areas of 840 and 1200 cm^2, respectively. A 4 % uptake is assumed, which gives an exposure of 3.4 and 4.8 mg/day, resulting in an estimated uptake of 0.05 and 0.07 mg/kg/day HBCDD in XPS/EPS-production and textile backcoating, respectively. The corresponding value for the use of granules in XPS/EPS-production is 0.002 mg/kg/day.

Repeated dose toxicity
The exposure level should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 327 for textile backcoating, 458 for use of standard powder and 11450 for use of granules/masterbatch in XPS/EPS-production.
When compared with a minimal MOS of 20 (see above) there is no concern for repeated dose toxicity after dermal exposure.

Reproductive toxicity/fertility
The exposure level should be compared with a NOAEL of 10 mg/kg/day for reproductive toxicity/fertility, giving a MOS of 143 for textile backcoating, 200 for use of standard powder and 5000 for use of granules/masterbatch in XPS/EPS-production.
When compared with a minimal MOS of 50 (see above) there is no concern for reproductive toxicity after dermal exposure.

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

Multiple routes
Occupational exposures to HBCDD mainly occur by inhalation and dermal contact. The extent of exposure by multiple exposure routes can be calculated as the sum of the highest exposure from each route during a working day, giving a total intake during adding operations of about 0.51-0.41 and 0.03 mg/kg/day for powder (textile backcoating and XPS/EPS-production) and granules/masterbatch (XPS/EPS-production), respectively.

Repeated dose toxicity
The exposure level should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 45-56 and 763 for powder and granules, respectively.
When compared with a minimal MOS of 20 (see above) there is no concern for repeated dose toxicity after multiple routes exposure.

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

Reproductive toxicity/fertility
The exposure level should be compared with a NOAEL of 10 mg/kg/day for repeated dose toxicity/fertility, giving a MOS of 20-24 and 333 for powder and granules, respectively.
When compared with a minimal MOS of 50 (see above) there is concern for reproductive toxicity after multiple routes exposure to fine and standard powder but not after exposure to granules.

Conclusion:

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

4.1.3.2.4 End use of HBCDD (professional)

It is assumed that the highest exposure to HBCDD during industrial use of end-products occurs principally during sewing textiles containing HBCDD.

Scenario: sewing (industrial)

Inhalation exposure
It is assumed that the concentration of airborne textile dust is 5 mg/m^3, and that the dust contains 10 % HBCDD. Assuming a pulmonary ventilation rate of 10 m^3 per 8 hours, and 100 % uptake, there will be an inhalation exposure to 0.5 mg/m^3 (8 hour TWA). Since most workers in this setting are women (with a median body weight of 60 kg), it results in a total intake of about 0.08 mg/kg/day. It is acknowledged that there is no measured data to support the modelling and that the assumptions really are of a worst-case character.

Repeated dose toxicity
The exposure level should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 286. When compared with a minimal MOS of 20 (see above) there is no concern for repeated dose toxicity after inhalation exposure.

Reproductive toxicity/fertility
The exposure level should be compared with a NOAEL of 10 mg/kg/day for reproductive toxicity/fertility, giving a MOS of 125. When compared with a minimal MOS of 50 (see above) there is no concern for reproductive toxicity after inhalation exposure.

Conclusion:

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

Dermal exposure
A dermal exposure to 1 mg/cm^2/day of textile dust on 840 cm^2 (both hands) is assumed to be possibly realistic at this work environment. If the concentration of HBCDD in the material and in the airborne dust is 10 %, and the body weight 60 kg, the dermal exposure will be 0.06 mg/kg bwt/day.

Repeated dose toxicity

The exposure level should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 382. When compared with a minimal MOS of 20 (see above) there is no concern for repeated dose toxicity after dermal exposure.

Reproductive toxicity/fertility
The exposure level should be compared with a NOAEL of 10 mg/kg/day for reproductive toxicity/fertility, giving a MOS of 167. When compared with a minimal MOS of 50 (see above) there is no concern for reproductive toxicity after dermal exposure.
Conclusion:

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

Multiple routes

The extent of exposure by multiple exposure routes is calculated as the sum of the highest exposure from each route during a day. Thus, the total intake via inhalation and dermal exposure will be 0.14 mg/kg bwt/day.

Repeated dose toxicity
The exposure level should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 164.
When compared with a minimal MOS of 20 (see above) there is no concern for repeated dose toxicity after multiple routes exposure.

Reproductive toxicity/fertility
The exposure level should be compared with a NOAEL of 10 mg/kg/day for reproductive toxicity, giving a MOS of 71.
When compared with a minimal MOS of 50 (see above) there is no concern for reproductive toxicity after multiple routes exposure.

Conclusion:

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

4.1.3.2.5 Summary of the risk characterisation for workers

For repeated dose toxicity, a NOAEL of 22.9 mg/kg/day is used for the MOS calculation. The minimal MOS is 20.

For reproductive toxicity/fertility, a NOAEL of 10 mg/kg/day is used for the MOS calculation. The minimal MOS is 50.

There are presently no occupational limit values for HBCDD. In view of the outcome of this risk assessment, it is concluded that there is a need to establish occupational exposure limit values for HBCDD.

Intakes for different routes of exposure are presented for different occupational scenarios in Table 4-33 (repeated dose toxicity) and Table 4-34 (reproductive toxicity/fertility).

Table 4-33. Compilation of data from the occupational risk characterisation for repeated dose toxicity, based on realistic worst-case exposure concentrations. For inhalation exposure estimates, the typical exposure (in mg/m³) is given within brackets.

Scenario	Product grade	Inhalation			Dermal			Multiple routes exposure		
		mg/m³ mg/kg/day	MOS rdt	Concl rdt	mg/day mg/kg/day	MOS rdt	Concl rdt	mg/kg/day	MOS rdt	Concl rdt
FILLING. Filling of bags at the production of HBCDD[a]	Fine powder	10 (5) 1.42	16	(iii)	4200 2.4	10	(iii)	3.82	6	(iii)
	Powder	1.9 (0.95) 0.27	85	(ii)	840 0.49	47	(ii)	0.76	30	(ii)
	Granules	0.19 (0.1) 0.03	763	(ii)	84 0.02	1145	(ii)	0.05	458	(ii)
ADDING. Industrial use of HBCDD as an additive[a]	Formulation of textile. Fine powder	3.1 (1.55) 0.44	52	(ii)	120 0.07	327	(ii)	0.51	45	(ii)
	Formulation of polystyrene (EPS, XPS and HIPS), standard grade powder	2.5 (1.25) 0.36	64	(ii)	84 0.05	458	(ii)	0.41	56	(ii)
	Formulation of polystyrene (EPS, XPS and HIPS), granules masterbatch	0.22 (0.11) 0.031	739	(ii)	8.4 0.002	1145 0	(ii)	0.033	763	(ii)
SEWING (Occupational). Industrial end-use[b]		0.5 (0.25) 0.08	286	(ii)	84 0.06	382	(ii)	0.14	164	(ii)

[a] When two numbers exist in a box, the first one gives the concentration (mg/m³ or mg/day) and the second, lower one the internal intake in mg/kg/day.

Table 4-34. Compilation of data from the occupational risk characterisation for reproductive toxicity, based on realistic worst-case exposure concentrations. For inhalation exposure estimates, the typical exposure (in mg/m³) is given within brackets.

Scenario	Product grade	Inhalation			Dermal			Multiple routes exposure		
		mg/m³ mg/kg/day	MOS repro	Concl repro	mg/day mg/kg/day	MOS repro	Concl repro	mg/kg/day	MOS repro	Concl repro
FILLING. Filling of	Fine powder	10 (5) 1.42	7	(iii)	4200 2.4	4	(iii)	3.82	3	(iii)

Scenario	Product grade	Inhalation			Dermal			Multiple routes exposure		
bags at the production of HBCDD[a]	Powder	1.9 (0.95) 0.27	37	(iii)	840 0.49	20	(iii)	0.76	13	(iii)
	Granules	0.19 (0.1) 0.03	333	(ii)	84 0.02	5000	(ii)	0.05	200	(ii)
ADDING. Industrial use of HBCDD as an additive[a]	Formulation of textile. Fine powder	3.1 (1.55) 0.44	23	(iii)	120 0.07	143	(ii)	0.51	20	(iii)
	Formulation of polystyrene (EPS, XPS and HIPS), standard grade powder	2.5 (1.25) 0.36	28	(iii)	84 0.05	200	(ii)	0.41	24	(iii)
	Formulation of polystyrene (EPS, XPS and HIPS), granules masterbatch	0.22 (0.11) 0.031	323	(ii)	8.4 0.002	5000	(ii)	0.033	333	(ii)
SEWING (Occupational). Industrial end-use[b]		0.5 (0.25) 0.08	125	(ii)	84 0.06	167	(ii)	0.14	71	(ii)

[a] When two numbers exist in a box, the first one gives the concentration (mg/m^3 or mg/day) and the second, lower one the internal intake in mg/kg/day.

4.1.3.3 Risk characterisation for consumers

HBCDD is used in several products, some of which are available to consumers, e.g. textiles in furniture, automobile interior textile, construction boards, mattress ticking and videocassettes. These are potential sources of consumer exposure to HBCDD. The concentrations of HBCDD in various products are given in chapter 2.2.

In most applications HBCDD is present non-bound within a polymer matrix, hence, it may migrate from the polymer and be released. Release of HBCDD from the surface of the product into atmosphere from plastic products may be a potential way of exposure. Due to the low vapour pressure the release to air from products is assumed to be relatively low. Direct contact with products containing HBCDD may give rise to dermal exposure.

Consumers may be exposed to HBCDD by dermal, oral and respiratory routes of exposure. Exposure by multiple exposure routes is considered to more accurately represent the intake for each population. The extent of exposure by multiple exposure routes is calculated as the sum of the highest exposure from each route during a day.

It is assumed that direct exposure to consumers occurs mainly during contact with textiles containing HBCDD, but exposure via indoor air is also a possibility. The information used for

4.1.3.3.1 Calculation of minimal MOS for consumers

The following assessment factors are applied in the setting of a minimal MOS for repeated dose toxicity;

- a factor of 10 for intraspecies differences; this covers the variation in sensitivity expected in the whole human population
- a factor of 4 for interspecies differences; a factor of 4 represents the difference in caloric demand between rats and humans. If speculating that enzyme induction explains the liver and thyroid weight increases, then humans are not expected to be more sensitive than rats to the enzyme induction/liver weight increase, and no factor for differences in sensitivity is thus needed.
- a factor 1 for differences between the experimental sub-chronic (28 days) and chronic exposure; Although a steady-state has not been reached in female rats in the 28 days study, and that extrapolation from 28 days to chronic exposure situation normally require an assessment factor, a factor of 1 is proposed as 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.

The resulting minimal MOS for comparison with the obtained MOS-values is 40.

The following assessment factors are applied in the setting of a minimal MOS for reproduction toxicity/fertility

- a factor of 10 for intraspecies differences; this covers the variation in sensitivity expected in the whole human population
- a total factor of 10 for interspecies differences; 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).

The resulting MOS for comparision with the obtained MOS-values is 100.

4.1.3.3.2 Scenario: textiles

The release of HBCDD from textiles during mouthing has been studied by immersion of a back-coated textile in surrogate biological media. Based on the study, 2.75 % of the HBCDD content is thought to be extractable when both sides are available for mouthing. When only the textile side is available, it is assumed that 0.27 % is extractable. The reasonable worst-case

intake during mouthing both sides of a back-coated textile is 30 µg/kg/day, and when only the textile side is available, 3 µg/kg/day.

Repeated dose toxicity

The exposure levels should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving MOS values of 760 and 7600, depending on if the mouthing includes the back-coating or not, respectively. When compared with a minimal MOS of 40 (see above) there is no concern for repeated dose toxicity.

Reproductive toxicity/fertility
The exposure levels should be compared with a NOAEL of 10 mg/kg/day for reproductive toxicity/fertility, giving MOS values of 330 and 3300, depending on if the mouthing includes the back-coating or not, respectively. When compared with a minimal MOS of 100 (see above) there is no concern for reproductive toxicity.

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

Combined (multiple) consumer exposure

This scenario applies for a person exposed to HBCDD or HBCDD-containing dust from building material in houses, from HBCDD-treated mattresses and textile in furniture. It also includes children's mouthing of HBCDD-treated textiles. However, there is no concern for the mouthing scenario, and the other exposure routes are considered to be insignificant.

There is no concern for repeated dose- or reproductive toxicity/fertilityafter combined consumer exposure.

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

4.1.3.3.3 Summary of the risk characterisation for consumers

For repeated dose toxicity, a NOAEL of 22.9 mg/kg/day is used for the MOS calculation. The minimal MOS is 40.
For reproductive toxicity/fertility, a NOAEL of 10 mg/kg/day is used for the MOS calculation. The minimal MOS is 100.
Exposure estimates and calculated margins of safety are presented in Table 4-35.

Table 4-35 Compilation of data used for the risk characterisation for consumers, including reasonable worst-case exposure concentrations, Margins of Safety (MOS) and conclusions (concl.).

Exposure scenario	total body burden (mg/kg/day)	MOS rtd	MOS_{rdt} (concl)	MOS repro	MOS_{repro} (concl)
Mouthing (only textile side	0.003	7600	(ii)	3300	(ii)
Mouthing (both sides)	0.03	760	(ii)	330	(ii)

4.1.3.4 Risk characterisation for man exposed indirectly via the environment

HBCDD may be released to the environment through wastewater and air effluents from production, formulation, industrial use, use and disposal of HBCDD containing products. Such indirect exposure routes are taken into account in chapter 4.1.1.4. Available information indicates that release from diffusive sources, such as use of end-products and from disposal, could be important.

It should be noted, that there is limited biodegradation of HBCDD in the environment and it has bioaccumulating properties. There are limited data on indirect exposure to HBCDD, with most monitoring/screening being performed in Scandinavia, and recently, in the UK and the Netherlands. Low levels of HBCDD has been found in different fish species and in food items, such as salmon, egg, milk, vegetable oils and fats, and fat from domestic animals (chicken, beef and lamb). Based on these data the intake of HBCDD via food has been estimated to be 0.00002 mg/kg/day, which will be used rather than the EUSES-prediction of regional exposure.

Predicted daily dose via the environment is estimated by EUSES in section 4.1.1.4. Total daily intakes from local generic sources are presented in 4.1.1.4. When compared with monitoring, where available, it seems that EUSES overestimates the exposure, for the local scenarios with at least a factor of 10. However, the discrepancy is also likely caused by differences in the composition of the food basket. Also, EUSES assumes that RWC-emissions from all life-cycle stages do occur in the hypothetical region, which is perhaps not that likely in the reality. Very high concentrations are predicted in root crops, especially in scenarios for industrial use of HBCDD. There are data on the occurrence of HBCDD in potatoes, but they do not support such a strong accumulation of HBCDD in root crops, and one may doubt this high uptake.

4.1.3.4.1 Calculation of minimal MOS for indirect exposure via the environment

The following assessment factors are applied in the setting of a minimal MOS for repeated dose toxicity;

- a factor of 10 for intraspecies differences; this will cover the expected variation in sensitivity in the total human population.
- a factor of 4 for interspecies differences; a factor of 4 represents the difference in caloric demand between rats and humans. If speculating that enzyme induction explains the liver and thyroid weight increases, then humans are not expected to be more sensitive than rats to the enzyme induction/liver weight increase, and a factor for differences in sensitivity is thus not needed.
- a factor 1 for differences between the experimental sub-chronic (90 days) and chronic exposure; Although a steady-state has not been reached in female rats in the 28 days study, and that extrapolation from 28 days to chronic exposure situation normally require a higher assessment factor, a factor of 1 is proposed as 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.

The resulting minimal MOS for comparison with the obtained MOS-values is 40 when the exposure is based on monitored data (i.e., the regional scenario).

The following assessment factors are applied in the setting of a minimal MOS for reproductive toxicity/fertility;

- a factor of 10 for intraspecies differences; this will cover the expected variation in sensitivity in the total human population.
- a total factor of 10 for interspecies differences; 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

The resulting minimal MOS for comparison with the obtained MOS-values is 100 when the exposure is based on monitored data (i.e., the regional scenario).

The conclusions are set based on a qualitative assessment, comparing EUSES data (the local scenarios) and measured data (site specific data), as EUSES seems to overestimate the exposure by at least a factor of 10. Although difficult to quantify, there is some remaining uncertainty that needs to be reflected on. Most importantly, whereas the α-diastereomer of HBCDD is a minor component of the technical HBCDD examined in the toxicity studies, it is the major diastereomer in the exposure of man via the environment due to its bioaccumulation

properties. Thus, the toxicity of the HBCDD component contributing most to exposure via the environment is not as thoroughly studied as the technical mixture.

4.1.3.4.2 Scenario: local and regional sources

The dominating foodstuff contributing to the human exposure via the environment is root-crops and fish. There is few measured data for root crops, and although root crops may take up HBCDD, a high accumulation is not expected. Fish is often contaminated with HBCDD, with the highest concentration of HBCDD found in fish locally downstream a production plant and lower concentrations in most fish.

Regional exposure
The regional exposure of man via the environment has been estimated to be 20 ng/kg/day, based on measured values in food basket studies, se chapter 4.1.1.5.
The EUSES model predicts a regional intake of approximately 5 µg/kg/day.

*Repeated dose toxicity*The exposure levels should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity. When comparing the MOS values with a minimal MOS of 40 (see above), there is no concern for repeated dose toxicity from regional exposure (Table 4-36).
Reproductive toxicity/fertility
The exposure levels should be compared with a NOAEL of 10 mg/kg/day for reproductive toxicity/fertility. When comparing the MOS values with a minimal MOS of 100 (see above), there is no concern for reproductive toxicity from regional exposure.

Table 4-36 Regional margin of safety (MOS), predicted daily intake and calculated daily intake from monitoring data, for man exposed indirectly via the environment .

Site	Total daily intake (mg HBCDD/kg/day)	MOS rdt	MOS repro
Regional (predicted)	0.0044	5200	2300
Regional (monitored)	0.00002	1145000	500000

Conclusion:

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

Local exposure

The highest local generic (worst case) indirect exposure is at industrial use of textile backcoating (5.1 mg/kg/day) (see section 4.1.1.4 and Tabell 4-14). For the other generic local scenarios, the exposure is <0.3 mg/kg/day. Site-specific information shows lower emissions. However, there are many uncertainties with respect to indirect exposure via the environment.
The exposure levels should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity and 10 mg/kg/day for reproductive toxicity.

Table 4-37 Margin of safety (MOS) and predicted daily intake for man exposed indirectly via the environment due to production and micronising of HBCDD.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/day)	MOS rdt	MOS repro
ProdB	Yes	0.00085	27000	12000
Micronising	Yes/No*	0.000043	530000	230000

*Connection to STP not known. However, no emissions to waste water.

Table 4-38 Margin of safety (MOS) and predicted daily intake for man exposed indirectly via the environment due to formulation of compound for EPS and HIPS.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/day)	MOS rdt	MOS repro
Site A+M	No	0.00019	1200000	53000
Site F+N	No	0.0013	17000	7700
Site I+O	No	0.00039	58000	26000
Site B	Yes	0.00024	94000	42000
Site C	No	0.00073	31000	14000
Site D	No	0.00056	41000	18000
Site E	No	0.00025	90000	40000
Site G	Yes	0.0050	4500	2000
Site H	No	0.00070	32000	14000
Site J	No	0.035	660	290
Site K	Yes	0.038	600	260
Site L	Yes	0.019	1200	530
Site P	No	0.0012	19000	8300
GEN_EPS_FORM	Yes	0.042	540	240
	No	0.087	260	110

Table 4-39 Margin of safety (MOS) and predicted daily intake for man exposed indirectly via the environment due to formulation of XPS compound.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/day)	MOS rdt	MOS repro
Masterb G	Yes	0.00047	49000	21000
Masterb H	Yes	0.00062	37000	16000
Masterb I	Yes	0.069	330	140
GEN_XPS_FORM	Yes	0.048	480	210
	No	0.10	230	100

Table 4-40 Margin of safety (MOS) and predicted daily intake for man exposed indirectly via the environment due to formulation of polymer dispersion for textiles.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/day)	MOS rdt	MOS repro
TexForm 1	Yes	0.00033	70000	30000
	No	0.00052	44000	19000
TexForm 3	Yes/No*	0.000022	1000000	450000
TexForm 4	Yes	0.00016	140000	63000
	No	0.00025	93000	40000
TexForm 5	Yes/No*	0.00011	200000	91000
TexForm A	Yes/No*	0.00002	1100000	500000
TexForm B	Yes	0.016	1500	630
	No	0.033	710	300
GEN_TEX_FORM	Yes	0.18	130	60
	No	0.37	63	30

*Connection to STP not known. However, no emissions to waste water.

Table 4-41 Margin of safety (MOS) and predicted daily intake for man exposed indirectly via the environment due to industrial use of EPS compound at the manufacture of flame retarded EPS.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/day)	MOS rdt	MOS repro
GEN_EPS_IndUse	Yes	0.001	23000	10000
	No	0.002	11000	5000

Table 4-42 Margin of safety (MOS) and predicted daily intake for man exposed indirectly via the environment due to industrial use of flame retarded HIPS.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/day)	MOS rdt	MOS repro
GEN_HIPS_IndUse	Yes	0.0018	13000	6000
	No	0.00063	36000	16000

Table 4-43 Margin of safety (MOS) and predicted daily intake for man exposed indirectly via the environment due to industrial use of EPS compound for flame retarded XPS.

Site	Connected to municipal STP	Total daily intake (mg HBCDD/kg/day)	MOS rdt	MOS repro

	(Yes/No)			
XPS 1	Yes	0.048	480	200
	No	0.00013	175000	80000
XPS 2	Yes	0.0007	32000	14000
XPS 3	Yes	0.045	510	220
	No	0.0017	14000	6000
XPS 11	Yes	0.076	300	130
	No	0.017	1400	600
GEN_XPS_IndUse	Yes	0.28	81	36
	No	0.031	730	320

Table 4-44 Margin of safety (MOS) and predicted daily intake for man exposed indirectly via the environment due to industrial use of HBCDD powder for flame retarded XPS.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/day)	MOS rdt	MOS repro
XPS 4	Yes	0.079	290	130
	No	0.017	1400	600
XPS 5	Yes	0.020	1100	500
	No	0.00019	120000	53000
XPS 6	Yes	0.018	1300	600
	No	0.00034	68000	29000
XPS 7	Yes	0.13	180	80
	No	0.00029	79000	34000
XPS 8	Yes	0.00010	220000	100000
	No	0.00012	18000	83000
XPS 9	Yes/No*	0.000045	510000	220000
XPS 10	Yes	0.21	110	48
	No	0.016	1400	600
XPS 13	Yes	0.00015	160000	70000
	No	0.000090	250000	100000
XPS 14	Yes	0.000059	390000	170000
XPS 16	Yes/No*	0.000032	710000	310000
XPS 17	No	0.00017	130000	59000
XPS 18	No	0.00018	130000	55000
XPS 20	Yes	0.0020	12000	5000
XPS 21	Yes	0.26	88	40
	No	0.0029	7900	3500
XPS 23	Yes	0.000071	320000	140000
XPS 24	Yes	0.000099	230000	100000

XPS 26	Yes	0.0011	21000	9000
XPS 27	Yes	0.089	260	110
	No	0.0096	2400	1000

*Connection to STP not known. However, no emissions to waste water.

Table 4-45 Margin of safety (MOS) and predicted daily intake for man exposed indirectly via the environment due to industrial use of HBCDD as textile back-coating agent.

Site	Connected to municipal STP (Yes/No)	Total daily intake (mg HBCDD/kg/day) (using modelling data)	MOS rdt (using modelling data)	MOS repro (using modelling data)	Total daily intake (mg HBCDD/kg/day) (using measured fish data)[1]	MOS rdt (using measured fish data)[1]	MOS Repro (using measured fish data)[1]
Backcoat 1	Yes	0.016	1500	630			
	No	0.027	850	370			
Backcoat 2	Yes	0.011	2000	900			
	No	0.0064	3600	1600			
Backcoat 3	Yes	2.1	11[2]	5[2]	1.16 0.02[3]	20 1150[3]	9 500
	No	1.7	13[2]	9[2]	0.016	1440	630
Backcoat 4	Yes	0.000049	470000	200000			
	No	0.000063	360000	150000			
Backcoat C	Yes	0.0036	6400	3000			
	No	0.0017	14000	6000			
GEN_TEX_IndUse	Yes	2.75	8.3[2]	4[2]	1.16 0.02[3]	20 1150[3]	9 500
	No	2.0	12[2]	5[2]	0.016	1400	630

1=The highest measured HBCDD concentration fish of 9.4 mg/kg has been used in the EUSES modelling.

2= The EUSES modelling overpredicts the concentration of HBCDD in root crops and in fish, the MOS and conclusion is unrealistic.

3= HBCDD in root crops contributes extensively to the total daily intake in those cases where STP has been accounted for. In this scenario measured HBCDD concentration in fish and the regional measured concentration in potatoes has been used ih the calculation.

For site specific local scenarios, backcoat 3, and generic local scenarios, the very low MOSes for textile backcoating (industrial use) could be worrying, but since EUSES overestimates the exposure, these MOSes are unrealisticly low. The calculated MOSes formally leads to concern (iii) but considering the overestimation by at least a factor of 10, there is no concern and conclusion (ii) applies for all identified scenarios. To demonstrate this reasoning, when the highest measured fish data have been used in the EUSES modelling the resulting MOS value is much higher, see Table 4-45. In those scenarios where STP has been accounted for, the regional measured concentration in potatoes has been used in the calculation and the resulting MOS values are much higher. As this value on HBCDD concentration in potatoes is

a regional value calculated from measured data in UK, its representativnes can be questioned.Although, values as high as 10 mg HBCDD/kg root crops can be used in the calculation and the MOS values are still high,,305,for repeated dose toxicity and 150 for reproductive toxicity/fertility.
Considering that the highest concentrations of HBCDD in fish were found in the samples caught closest to the Newton Aycliffe plant and that the production of HBCDD in the UK by Great Lakes ceased in December 2003, this is really a worst case estimation.

Conclusion: There is no concern for reproductive or repeated dose toxicity to man exposed via the environment from local or regional sourses.

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

4.1.3.4.3 Exposure via breast milk

Exposure from breast milk will greatly vary within the population depending on the childs/mothers individual feeding habits and also on cultural practices associated with different regions and countries. The worst case average daily uptake of the breast-feeding infant, estimated for 0-3 month of age, is 0.015 µg HBCDD/kg/day (see section 4.1.1.5.1), based on Swedish breast milk data.

Repeated dose toxicity
The exposure level should be compared with a NOAEL of 22.9 mg/kg/day for repeated dose toxicity, giving a MOS of 1.5×10^6. When compared with a minimal MOS of 40, there is no concern for repeated dose toxicity for breast feeding infants.

Reproduction toxicity/fertility
The exposure level should be compared with a NOAEL of 10 mg/kg/day for reproductive toxicity/fertility, giving a MOS of 0.7×10^6. When compared with a minimal MOS of 100, there is no concern for reproductive toxicity for breast feeding infants.

Conclusion:

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

For illustrative purposes, exposure via milk is also compared with the indicative LOAEL of 0.9 mg/kg/day for developmental neurotoxicity, giving a MOS of 60,000.

4.1.3.4.4 Summary of the risk characterisation for man exposed indirectly via the environment

There is neither concern (ii) for repeated dose toxicity or for reproductive toxicity/fertility to man exposed via the environment from local or regional sources nor from exposure via breast milk (ii).

4.1.3.4.5 Risk characterisation for combined exposure

Due to the use of HBCDD in the society and the diffuse emissions from these products, humans may be exposed from different sources (see section 4.1.1.1). The total exposure (body burden) is the summary of all the specific exposures. There is a big uncertainty in the future human exposure to HBCDD because of uncertainties in how the demolition of buildings with insulation made of flame-retarded polystyrene (EPS and XPS) will be performed. This handling may contribute both to direct occupational exposure and consumer exposure, but also to exposure of man via the environment.
In calculating the combined exposure, only occupational exposure and exposure via the local environment (i.e., by food produced nearby plants emitting HBCDD) need to be considered. The contribution to exposure from consumer articles and via regional exposure of man via the environment is too low to affect the risk from combined exposure. Additions of individual scenarios are not considered to change any of the conclusions, and no calculation on combined exposure has therefore been performed.

4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

Flammability, explosive and oxidising properties are not considered to represent a hazard, and hence, further characterisation is not undertaken in this report. In addition, there is no need for further information and/or testing with regard to physico-chemical properties.

Conclusion:

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

5 RESULTS

5.1 ENVIRONMENT

The results are presented in more details in section 3.

5.1.1 Aquatic compartment

STP

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 (ii) applies to production, micronising, EPS formulation, XPS formulation, Textile formulation, Industrial use of EPS, industrial use of HIPS, most sites involved in industrial use of XPS compound, sites involved in industrial use of HBCCD powder for XPS and individual sites involved in textile backcoating.

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

Conclusion (iii) applies to some sites with industrial use of XPS having intermittent releases to waste water and for 1 textile backcoating site including the generic textile backcoating scenario.

Surface water

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 (ii) applies to production and micronising, industrial use of EPS and HIPS and for most sites in the other use areas.

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

Conclusion (iii) applies to some sites involved in EPS formulation including the generic scenario, one site involved in formulation of XPS compound and the generic scenario, the generic local scenario for formulation of polymer dispersions for textiles, individual sites involved in industrial use of XPS compound and HBCDD powder including the generic local scenario for industrial use of XPS compound and finally, sites involved in textile backcoating including the generic scenario.

Freshwater sediment

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 (ii) applies to production, micronising and industrial use of EPS, industrial use of HIPS and to most sites for industrial use of HBCDD powder for XPS and for individual sites in the other use areas.

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

Conclusion (iii) applies to some sites involved in EPS formulation including the generic scenario, onesite involved in XPS formulation including the generic scenario, one site involved in formulation of polymer dispersions for textiles including the generic scenario, individual sites involved in industrial use of XPS compound and HBCDD powder including the generic local scenario for industrial use of XPS compound and sites involved in textile backcoating including the generic scenario. A general conclusion (iii) is drawn for textile backcoating. based on measured concentrations in sediment downstream three different locations giving RCRs >1.

5.1.2 Terrestrial compartment

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 (ii) applies to production, micronising, EPS formulation, XPS formulation, Textile formulation, industrial use of EPS, industrial use of HIPS, all sites involved in industrial use of XPS compound for which site specific information was available, most of the sites involved in industrial use of HBCDD powder for XPS, and individual sites involved in textile backcoating.

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

Conclusion (iii) applies to the generic scenario for industrial use of XPS compound, three sites using HBCDD powder in the production of XPS and one site involved in textile backcoating including the generic scenario.

5.1.3 Atmosphere

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

This conclusion applies to all life cycle stages and scenarios.

5.1.4 Secondary poisoning

Aquatic predators, Terrestrial predators, Marine predators, Marine top predators

In the light of HBCDD being a PBT substance (c.f. 3.4.6) and considering the large uncertainties both in the derivation of PECs and in the derivation of PNEC it is not considered appropriate to draw conclusions for the individual sites. Since for PBT-substances the major concern is that accumulation of such substances in the foodchain may result in unpredictable effects in the long term it is appropriate to draw an overall conclusion iii) for secondary poisoning.

5.1.5 Marine environment

Marine Surface water

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 (ii) applies to production, micronising and industrial use of EPS and HIPS and for individual sites in the other use areas.

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

Conclusion (iii) applies to some sites involved in EPS formulation including the generic scenario, one site and the generic scenario for XPS formulation, one site involved in formulation of polymer dispersions for textiles including the generic scenario, individual sites involved in industrial use of HBCDD powder in XPS and use of XPS compound including the local generic scenario for industrial use of XPS compound, and some sites involved in textile backcoating including the generic scenario.

Marine Sediment

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 (ii) applies to production, micronising and industrial use of EPS and HIPS and for individual sites in the other use areas.

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

Conclusion (iii) applies to some sites involved in EPS formulation including the generic scenario, one site and the generic scenario for XPS formulation, one site involved in formulation of polymer dispersions for textiles including the generic scenario, individual sites involved in industrial use of HBCDD powder in XPS and use of XPS compound including the generic local scenario for industrial use of XPS compound, and some sites involved in textile backcoating including the generic scenario. In addition, measurements in marine sediment associated to a producer of EPS beads (EPS formulation) gives a RCR >1which indicates that there are concerns for this site and that there may be a general concern for this use area.

5.1.6 PBT-assessment

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

HBCDD does not unequivocally fulfil the specific P-criterion, with some reliable studies indicating that biodegradation can occur. It does however not degrade rapidly and monitoring data indicate a significant degree of environmental transport and overall stability. The BCF of HBCDD is 18 100 and thus the vB criterion is fulfilled. Also the T-criterion is fulfilled according to available data. HBCDD is ubiquitous in the environment, being also found in remote areas far away from point sources. The highest concentrations of HBCDD are detected in marine top-predators such as porpoise and seals showing that HBCDD bioaccumulates up the foodchain. Based on an overall assessment the TCNES subgroup on identification of PBT and vPvB substances have concluded that HBCDD has PBT properties according to the PBT criteria of the TGD.

5.2 HUMAN HEALTH

The results summarised here are presented in detail in section 4.1.3, ”Risk characterisation for Human Health”.

Exposed human populations include workers, consumers, and humans via the environment. In the case of HBCDD measured data from occupational-,consumer- and indirect exposure exists.

The endpoint of concern are:

- **repeated dose toxicity** for which a NOAEL/BMD-L of 22.9 mg/kg/day based on an increased liver weight is deduced from an 28 days oral study using a benchmark

model design.Worst-case exposures are assumed for all exposure scenarios and for a few occupational exposure scenarios, **conclusion (iii)** applies.

- **reproductive toxicity** for which a NOAEL of 10 mg/kg/day based on a dose-dependent decrease in fertility index and a reduced number of primordial follicles is deduced from a two generation reproductive toxicity studie in rats. Worst-case exposures are assumed for all exposure scenarios and for a few occupational exposure scenarios, **conclusion (iii)** applies.

There are indications of developmental neurotoxicity in adult mice exposed to HBCDD as pups. However, this study by Eriksson et al (2006) is not performed according to current guideline and GLP and therefore this potential developmental neurotoxicity needs to be examined further and conclusion (i) is reached for all exposure scenarios.

However, similar results on developmental neurotoxicity have been published for decabromodiphenyl ether by the same authors using the same metod. For decabromodiphenyl ether it has been agreed to perform a new toxicokinetics/developmental neurotoxicity study according to a modified OECD guideline and GLP. The results from this new decabromodiphenyl ether study will serve as a guidance on how to interpret the data from the Eriksson study, and may also serve as a basis on how to proceed with further testing of neurotoxicity. While awaiting this result, a **conclusion (i) on hold** with regard to a developmental neurotoxicity study applies.

5.2.1 Workers assessment

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

There are indications of developmental neurotoxicity in adult mice exposed to HBCDD as pups. However, this study by Eriksson et al (2006) is not performed according to current guideline and GLP and therefore this potential developmental neurotoxicity needs to be examined further and conclusion (i) is reached for all exposure scenarios.

However, similar results on developmental neurotoxicity have been published for decabromodiphenylether by the same authors using the same method. For decabromodiphenylether it has been agreed to perform a new toxicokinetics/developmental neurotoxicity study according to a modified OECD guideline and GLP. The results from this new decabromodiphenylether study will serve as guidance on how to interpret the data from the Eriksson study, and may also serve as a basis on how to proceed with further testing of neurotoxicity. While awaiting these results a **conclusion (i) on hold** with regard to a developmental neurotoxicity study applies.

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

Conclusion (ii) applies to repeated dose toxicity for workers during filling of HBCDD powder and granules in the production, adding of HBCDD fine powder, powder and granules in industrial use and sewing of HBCDD fine powder, powder and granules in industrial end use.
Conclusion (ii) also applies for toxicity on reproductive toxicity/fertility for workers during filling of HBCDD granules in the production, adding HBCDD granules in industrial use and sewing of HBCDD in industrial end-use.

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

There are three occupational exposure scenarios; exposure during filling (production), adding (industrial use), and sewing (end use by professionals). The results of the occupational assessment are presented in Table 5-1.

There is concern for repeated dose toxicity for workers during filling HBCDD fine grade powder in production. Therefor it is considered that risk reduction measures are required and **conclusion (iii)** applies.

There is concern for reproductive toxicity/fertility for workers during filling HBCDD fine and standard grade powder in production and during adding HBCDD fine and standard powder at industrial use of HBCDD as an additive. Therefor it is considered that risk reduction measures are required and **conclusion (iii)** applies.

Table 5-1. Overview of conclusions with respect to occupational risk characterisation.

End point	**Conclusions for the occupational scenarios**					
	Production		**Industrial use**		**End use, professional**	
	MOS	**conclusion**	**MOS**	**conclusion**	**MOS**	**conclusion**
Repeated dose toxicity; fine grade powder						
-inhalation	16	**(iii)**	52	(ii)	286	(ii)
-dermal	10	**(iii)**	327	(ii)	382	(ii)
-multiple exposure	6	**(iii)**	45	(ii)	164	(ii)
Repeated dose toxicity; standard grade powder						

End point	Conclusions for the occupational scenarios					
	Production		Industrial use		End use, professional	
	MOS	conclusion	MOS	conclusion	MOS	conclusion
-inhalation	85	(ii)	64	(ii)		
-dermal	47	(ii)	458	(ii)		
-multiple exposure	30	(ii)	56	(ii)		
Repeated dose toxicity; granules/masterbatch -all routes and multiple	>458	(ii)	>739	(ii)		
Reproductive toxicity; fine grade powder						
-inhalation	7	**(iii)**	23	**(iii)**	125	(ii)
-dermal	4	**(iii)**	143	(ii)	167	(ii)
-multiple exposure	3	**(iii)**	20	**(iii)**	71	(ii)
Reproductive toxicity; standard grade powder						
-inhalation	37	**(iii)**	28	**(iii)**		
-dermal	20	**(iii)**	200	(ii)		
-multiple exposure	13	**(iii)**	24	**(iii)**		
Reproductive toxicity; granules/masterbatch -all routes and multiple	>200	(ii)	>323	(ii)		

5.2.2 Consumers assessment

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

One exposure scenario for consumers have been considered; mouthing of textile. The results of the consumer assessment are presented in Table 5-2. There is no concern with respect to repeated dose toxicity or reproductive toxicity/fertility and conclusion (ii) **applies.Table 5-2. Overview of conclusions with respect to risk characterisation for consumers.**

Endpoint	Mouthing of textile (both sides)	
	MOS	Conclusion
Repeated dose toxicity	760	(ii)
Reproductive toxicity	330	(ii)

5.2.3 Humans exposed via the environment assessment

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

The EUSES modelling predicts too high concentrations of HBCDD in root crops and fish locally around plants formulating or using HBCDD. Based on data calculated from EUSES modelling, there is concern for reproductive and repeated dose toxicity in one site-specific local scenario relating to textile backcoating and the generic textile industrial use scenario. However, these conclusions are unrealistic. Instead, if using the highest measured HBCDD concentration in fish and the measured value of HBCDD concentration in potatoes, the result seems more realistic. Consequently, the MOS values are approximately 100-1000 times higher than those based on calculated data. Usually, calculated data are used. However, in this case where we are able to compare calculated with measured values, it is obvious that the model overpredicts the HBCDD concentration in root crops and in fish. Consequently, there is no concern with respect to repeated dose toxicity for humans exposed via the environment and conclusion (ii) applies for all identified scenarios.

No calculations concerning combined exposure have been performed.

5.2.4 Human health (physico-chemical properties)

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

Flammability, explosive and oxidising properties are not considered to be of concern and conclusion (ii) applies.

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ABBREVIATIONS

ABS	Acrylonitrile butadiene styrene (polymer)
ADI	Acceptable Daily Intake
AF	Assessment Factor
ASTM	American Society for Testing and Materials
ATP	Adaptation to Technical Progress
AUC	Area Under The Curve
B	Bioaccumulation
BBA	Biologische Bundesanstalt für Land- und Forstwirtschaft
BCF	Bioconcentration Factor
BMC	Benchmark Concentration
BMD	Benchmark Dose
BMF	Biomagnification Factor
BUA	Beratergremium für umweltrelevante Altstoffe
bw	body weight / *Bw*, *b.w.*
C	Corrosive (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
CA	Chromosome Aberration
CA	Competent Authority
CAS	Chemical Abstract Services
CEC	Commission of the European Communities
CEN	European Standards Organisation / European Committee for Normalisation
CMR	Carcinogenic, Mutagenic and toxic to Reproduction
CNS	Central Nervous System
COD	Chemical Oxygen Demand
CSTEE	Scientific Committee for Toxicity, Ecotoxicity and the Environment (DG SANCO)
CT_{50}	Clearance Time, elimination or depuration expressed as half-life
Dwt	Dry weight
d.wt	dry weight / dw
Dfi	daily food intake
DG	Directorate General
DIN	Deutsche Industrie Norm (German norm)
DNA	DeoxyriboNucleic Acid
DOC	Dissolved Organic Carbon
DT50	Degradation half-life or period required for 50 percent dissipation / degradation
DT90	Period required for 50 percent dissipation / degradation

E	Explosive (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
EASE	Estimation and Assessment of Substance Exposure Physico-chemical properties [Model]
EbC50	Effect Concentration measured as 50% reduction in biomass growth in algae tests
EC	European Communities
EC10	Effect Concentration measured as 10% effect
EC50	median Effect Concentration
ECB	European Chemicals Bureau
ECETOC	European Centre for Ecotoxicology and Toxicology of Chemicals
ECVAM	European Centre for the Validation of Alternative Methods
EDC	Endocrine Disrupting Chemical
EEC	European Economic Communities
EINECS	European Inventory of Existing Commercial Chemical Substances
ELINCS	European List of New Chemical Substances
EN	European Norm
EPA	Environmental Protection Agency (USA)
EPS	Expanded polystyrene
ErC50	Effect Concentration measured as 50% reduction in growth rate in algae tests
ESD	Emission Scenario Document
EU	European Union
EURO	The European Union before the enlargement 2004
EUSES	European Union System for the Evaluation of Substances [software tool in support of the Technical Guidance Document on risk assessment]
EXIBA	European Extruded Polystyrene Insulation Board Association
F(+)	(Highly) flammable (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
FAO	Food and Agriculture Organisation of the United Nations
FELS	Fish Early Life Stage
GLP	Good Laboratory Practice
HBCDD	Hexabromocyclododecane
HEDSET	EC/OECD Harmonised Electronic Data Set (for data collection of existing substances)
HELCOM	Helsinki Commission -Baltic Marine Environment Protection Commission
HIPS	High-impact polystyrene
HPLC	High Pressure Liquid Chromatography
HPVC	High Production Volume Chemical (> 1000 t/a)
IARC	International Agency for Research on Cancer
IC	Industrial Category
IC50	median Immobilisation Concentration or median Inhibitory Concentration

ILO	International Labour Organisation
IPCS	International Programme on Chemical Safety
ISO	International Organisation for Standardisation
IUCLID	International Uniform Chemical Information Database (existing substances)
IUPAC	International Union for Pure and Applied Chemistry
JEFCA	Joint FAO/WHO Expert Committee on Food Additives
JMPR	Joint FAO/WHO Meeting on Pesticide Residues
Koc	organic carbon normalised distribution coefficient
Kow	octanol/water partition coefficient
Kp	solids-water partition coefficient
Lwt	Lipid weight
L(E)C50	median Lethal (Effect) Concentration
LAEL	Lowest Adverse Effect Level
LC50	median Lethal Concentration
LD50	median Lethal Dose
LEV	Local Exhaust Ventilation
LLNA	Local Lymph Node Assay
LOAEL	Lowest Observed Adverse Effect Level
LOEC	Lowest Observed Effect Concentration
LOED	Lowest Observed Effect Dose
LOEL	Lowest Observed Effect Level
MAC	Maximum Allowable Concentration
MATC	Maximum Acceptable Toxic Concentration
MC	Main Category
MITI	Ministry of International Trade and Industry, Japan
MOE	Margin of Exposure
MOS	Margin of Safety
MS	Mass spectrometry
MSDS	Material safety data sheet
MW	Molecular Weight
N	Dangerous for the environment (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC
NAEL	No Adverse Effect Level
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
NOEC	No Observed Effect Concentration
NTP	National Toxicology Program (USA)
O	Oxidizing (Symbols and indications of danger for dangerous substances and preparations

	according to Annex III of Directive 67/548/EEC)
OECD	Organisation for Economic Cooperation and Development
OEL	Occupational Exposure Limit
OJ	Official Journal
OSPAR	Oslo and Paris Convention for the protection of the marine environment of the Northeast Atlantic
P	Persistent
PBDD	Polybrominated dibenzodioxins
PBDF	Polybrominated dibenzofurans
PBT	Persistent, Bioaccumulative and Toxic
PBPK	Physiologically Based PharmacoKinetic modelling
PBTK	Physiologically Based ToxicoKinetic modelling
PE	Polyethylene
PEC	Predicted Environmental Concentration
PET	Polyethylene terephthalate
pH	logarithm (to the base 10) (of the hydrogen ion concentration $\{H^+\}$
pKa	logarithm (to the base 10) of the acid dissociation constant
pKb	logarithm (to the base 10) of the base dissociation constant
PNEC	Predicted No Effect Concentration
POP	Persistent Organic Pollutant
PP	Polypropylene
PS	Polystyrene
PPE	Personal Protective Equipment
QSAR	(Quantitative) Structure-Activity Relationship
R phrases	Risk phrases according to Annex III of Directive 67/548/EEC
RAR	Risk Assessment Report
RC	Risk Characterisation
RfC	Reference Concentration
RfD	Reference Dose
RHO	Bulk density of the solid phase (soil, sediment, susp. matter)
RNA	RiboNucleic Acid
RPE	Respiratory Protective Equipment
RWC	Reasonable Worst Case
S phrases	Safety phrases according to Annex III of Directive 67/548/EEC
SAN	Styrene-acrylonitrile copolymer
SAR	Structure-Activity Relationships
SBR	Standardised birth ratio
SCE	Sister Chromatic Exchange

SDS	Safety Data Sheet
SETAC	Society of Environmental Toxicology And Chemistry
SIMPLETREAT	Fugacity model for simulation of the fate of chemicals in wastewater treatment plants. Based on partition coefficient octanol-water, vapour pressure and biodegradability.
SNIF	Summary Notification Interchange Format (new substances)
SSD	Species Sensitivity Distribution
STP	Sewage Treatment Plant
STW	Sewage Treatment Water
T(+)	(Very) Toxic (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
TDI	Tolerable Daily Intake
TG	Test Guideline
TGD	Technical Guidance Document
TLV	Threshold Limit Value
TNsG	Technical Notes for Guidance (for Biocides)
TNO	The Netherlands Organisation for Applied Scientific Research
TWA	Time Weighted Average
UC	Use Category
UDS	Unscheduled DNA Synthesis
UN	United Nations
UNEP	United Nations Environment Programme
US EPA	Environmental Protection Agency, USA
UV	Ultraviolet Region of Spectrum
UVCB	Unknown or Variable composition, Complex reaction products of Biological material
vB	very Bioaccumulative
VCR	Video cassette recorder
vP	very Persistent
vPvB	very Persistent and very Bioaccumulative
v/v	volume per volume ratio
w/w	weight per weight ratio
Wwt	Wet weight
WHO	World Health Organization
WWTP	Waste Water Treatment Plant
Xi	Irritant (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
Xn	Harmful (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
XPS	Extruded polystyrene

Appendix 1. Modified PECs

This appendix contains modified PECs further used in the risk characterisation. "$PEC_{agricultural\ soil}$ 30 day average" and "$PEC_{oral\ predator.earthworm}$ " presented here is the result of using slower degradation in soil and sediment as descibed in chapter *3.1.3.1 Degradation and transformation in the environment*, and chapter *3.4.4. The use of alternative biodegradation scenarios.*

"$PEC_{oral\ predator\ .\ fish}$", "$PEC_{oral\ marinepredator\ (fish)}$", and "$PEC_{oral\ marine\ top\ predator}$" is the result of the use of measured values of the concentration in fish and top predators as described in chapter 3.2.4.3 Comparison between predicted and measured levels.

X = PECs not relevant

Site	**Connected to municipal STP (Yes/No)**	**$PEC_{agricultural\ soil}$ 30 day average**	**$PEC_{oral\ predator\ earthworm}$**	**$PEC_{oral\ predator\ fish}$**	**$PEC_{oral\ marine\ predator\ (fish)}$**	**$PEC_{oral\ marine\ top\ predator}$**
		mg/kg dw	mg/kg	mg/kg ww		
ProdA	Yes	9.6	17	4800	X	X
ProdB	Yes	0.0036	0.0065	X	0.019	0.37
Micronіz	Yes/No	X	0.0004	0.020	0.0018	0.34
Site A	No	X	X	0.44	X	X
Site F	No	X	X	3.5	X	X
Site I	No	X	X	0.58	X	X
Site B	Yes	0.00072	0.0016	0.023	X	X
Site C	No	0.0048	0.0086	1.7	1.6	3.6
Site D	No	X	X	1.3	X	X
Site E	No	X	X	0.67	X	X
Site G	Yes	0.015	0.027	6.3	0.63	1.6
Site H	No	X	X	2	X	X
Site J	No	X	X	110	X	X
Site K	Yes	0.12	0.20	49	4.9	10
Site L	Yes	0.060	0.10	25	2.5	5.2
Site P	No	X	X	3.5	0.35	1.0
GEN_EPS_FORM	Yes	0.12	0.23	55	5.5	11
	No	X	X	260	26	53
MasterbG	Yes	0.00070	0.0016	0.31	0.031	0.39
MasterbH	Yes	0.0016	0.0031	0.67	0.66	X
MasterbI	Yes	0.15	0.26	62	6.2	13
GEN_XPS_FORM	Yes	0.13	0.26	62	6.2	13
	No	X	X	300	30	61

Site	Connected to municipal STP (Yes/No)	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{oral\ predator\ earthworm}$	$PEC_{oral\ predator\ fish}$	$PEC_{oral\ marine\ predator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
TexForm1	Yes	0.00058	0.0014	0.26	0.025	0.38
	No	X	X	1.2	0.12	0.57
TexForm3	Yes/No*	X	0.00040	0.020	X	X
TexForm4	Yes	0.00027	0.00086	0.13	0.013	0.36
	No	X	X	0.54	0.054	0.44
TexFormB	Yes	0.049	0.085	20	2.0	4.4
	No	X	X	98	9.8	20
GEN_TEX_FORM	Yes	0.49	0.96	230	23	46
	No	X	X	1100	110	220
GEN_EPS_IndUse	Yes	0.0026	0.0055	1.2	0.12	0.58
	No	X	X	5.9	0.59	1.5
GEN_HIPS_IndUse	Yes	0.0081	0.016	0.4	0.040	0.41
	No	X	X	1.9	0.19	0.70
XPS 1	Yes	0.26	0.45	0.074	0.055	0.44
	No	X	X	0.28	0.26	0.86
XPS 3	Yes	0.24	0.41	0.30	0.28	0.90
	No	X	X	1.4	1.4	3.1
XPS 11	Yes	0.80	1.4	10	1.0	2.4
	No	X	X	49	4.9	10
GEN_XPS_IndUse	Yes	1.3	2.6	19	1.9	4.1
	No	X	X	91	9.1	18
XPS 4*	Yes	0.42	0.72	10	1.0	2.4
	No	X	X	51	5.1	11
XPS 5	Yes	0.11	0.19	0.047	X	X
	No	X	X	0.15	X	X
XPS 6	Yes	0.011	0.019	0.021	X	X
	No	X	X	0.026	X	X
XPS 7	Yes	0.70	1.2	0.11	X	X
	No	X	X	0.45	X	X
XPS 8	Yes	0.00024	0.00083	0.026	0.0024	0.34
	No	X	X	0.048	0.0046	0.34
XPS 10	Yes	1.1	2.0	10	X	X
	No	X	X	50	X	X
XPS 13	Yes	0.00057	0.0014	0.027	X	X
	No	X	X	0.23	X	X
XPS 14***	Yes	0.00020	0.00074	-	0.0023	0.34

Site	Connected to municipal STP (Yes/No)	$PEC_{agricultural\ soil}$ 30 day average	$PEC_{oral\ predator\ earthworm}$	$PEC_{oral\ predator\ fish}$	$PEC_{oral\ marine\ predator\ (fish)}$	$PEC_{oral\ marine\ top\ predator}$
XPS 16	Yes/No**	X	0.00041	0.020	X	X
XPS 17	No	X	X	0.020	X	X
XPS 18	No	X	X	0.020	X	X
XPS 20	Yes	0.010	0.018	0.023	X	X
XPS 21	Yes	1.4	2.5	1.7	X	X
	No	X	X	8.4	X	X
XPS 23	Yes	0.000012	0.00042	0.12	0.012	0.34
XPS 24	Yes	0.000011	0.00042	0.021	0.0019	0.34
XPS 26	Yes	0.0040	0.0074	0.071	X	X
XPS 27	Yes	0.48	0.82	6.0	0.60	1.5
	No	X	X	29	2.9	6.2
Backcoat.1	Yes	0.055	0.094	17	1.7	3.7
	No	X	X	82	8.2	17
Backcoat.2	Yes	0.054	0.094	4.0	0.40	1.1
	No	X	X	20	2.0	4.2
Backcoat.3	Yes	9.3	16	1100	110	220
	No	X	X	5200	520	1040
Backcoat.4	Yes	0.00011	0.00060	0.047	0.0045	0.34
	No	X	X	0.15	0.015	0.36
BackcoatC	Yes	0.018	0.031	1.1	0.11	0.54
	No	X	X	5.0	0.50	1.3
GEN_TEX_IndUse	Yes	8.2	16	3200	320	640
	No	X	X	6000	1600	3100

Appendix 2. EASE - modelled occupational exposure data.

Modelling is performed for;

- **production of HBCDD** and for
- **industrial use of HBCDD as an additive**.

HBCDD is handled either as fine powder, standard grade powder, granules, or XPS/EPS-masterbatch. Based on job descriptions in production and industrial use, respectively, separate modelling for fine and standard grade HBCDD is performed for production, whereas one common modelling for fine and standard grade is performed for industrial use.
No modelling is performed for use of granules or master-batch.

Production of HBCDD (filling of bags).

Particle size	Inhalation exposure	Dermal exposure
Fine	No modelling is performed as measured data exists, and indicates a very high degree of dusting.	The temperature of the process is 20 The physical-state is solid dust-inhalation is false mobile-solid is true solid-vp is false The exposure-type is dermal The use-pattern is Dispersive use The pattern-of-control is Direct handling The contact-level is Intermittent CONCLUSION: The predicted dermal exposure to HBCDD is 1-5 mg/ cm^2 /day
Standard	The name of the substance is HBCDD The temperature of the process is 25 The physical-state is solid dust-inhalation is true mobile-solid is true solid-vp is false The exposure-type is dust The particle-size is Inhalable *(Standard* The operations is Dry manipulation The dust-type is Non-fibrous aggregates is false The pattern-of-control is LEV present CONCLUSION: The predicted dust exposure to HBCDD is 2-5 mg/ m^3	The temperature of the process is 20 The physical-state is solid dust-inhalation is false mobile-solid is true solid-vp is false The exposure-type is dermal The use-pattern is Non-dispersive use The pattern-of-control is Direct handling The contact-level is Intermittent CONCLUSION: The predicted dermal exposure to HBCDD is 0.1-1 mg/cm^2 /day

Industrial use of HBCDD as an additive

Particle size	Inhalation exposure	Dermal exposure
Fine and standard grade	The name of the substance is HBCDD The temperature of the process is 25 The physical-state is solid dust-inhalation is true mobile-solid is true solid-vp is false The exposure-type is dust The particle-size is Respirable-inhalable The operations is Dry manipulation The dust-type is Non-fibrous aggregates is false The pattern-of-control is LEV present CONCLUSION: The predicted dust exposure to HBCDD is 2-5 mg/m^3	The name of the substance is HBCDD The temperature of the process is 25 The physical-state is solid dust-inhalation is false mobile-solid is true solid-vp is false The exposure-type is dermal The use-pattern is Non-dispersive use The pattern-of-control is Direct handling The contact-level is Incidental CONCLUSION: The predicted dermal exposure to HBCDD is 0-0.1 mg/c m^2 /day

The report provides the comprehensive risk assessment of the substance Hexabromocyclododecane It has been prepared by Sweden in the frame of Council Regulation (EEC) No. 793/93 on the evaluation and control of the risks of existing substances, following the principles for assessment of the risks to man and the environment, laid down in Commission Regulation (EC) No. 1488/94.

The evaluation considers the emissions and the resulting exposure to the environment and the human populations in all life cycle steps. Following the exposure assessment, the environmental risk characterisation for each protection goal in the aquatic, terrestrial and atmospheric compartment has been determined.

The environmental risk assessment for Hexabromocyclododecane concludes that the substance has PBT properties. There is additional concern for the freshwater and marine environment due to the use of HBCDD in the formulation of EPS, XPS, and polymer dispersions for textiles; the industrial use of XPS and textile backcoating. A risk is also identified for the functioning of waste water treatment plants of sites with industrial use of XPS with intermittent releases and due to textile backcoating.
For human health the scenarios for occupational exposure, consumer exposure and humans exposed via the environment have been examined and the possible risks have been identified. The human health risk assessment concludes that there is concern for workers with regard to repeated dose toxicity and reproductive toxicity/fertility during production and industrial use of standard and fine grade powder. There is a need for further information and testing (on hold) for developmental neurotoxicity. For all other worker exposure scenarios, for consumers, for humans exposed via the environment and for human health (physico-chemical properties) there is no concern.