An evaluation of hexabromocyclododecane (HBCD) for Persistent Organic Pollutant (POP) properties and the potential for adverse effects in the environment

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European Brominated Flame Retardant Industry Panel (EBFRIP)

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EXECUTIVE SUMMARY

This document is an environmental risk assessment of hexabromocyclododecane (HBCD). HBCD is also assessed with regards to persistent organic pollutant (POP) and persistent, bioaccumulation and toxicity (PBT) categorization criteria. Mass balance model simulations are used to compare HBCD with benchmark chemicals including listed POPs, non-POPs, and candidate POPs. Fate and exposure model predictions are compared with available monitoring data in "source" and "remote" regions to corroborate likely patterns for emissions and mode-of-entry to the environment. Body/tissue-based effects and no effects thresholds (PNECs) and total daily intake (TDI) rates associated with no observed adverse effects levels (NOAELs) are combined with exposure data for screening level assessments of risk in "source" and "remote" environments.

The findings in the present study show that HBCD fulfils some, but not all, of the UN-ECE and UNEP Stockholm Convention POP criteria. For example, based on UN-ECE POP criteria, HBCD shows the potential for long range transboundary atmospheric transport (1a), and bioaccumulation (1d); however, the available environmental half-life data suggest an uncertain categorization for persistence (1c), and current monitoring and toxicity data indicate that HBCD levels in remote regions are below those associated with the likelihood of adversely affecting human health and/or the environment (1b). Furthermore, available monitoring and modelling data suggest long range transboundary atmospheric transportation potential (2a); however, based on current data, HBCD is not likely to have adverse human health and/or environmental effects as a result of its long range transboundary atmospheric transport (2b).

Commercial (technical) t-HBCD is a chemical mixture, primarily consisting of the three diastereomers α-, β-, and γ-HBCD. Mixtures and chiral substances need to be evaluated on an individual basis because each structure has unique properties. The present assessment includes a comprehensive collection and critical review of available physical-chemical property and transformation data, monitoring data, novel model simulations, and toxicity data for t-HBCD and α-, β-, and γ-HBCD. Individual diastereomers were evaluated to the greatest extent possible based on the available data. Due to the limited availability of data for the diastereomers, particularly with regards to transformation and isomerization, it was ultimately necessary to evaluate HBCD based on representative properties selected for t-HBCD. In some cases, model predictions for the diastereomers were compared with t-HBCD predictions.

Physical-chemical property measurements and estimates were critically reviewed and thermodynamically consistent property values were calculated (Final Adjusted Values, FAVs). Measurements and model estimates for environmental degradation and metabolic biotransformation half-lives were critically evaluated. Median half-lives and the expected range of plausible values (lower and upper bound estimates) were selected for air, water, soil, sediment, fish, birds and mammals. Efforts were made to reduce uncertainty in the estimate of these key properties, while recognizing that uncertainty in these estimates cannot be eliminated entirely. Thus, many mass balance calculations also consider uncertainty for the required model input parameters.

Available monitoring data were separated into general categories of "local/near-point source", "source" and "remote" regional scales based on distances, corresponding with production and industrial facilities, urban/rural areas, and Arctic regions, respectively. HBCD is detected in each of these regions indicating the potential for long range transport (i.e. 1a and 2a of the UN-ECE POP criteria). However, spatial trends show decreasing concentrations with increased distance from known point sources. Furthermore, remote regions are shown to have environmental concentrations that are orders of magnitude lower than those near-point sources. Temporal trends in the monitoring data were explored but no consistent trends could be identified when considering all of the available studies. There appear to be slight increases in certain compartments in the past 15-20 years; however, other compartments show no noticeable trends. The slight increase is primarily observed in compartments that require longer times to reach steady state and may reflect an approach to steady state levels.

Mass balance model simulations compare HBCD to classified POPs, substances that are not considered to be POPs (non-POPs), and substances that are presently under review as candidate POPs (candi-POPs) in a variety of benchmarking exercises. HBCD is shown to have some potential for long range transport and overall persistence (P_{OV}); however, other non-POP chemicals also show these properties. These properties for HBCD are generally found to be lower than POPs and candidate POPs, particularly when median and lower bound degradation half-lives for HBCD are considered The benchmark comparisons do not provide clear evidence for assigning HBCD as a "POP" or a "non-POP" largely because of the uncertainties in the half-life data and the wide range of LRT and P_{OV} values for POPs and non-POPs. Uncertainty for HBCD mode-of-entry to the environment is also a factor. These findings and comparisons of the plausible range of half-life values for HBCD with media specific half-life persistence criteria result in an ambiguous persistence categorization (i.e. 1c of the UN-ECE and UN Stockholm Convention criteria).

Realistic emissions estimates were used to model steady state concentrations in a range of representative species of varying trophic position including fish, birds, marine mammals, and humans in a regional environment. These predictions were compared with monitoring data representative of regions in Northern Europe (Sweden, Norway). Reasonable agreement of the predicted concentrations with the available monitoring data (i.e. typically within a factor of 3) suggests that HBCD is near steady state conditions in the environment and that the selected model input parameters for HBCD are reasonable. The model predicted upper trophic level organisms, particularly species that consume fish such as marine mammals and piscivorous birds, to have the highest exposure potential. The model predictions are corroborated by the available monitoring data. These findings support selected estimates for slow biotransformation rates in fish and the bioaccumulation potential categorization for HBCD (i.e. 1d of the UN-ECE criteria). A sensitivity analysis provides recommendations to reduce uncertainty in model predictions. For exposures to marine mammals biotransformation half-lives in biota are the most sensitive parameters followed by degradation half-lives in water and sediment.

This largely reflects the primary route of dietary exposure to upper trophic level organisms from the consumption of fish.

Global scale mass balance model predictions using spatially resolved estimates for realistic emission rates and assumed mode-of-entry scenarios were also found to be in reasonable agreement with available monitoring data from different regions. The model predicts lower concentrations in remote regions than in source regions also reflecting the observed spatial trends in the monitoring data.

Regional scale dynamic mass balance calculations were used to estimate response times for HBCD in the environment. Response times in most environmental media are predicted to range from a few days (air) to about 5 years (soil). In comparison, listed POPs such as PCB 180 have much longer response times in the environment, of the order of years to decades. Thus, the model results indicate that concentrations of HBCD in the environment will decline faster in response to reduced emissions than those of many listed POPs. The relatively short response times for HBCD also partly explain why steady state model predictions are in good agreement with the monitoring data.

A toxicological evaluation of HBCD was carried out with respect to a PBT classification based on European Commission "T" criteria and for the basis of conducting a risk assessment using available monitoring data. We first discuss the findings for the "T" categorization followed below by the findings for the risk assessment. The aquatic exposure-based ecotoxicity data available for HBCD are generally confounded by either the use of a cosolvent or a generator column. OECD testing guidance for difficult substances and mixtures (HBCD is both) specifically recommends that such practices be avoided because of the uncertainties associated with interpreting such exposure-based test results. Therefore, these aquatic exposure-based results that were used in previous assessments are considered herein to be uncertain and of questionable reliability, particularly for comparison to the aquatic exposure-based "T" criteria.

The predominant isomer in t-HBCD is γ-HBCD, which has a water solubility limit of about 2 to 3 $\mu g \cdot L^{-1}$. For exposure concentrations of t-HBCD greater than about 3 $\mu g \cdot L^{-1}$ precipitated γ-isomer in the water column make it impossible to produce a purely dissolved aqueous t-HBCD exposure. The low water solubility of t-HBCD brings into question the validity of using the European Commission NOEC "T" criterion of 10 $\mu g \cdot L^{-1}$ for toxicity assessments, or for that matter, other aquatic exposure-based criteria greater than a substance's water solubility limit. It is likely that this "T" criterion is inappropriate and unsuitable for the PBT classification of HBCD and other substances with very low water solubility. Importantly, whereas some of the aquatic toxicity data are above the water solubility limit for γ-HBCD, those data that are at, or just below, the water solubility limit show no effects. Thus, the positive "T" assessment conclusion in the previous risk assessment by the European Commission is not supported in the present assessment. The present detailed interpretation and evaluation indicates that available key aquatic toxicity data for HBCD show low toxicity.

The present risk assessment was used to assess HBCD with respect to POP evaluation criteria (e.g. UN-ECE 1b and 2b, the potential for significant adverse effects as a result of long range atmospheric transport). To conduct this assessment reliable toxicity endpoints for possible effects and no effects are needed. Aquatic exposure-based toxicity endpoints, such as those used in previous risk assessments, are not readily applicable for multimedia-based exposures to HBCD in the environment, particularly for higher trophic level organisms that are exposed to HBCD by the consumption of food. Alternative methods for toxicity and risk evaluations that are not affected by the confounding issues related to the aqueous-exposure toxicity endpoints are the body/tissue-residue and total daily intake (TDI) approaches. Illustrative examples of these alternative approaches for HBCD risk evaluation were prepared to address the UN-ECE POPs evaluation (i.e. 1b and 2b). For the body/tissue-residue approach, dose-response data based on measured amounts of HBCD in the organism are used to establish thresholds that can be directly compared to organism monitoring data. As discussed below, the present study adopts two residue-based PNECs; one for baseline narcosis and one for an assumed and unidentified more specific mode of action ("worst-case"). For the TDI approach, HBCD exposure dose levels associated with laboratory NOAELs are compared with estimated HBCD intake rates in the environment. These two general approaches are considered viable methods to estimate the likelihood of significant adverse effects in upper trophic level organisms in source and remote regions.

Available residue-based toxicity data suggest that HBCD toxicity to freshwater and marine fish and earthworms is due to baseline narcosis, the least toxic mode of action exhibited by organic chemicals. A residue-based PNEC was established for this mode of action. Some recent work with rodents exposed to t-HBCD includes residue-effect estimates for several response endpoints. It is uncertain if some of the observations in the rat data (i.e. detectable physical and/or biochemical changes but no organism level survival, growth, or reproduction effects) are due to effects at the extreme tail of baseline narcosis toxicity or that a separate, more specific, yet unknown mode of toxic action is operational. As a "worst case" risk assessment exercise, another residue-based PNEC was assumed at a tissue level 100 times lower the baseline narcosis PNEC to address the uncertainties associated with the mammalian toxicity data. This "worst case" toxicity PNEC is considered to be a more stringent and conservative threshold for the present screening assessment; however, it is unclear whether this "worst case" PNEC is truly indicative of a significant adverse effect.

Available monitoring data were separated into general categories of "local/near-point source", "source" and "remote" regional scales and subdivided into organism groupings. For the baseline narcosis mode of toxic action residue-based PNEC there is no indication of potential adverse effects based on any of the available monitoring data for invertebrates, fish, marine mammals, or birds. For the more stringent "worst case" PNEC there was no indication of exposures exceeding this threshold for any of these organism groups at "remote" locations. For "source" regions, the upper end of the marine mammal monitoring concentration distribution exceeds the "worst case" PNEC threshold while the upper end of the bird monitoring data enters the "worst-case" PNEC threshold. For "local/near point source" regions (i.e. sites near known point source emissions of

HBCD), the majority of the marine mammal and fish monitoring concentration distributions exceed the "worst case" PNEC threshold and the upper end of the bird monitoring data enters the "worst-case" PNEC threshold. Based on the "worst case" body/tissue-residue PNEC threshold, present HBCD concentrations in biota at local/source regions approach levels warranting a more comprehensive risk assessment as is typically conducted for non-POP substances.

The potential for significant adverse effects was also evaluated by comparing TDI exposure estimates of HBCD for upper trophic level species in the environment (i.e. marine mammals) against a TDI NOAEL of 10 $mg \cdot (kg\text{-}bw \cdot d)^{-1}$ derived from laboratory testing data. This NOAEL was recommended in the European Commission Draft Risk Assessment Report. The TDI required to obtain a steady state concentration of HBCD corresponding with the highest measured concentration in a marine mammal in a remote region is about 5 orders of magnitude below the NOAEL. The TDI of HBCD corresponding to a steady state concentration at the 95th percentile of measured concentrations in marine mammals in "source" and "local/near point source" regions ranges from about 2 to 3 orders of magnitude below the NOAEL. Based on this endpoint for risk characterization, TDIs for piscivorous marine mammals in all three geographic regions were well below the NOAEL suggesting no potential for significant adverse effects (i.e. 1b and 2b of the UN-ECE POP criteria).

In summary, simply because a substance meets screening level hazard categorization criteria and can be detected in remote environments, does not constitute sufficient justification for concluding that there is a likelihood of significant adverse effects in remote environments. The final judgement on whether a candidate substance such as HBCD should be categorized as a POP requires a more detailed and thorough risk-based assessment. A risk-based approach using organism-based dose metrics is suggested to evaluate exposure levels in the environment by comparing them with levels associated with effects and no effects.

Three evaluations using these methods were illustrated in the present study using available data. These evaluations do not support the classification of HBCD as a POP since there are no indications that significant adverse effects are likely to occur in organisms living in remote areas distant from known point-source emissions of HBCD. The present assessment does not include a thorough uncertainty analysis; however, conservative values have been selected. A comprehensive risk assessment fully characterizing the uncertainty in the present findings is not possible at this time due to isomer specific data limitations for HBCD, in particular for the diastereromers (i.e. isomerization rates, isomer specific degradation rates, and potential differences in isomer toxicity) Recommendations are provided in the report to reduce uncertainty in further risk-based evaluations of HBCD.

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1.0 INTRODUCTION

1.1 BACKGROUND

Chemical assessment methods and criteria have been developed over the last 40 years and are now being applied broadly in regulatory programs such as the United Nations Stockholm Convention on Persistent Organic Pollutants (POPs) (UNEP 2001). Programs such as these often screen intrinsic chemical properties against selected criteria or "trigger values" in specified and separate categories such as persistence (P), bioaccumulation (B), and toxicity (T). Often few experimental and monitoring data are available for current chemical regulatory assessment needs, necessitating the use of Quantitative Structure-Activity Relationships (QSARs) to obtain chemical property estimates for mass balance models to enable environmental fate and exposure prediction (Muir and Howard 2006).

Hexabromocyclododecane (HBCD) is used as a flame retardant primarily in building insulation composed of extruded or expanded polystyrene foam but also in textiles and other products. Technical (commercial) t-HBCD is actually a mixture of primarily three diastereomers: α-, β-, and γ-HBCD (Heeb et al. 2005; Heeb et al. 2008). In the present study t-HBCD refers explicitly to the technical mixture, α-, β-, and γ-HBCD refer to the three diastereomers, and HBCD refers to cases in which the reference has been unclear in previous reports or distinctions are not considered relevant for the discussion. The Swedish Chemicals Agency has conducted a risk assessment for t-HBCD for the European Commission (EC 2008) and the Nordic Council of Ministers has completed a report in which information on environmental properties for t-HBCD were compared with the criteria for POP categorization (TemaNord 2008). The TemaNord document is being used to recommend adding t-HBCD to Annex A of the United Nations Stockholm Convention on POPs and Annex 1 of the United Nations Economic Commission for Europe (UN-ECE) Protocol on POPs under the Convention on Long range Transboundary Air Pollution.

The European Commission Draft Risk Assessment Report (RAR) for t-HBCD was carried out in accordance with Council Regulation (EEC) 793/93 on the evaluation and control of the risks of existing substances using a method supported by a Technical Guidance Document (TGD) (EC 2003; EC 2008). The RAR concludes that t-HBCD meets PBT criteria as outlined in the TGD; however, as noted in the draft report, the information contained therein does not necessarily provide a sufficient basis for decision-making regarding the hazards, exposures or the potential risks associated with t-HBCD (EC 2008). In the RAR, t-HBCD was considered a T-substance largely because of the 21-day Daphnia No Observed Effect Concentration (NOEC) of 3.1 $\mu g \cdot L^{-1}$ (EC criterion for NOEC <10 $\mu g \cdot L^{-1}$). The RAR indicated an absence of persistence based on degradation half-lives of 1.5 and 7 days for anaerobic sediments and soils, respectively; however, t-HBCD was considered to be "P" because it could be found in biota of remote regions such as the Arctic. In the TemaNord report (2008), t-HBCD was classified as "P" based on temperature-adjusted degradation half-lives of α- and γ-HBCD in aerobic sediments

(12 ^{o}C) and negligible degradation in soil from one particular study (Davis et al. 2004). Half-life data documented in the RAR and TemaNord reports are largely contradictory and uncertain.

There are uncertainties with the available data (e.g. degradation half-lives) and the interpretation of these data such that the conclusions in the previous assessments may be inappropriate. In particular, as to whether or not HBCD is likely to have significant adverse human health and/or environmental effects as a result of its long range transboundary atmospheric transport. HBCD is very hydrophobic and complications related to conducting water-based tests and uncertainties interpreting the data for hydrophobic substances are well recognized. The PBT hazard categories are intended to identify chemicals that may pose potential risks to humans and the environment. These screening level methods and criteria may not effectively identify and prioritize chemicals of concern and complementary holistic methods are recommended (Arnot and Mackay 2008). Wu and colleagues (2008) and Leonards and colleagues (2008) have also argued that more comprehensive POP assessments should compare threshold body burdens associated with measured or expected concentrations in the environment. The combination of sophisticated environmental fate and exposure models with organism-based toxicity levels has actually been advocated for a number of years (McCarty and Mackay 1993). Combining monitoring data and mass balance models maximizes theoretical and empirical knowledge for comprehensive chemical assessments (McKone et al. 2007; Cowan-Ellsberry et al. 2009).

1.2 REPORT OBJECTIVES

The primary objective of the present study is to provide a scientifically defensible assessment of t-HBCD, and the three diastereomers where possible, using physical-chemical property data, emission estimates, multimedia fate, bioaccumulation and exposure models, toxicity and effects information, monitoring data, and spatial and temporal trends of environmental concentrations. This assessment includes the collection and critical evaluation of Final Adjusted Values (FAVs) for physical-chemical properties used throughout the assessment and the collection and critical review of available monitoring and testing data. The study uses state of the science mass balance models and QSARs to compare HBCD with selected POPs, candidate POPs currently under risk review, and non-POPs. Mass balance models seek to corroborate emissions estimates, half-life degradation data and monitoring data including estimated response times ot HBCD emission reductions in various environmental compartments. The model-monitoring comparisons can also provide justification for the selected degradation half-lives. The present assessment also serves as a critical appraisal of the TemaNord (2008) recommendation to categorize HBCD as a POP and provides a more comprehensive perspective in relation to the proposed POP categorization of HBCD according to the UN Stockholm Convention and UN-ECE POP criteria. The present study also highlights key issues related to the RAR.

The focus of the present study is on an ecological risk assessment; however, human farfield exposures to POPs, non-POPs, and candidate POPs are also considered when

comparing model predictions with monitoring data. There are uncertainties associated with indoor (nearfield) exposures; therefore, a comprehensive human health risk assessment is not considered in the present study. The RAR found no need for further information or for risk reduction measures beyond those, which are being applied already for humans exposed via the environment (EC 2008).

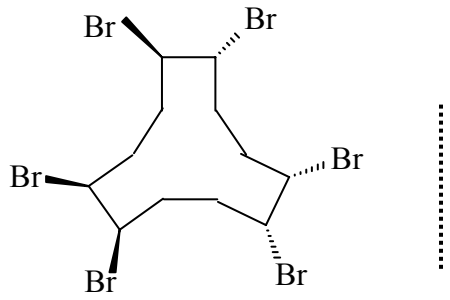
2.0 SUBSTANCE INFORMATION

Details regarding t-HBCD and the different isomers that comprise the mixture can be found elsewhere (Heeb et al. 2005; Koppen et al. 2008) and in the RAR (EC 2008). The general objective in this section is to recognize there are differences between t-HBCD (mixture) and the diastereomers and to review and select physical-chemical properties and half-life data for applications in the present assessment.

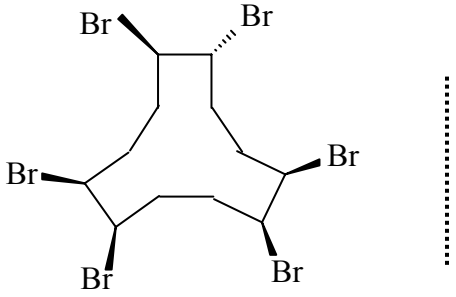
2.1 IDENTITY AND STEREOCHEMISTRY

The CAS Registry contains two numbers representing the undefined mixtures of t-HBCD: 25637-99-4 (without numbering for the position of the bromine substitution pattern) and 3194-55-6 for 1,2,5,6,9,10-HBCD (Becher 2005). It is noted that the differences in the SMILES notation for the two CAS numbers result in different representative structures and can therefore lead to different estimates in chemical properties. For example, the SMILES for CAS number 3194-55-6 in EPI Suite™ (U.S. EPA 2009) is consistent with the structures in Figure 1-1 of the RAR; whereas the SMILES in EPI Suite™ for the CAS number 25637-99-4 shows a substitution pattern of 1,3,5,7,9,11-HBCD which is inconsistent with Figure 1-1 of the RAR. Thus, for the present study CAS number 3194-55-6 is considered to represent the appropriate substitution pattern. The CAS numbers reflect undefined mixtures of isomers each with differences in chemical properties; therefore, certain properties cannot be reliably defined for t-HBCD.

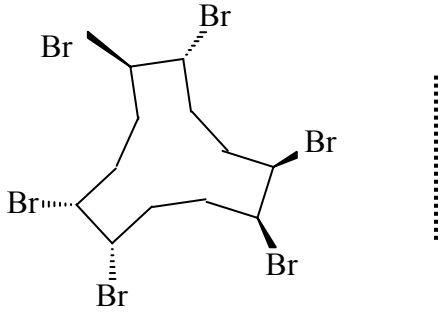
The stereochemistry of HBCD is complex including 16 stereoisomers (Law et al. 2005) and HBCD is subject to isomerization during product synthesis and in the environment. These elements and the general lack of measured data provide a challenge for robust chemical assessments. CAS numbers have been assigned to the three main diastereoisomers comprising the majority of t-HBCD as 134237-50-6, 134237-51-7, 134237-52-8 for α- β- and γ-HBCD, respectively (Janák et al. 2004). As discussed below, these three diastereomers have different physical-chemical properties (MacGregor and Nixon 2004; Goss et al. 2008) and will therefore display distinct fate and transport characteristics in the environment. Figure 2.1 illustrates the three main diastereoisomers α-, β- and γ-HBCD found in t-HBCD. These three diastereoisomers are chiral and exist as pairs of enantiomers in t-HBCD for a total of six stereoisomers (+/-)α-, (+/-)β- and (+/-)γ-HBCD. α- and γ-HBCD have a C2 axis of symmetry, but β-HBCD does not (Smith et al. 2005). Studies from thermal rearrangement indicate that α-HBCD is the most thermodynamically stable isomer of the three (Smith et al. 2005). There are two achiral diastereoisomers that can be found in t-HBCD at levels usually less than 1% each, termed δ- and ε-HBCD (Law et al. 2005; EC 2008).



α-HBCD (CAS 134237-50-6), the line indicates a mirror plane



β-HBCD (134237-51-7), the line indicates a mirror plane



γ-HBCD (134237-52-8), the line indicates a mirror plane

Figure 2.1. The three main diastereoisomers (pairs of enantiomers) in t-HBCD (recreated from (EC 2008); data from (Heeb et al. 2005)).

2.2 Analytical Methods and Isomerization

The challenges of analyzing brominated flame retardants are well recognized (Law et al. 2005; de Boer and Wells 2006). Analysis and modelling of HBCD are further complicated by the possibility of interconversion of the different isomers (isomerization) (Zegers et al. 2005; Law et al. 2006b; Koppen et al. 2008). Thermal isomerization has resulted in LC-MS methods being advocated over GC-MS methods whenever diastereomeric composition has to be preserved during analysis. Sensitivities of the methods are different; however, as illustrated in Figure 2.2, quantification of total HBCD by the two methods is comparable (Petersen et al. 2004).

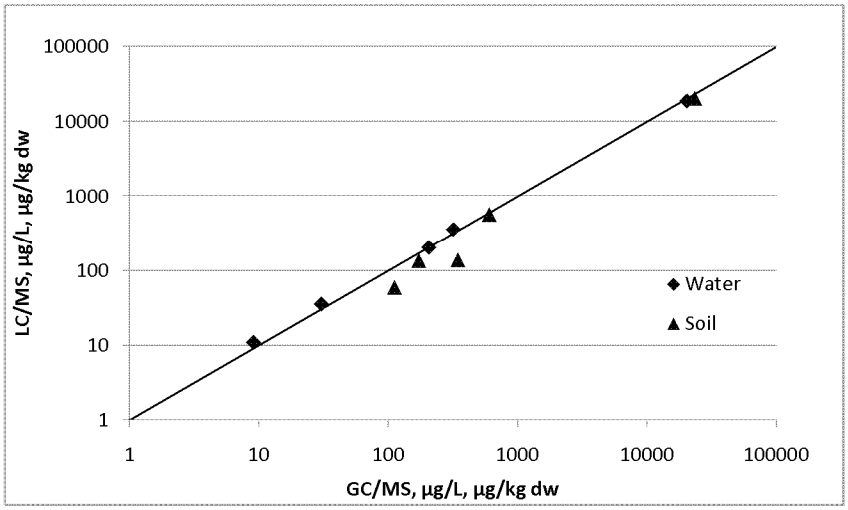


Figure 2.2. Illustration of analytical methods comparison (data from Petersen et al. 2004).

The complex competing interconversion pathways of α-, β- and γ-HBCD at increased temperatures illustrated by Koppen et al. (2008) may also occur at lower temperatures, but at slower rates and possibly following different isomerization mechanisms (e.g., enzymatic conversions, photoisomerization). Heeb et al. (2008) have elucidated kinetics and mechanisms for isomerization and noted the possibility of isomerization occurring at lower temperatures. This study also noted that the highly symmetrical conformation of α-HBCD is thermodynamically favored and adopts optimized and less reactive conformations compared to β- and γ-HBCD. Both α-HBCD enantiomers thermally degraded rapidly with no detectable isomerization reactions. Heeb et al. (2008) also show the stereoselective formation of α-HBCD from γ-HBCD. Heeb et al. (2008) advocate the need to elucidate the fate of HBCDs in the environment as individual stereoisomers.

Two recent studies suggest further uncertainties in HBCD measurements and data analysis. Kajiwara and colleagues (2009) have shown that soxhlet extraction methods can result in isomerization during sample preparation (predominantly γ-HBCD to α-HBCD). This was also postulated by Heeb et al. (2008). The Kajiwara study also noted changes in isomeric patterns from t-HBCD to those in textiles. In studies of house dust comparing isomeric patterns in the presence and absence of light Harrad and colleagues (2009) have

shown that HBCD isomers are subject to photoisomerization. This study suffers somewhat from an incomplete account of the HBCD mass balance; however, if verified, photoisomerization may also occur in various environmental compartments (atmosphere, soils, surfaces). The isomerization of HBCD at lower temperatures than previously considered (e.g. solvent extraction) and in the presence of light suggests that isomerization could be occurring during sampling and analysis and perhaps while in storage. These issues have implications for the QA/QC of HBCD data that have not been fully considered in previous assessments, and while recognized here, these issues can neither be fully rectified in the present study.

2.3 PHYSICAL-CHEMICAL PROPERTIES

Measured and modelled estimates of physical-chemical properties for t-HBCD and the three main diastereoisomers (α-, β- and γ-HBCD) relevant for the present study were compiled and critically reviewed as detailed in Section 9.1. Based on the critical review, physical-chemical property values were selected. The physical-chemical properties in the present study include molar mass (M; $g \cdot mol^{-1}$), melting point (T_M; ^oC or K), water solubility (S; $mol \cdot L^{-1}$), vapor pressure (P; Pa), Henry's law constant (H; $Pa \cdot m^3 \cdot mol^{-1}$), and the dimensionless equilibrium partition coefficients between air and water (K_{AW}), octanol and water (K_{OW}), and octanol and air (K_{OA}). For some parameters, such as the K_{OW}, there are many measurements and estimates available, whereas for other parameters, such as the K_{OA}, the number of studies reporting measurements/estimates is limited. Methods to seek thermodynamically consistent and thus more accurate solubility and partitioning properties are applied to obtain Final Adjusted Values (FAVs) for the evaluation of testing and mass balance modelling.

2.3.1 Addressing Isomerization

Initial efforts were made to collect, review and select monitoring data and model input parameters for diastereomer specific analyses. Through the course of the research it was recognized that diastereomer data analyses and modelling would be limited largely as a result of unknown isomerization rates and processes that may occur in most, if not all, environmental compartments. Many of the reported measured properties and tests are for t-HBCD, which is a mixture of isomers, each with unique properties. The poorly characterized, and seemingly competing, isomerization processes and rates occurring in the natural environment and during analytical procedures complicates the interpretation of monitoring and testing data and the application of mass balance models. For example, when t-HBCD is exposed to an organism in a test the different isomers may be subject to different rates of interconversion during exposure and within the organism and the different isomers (and enantiomers) may also have different biological activities. It therefore often remains uncertain which isomers (and enantiomers) are actually responsible for the perceived biological activities. Previous studies have used mass balance models to evaluate t-HBCD as a mixture; therefore, due to a general lack of information to assess the isomers specifically and to compare model estimates with limited monitoring and testing data reported as ΣHBCD we include representative properties selected for t-HBCD recognizing that this is not ideal.

The uncertainties associated with isomerization, in particular unknown isomerization rates in the multiple compartments of the environment, not only limit our ability to interpret the time trend and monitoring data, but also our capacity to model the behaviour of specific isomers. Furthermore, existing fate and bioaccumulation models are not capable of accounting for isomerization. If the required input data for isomerization rates and pathways were available, models used in the present study could be revised to improve fate and exposure assessments.

2.3.2 Selection of Reliable Estimates for Solubility and Partitioning

Reliable estimates of solubility and partitioning for commercial HBCD and the three diastereoisomers are required to improve the interpretation of test data and to input into mass balance models. One objective of the present study is to obtain thermodynamically consistent physical-chemical properties as suggested by various authors (Cole et al. 2000; Beyer et al. 2002; Schenker et al. 2005). These FAVs were obtained from the selection of critically evaluated property estimates and the application of the least squares adjustment procedure for property harmonization suggested by Schenker et al. (2005). The fugacity ratio F (unitless) is required to correct for the influence of intermolecular interactions of the solid phase, which are not relevant to chemical partitioning when dissolved in solutions or phases. The solid state property data (subscript S) were corrected to the subcooled liquid state values (subscript L) using

$$P_L = P_S/F \quad (2.1)$$

$$S_L = S_S/F \quad (2.2)$$

where F is estimated to be 0.023 based on a T_M of 190^oC for commercial (technical) HBCD and the assumption that the entropy of fusion is 56.5 $J \cdot K^{-1} \cdot mol^{-1}$ (Mackay 2001). F is expected to be different for the diastereoisomers due to different melting points and different entropies of fusion; however, for the screening level calculations in the present study the same value of F is also applied to the diastereoisomers. F cancels out in the calculation of partition coefficients.

Some of the physical-chemical property estimates selected for the calculation of FAVs are different from the values selected in the RAR. These discrepancies are detailed in Section 9.1 and summarized below.

The RAR discusses the selection of a solid state water solubility S_S for t-HBCD of 66 $\mu g \cdot L^{-1}$ (EC 2008). When there is 65.6 $\mu g.L^{-1}$ of dissolved t-HBCD in pure water, the total content of HBCD in the water is 610 $\mu g.L^{-1}$ including 544.4 $\mu g.L^{-1}$ of non-dissolved γ- and β-HBCD diastereoisomers (EC 2008). The predominant diastereoisomer in t-HBCD is γ-HBCD which has a water solubility about 20 times lower than t-HBCD. This is an important issue with regards to the interpretation of toxicity information derived from in experiments with water-based exposures; therefore, for consistency in the report we selected a value of 3.0 $\mu g.L^{-1}$ for S_S to represent t-HBCD.

The RAR discusses the selection of a solid state vapor pressure P_S of 6.3×10^{-5} Pa at 21°C for t-HBCD from two reported measurements (EC 2008). According to OECD guidelines both of the methods are not recommended for substances with vapor pressures as low as the values measured for HBCD (OECD 2002). Wania (2003a) determined a subcooled liquid state vapor pressure P_L of 2.41×10^{-5} Pa at 25 °C for t-HBCD relying on the gas chromatographic retention time method by Bidleman (1984) and using *p,p*'-DDT as the reference compound and a suite of organochlorine pesticides as the calibration compounds. This value was selected for the present study.

The RAR calculates a Henry's law constant of 0.75 $Pa\cdot m^3\cdot mol^{-1}$ for t-HBCD as the ratio of their selected values for P_S and S_S (EC 2008). The different values for P_S and S_S selected in the present study result in an estimate of 0.120 $Pa\cdot m^3\cdot mol^{-1}$. The EPI Suite™ bond method estimate (U.S. EPA 2009) of 0.174 $Pa\cdot m^3\cdot mol^{-1}$ provides an independent and consistent estimate for inclusion in the least squares adjustment calculations.

2.3.3 Thermodynamic Consistency Calculations for Chemical Properties

The methods of Schenker et al. (2005) were used to obtain thermodynamically consistent estimates for the solubility and partitioning properties of t-HBCD and the three diastereoisomers. The data selected as input for the calculation of thermodynamically consistent FAVs are listed in Table 2.1. An effort was made to maximize input parameters, but to ensure that data sources were reliable and independent. In this context "independent" means that physical-chemical properties used in the harmonization calculations were obtained from distinct measurements or estimates. For example, K_{AW} estimates derived from ratios of P_S and S_S were not included in the calculations because the selected estimates of P_S and S_S were already used as input. Reliability was considered high if multiple data sources provided similar values. Model estimates were considered as well as measured data. The harmonization method also requires and provides relative variance values for chemical properties. These values provide guidance as to the relative uncertainty of the different properties. The relative variance value inputs were selected based on professional judgement of the general reliability of the various parameters.

The issue of the mutual solubility of water and octanol can be important when seeking to obtain thermodynamic consistency in solubility and partitioning parameters (Cole et al. 2000; Beyer et al. 2002). Typical K_{OW} measurement techniques, such as the shake-flask and slow stir methods, result in the mixing of water and octanol phases, which results in the presence of some octanol in the water phase and of some water in the octanol phase. Measurements of the octanol-air partition coefficient (K_{OA}) and of the solubility in octanol, on the other hand, are obtained using "dry" octanol, i.e. octanol that has not been saturated with water. Similarly, measurements of water solubility and air-water partition coefficients are typically performed with pure water. To account for the possible influence of the mutual solubility of water and octanol on the phase partitioning of highly hydrophobic substances, relationships have been proposed for converting measured K_{OW} values to ratios of concentrations in pure octanol and pure water C_O/C_W. For the thermodynamic consistency analysis, the equation recommended by Beyer et al. (2002) was used to convert K_{OW} to C_O/C_W prior to adjustment and to convert the adjusted C_O/C_W back to K_{OW} after adjustment.

Table 2.2 lists the thermodynamically consistent FAVs for the relevant solubility and partitioning properties. The percentages of adjustments were modest for changes from the selected input values to the calculated FAVs for most properties suggesting that the data are generally consistent and reasonable. Notably, all of the K_{OW} values were slightly increased and the solubility in octanol decreased in all cases except for α-HCBD for which it increased (largest percentage adjustment). Notably, the adjustment suggests that α-HCBD is about 4 times more soluble in octanol than γ-HCBD. This may be partially explained by the high symmetry of the α-isomer (Heeb et al. 2008) and may partially explain some of the observed increases in bioaccumulation patterns in the environment for α-HCBD. The relative ranking of the FAVs for K_{OW} are consistent with the rankings of the measured water solubilities and the independent K_{OW} estimates by Hayward et al. (2006) that were not used in this procedure. The FAVs for K_{OA} are also comparable to the estimates that were not included in the consistency calculations.

The method was applied to t-HBCD in the current study; however, a thermodynamic consistency calculation should generally not be used for mixtures, but rather for discrete substances with distinct properties. The method was applied for consistency with diastereomer methods and for comparing model estimates for t-HBCD and individual diastereomers with monitoring data for t-HBCD. Considering the uncertainty in the FAVs for t-HBCD in the context of the greater uncertainties associated with actual emissions, mode-of-entry, isomerization and degradation in the environment, the errors in calculating "representative" FAVs for t-HBCD are considered to be comparatively low.

Table 2.1. Selected values for physical-chemical properties (at 25 ^{o}C) and relative variance values used as inputs for thermodynamic consistency calculations.

Substance	P_L, Pa	S_L, mol·m^{-3}	S_{OL}, mol·m^{-3}	Log K_{AW}	Log K_{OW} [a]	Log K_{OA}
t-HBCD	2.41×10^{-5}	2.01×10^{-4}	698	-4.15	5.63	10.71 [b]
α-HBCD	2.93×10^{-4}	3.27×10^{-3}	698	-4.35	5.59	9.62 [b]
β-HBCD	3.86×10^{-5}	9.86×10^{-4}	698	-4.71	5.44	10.50 [b]
γ-HBCD	2.00×10^{-5}	1.41×10^{-4}	698	-4.15	5.53	10.79 [b]
			Relative variance			
t-HBCD	3	2	4	4	3	
α-HBCD	5	1	4	5	4	
β-HBCD	5	1	4	5	4	
γ-HBCD	5	1	4	5	4	

[a] log K_{OW} values corresponding with log (Co/Cw) values of 6.02, 5.97, 5.76, and 5.89 for t-, α-, β-, and γ-HBCD, respectively

[b] values not used as inputs for calculation of FAVs because they are not "independent" values, i.e., they were derived from vapor pressure and octanol solubility estimates (see Appendix). The values are included for comparisons with FAVs

Table 2.2. Final adjusted values (FAVs at 25 °C), relative variance values, percent adjustments, and assigned confidence factors used in the present study for mass balance modelling and data analyses obtained from the thermodynamic consistency calculations (Schenker et al. 2005).

Substance	P_L, Pa	S_L, mol·m^{-3}	S_{OL}, mol·m^{-3}	Log K_{AW}	Log K_{OW} [a]	Log K_{OA}
t-HBCD	3.03×10^{-5}	2.33×10^{-4}	380	-4.28	5.77	10.46
α-HBCD	3.00×10^{-4}	2.77×10^{-3}	1340	-4.36	5.38	9.96
β-HBCD	4.29×10^{-5}	9.89×10^{-4}	630	-4.76	5.47	10.47
γ-HBCD	2.42×10^{-5}	1.68×10^{-4}	300	-4.23	5.80	10.40
			Relative variance			
t-HBCD	1.95	1.27	2.13	2.13	1.95	3.45
α-HBCD	2.70	0.82	2.20	2.70	2.20	4.50
β-HBCD	2.70	0.82	2.20	2.70	2.20	4.50
γ-HBCD	2.70	0.82	2.20	2.70	2.20	4.50
			Percent adjusted			
t-HBCD	26%	16%	-45%	-26%	39%	26%
α-HBCD	2%	-15%	92%	-2%	-38%	2%
β-HBCD	11%	0%	-9%	-10%	7%	11%
γ-HBCD	21%	19%	-57%	-18%	87%	21%
			Confidence factor (*Cf*)			
t-HBCD	3	2	3	3	3	3
α-HBCD	3	1.5	3	3	3	3
β-HBCD	3	1.5	3	3	3	3
γ-HBCD	3	1.5	3	3	3	3

[a] log K_{OW} values corresponding with log (Co/Cw) values of 6.22, 5.68, 5.81, 6.25 for t-, α-, β-, and γ-HBCD, respectively

2.3.4 Uncertainty in FAVs

The FAVs have been carefully selected to reduce uncertainty; however, it is recognized that uncertainty in model input parameter values cannot be completely eliminated. Uncertainty in model input parameters such as chemical properties and half-lives propagate uncertainty in environmental fate and exposure model predictions. Monte Carlo analysis and analytical methods can be used to assess the sensitivity and uncertainty of model input parameters on model output (MacLeod et al. 2002). The analytical method uses a confidence factor *Cf* (or more appropriately an uncertainty factor) to express the degree to which parameter X may deviate from the median M in a lognormal distribution. For example, a *Cf* of 2 suggests that 95% of all of the values in the distribution are within 2 and 0.5 times M. A 95% probability can be expressed as:

$$\text{probability}\left\{\frac{M}{Cf} < X < Cf \cdot M\right\} = 0.95 \quad (2.3)$$

Thus, the variance in the lognormal distribution increases with an increase in *Cf*. Values of confidence can be derived empirically or subjectively or in combination. There is a degree of co-dependence between the three partition coefficients, but the three solubility parameters are independent. It is recognized that certain properties are often used as surrogates in the models (e.g., K_{OW} for lipid-water partitioning and organic carbon-water partitioning, K_{OA} for lipid-air partitioning) and that variable conditions occur in the environment. Based on these considerations, the data review, the relative variance values calculated by the harmonization procedure, and professional judgement, *Cf*s were selected for the FAVs as listed in Table 2.2.

No estimates for energies of phase transition are available for technical HBCD or the diastereoisomers.

2.3.5 Physical-Chemical Properties for Benchmarking

The chemicals selected for this preliminary benchmarking exercise representing POPs were as follows: HCB, PCB28, PCB101, PCB180, Aldrin and Heptachlor. Non-POPs include biphenyl, p-cresol, atrazine and tetrachloromethane and candidate POPs (Candi-POPs) include α-, β-, γ-HCH, and PBDE-99. These substances represent a range of physical-chemical properties. Most of these substances have been used in previous POPs benchmarking assessments (Cowan-Ellsberry et al. 2009). Aldrin and heptachlor were selected because these POPs have shorter half-lives in air than most POPs and these half-life estimates are comparable to those for HBCD.

Table 2.3. Selected physical-chemical properties for the model benchmarking simulations. Data are from (Cowan-Ellsberry et al. 2009) except for Aldrin and Heptachlor which are from (Schenker et al. 2005; Mackay et al. 2006).

Status	**Name**	**Abbrev**	**MW ($g.mol^{-1}$)**	**log K_{OW}**	**log K_{AW}**	**log K_{OA}**
POP	Hexachlorobenzene (HCB)	HCB	284.8	5.61	-1.51	7.12
POP	2,4,4'-trichlorobiphenyl (PCB 28)	PCB28	257.6	5.66	-1.93	7.86
POP	2,2',4,5,5'-pentachlorobiphenyl (PCB 101)	PCB101	326.4	6.38	-2.08	8.83
POP	2,2',3,4,4',5,5'-hexachlorobiphenyl (PCB 180)	PCB180	395.3	7.15	-2.51	10.17
POP	Aldrin	Ald	364.9	6.25	-2.01	8.26
POP	Heptachlor	Hept	373.4	5.96	-1.78	7.74
NOP	Biphenyl	Biphenyl	154.2	4.06	-1.96	6.02
NOP	*p*-cresol	*p*-cresol	108.1	1.97	-4.26	6.23
NOP	Atrazine	Atrazine	215.7	2.73	-6.84	9.57
NOP	Tetrachloromethane	CCl4	153.8	2.83	0.19	2.64
Candi-POP	alpha-hexachlorocyclohexane	α-HCH	290.8	3.88	-3.59	7.48
Candi-POP	beta-hexachlorocyclohexane	β-HCH	290.8	3.91	-4.83	8.74
Candi-POP	gamma-hexachlorocyclohexane	γ-HCH	290.8	3.76	-3.96	7.72
Candi-POP	2,2',4,4',5-pentabromodiphenyl ether	PBDE99	564.7	6.76	-3.67	11.26

2.4 DEGRADATION, BIOTRANSFORMATION AND HALF-LIFE DATA

Reliable estimates for environmental and biological compartment half-lives are required for mass balance model calculations and environmental half-lives are used for screening level hazard categorizations of chemical Persistence (P). The primary objective of this section is to describe the compilation, review and selection of primary transformation half-life (HL) estimates for the bulk physical compartments of the environment (atmosphere, water, soil and sediment) and for biological species (fish, mammals). This HL estimate is defined as the time it takes for half of the chemical amount present to undergo a change in structure. Distinctions between transformation from HBCD to a degradation product and isomerization will be discussed (e.g. biotransformation resulting in elimination from an organism and bioisomerization that does not result in chemical elimination from an organism). Measurements and model estimates are considered.

2.4.1 Environmental Degradation Half-lives

Degradation half-lives in physical compartments of the environment need to be distinguished from experimentally-derived dissipation or disappearance times since the latter two include losses attributable to processes other than transformation of the parent compound e.g. adsorption to surface of glassware, volatilization out of test system. In practice, distinguishing between degradation and dissipation is often difficult.

Data characterizing the degradation potential of HBCD has been compiled and reviewed in the RAR (EC 2008) and the TemaNord report (2008). Experimental data are available for an aqueous test system, sediments and soils (aerobic, anaerobic) and sewage sludge. Here we briefly discuss the available data and then present the values selected for the current model simulations along with estimates of the associated uncertainties. Data from sewage sludge are not discussed here since they are the least relevant for the model simulations conducted for this study.

Atmosphere (air)

For most semi-volatile organic compounds, reactions in the gas phase with OH radicals are considered to be the most important degradation pathways in the atmosphere. Hence, a typical approach to estimate atmospheric degradation half-life (HL_{air}, s or d) is to use a hydroxyl radical reaction rate constant (k_{OH}, $cm^3 \cdot molecule^{-1} \cdot s^{-1}$) and an assumed OH radical concentration ([OH], $molecules \cdot cm^{-3}$) according to the following pseudo-first order equation:

$$HL_{air} = \frac{\ln 2}{k_{OH}\,[OH]} \tag{2.4}$$

k_{OH} may be derived from experiments or field observations but due to lack of data is more frequently calculated using estimation software. For example, the k_{OH} of HBCD estimated using AOPWIN v1.92 (U.S. EPA 2009) is $6.12 \times 10^{-12} cm^3 \cdot molecule^{-1} \cdot s^{-1}$. Based on the reported model performance, this estimate is expected to be accurate within a factor of 3

in either direction (U.S. EPA 2009). Since AOPWIN v1.92 does not distinguish between α-, β- and γ-HBCD, we assume the same value for all isomers. In EPI Suite v4.0, the [OH] is assumed to be 1.5×10^6 molecules·cm^{-3} whereas a value of 5×10^5 molecules·cm^{-3} was assumed in the TemaNord report (2008). The value of 1.5×10^6 molecules·cm^{-3} is meant to represent a 12-hour (daylight) average whereas the value of 5×10^5 molecules·cm^{-3} represents a 24-hour average. When converting HL_{air} from seconds into days, it is therefore important to select a value of [OH] that is consistent with the assumptions regarding the time period over which the reactions occur.

Bahm and Khalil (2004) derived a 24 hour global annual average [OH] of 9.2×10^5 molecules·cm^{-3}, with a value of 9.8×10^5 molecules·cm^{-3} for the northern hemisphere and 8.5×10^5 molecules·cm^{-3} for the southern hemisphere. These values are consistent with (Prinn et al. 1995) and (Montzka et al. 2000) who deduced [OH] from atmospheric measurements of methyl chloroform, reporting 24 hour global annual average values of $9.7(\pm0.6)\times10^5$ and $1.1(\pm0.2)\times10^6$ molecules·cm^{-3} respectively. Using the 24-hour average [OH] value calculated by Bahm and Khalil (2004) for the northern hemisphere and the default k_{OH}, the estimated atmospheric half-life is ~ 1.3 days. Using the 12 hour (daylight) [OH] assumed in AOPWIN v1.92 and assuming a night time [OH] of 0 the estimated atmospheric half-life is ~ 1.8 days. Considering the uncertainty in model estimates of k_{OH}, the range is 0.4 to 4 days and 0.6 to 5.4 days respectively, meaning that it is not possible to unambiguously conclude that HBCD exceeds (or does not exceed) the atmospheric half-life criterion for persistence in air (> 2 days). With respect to POP benchmarking it is important to standardize the relevant assumptions for all chemicals considered, particularly if HL_{air} values rather than k_{OH} rates are being compared.

Water

Biodegradation of HBCD in an aqueous test system was investigated by Schaefer and Haberlein (1996), who conducted a Closed Bottle Test (OECD Guideline 301D, (OECD 1992)) to assess ready biodegradability under aerobic conditions. The results of these data were used to conclude that no biodegradation of HBCD occurred over a 28-day period (EC 2008; TemaNord 2008). However, according to Shaefer and Haberlein (1996), the concentration of HBCD in the test solution was 7.7 mg·L^{-1}, which is at least three orders of magnitude greater than the estimated water solubility of this compound (~3 µg·L^{-1}). This discrepancy introduces a bias in the interpretation of the experimental results since the concentration of the test substance is used to estimate biochemical oxygen demand (BOD, mg O^2·mg test substance^{-1}). BOD is then compared to theoretical oxygen demand (ThOD) to calculate % degradation. While it is true that OECD Guidelines for the 301D test recommend that the test substance be present at concentrations in the range of 2 to 10 mg·L^{-1} (OECD 1992), this recommendation implies that the 301D test is inappropriate for HBCD since this compound has a low water solubility. Therefore, in contrast to the RAR (EC 2008) and TemaNord report (2008), we do not consider the Schaefer and Haberlein (1996) results to be reliable and instead characterize them as inconclusive. Note that concerns about the validity of this test have been previously raised (Davis et al. 2005).

Model output characterizing ready biodegradability generated using BIOWIN^{TM} (EPI Suite v4.0) was reported in (TemaNord 2008) and is summarized in Section 9.2. HBCD is classified as not readily biodegradable under aerobic conditions according to the criteria developed for the model. Based on BIOWIN4 (Expert Survey, Primary Biodegradation Model), the expected time frame for primary degradation to an initial metabolite is on the order of weeks. However, as noted in the TemaNord report (2008), the cyclic structure of HBCD is not explicitly accounted for by the fragment approach applied in the BIOWIN4 model.

Potential abiotic degradation pathways for HBCD were presented in the RAR (EC 2008). Abiotic degradation of HBCD in surface waters is also possible via the indirect photolysis mechanisms, which result from the generation of photooxidants including OH radicals in the water column. However, in addition to knowledge of HBCD's 2nd-order rate constant for reaction with OH radicals in aqueous solution, a representative daylight or 24-hour average [OH] in water would also have to be estimated. HBCD is not expected to be subject to hydrolysis reactions in the water.

In summary, there are no reliable empirical data characterizing the degradation kinetics of HBCD in water. Methods to estimate degradation half-lives from QSAR model output are discussed below.

Sediment

Based on the sediment degradation studies cited in the RAR (EC 2008), it can be concluded that i) degradation of HBCD proceeds at a faster rate under anaerobic conditions than under aerobic conditions ii) based on experimental data from sterilized test systems, abiotic reaction pathways support a degree of baseline degradation in the environment but iii) degradation of HBCD in the presence of a viable microbial community proceeds at a faster rate than in the absence of microbes (i.e. sterilized test system)

Table 2.4 lists two sets of degradation half-lives that were used for total HBCD (Simulation Study 1 and 2) in the RAR (EC 2008). These values include the extrapolation of measured half-lives at 20 ^{o}C to 12 ^{o}C (EC 2008). The adopted approach for scaling degradation half-lives for POP and PBT screening level assessments is fundamentally wrong however because it is only applicable to abiotic extrapolations. This method is not recommended by recognized experts in the field due to a lack of a scientific justification (Boethling et al. 2009). Furthermore, the presumed adjustments do not consider the adaptive nature of microbial populations in different environments.

Table 2.4. Degradation half-lives for total HBCD proposed in the European Risk Assessment Report (RAR) (EC 2008).

Test System	Simulation Study 1	Simulation Study 2
Aerobic freshwater sediments		
20 ^{o}C	11 days	101 days
12 ^{o}C	21 days	191 days
Anaerobic freshwater sediments		
20 ^{o}C	1.5 days	66 days
12 ^{o}C	2.8 days	125 days

Data used to derive estimates for "Simulation Study 1" were taken from Davis et al. (2003a). These data are now published in the peer-reviewed literature as Davis et al. (2005)). Data used to derive estimates for "Simulation Study 2" were taken from Davis et al. (2004), now published as Davis et al. (2006). Note that the degradation half-lives calculated directly from the rate constants presented in the Supporting Information of Davis et al. (2006) are somewhat different than the half-lives presented in the RAR (EC 2008). For example, the average degradation half-life of the α-, β- and γ-HBCD isomers is 94 days whereas a value of 101 days was reported in the RAR (European Commission 2008) These discrepancies are presumably related to different statistical treatment of the underlying data.

In the studies conducted in Davis et al. (2006), the apparent half-life of α-HBCD tended to be longer than that of β- and γ-HBCD. For example, in aerobic freshwater sediments (20 to 22 ^{o}C), the estimated degradation half-life of α-HBCD was 128 days versus 72 and 92 days for β- and γ-HBCD respectively (Davis et al. 2006). These differences were not found to be statistically significant ($p > 0.05$), in part due to the high level of variance in replicate observations. In digester sludge, the degradation half-life of β-HBCD was significantly shorter than that of α- and γ-HBCD but no statistically significant difference was found between the latter two isomers. In contrast, Gerecke et al. (2006) reported slower degradation kinetics for α-HBCD (~ 50%) in digester sludge but no difference between β- and γ-HBCD. Hence while there is some evidence to support the claim that α-HBCD is more recalcitrant than other isomers, the uncertainty in the available data does not allow a definitive conclusion to be drawn.

While differences in susceptibility to degradation among HBCD isomers is an important consideration, the more relevant issue to be addressed is the apparent discrepancy in the magnitude of the degradation rate constants between Simulation Study 1 (Davis 2003a; Davis et al. 2005) and 2 (Davis et al. 2004; Davis et al. 2006). Berg and Nyholm (1996)

discussed the fact that industrial chemicals are typically present in the environment at such low concentrations that they can be expected to be degraded as 'secondary substrates' (i.e. marginal contribution to energy flow in system) as opposed to primary substrates. This mode of degradation is characterized by first order kinetics with a constant rate parameter and it is therefore important that chemical concentrations used in test systems be low enough to ensure that the 'secondary substrate' degradation regime predominates (Berg and Nyholm 1996). Note that this does not necessarily mean that test concentrations must be similar to environmentally-relevant concentrations to yield representative results, only that concentrations should not be elevated to the point that the 'secondary substrate' degradation regime no longer dominates. Davis et al. (2003a; 2005) conducted the degradation studies using nominal HBCD concentrations in the range of 25 to 90 $\mu g \cdot kg^{-1}$ dw whereas the degradation studies in Davis et al. (2006) had nominal concentrations in the range of ~ 3 to 5 $mg \cdot kg^{-1}$ dw. Davis et al. (2006) suggested that the apparent degradation rates from their studies could be biased low due to a shift from first- to zero-order degradation kinetics at the high concentrations used. At elevated concentrations, degradation kinetics may become limited by processes such as mass transfer into the microbes rather than transformation of the parent compound itself. While these considerations are important, it is also worth noting that the experimental data in all test systems (sludge, aerobic and anaerobic sediments) were described reasonably well assuming a first-order decay model (Davis et al. 2006).

With respect to mass balance modelling and exposures to air-respiring organisms, the most relevant degradation half-lives are those derived from tests in viable aerobic sediments, since these sediments are most representative of the "active sediment layer" included in the model domain (upper 0 to 5 cm). Aerobic/oxic conditions may or may not prevail in this upper layer, largely depending on the condition of the overlying water column. Sediment burial is included in fate models as a mechanism of removal, transporting substances to deeper sediment layers in which their subsequent fate is no longer explicitly considered, i.e., they are "lost" from the system. Selecting appropriate half-life values for model purposes is hindered by the recognized uncertainties in the data and variability in the natural environment. The reported degradation half-lives in viable aerobic sediments (20 ^{o}C), ranging from 11 to 128 d represent the range of measured values that are considered to be generally reliable There are three reported aerobic sediment degradation half-lives for commercial HBCD, 11, 32, and 94 d (or 11, 32 and 101 d, if the values in Table 2.4 are preferred). The median value in both cases is 32 and the two other data points are within approximately a factor of 3. This distribution is consistent with a log normal distribution for degradation half-lives measured in test data and expected in the environment. Different sediments in the natural environment are expected to have a range of genetic competence for degrading HBCD (variable microbial communities) and it is plausible that some reduced oxygen conditions may occur. Thus data from the laboratory studies were extrapolated to environmental conditions using a confidence (uncertainty) factor of 6 in the present study. Based on a median value of ~ 32 d, 95% of the expected values fall between 5.3 and 194 d. This upper bound level is also consistent with abiotic half-lives (sterile aerobic sediment environments). The lower bound can be considered more representative of aquatic systems where anaerobic conditions tend to dominate.

Soil

Degradation studies in soils were reported in Davis et al. (2003b) and Davis et al. (2004). Data from Davis et al. (2003b) were published in the peer-reviewed literature as Davis et al. (2005). Note that the degradation studies presented in this study was conducted at more environmentally-relevant concentrations and hence, in principal, are expected to be more representative, all other factors being equal (Davis 2003b; Davis et al. 2005). Similar to the sediment studies, there was a substantial difference in the degradation kinetics between the tests conducted at more environmentally-relevant concentrations ($\mu g \cdot kg^{-1}$ dw) (Davis 2003b) and higher concentrations ($mg \cdot kg^{-1}$ dw) (Davis et al. 2004). Hence the issues regarding elevated concentrations and apparent degradation kinetics discussed above and in Davis et al. (2006) also apply to the soil studies presented in Davis et al. (2004).

For the EUSES model simulations conducted in the RAR (EC 2008), two scenarios were again considered. One scenario assumed an aerobic (viable) soil degradation half-life of 63 days (20 ^{o}C, based on Davis et al. (2003b)) while the other assumed negligible degradation (i.e. infinite half-life, based on (Davis et al. 2004)). The assumption of negligible degradation in aerobic soils seems overly conservative or at least atypical, given the range of values considered for aerobic sediments. For example, estimated degradation half-lives in property handbooks and screening level QSAR model predictions for both persistent and non-persistent chemicals generally follow the pattern $HL_{water} \leq HL_{soil} \leq HL_{sed}$ (Howard et al. 1991; Mackay et al. 2006; U.S. EPA 2009). Sinkkonen and Paasivirta (2000) also proposed that, as a first approximation, the same empirically derived first-order biodegradation rate constant (anaerobic, aerobic) could be assumed for soils and sediments when estimating degradation half-lives for PCBs and PCDD/Fs. Hence there are precedents in the scientific literature for interrelating degradation half-lives in different environmental media although no definite conclusions can be made for HBCD. Overall, since there is no conclusive evidence upon which to exclude the data from Davis et al. (2004), half-lives in excess of those estimated from Davis et al., (2003b; 2005)) will also be considered in the model simulations for HBCD.

Model Estimated Aerobic Biodegradation Half-lives

The standard BIOWIN™ model output provides a qualitative assessment of biodegradability (bins based on numerical output) rather than quantitative estimates of degradation half-lives (U.S. EPA 2009). For example, bins include “ready” or “not ready” for general degradability and “days-weeks”, “weeks-months” for primary and ultimate degradation times. Arnot et al. (2005) proposed a method to derive environmental biodegradation half-lives for screening level assessments from numerical BIOWIN™ model output. The approach calibrated BIOWIN™ numerical model output to empirical biodegradation half-lives through linear regression. These linear regression equations along with the necessary BIOWIN™ input are presented in the Appendix (Table 9.6). Based on this approach, an aerobic biodegradation half-life in the range of 85 to 130 days is estimated for HBCD (not isomer specific). Aronson et al. (2006) also

utilized similar correlation approaches to estimate biodegradation half-lives from BIOWIN^{TM} model output. For example, based on BIOWIN3 model output for HBCD (ultimate biodegradation survey model), the recommended default degradation half-life (primary degradation) in the water column is 60 to 120 days (Aronson et al. 2006). However, it should be noted that the range of experimentally derived degradation half-lives correlated to this category of BIOWIN3 model output is 1 to 1420 days, indicative of the high degree of variability and uncertainty in the experimental data.

We recommend that further studies be conducted for biodegradation half-life estimation in soil and sediment. Studies on the atmospheric fate of HBCD, particularly with respect to reactions with OH radicals, would also be valuable since the efficiency of this degradation pathway can have a major influence on atmospheric long range transport potential

Selected Degradation Half-lives for Model Simulations

The total degradation half-lives (*HL*; d) assumed for the main bulk environmental media are presented in Table 2.5. These values are based on the available data and consideration of BIOWIN model output. Confidence factors (*Cf*) were also selected based on a review of the available data and professional judgment.

Table 2.5. Total degradation half-lives (d) assumed for all model simulations

Media	Default *HL* (d)	*Cf*	95% Range
Air*	1.3	3	0.4 to 4.0
Water (fresh, marine)	85	10	8.5 to 850
Sediments (fresh, marine)	35	6	6.0 to 210
Soil	85	10	8.5 to 850

* refers to gas-phase reactions only

Air

The default value for models requiring HL_{air} as model input of 1.3 d is based on AOPWIN v1.92 model output for the hydroxyl radical rate constant (k_{OH}) and the 24 hour average [OH] suggested by (Bahm and Khalil 2004) for the northern hemisphere. The *Cf* reflects the expected uncertainty in the hydroxyl radical rate constant only. For models requiring k_{OH} only, the default value of 6.12×10^{-12}cm^3·molecule^{-1}·s^{-1} is used instead (same *Cf*).

Water

The default value selected of 85 d for HL_{water} is based on aerobic degradation half-lives derived from BIOWIN^{TM} model output following Arnot et al. (2005) and Aronson et al. (2006). The larger *Cf* reflects uncertainty in the methods applied to arrive at the default value and the absence of empirical measurements.

Sediment

The default selected value of 35 d for HL_{sed} is justifiable as either the median degradation half-life based on available empirical data for total HBCD or the geometric mean of the range (~ 10 to 130 d). The *Cf* was selected to arrive at lower and upper bounds consistent with the RAR (EC 2008) and the available data.

Soil

The default selected value of 85 d for HL_{soil} was based on consideration of data presented in Davis (2003b) and BIOWIN™ model-derived estimates of aerobic biodegradation half-lives. The selected *Cf* allows the longer degradation half-lives implied by data in Davis et al. (2004) to be addressed in the model simulations.

In contrast to typical default extrapolation values for half-lives (e.g. water:soil:sediment; 1:2:9 (Mackay et al. 2006; U.S. EPA 2009)), the half-lives selected in the present study follow the pattern $HL_{water} = HL_{soil} \leq HL_{sed}$. Thus, the trend in selected degradation half-lives diverges from the usual pattern; however, it is more consistent with the available data.

2.4.2 Biotransformation Half-lives

The RAIDAR and CoZMoMan models require the whole body primary biotransformation half-life (*HL*; d) as an input parameter; otherwise biotransformation is assumed negligible in fish, birds and mammals. These biotransformation half-lives are assumed to be first order processes and thus relate to whole body biotransformation rate constants (d^{-1}) as $HL = \ln 2/k_M$. Biotransformation half-lives and rate constants are dependent on body mass and to a lesser degree temperature (Hu and Hayton 2001; Nichols et al. 2007). Biotransformation half-lives for organisms of specific body sizes and temperatures (HL_X) need to be calculated from the selected normalized half-life (HL_N) as (Arnot et al. 2008b; Cowan-Ellsberry et al. 2009)

$$HL_X = HL_N \, (W_X/W_N)^{-0.25} \qquad (2.5)$$

where W_X is the specific mass of the organism (kg) in the model and W_N is the selected normalized mass of the organism (e.g., 1 kg fish). Temperature differences are considered negligible in the present study. Five sets of data, including in vitro and in vivo studies, were reviewed to select estimates for whole body biotransformation rate constants in fish and mammals.

Fish Biotransformation

Law et al. (2006b) conducted a dietary uptake (feeding) and depuration study. Fish (~0.25 kg) were fed diastereomer specific diets and first-order "depuration" rate constants of 0.0044 and 0.0048 d^{-1} with respective half-lives of 157 d (±71 SE) and 144 d (±60 SE) were reported for β- and γ-HBCD. Estimates for α-HBCD could not be determined because "elimination" did not follow first-order kinetics. The study shows the potential

for bioisomerization of the different isomers whereby the isomers are apparently converted from one isomeric form to another. Some of the HBCD is not being eliminated or depurated from the organism but merely transformed into another diastereomer; therefore these "elimination" rates do not necessarily account for the loss of HBCD from the organism via transformation to a metabolite.

Table 2.6 summarizes the molar elimination and formation of the diastereoisomers during the "elimination" phase based on the data reported in Table 2 of Law et al. (see also Figure 1 from that study) (2006b). The data suggest that γ-HBCD is predominantly converted to α-HBCD (70%) and only about 30% of the total elimination half-life is actually attributable to "elimination of HBCD" from the organism through the formation of a metabolite. The data suggest that β-HBCD is predominantly converted to α-HBCD; however, the molar change during the depuration period actually suggests a net "gain" of total HBCD. It is not possible for the fish to create HBCD; therefore, based on the general uncertainty of the reported measurements it can be assumed that there is no apparent "loss of HBCD" from the organism for β-HBCD. The data also suggest that α-HBCD is predominantly eliminated with minimal conversion to the other diastereoisomers. Based on the data for γ-HBCD (high conversion to α-HBCD), some of the γ-HBCD isomer being formed from α-HBCD in the α-HBCD exposure study is expected to be converted back to α-HBCD during the experiment. It would seem that γ-HBCD is converted to α-HBCD faster than α-HBCD is converted to γ-HBCD, which would support the overall observation for the attenuation of γ-HBCD isomers in food webs. Furthermore, if relative bioisomerization rates approximate the relative rates of thermal isomerization, γ-HBCD would be converted to α-HBCD at rates approximately 10 times faster than α-HBCD is converted to γ-HBCD (Koppen et al. 2008).

Table 2.6. Molar mass balance for diastereoisomer bioisomerization and actual total elimination (fecal egestion, gill respiration and biotransformation).

Isomer fed to fish	Elimination phase		Parent "lost" during this time period	Other isomers formed (+) or lost (-) during this time period			Net loss of parent isomer	% actual elimination of parent isomer
	Day 56	Day 168		gamma	beta	alpha		
alpha	1.4	0.5	0.9	-0.23	-0.1	N/A	0.9	~100
beta	0.4	0.3	0.1	0	N/A	+0.2	-0.1	~0
gamma	0.4	0.17	0.23	N/A	-0.01	+0.16	0.07	~30.4

Table 2.6 suggests that α-HBCD is biotransformed primarily to a metabolite, whereas γ-HBCD is predominantly biotransformed to α-HBCD. β-HBCD does not appear to be biotransformed to a metabolite. Our interpretation of the data is somewhat different from the interpretation of Law et al. (2006b) who suggested that α-HBCD is recalcitrant to bioisomerization. Non-first order elimination kinetics were reported for α-HBCD. Indeed non-first order kinetics may be the result of the relatively faster rates of conversion of γ-HBCD back to α-HBCD. A "pseudo-elimination rate constant" based on the molar quantity at the start of the elimination (Day 56) and the molar quantity at the end of the elimination phase (Day 168) is estimated as 0.009 d^{-1} for a 0.25 kg fish. From the net loss

it appears that most of α-HBCD is actually biotransformed to a metabolite and eliminated from the fish, corresponding to an elimination half-life of about 75 d for α-HBCD.

Table 2.7 summarizes the parameters required to estimate biotransformation and bioisomerization rates using a mass balance estimation method for fish when applied to the Law et al. dataset for Rainbow trout (Arnot et al. 2008a). There are three possible biotransformation rate constant (k_M) estimates that could be considered. The first is "loss of parent" diastereomer. This includes bioisomerization from one diastereomer to a different diastereomer (and subsequent bioisomerization reactions) and the formation of metabolites (elimination). This would be calculated based on the "total depuration" rate constant (k_T) reported by Law et al. (2006b). The second is biotransformation to a metabolite that is no longer HBCD. This value is based on the reported total depuration rate constant (or estimated depuration rate constant for α-HBCD) and a correction for the actual loss of parent diastereomer (Table 2.6), i.e., 30% of the reported total depuration rate constant for γ-HBCD. The third transformation rate constant is the difference between the two estimated rate constants which is assumed to be the result of bioisomerization to another isomeric form of HBCD but without elimination from the fish, i.e., 70% of the reported total depuration rate constant for γ-HBCD. It must be recognized that the measured data are uncertain and that possible interconversion back and forth between the different isomeric forms at competing, and presumably different rates, further confounds the interpretation of the data and the ability to accurately estimate bioisomerization and biotransformation (elimination) rate constants. Since the bioaccumulation models used in the present study do not currently account for bioisomerization only the second rate constant (loss of HBCD from the organism) is considered relevant for exposure modelling at this time.

The estimated total elimination rate is so slow for β-HBCD, that no estimate for biotransformation is possible using the mass balance method. The estimated biotransformation rate constants for Rainbow trout (~0.25 kg) were 1.7×10^{-3} and 3.0×10^{-4} d^{-1} for α-, and γ-HBCD, respectively. The estimation method provides screening level information regarding confidence in the estimated k_M values (Arnot et al. 2008a; Arnot et al. 2008b). Although an estimate for γ-HBCD is calculated, the percentage of positive values is less than 25% corresponding to a low confidence category assignment for the estimated k_M value (Arnot et al. 2008b). Although an estimate for α-HBCD is calculated, the output confidence factor is greater than 10.3 corresponding to a "low" confidence category assignment for the estimated k_M value (Arnot et al. 2008b). Using a rearrangement of Equation 2.5, the corresponding primary biotransformation half-lives normalized HL_N to a 1 kg fish are 3330 and 720 d for α-, and γ-HBCD, respectively. Thus, the analysis from the measured data from Law et al. (2006b) suggest "very slow" (Arnot et al. 2009) and low confidence primary biotransformation half-lives for α-, and γ-HBCD.

Table 2.7. Mass balance model summary for estimating biotransformation and bioisomerization rate constants from a dietary feeding study in rainbow trout (Law et al. 2006b).

	Diastereomer					
Parameter	**α-HBCD**		**β-HBCD**		**γ-HBCD**	
	Value	*Cf*	Value	*Cf*	Value	*Cf*
W (kg)	0.25	1.5	0.25	1.5	0.25	1.5
L_F (kg/kg)	0.045	1.5	0.045	1.5	0.045	1.5
T (°C)	11.5	1.05	11.5	1.05	11.5	1.05
G_D (kg/d)	$0.005W$	2	$0.005W$	2	$0.005W$	2
C_{OX} (mg/L)	9.9	1.25	9.9	1.25	9.9	1.25
E_D	N/R [a]	N/A	0.41	2	0.41	2
K_{OW}	$2.40\text{x}10^5$	3	$2.95\text{x}10^5$	3	$6.31\text{x}10^5$	3
k_T (d^{-1}) [b]	$9.0\text{x}10^{-3}$	4.4 [c]	$4.41\text{x}10^{-4}$	4.6 [d]	$1.44\text{x}10^{-3}$	4.2 [d]
k_M (d^{-1})	$1.7\text{x}10^{-3}$	13	No estimate possible		$3.0\text{x}10^{-4}$	7

Cf: confidence factor; k_T: total elimination rate constant; W: whole body weight of the fish; L_F: whole body lipid fraction of the fish; T: water temperature; G_D: feeding rate; C_{OX}: dissolved oxygen concentration; E_D: chemical transfer efficiency between the gastrointestinal tract and the organism; K_{OW}: octanol-water partition coefficient; k_M: primary biotransformation half-life (loss of NA: not applicable; NR: not reported.

[a] Model default calculation

[b] Reported or calculated elimination rate constants for "net loss of parent isomer". For α-HBCD, the rate constant described in the text is assumed first-order, for β-HBCD and γ-HBCD, the reported rate constants are corrected for the % of actual elimination of the parent isomer as reported in Table 2.6. Since the value for β -HBCD is apparently "no net loss" a value of 10% of the reported value was assumed

[c] Assumed

[d] Calculated from the coefficient of variation from the empirical elimination half-life estimates following (Slob 1994).

There are complicating factors with these data other than bioisomerization. The Law et al. (2006b) data are sampled from muscle tissue and not whole body elimination rate constant estimates. The fish were being dosed via the food and HBCD would be subject to first-pass at the liver, which could be the predominant site for bioisomerization as well as biotransformation of the HBCD isomers to metabolites. Whole body distribution (toxicokinetics) is expected to be slow and changes in the liver are expected to be faster than changes in the muscle tissue. The preferential solubility in storage lipids (solubility in octanol) for α-HBCD may be another complicating factor in the analysis. Furthermore, the study recognized that steady state was not reached.

Drottar and Krueger (2000) conducted a flow through bioconcentration test for t-HBCD at two different exposure concentrations for Rainbow trout (0.035 kg). The measured average concentrations were 0.18 and 1.8 $\mu g \cdot L^{-1}$, for the low and high levels, respectively. The whole body bioconcentration factors (BCFs) were 13,000 (not at steady

state) and 9,000 $L \cdot kg^{-1}$, respectively. This study reports total elimination rate constants of 0.0228 and 0.0359 d^{-1} for t-HBCD for the low and high levels, respectively. It is noted that the sponsor of the study did not want the first two sample times during the depuration phase (days 1 and 3) analyzed and thus these time points were not included in the elimination rate constant estimates. The implications are unclear. The total uptake and elimination rate constants were calculated by BIOFAC and were used as inputs into the mass balance calculations for estimating k_M. The mass balance calculations for primary biotransformation of t-HBCD to metabolites provide k_M estimates of 0.0027 d^{-1} (Cf = 7.4) and 0.012 d^{-1} (Cf = 6.7) for the low and high levels, respectively. According to the screening level information regarding confidence in the estimated k_M values, both estimates are considered "high" (Arnot et al. 2008b). The study documents some experimental difficulties maintaining a constant water concentration for the low level exposure test. These difficulties may result in errors in the BIOFAC rate constant estimates which are derived by fitting a model to the chemical concentration time trends. The biotransformation rate constants from both exposures correspond to primary biotransformation half-lives of 260 and 56 d for the mass of the fish in the study (0.035 kg). Using a rearrangement of Equation 2.5, the corresponding primary biotransformation half-lives normalized HL_N to a 1 kg fish are 1080 and 235 d for t-HBCD for the low and high levels, respectively. These biotransformation rates are considered "very slow" and "slow", respectively (Arnot et al. 2009).

Nyholm et al. (2009) evaluated the dietary exposure and biotransformation of various brominated flame retardants, including HBCD, in Zebrafish. There was low recovery for HBCD in this study and only relative concentrations could be determined. Thermal degradation during the analysis is considered to be a plausible explanation for the low recovery. A "high bioaccumulation potential" was suggested by the authors, although bioaccumulation parameters such as elimination rate constants were not reported.

A QSAR for predicting screening level primary biotransformation rates in fish estimates a k_M of 0.0071 d^{-1} for a 1 kg fish (Arnot et al. 2009; U.S. EPA 2009). This rate constant corresponds to a HL_N of about 100 d.

In consideration of all of the factors for the biotransformation data available for fish a HL_N of 250 d is selected for mass balance modelling with a Cf of 7. Thus, 95% range of the generated estimates for HL_N will be between 35 and 1750 d.

Mammalian Biotransformation

Geyer et al. (2004) reported total elimination half-life estimates for rats and humans for t-HBCD. The data for humans were derived from body burdens and daily intake rates. It is unclear how the data for rats were determined. The reported total elimination half-lives are 64 d (reported range of 23 to 219) and 8 d for humans and rats, respectively. The total elimination half-life includes elimination processes such as respiration, fecal egestion, urinary excretion and biotransformation. The mass balance method for fish was refined for application to mammalian half-life data assuming 70 kg for the human and 0.25 kg for the rat. The estimated HL_N (normalized to 1 kg) are 25 d (Cf = 3.4) and 13 d (Cf = 3.2) from the reported human and rat total elimination half-life data. A HL_N value of 20 d (Cf

= 5) was selected for the mass balance exposure modelling for t-HBCD. The 95% range of the generated estimates for HL_N will be between 4 and 100 d. This HL_N value is about 1 order of magnitude faster than the HL_N value selected for fish.

In vitro biotransformation studies have been conducted for laboratory rats and a harbour seal (sampled shortly after death) using microsomal liver preparations (Zegers et al. 2005). Significant reductions in LC-MS chromatogram peaks in the in vitro assays for rats (incubations of 1:1:1 isomeric mixture of α-, β- and γ-HBCD) showed that β- and γ-HBCD diastereromers were biotransformed; however, the peak of the α-HBCD did not decrease "significantly". The incubations of a 1:1:1 mixture of the HBCD isomers with the harbour seal microsomes showed average decreases of the parent isomers by 69, 60, and 17% for the β-, γ-, and α-HBCD isomers, respectively. β- and γ-HBCD were also tested separately and new peaks of brominated compounds (indicative of metabolites) were only formed when NADPH was added confirming a cytochrome P-450 mediated biotransformation (Zegers et al. 2005). Unfortunately, it does not appear that α-HBCD was analyzed for the possibility of forming peaks in the same isolated manner. Therefore the possible biotransformation of α-HBCD by cytochrome P-450 cannot be confirmed or refuted. Although α-HBCD was reduced to a lesser degree some formation of α-HBCD from γ-HBCD (bioisomerization) may have been possible during the simultaneous incubations.

Invertebrate Biotransformation

For earthworms, estimated biota-soil accumulation factors (wet weight-soil/wet weight-worm) ranged between 0.03 and 0.08 based on the total concentration of HBCD in worm tissue and estimates for the wet weight concentration in soil after 28 day of exposures to a range of soil concentrations (EC 2008). The diastereomer specific biota-soil accumulation factor for α-HBCD is more than one order of magnitude higher than the value for γ-HBCD (EC 2008). The study suggests a degree of bioisomerization of γ-HBCD to α-HBCD or preferential biotransformation of γ-HBCD. It is recognized that this is not a valid BCF or BSAF study.

Biotransformation Rate Summary

The selected half-life values are based on a critical review of the limited available data and thus require a degree of expert judgement. It is noteworthy that the primary transformation half-lives in water, soil, and sediment are shorter than the half-lives in fish. While this may be possible, it is counterintuitive, especially with regards to the degree in which the values differ (e.g. aerobic sediment – 35 d; 1 kg fish – 250 d). The data for fish are not definitive, but are supported by bioaccumulation measurements in the environment showing biomagnification in food webs (low biotransformation rates). This suggests that the environmental half-lives may be underestimates. Therefore, some of the mass balance model simulations will explore the possibility of half-lives in water, soil and sediment that are greater than median values.

In summary, there is conflicting evidence for diastereomer-specific biotransformation and bioisomerization rates and pathways based on in vitro, laboratory and monitoring data. These data are for different species under different conditions and natural variability is

expected among and between individuals and species. It is recognized that α-HBCD is usually the predominant diastereoisomer in monitoring data particularly for upper trophic level species (e.g. marine mammals, birds), but the precise reason for this cannot be elucidated from the data. It may be a result of preferential biotransformation to metabolites of the β- and γ-HBCD isomers and a limited capacity for biotransformation for α-HBCD in the food webs. It could also be that bioisomerization rates for γ-HBCD to α-HBCD are relatively faster than biotransformation rates for α-HBCD resulting in the preferential accumulation of α-HBCD. Different relative solubilities for the diastereomers in storage lipids may also be a factor. The uncertainty associated with bioisomerization and biotransformation limits the ability to accurately model isomer-specific HBCD bioaccumulation and subsequent exposures to organisms in the environment.

It is recommended that more comprehensive studies on bioisomerization and biotransformation in various species be conducted.

2.4.3 Halogen Bond Strength and Persistence

Table 2.8 compares general properties of different halogens (F, Cl, Br, I), in particular with regards to halogen-carbon bonds. With respect to reactivity, the relative bond strength is an important consideration as is electronegativity. The order of bond strength is C-F > C-Cl > C-Br > C-I and the order of electronegativity is F > Cl > Br > I. These general trends support the hypothesis that organobrominated compounds are less persistent than chlorinated and fluorinated analogues (Neilson 2003), although steric hindrance and halogen substitution patterns are also important considerations.

Table 2.8. Selected properties of fluorine, chlorine, bromine and iodine atoms where X is the halogen (Lide 2000; Neilson 2003).

Property	**Fluorine (F)**	**Chlorine (Cl)**	**Bromine (Br)**	**Iodine (I)**
Atomic Mass	18.998	35.443	79.904	126.904
Ionic radius (nm)	0.133	0.181	0.196	0.198
Bond length in CX_4 (nm)	0.132	0.177	0.194	0.213
C-X bond energy (kJ mol^{-1})	552	397±29	280±21	209±21
Electronegativity	4.0	3.0	2.8	2.7

2.4.4 Half-Lives for Benchmarking

Table 2.9 lists half-life values selected for the benchmark chemicals.

Table 2.9. Selected primary transformation half-lives (HL; days) for the model benchmarking simulations. Data are from (Cowan-Ellsberry et al. 2009) except for *, in which environmental HLs were selected based on (Arnot et al. 2005; Mackay et al. 2006; U.S. EPA 2009) and fish HLs are from (Arnot et al. 2009; U.S. EPA 2009). Avian and mammalian HLs (Av/Mam) for * were assumed to be 1/3 estimates for fish. Transformation half-lives (HL_N) in biota are listed normalized to 1 kg body size and scaled to mass (M_i; kg) specific values (HL_i) for different species in the models as $HL_i = HL_N \times (M_i/1)^{-0.25}$.

Status	Abbrev	HL_Air	HL_Water	HL_Soil	HL_Sediment	HL_Fish (1 kg)	HL_Av/Mam (1 kg)
POP	HCB	650	1000	1500	9000	860	290
POP	PCB28	6	300	450	2700	190	8.3
POP	PCB101	20	1200	1800	10800	370	44
POP	PCB180	80	2200	3300	19800	1100	29000
POP	Ald *	0.2	1000	1000	2000	75	25
POP	Hept *	1.5	25	50	150	90	30
NOP	Biphenyl	3	15	22.5	135	5.2	0.2
NOP	*p*-cresol	0.3	5	7.5	45	0.35	0.2
NOP	Atrazine	0.4	50	75	450	0.3	0.2
NOP	CCl4	900	150	225	1350	0.2	0.2
Candi-POP	α-HCH	50	440	660	3960	55	20
Candi-POP	β-HCH	50	900	1350	8100	90	30
Candi-POP	γ-HCH	50	400	600	3600	120	40
Candi-POP	PBDE99	20	850	1275	7650	285	95

3.0 EXPOSURE CHARACTERIZATION

3.1 PRODUCTION AND USE

HBCD has been on the world market since the 1960s but its use in the production of flame-retarded polystyrene materials only began in the 1980s (EC 2008). The estimated total production volume of HBCD in 2001 was 16 700 *t* with approximately 57% of global distribution occurring in Europe, 23% in Asia-Pacific region, 17% in North America (5% in other regions) (see (TemaNord 2008)). Estimated HBCD production in 2003 was 21 900 *t*, suggesting that global production of this substance is still increasing.

HBCD is used primarily as an additive flame retardant (EC 2008). The four main products in which HBCD is used are:

i) Expandable polystyrene (EPS)
ii) Extruded polystyrene (XPS)
iii) High Impact Polystyrene (HIPS)
iv) Polymer dispersion for textiles

End-product uses include insulation and packing materials (EPS, XPS), electrical and electronic parts (HIPS) and textile coating agents (polymer dispersions). According to industry information, the main use (90%) of HBCD is for flame-retarded polystyrenes (EC 2008), predominantly EPS and XPS. While the production of polymer dispersions for textiles may be more limited, this category is important to consider since textile backcoating (industrial) was estimated to be the dominant release category to waste- and surface waters (EC 2008).

3.2 EMISSIONS AND MODE-OF-ENTRY

Several considerations are required for the evaluation of chemical concentration trends in environmental and biological compartments. Predicted concentration trends and values of P and LRT are dependent on mode of entry of substances into the environment and the parameters of the modelled environment. Further, the emission history and the location of emissions (source area) can determine if pseudo-steady state conditions are expected. This is particularly important for substances with high P_{OV}, since they will take a long time to reach steady state, especially in soil and sediment compartments where degradation is usually slower. Since P_{OV}, B, T and LRTP are estimated as intrinsic properties independent on the amount of substance released, an 'indirect' model evaluation can be conducted using relative concentrations.

Table 3.1 summarizes initial estimates of emissions and mode of entry to the environment for HBCD (EC 2008). Alternative plausible emission regimes were explored by corroborating mass balance model estimates with available monitoring data. Mode of entry information was used to determine the ratio of emissions occurring to air, water and soil (EC 2008). These assumptions were considered a starting point but variations were

also explored using model simulations and comparisons to available monitoring data. For HBCD simple assumptions are necessary based on the uncertain use and expected release scenarios.

Table 3.1. Total estimated European Union (EU-Releases) of HBCD by emission compartment and emission source category (point sources versus diffuse sources). Adapted from TemaNord (2008).

Compartment	Estimated Release ($kg \cdot yr^{-1}$) (% of total)	Attributed to Point Sources	Attributed to Diffuse Sources
Air	508 (6%)	53.6%	46.4%
Wastewater (to STP)	6251 (72%)	98.1%	1.9%
From STP to Recipient water*	1250		
Surface Water (direct)	1933 (22%)	89.2%	10.8%

* ~ 20% of incoming HBCD was estimated to be released from STP, ~ 80% is associated with sludge (EC 2008). Total degradation rate constant is assumed to be zero however.

The total estimated European Union emissions presented in Table 3.1 were based on an analysis of current production sites and do not include emission estimates from a major HBCD production facility in the UK which closed at the end of 2003 (EC 2008). According to estimates, this production facility used to be the largest point source of HBCD emissions in Europe. Another production facility, located in Germany, closed in 1997 but no emission data are available from this site.

Emission estimates from the UK production site were assessed in two different surveys (EC 2008). In the first survey, emissions to air and surface water were estimated to be approximately 600 and 1335 $kg \cdot yr^{-1}$ respectively for the year 2000. In the second survey, performed in 2001, emissions to air were estimated to be 3400 $kg \cdot yr^{-1}$ whereas emissions to wastewater (following onsite sewage treatment) were estimated to be 2000 $kg \cdot yr^{-1}$. These data suggest that i) emissions to air were proportionally higher for the EU region than indicated by Table 3.1, and ii) total emissions for the EU region were higher in absolute terms in the past.

HBCD is an additive BFR (physically mixed into polymeric products) and is not covalently bonded to the products as some other BFRs. HBCD is therefore expected to have higher leaching and emissions rates during the product life cycle compared to other BFRs that are chemically integrated (Law et al. 2005; Nyholm et al. 2009).

3.3 MONITORING DATA

Monitoring data from industrialized source regions and remote (Arctic) regions have been used as an indicator of LRT potential; however, the mere presence of a substance in a remote area is not evidence for a high LRT potential in itself. Modern analytical

methods can measure certain substances at concentrations of femtograms per cubic meter (10^{-15} g·m^{-3}). An important consideration is potential concentration gradients from sources in which the transport potential relative to other chemicals can be determined.

There are limited monitoring data available, particularly for the abiotic environment. The uncertainties associated with isomerization processes and rates in the environment further complicate any potential for isomeric-specific analysis at this time.

3.3.1 Air

Monitoring of HBCD in air has taken place in both remote and source regions of Sweden, the United States and China since the early 1990s. Early atmospheric measurements made in 1990-1991 at Ammarnäs and Hoburgen, Sweden showed that HBCD was present in ambient air in remote locations at picogram per cubic metre concentrations (Bergander et al. 1995). Measurements later reported at Rörvik, Sweden and Pallas, Finland, in 2000 and 2001 showed that rural Scandinavian locations were still exhibiting similar levels 10 years later, in fact Aspvreten, south of Stockholm exhibited concentrations up to 100 times the other background sites (25 and 280 pg·m^{-3}) (Remberger et al. 2004). Aspvreten is 80 km NE of the XPS facility and may be influenced by those emissions, although is termed "remote" in the literature. This study also reported HBCD levels in air in urban areas (76 and 610 pg·m^{-3}) as well as by point sources (19 to 1,070 pg·m^{-3}). Figure 3.1 shows that urban areas have similar atmospheric levels of HBCD as textile processing facilities and demolition landfill sites.

HBCD is present in air predominantly in the particulate phase. Yu et al. (2008) show concentrations in Guangzhou in Southern China to range from 0.7 to 3.1 pg·m^{-3} total, with 85 to 95% associated with particulates. This study also shows the importance of the urban environment with respect to atmospheric concentrations of HBCD as the highest level recorded was at an urban site. The materials used in the construction/renovation of urban areas as well as the treated consumer products utilized by the population are probable sources of HBCD to the urban atmosphere.

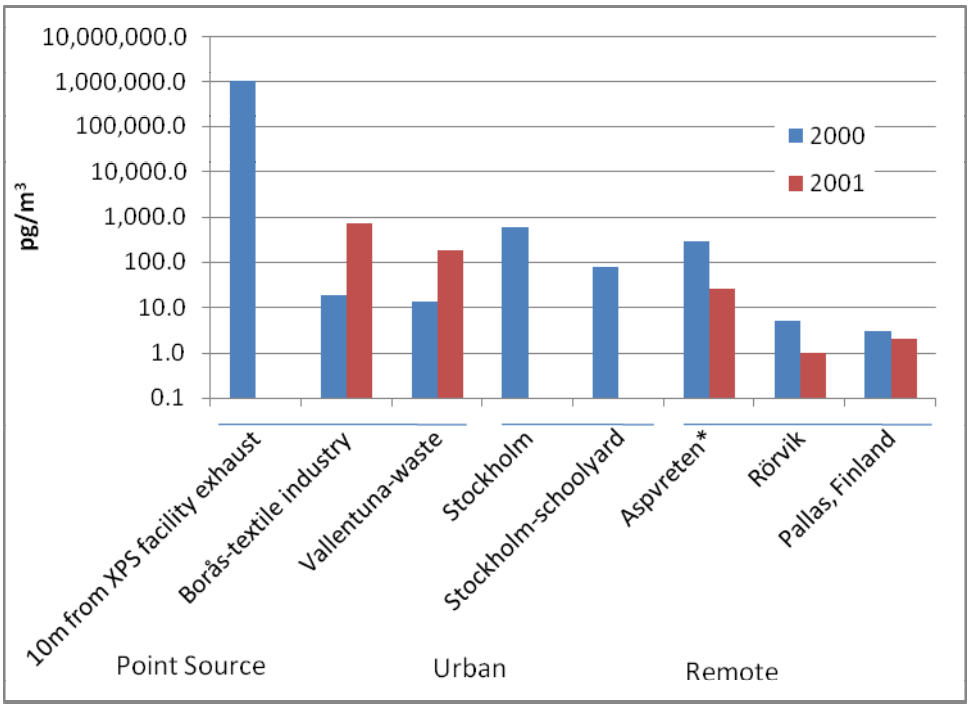


Figure 3.1. Air concentrations in Sweden and Finland in 2000-2001. *Aspvreten is 80 km NE of the XPS facility and may be influenced by those emissions, although is termed "remote" in the literature.

Hoh and Hites (2005) show the influence of urban areas as well as the influence of production facilities on atmospheric concentrations of BFRs in the atmosphere in the United States. Levels at the Arkansas site, approximately 150 km from Great Lakes Chemical (El Dorado, AR) and Albemarle (Magnolia, AR) plants where BFRs are manufactured were about 10 pg·m^{-3}, similar to those measured in Chicago (Hoh and Hites 2005). The background regional sites in northern Michigan and southern Louisiana showed concentrations between 0.16 and 8 pg·m^{-3}, similar to the industrialized and urbanized sites. The similarity of the background sites to "near source" regions suggests that HBCD is emitted, presumably from non-point sources (diffuse), in sufficient quantities to have become detectable in many populated areas of the global environment.

It is interesting to note is that at each of the sites in Guangzhou, the isomeric distribution is at least 50% α-HBCD (Figure 3.2). The similar profiles are in contrast to what was observed by Hoh and Hites (2005) in the US where the isomeric distribution is dissimilar across space and time (Figure 3.3). The spatial and temporal proximity of the Chinese samples to one another may explain their similarities. Atmospheric degradation is an important loss process and photoisomerization may also occur, therefore, differences in latitude and cloud-cover could play a role in the variation in the American data.

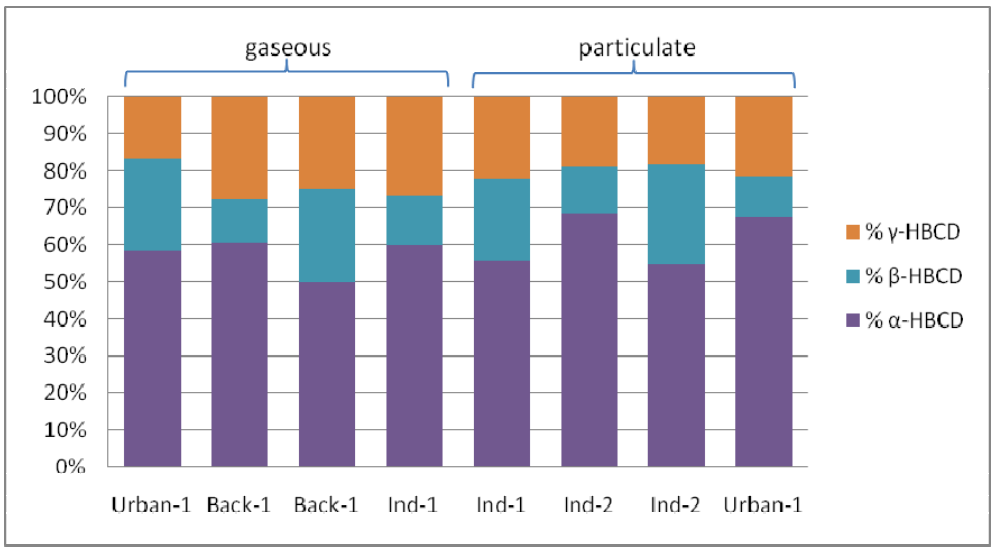


Figure 3.2. Isomeric distribution in gaseous and particulate air samples in Guangzhou, China (Yu et al., 2008).

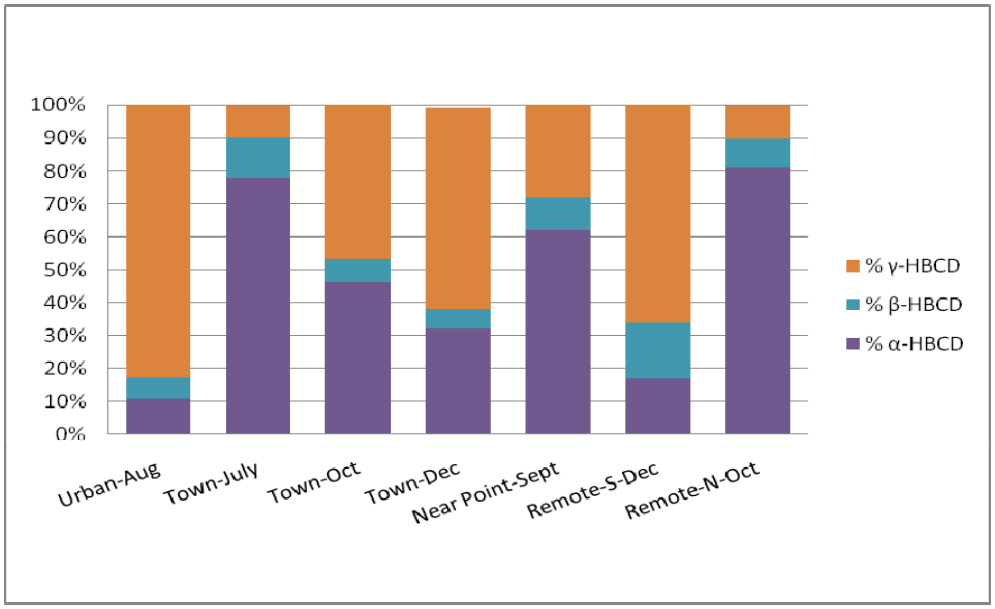


Figure 3.3. Isomeric distribution in particulate air samples from Central USA (Hoh and Hites 2005).

3.3.2 Sediment

Sediment is the most frequently monitored abiotic environmental compartment for HBCD. Sediment is a sink for hydrophobic chemicals such as HBCD. Due to the relatively high levels of organic matter present in the sediments and emissions to water, it is expected that HBCD will be present in sediments near source regions. A complication arises as aerobic degradation of HBCD is apparently much slower than anaerobic degradation, thus sediment analysis requires information on the aerobic status. Marine

sediments are reducing environments covered by an oxic surface layer of variable depth. For example, oxygen penetration depth in sediments from productive shallow coastal waters may only reach a few millimeters compared with cm or dm scales in oceanic sediments underlying a deep oligotrophic water column (Kristensen 2000). Estuarine and coastal sediments may also be anaerobic due to high organic input and pronounced stratification due to freshwater inflows (Drever 1997). Studies of fresh and estuarine sediments up and downstream from point sources such as textile plants and waste water treatment plants show the influence of these point sources to water systems. Studies involving point sources in fluvial and estuarine systems in Scandinavia, Switzerland, the Netherlands, Belgium and the UK, as well Tokyo Bay in Japan and the highly industrialized Detroit River in the USA/Canada show spatial sediment contamination trends. Figure 3.4 shows the downstream trend in average reported sediment concentrations.

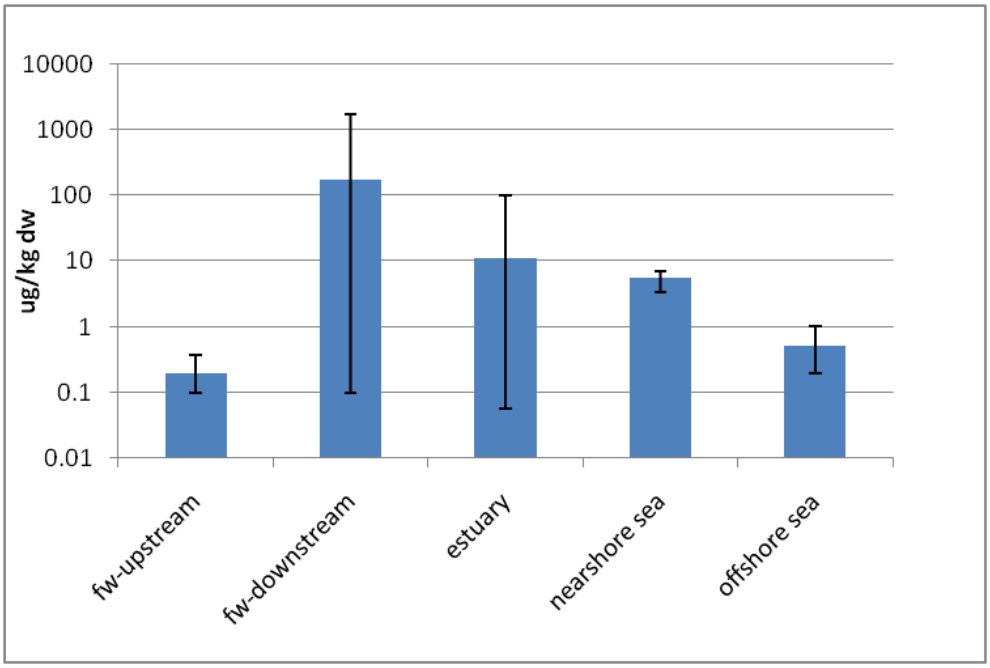


Figure 3.4. Downstream trend for mean sediment concentrations reported from Europe, Asia and North America. Error bars represent minimum and maximum values for each type of sediment (Sellström et al. 1998; Eljarrat et al. 2004; Morris et al. 2004; Remberger et al. 2004; Schlabach et al. 2004a; Schlabach et al. 2004b; Klamer et al. 2005; Verslycke et al. 2005; Minh et al. 2007).

Contaminant levels in isolated sediments in remote locations can implicate long range transport potential. Christensen et al. (Christensen et al. 2004) examined sediment cores from Lake Ellasjøen on Bear Island (Bjørnøya), a remote island in the Arctic Ocean (Latitude: 74.517 N, Longitude: 19.017 E). Lake Ellasjøen, which is on the southern end of the island, is highly productive due to inputs of seabird guano and it receives relatively high rates of precipitation for the island. Relatively high levels of contaminants are

observed due to bio-transport and precipitation (Christensen et al. 2004). Sediment cores reveal that HBCD was also detected (4.34 μg·kg^{-1} dw) in a subsurface layer dating from 1973 to 1987 with isomer ratios at ~10% α-HBCD, ~1% β-HBCD and ~89% γ-HBCD. This sediment core suggests a historical use pattern for HBCD and an isomeric pattern similar to t-HBCD.

Surface sediment monitoring data show that releases to river systems migrate downstream. Eljarrat et al. (2004), Sellström et al. (1998) and Schlabach et al. (2004b) studied European rivers in which there is a mix of possible industrial and urban sources. All studies used monitoring stations upstream of the impacted areas and then at sites downstream. Figure 3.5 shows that although there is variation in the concentration of HBCD in sediments along the watercourses, there is a general increase as the sampling proceeds downstream. It should also be noted that the levels observed in the upstream portions of these rivers (first point on each series) were below the detection limits of the studies and that HBCD levels increased between 1 and 4 orders of magnitude downstream. Figure 3.6 expands on the Drammen River and Fjord data and shows the concentrations and the isomeric breakdown at each site. Note that β-HBCD is reported below detection limits in most samples (MDL = 0.08 μg·kg^{-1} dw).

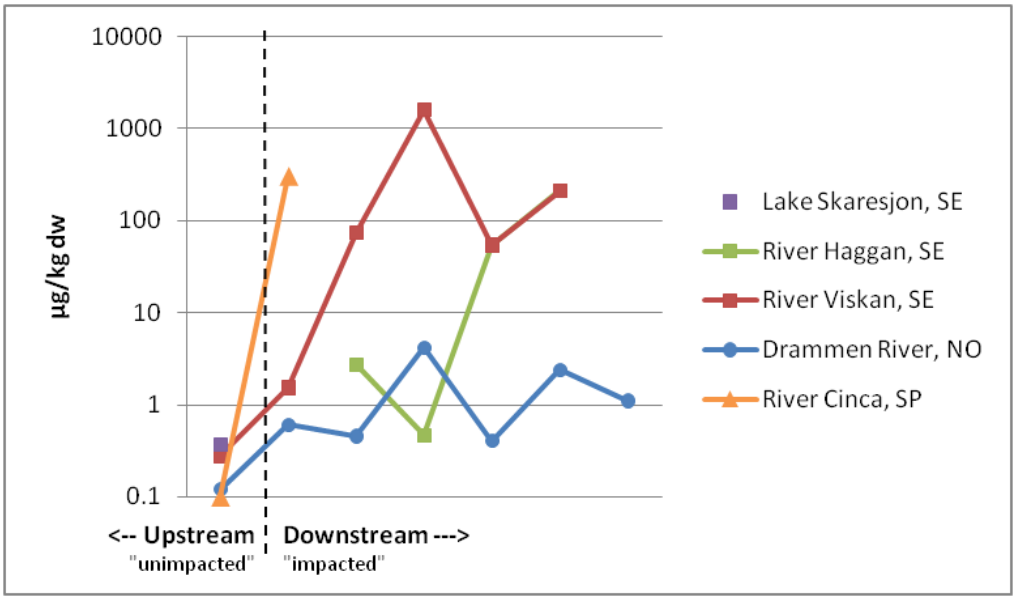


Figure 3.5. Surface sediment concentrations in two European river systems. The River Haggan is a tributary of the River Viskan. Lake Skaresjon is an unimpacted lake that is nearby the River Viskan. Dashed line represents relative location of initial HBCD release site. Location data are not to scale. Data are from Sellström et al. 1998 (Sweden) and Schlabach et al. 2004 (Norway), Eljarrat et al., 2004 (Spain).

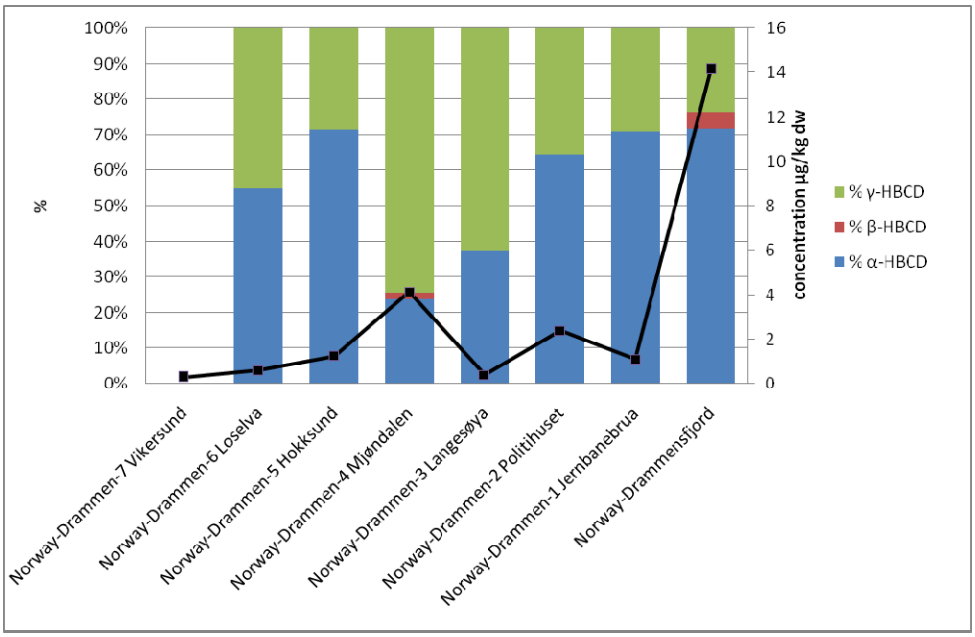


Figure 3.6. Proportion of isomers in Drammen River and Fjord sediment samples and concentration of ΣHBCD at each location. Only site 7-Vikersund is non-impacted. Locations of sources of HBCD were not included in the study (Schlabach et al. 2004a).

Estuarine environments are susceptible to contamination due to high sedimentation rates and current and historical industrial activity. Several studies have documented the levels of HBCD in sediments in the Scheldt Estuary (NL, BE), the Humber Estuary (UK), Dublin Bay (UK), Drammenfjord (NO) and Tokyo Bay (JP) (Morris et al. 2004; Schlabach et al. 2004a; Verslycke et al. 2005; Minh et al. 2007). Of these locations, the Scheldt has the highest average level of HBCD: 41 (range 0.7-99) $\mu g \cdot kg^{-1}$ dw and Tokyo Bay has consistently the lowest average: 0.76 (range 0.056 – 2.1) $\mu g \cdot kg^{-1}$ dw. Figure 3.7 shows the concentrations reported in these studies and the isomeric breakdown at each site. In Tokyo Bay, Minh et al. (2007) show the spatial variation of contaminant within the bay, ranging from 2.1 $\mu g \cdot kg^{-1}$ dw at the highly urban and industrial end of the bay to significantly lower concentrations nearing the mouth of the bay (Figure 3.8). This suggests that contaminant reaching the bay through riverine and direct inputs is being transported in small but detectable quantities to the oceans. Schlabach et al. (2004a) show similar results in Drammensfjord, NO.

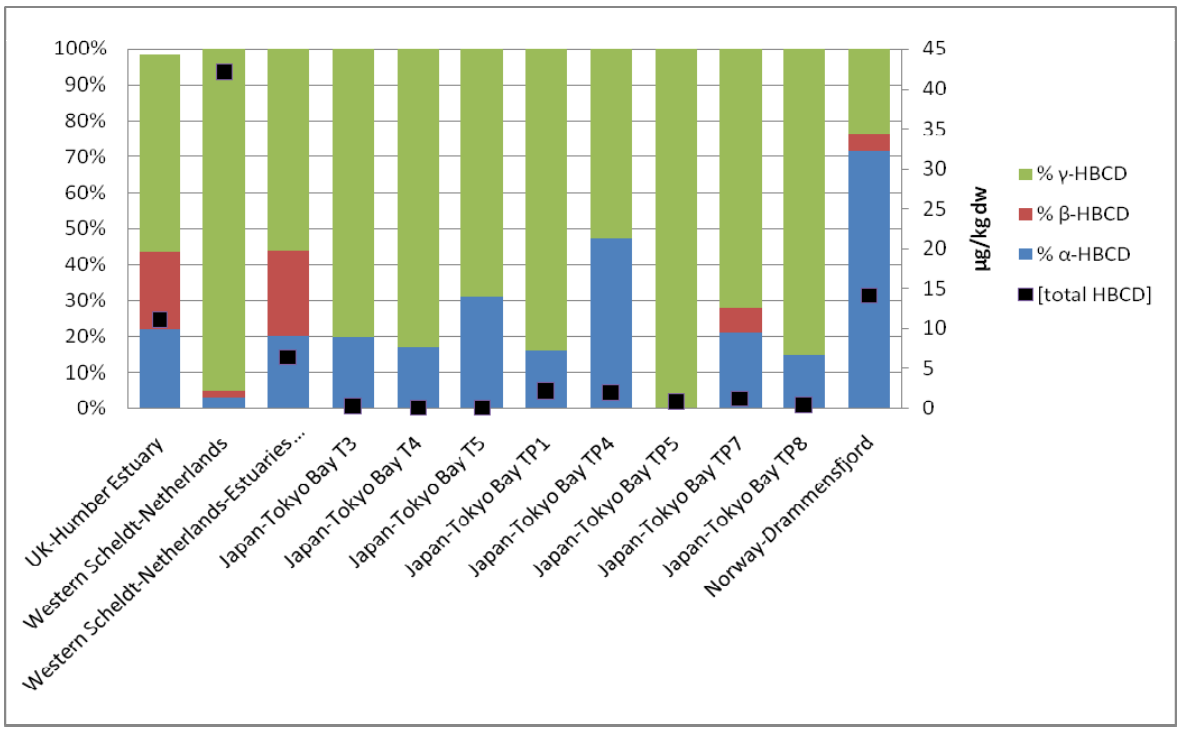


Figure 3.7. Estuarine concentrations and isomeric breakdown.

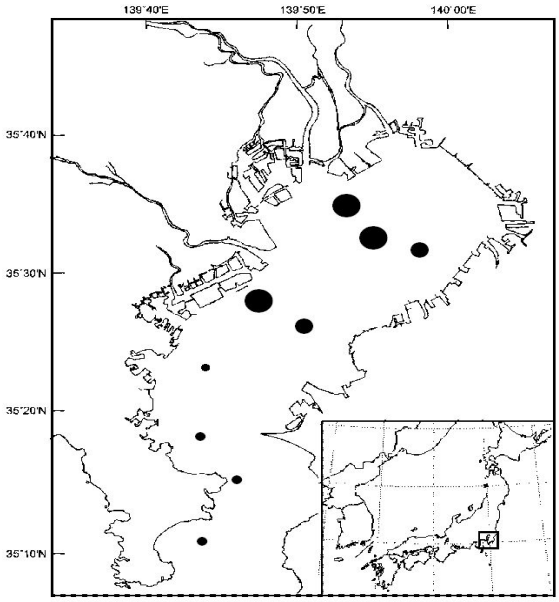


Figure 3.8. Spatial Trends: HBCD in Tokyo Bay surface sediments from Minh et al. (2007).

In the North Sea off the Netherlands and Germany, both nearshore and offshore measurements of surface sediment concentrations were made (Klamer et al. 2005; Lepom et al. 2007). The nearshore sediments ranged between 3.4 to 6.9 $\mu g \cdot kg^{-1}$ and the offshore sediments ranged from 0.2 (detection limit) to 1 $\mu g \cdot kg^{-1}$ dw. These offshore measurements in the North Sea show that HBCD sediment concentrations are detectable; however, at lower concentrations than those nearer to point sources.

3.3.3 Water and Soil

Remote measurements in soil and the water phases for HBCD are scarce. Studies reporting on these media are either centered in urban areas (Morris et al. 2004; Marvin et al. 2006) for water, or close to point source industrial facilities (Petersen et al. 2004; Remberger et al. 2004) for water and soil. These samples are often taken on the premises or a few hundred metres from the facilities. A summary of these data is presented in Table 3.2.

Table 3.2. Average water, suspended sediment and soil samples at point and urban sources in Europe.

Compartment	Point	Urban	n	Ref.
Water	141		8	Petersen et al. 2004
$\mu g \cdot L^{-1}$	0.006		2	Remberger et al. 2004
Susp Sed		1.6	4	Remberger et al. 2004
$\mu g \cdot kg^{-1}$ dw		1.3	8	Marvin et al. 2006
Soil	813		3	Remberger et al. 2004
$\mu g \cdot kg^{-1}$ dw	265		40	Petersen et al. 2004

It is recommended that more monitoring data be obtained, particularly in regional (non-point source) environments. Since air and water are the receiving compartments, these environments should be considered a priority.

3.3.4 Plants

Only one study documenting measured concentrations in HBCD in plants was cited in (EC 2008). The study reported measurements in stair-step moss (*Hylocomium splendens*) from locations in southern and northern Norway (Schlabach 2002), ranging from below the limit of detection (3 $\mu g \cdot kg^{-1}$ ww) to 11 $mg \cdot kg^{-1}$ ww for total HBCD. The most interesting aspect of this study was the spatial trend in reported concentrations; higher concentrations were generally reported for the more southern locations in Norway, falling below detection limits at the more northern sites. No additional studies reporting HBCD concentrations in plants have been located in the available literature.

3.3.5 Invertebrates

Monitoring data are available for both freshwater and marine invertebrates and include samples "near point sources", "regional source" and "remote" regions. Reported concentrations in marine invertebrates range over four orders of magnitude (EC 2008), indicative of strong spatial gradients in exposure related to proximity to point sources. For example, (Fjeld et al. 2005) reported concentrations of total HBCD in blue mussels (*Mytilus edulis*) from Åsnefjord, Norway, site of an EPS bead production facility, range from ~3,125 to 17,335 $\mu g \cdot kg^{-1}$ lw. Concentrations in blue mussels sampled along the Norwegian coast were substantially lower, ranging from ~50 to 400 $\mu g \cdot kg^{-1}$ lw. These latter concentrations are similar to those reported for the Kattegatt (Swedish Baltic coast) which ranged from 20 to 300 $\mu g \cdot kg^{-1}$ lw. In this case, the highest value was detected near a sewage treatment plant (Göransson et al. 2004); concentrations from the other sites ranged from 20 to 70 $\mu g \cdot kg^{-1}$ lw. Reported concentrations in tissues from other invertebrate species (e.g. hermit crab, lobster, oysters, prawns, scallops, starfish) harvested in EU and Norwegian waters that are not near known point sources for emissions tend to fall into a similar range range, i.e., 10 to 100 $\mu g \cdot kg^{-1}$ lw (EC 2008). However, it is difficult to compare concentrations across species since habitat use (benthic vs. pelagic), trophic position, and possibly biotransformation capacity all influence exposure and corresponding body burdens of hydrophobic substances such as HBCD.

Studies reporting concentrations of HBCD in invertebrates inhabiting remote regions are scarce. Tomy et al. (2008) reported concentrations in zooplankton (mixed, n = 5), shrimp (*Pandalus borealis* and *Hymenodora glacialis*, n = 5) and clams (*Mya truncate* and *Serripes groenlandica*, n = 5) from the Eastern Arctic sampled in 2000/2001; geometric mean values were 1.1, 1.9 and 1.4 $\mu g \cdot kg^{-1}$ lw total HBCD respectively. These relatively low values are consistent with the studies conducted by Sormo et al. (2006) and Morris (2007) which sampled invertebrates from the Norwegian Arctic and Barrow Strait (Canada) but reported that concentrations did not exceed detection limits.

3.3.6 Fish

Monitoring data are available for both freshwater and marine fish and also include samples from industrialized and remote regions (marine only). As with invertebrates, the range of reported values is high, spanning 5 to 6 orders of magnitude (EC 2008). This variability is again related to proximity to sources but may also be influenced more by trophic position of sampled species in comparison to invertebrates.

The highest reported concentrations of total HBCD in freshwater fish were reported for Brown trout (*Salmo trutta*) and eel (*Anguilla anguilla*) sampled in close proximity to known sources in the UK, Belgium and Sweden. In these studies, concentrations in muscle tissue as high as 160 $mg \cdot kg^{-1}$ lw were reported (downstream HBCD production plant) and values typically ranged from ~ 1 to 50 $mg \cdot kg^{-1}$ lw (EC 2008). Substantially lower muscle tissue concentrations ($\leq$ 35 $\mu g \cdot kg^{-1}$ lw) were reported in e.g. Brown trout inhabiting lakes in Switzerland (Schmid et al. 2004), perch (*Perca fluviatilis*) from lakes in Sweden ($\leq$ 25 $\mu g \cdot kg^{-1}$ lw) (Sternbeck et al. 2004) and pike (*Esox lucius*) from freshwater bodies in Finland (< 5 $\mu g \cdot kg^{-1}$ lw, (Peltola unpublished)). Concentrations in

fish sampled upstream of known sources are also substantially lower (on average) than downstream (EC 2008). These observations are broadly consistent with the emission mode of entry scenario presented in the (EC 2008); emissions to wastewater and surface water (confined to downstream areas of particular drainage basins, given the low log K_{AW} estimated for HBCD) dominate over atmospheric emissions (which tend to disperse more rapidly and lead to reduced spatial gradients in exposure). Comparisons between freshwater fish inhabiting industrialized areas and fish inhabiting remote regions (i.e. Arctic) are not possible due to lack of data.

Reported concentrations in marine fish included in (EC 2008) are lower on average and also span a narrower range of values in comparison to freshwater fish. As expected, fish species inhabiting marine environments impacted by point source discharges such as the Western Scheldt estuary tend to have higher reported concentrations. For example, measured conconcentrations of HBCD in sole (*Solea vulgaris*) and whiting (*Merlangius merlangus*) muscle tissue ranged from approximately 100 to 1110 $\mu g \cdot kg^{-1}$ lw and 20 to 275 $\mu g \cdot kg^{-1}$ lw respectively (de Boer et al. 2002). In comparison, the reported concentration of total HBCD in whiting analyzed as part of a UK market survey (pooled sample, n = 60) was 28 $\mu g \cdot kg^{-1}$ lw (EC 2008). Reported concentrations of eel (muscle) sampled in the Western Scheldt estuary ranged from 10 to 310 $\mu g \cdot kg^{-1}$ lw (de Boer et al. 2002; Janák et al. 2005), far below levels reported in more impacted freshwater regions (see above). An extensive database is available for Baltic herring (*Clupea harengus*) muscle tissue, covering the years 1999–2007 (Naturhistoriska riksmuseet 2008). These data are noteworthy because the Baltic Sea environment is often more seriously impacted by anthropogenic pollution due to the slow turnover of these waters and extensive drainage basin (Swedish Environmental Protection Agency 2009). Geometric mean concentrations in these fish over this time period range from approximately 5 to 25 $\mu g \cdot kg^{-1}$ lw. These concentrations are similar to levels reported for herring (muscle) available on the UK market (9.5 $\mu g \cdot kg^{-1}$ lw), assumed to be caught in UK waters (EC 2008).

Limited data are available for marine fish inhabiting the waters of remote regions. Mean reported concentrations in polar cod (*Boreogadus saida*) and redfish (*Sebastes mentella*) muscle sampled in the Eastern Arctic (Davis Strait) in 2000/2001 were 0.4 and 2 $\mu g \cdot kg^{-1}$ lw respectively (Tomy et al. 2008). These measured concentrations in polar cod are substantially lower than values reported by (Jenssen et al. 2004) for the Svalbard area (range 5 to 25 $\mu g \cdot kg^{-1}$ lw, liver tissue) and the North-East Atlantic (range 7 to 23 $\mu g \cdot kg^{-1}$ lw, liver tissue) (Bytingsvik 2004). This apparent discrepancy could reflect spatial patterns in exposure (Svalbard is much closer to sources in Europe) although the fact that different tissues were analyzed in different laboratories could also be an important consideration.

3.3.7 Birds and Mammals

Tissue concentrations of HBCD have been reported for bird species (terrestrial and marine) and marine mammals from industrialized and remote regions. Concentrations in polar bears from various regions of the Arctic are also available.

Measured concentrations of HBCD have been measured in Peregrine falcon (*Falco peregrinus*) eggs sampled in Sweden, the UK and Greenland (EC 2008). Reported concentrations of total HBCD in eggs sampled from wild populations ranged from approximately 50 to 2400 μg·kg^{-1} lw in the southern population and 35 to 500 μg·kg^{-1} lw in the northern population (Sellström et al. 2001; Lindberg et al. 2004). HBCD was detected above detection limits in only 12 of 51 eggs sampled in the UK (Leslie et al. 2004). However, detection limits were unusually high in this study; when above the limits of detection, the reported concentrations were up to 780 μg·kg^{-1} lw, i.e. similar to concentrations reported for Sweden. Reported concentrations in eggs sampled in Greenland over a similar time period (1995–2000) were generally much lower, ranging from < 0.1 to 30 μg·kg^{-1} lw (Vorkamp et al. 2005).

Sellström et al. (2003) presented a series of measurements in guillemot (*Uria aalge*) eggs collected over the period 1969–2001. More recent measurements extending this dataset are presented in (Naturhistoriska riksmuseet 2008). The increasing temporal trend in these data is discussed in more detail in Section 3.4. Reported concentrations in eggs over the period 1995–2000 (pooled samples, n = 10) range from 110 to 170 μg·kg^{-1} lw, broadly similar to peregrine falcons. Concentrations of total HBCD in guillemot muscle issue sampled in the same area in 2000 are generally lower, ranging from 25 to 148 μg·kg^{-1} lw (geometric mean = 65 μg·kg^{-1} lw) (Lundstedt-Enkel et al. 2006). No comparisons to guillemot populations inhabiting other regions can be made at this time due to lack of data.

Yolk sac from newly hatched European shag (*Phalacrocorax carbo*) inhabiting the mid-Norwegian coast was also analyzed recently, with total HBCD concentrations reported at levels of 417±208 μg·kg^{-1} lw (Murvoll et al. 2006). Measured concentrations of HBCD in birds inhabiting more remote regions (e.g. Svalbard, Bjørnøya) have been reported for several species including glaucous gulls (*Larus hyperboreus*; plasma, eggs, dead/dying birds), herring gulls (*Larus hyperboreus*; plasma, eggs, dead/dying birds), Atlantic puffin (*Fratercula artica*; eggs), kittiwake (*Rissa tridactyla*; eggs) and Northern Fulmar (*Fulmarus glacialis*; liver). The highest concentrations in birds inhabiting these areas were presented in Knudsen et al. (2007) for glaucous gulls. Here concentrations in brain and liver tissue were reported as 98.9±136 and 3026±4322 μg·kg^{-1} lw α-HBCD respectively. However, of the 21 birds sampled, nearly 50% were classified as completely or severely emaciated (total or near-complete lack of abdominal fat) and 30% as emaciated (substantial loss of abdominal fat) (Knudsen et al. 2007). In these animals, hydrophobic compounds accumulated over the life-time of the animal have been redistributed into remaining body lipids as abdominal fat was consumed to meet metabolic demands. Since faecal egestion is limited during starvation episodes, elimination of hydrophobic compounds is extremely inefficient and it is therefore not surprising that elevated lipid-normalized concentrations are reported for brain and liver tissues in these animals. These levels cannot be considered representative however and should be disregarded in the context of risk or hazard assessment. Reported concentrations in glaucous gulls sampled for other studies are far lower. For example, Verrault et al. (2004) reported mean total HBCD concentrations of approximately 20 μg·kg^{-1} lw in plasma samples from glaucous gulls from Svalbard for the year 2004. These

concentrations are similar to the mean total HBCD values reported in Verrault et al. (2004) for plasma samples from glaucous gulls inhabiting Bjørnøya (35 to 50 μg·kg^{-1} lw). Higher concentrations were reported in glaucous gull egg samples from the same island (140 μg·kg^{-1} lw). These concentrations are comparable to levels reported in eggs of herring gulls, Atlantic Puffins and Kittiwake from different remote locations in 2003 (Knudsen et al. 2005), which ranged from 45 to 175 μg·kg^{-1} lw total HBCD.

The most extensive dataset available for marine mammals in industrialized regions is for harbour porpoises (*Phocoena phocoena*) stranded or bycaught in the UK. Geometric mean concentrations in blubber tissue sampled over the period 2000–2006 range from approximately 135 to 1705 μg·kg^{-1} lw with peak values observed for the years 2001 and 2002 (Law et al. 2006c; Law et al. 2008). The temporal trends in these data are discussed in further detail in Section 3.4. Zegers et al. (2005) also reported measurements for harbour porpoises stranded over the period 2000–2003 which confirm the high levels reported by Law et al. (2006c; 2008) for UK waters. Harbour porpoises stranded or bycaught in Galicia, Spain were also included in this monitoring survery. As shown in Figure 3.9, the geometric mean total HBCD concentrations in blubber are much lower (a factor of 10 or more) in this region compared to locations in the UK. These findings have been attributed to point sources such as an HBCD production plant in NE England (Law et al. 2006c; Law et al. 2008).

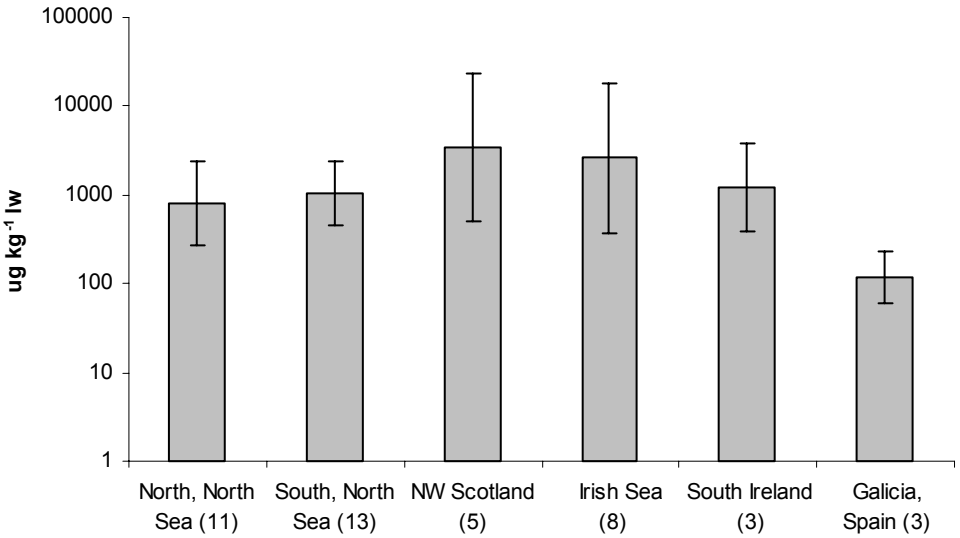


Figure 3.9. Geometric mean total HBCD concentrations (95% CIs indicated by error bars) in harbour porpoise blubber (μg·kg^{-1} lw) from various locations over the period 2000–2003 (Zegers et al. 2005).

Data from grey seals (*Halicoerus grypus*) inhabiting the Baltic Sea are also available (EC 2008). The reported mean (arithmetic) total HBCD concentration in blubber of these animals for the time period 2000–2002 is approximately 100 μg·kg^{-1} lw (range 31 to 554 μg·kg^{-1} lw), similar to harbour porpoises from Galicia, Spain (i.e. not indicative of highly

impacted environment). Total HBCD concentrations in muscle tissue ranged from 30 to 87 μg·kg^{-1} lw, somewhat lower than lipid-normalized blubber concentrations. Reported concentrations in marine mammals in industrialized regions of North America also appear to be lower than from harbour porpoises inhabiting the waters of the UK. For example, reported α-HBCD concentrations in blubber of male white-sided dolphins from the US East Coast sampled over the period 2000–2004 range from approximately 35 to 350 μg·kg^{-1} lw (Peck et al. 2008) while reported concentrations of total HBCD in blubber from California sea lions sampled over the period 2000–2003 range from approximately 4 to 95 μg·kg^{-1} lw with the majority of individuals below 20 μg·kg^{-1} lw (Stapleton et al. 2006).

Concentrations of HBCD in mammals inhabiting the remote Arctic regions have been reported for walrus, beluga and narwhal (Tomy et al. 2008), ringed seal (Sormo et al. 2006) and polar bears (Gabrielsen et al. 2004; Jenssen et al. 2004; Muir et al. 2006; Sormo et al. 2006). Reported total HBCD concentrations in walrus, beluga and narwhal blubber inhabiting the eastern Arctic (e.g. Davis Strait) sampled in 1998 were 0.6, 1.4 and 3.4 μg·kg^{-1} lw respectively (Tomy et al. 2008). These values are lower than those reported in mammals from Svalbard. For example, the total mean HBCD concentrations in blubber of ringed seal sample in 2003 was 20 μg·kg^{-1} lw whereas mean total HBCD concentrations in polar bears ranged from approximately 12 to 45 μg·kg^{-1} lw sampled in 2002–2003. Interestingly, mean total HBCD concentrations in polar bears inhabiting Greenland were similar to Svalbard (Muir et al. 2006) but reported concentrations in polar bears from the Bering Strait-Chukchi Sea area were substantially lower with a mean reported value of 0.4 μg·kg^{-1} lw (range <0.1 to 35.1 μg·kg^{-1} lw). If these concentrations are representative, it implies that i) emissions from Europe have a limited impact on the western Arctic and ii) HBCD inputs to the Western Arctic, likely emanating from North American and Asia-Pacific sources, are substantially lower than inputs impacting Svalbard. Hypothesis ii) is at least consistent with available information on the distribution of global HBCD production and use.

3.3.8 Humans

HBCD levels have been reported in human breast milk (e.g. (Aune et al. 2002; Lignell et al. 2003; Kakimoto et al. 2008; Eljarrat et al. 2009)) and adipose tissue (Pulkrabová et al. 2009). Kakimoto et al. (2008) analyzed samples in Japanese women (age 25–29) over the period 1973–2006; total mean HBCD concentrations over the period 2000 – 2006 ranged from 1 to 4 μg·kg^{-1} lw. These levels are higher than values reported for women in the Tromsø area (northern Norway); HBCD was detected in only 1/10 samples, at a concentration of 0.13 μg·kg^{-1} lw (Polder et al. 2008a). In contrast, substantially higher values were reported in a survey of women living in Northwestern Spain conducted in 2006/2007 (Eljarrat et al. 2009). In this location, the geometric mean total HBCD concentration was 27 μg·kg^{-1} lw (range 3 to 190 μg·kg^{-1} lw). It is also interesting to note that the γ-HBCD isomer dominated in the majority of these samples, as opposed to the α-HBCD isomer. Considering a wider range of studies from different countries however (Aune et al. 2002; Lignell et al. 2003; Ryan et al. 2006; Antignac et al. 2008; Polder et al. 2008a), the typical range of total HBCD concentrations in human breast milk in populations inhabiting industrialized areas appears to be <1 to 5 μg·kg^{-1} lw. No data on

concentrations of total HBCD in humans inhabiting the high Arctic are currently available for comparison.

HBCD has also been detected in human adipose tissue obtained by liposuction in the Czech Republic (Pulkrabová et al. 2009). Reported concentrations ranged from <0.5 to 7.5 μg kg^{-1} lw. No other human adipose tissue data are available for comparison.

Table 3.3. Summary of monitoring data collected near known point sources including the range of reported values and selected median values.

Compartment	**Units**	**Range**	**Median (*Cf*)**
Air	$pg \cdot m^{-3}$	10 - >1,000	100 (10)
Water	$\mu g \cdot L^{-1}$	0.006 - 141	5 (30)
Soil	$\mu g \cdot kg^{-1}$-dw	265 - 813	300 (3)
Sediment	$\mu g \cdot kg^{-1}$-dw	1 – 1,000	10 (80)
Aquatic invertebrates	$\mu g \cdot kg^{-1}$-lw		
Benthic invertebrates	$\mu g \cdot kg^{-1}$-lw	3,100 – 17,335	7,000 (2.5)
Fish - lower TL	$\mu g \cdot kg^{-1}$-lw		
Fish – upper TL	$\mu g \cdot kg^{-1}$-lw	1,000 – 50,000	7,000 (7)
Birds	$\mu g \cdot kg^{-1}$-lw		
Marine mammals	$\mu g \cdot kg^{-1}$-lw	130 - 1700	450 (4)
Terrestrial mammals	$\mu g \cdot kg^{-1}$-lw		
Humans	$\mu g \cdot kg^{-1}$-lw		

Table 3.4. Summary of monitoring data collected in "source" regional environments including the range of reported values and selected median values.

Compartment	**Units**	**Range (*n*)**	**Median (*Cf*)**
Air	$pg \cdot m^{-3}$	0.1 - 600	10 (10)
Water	$\mu g \cdot L^{-1}$		
Soil	$\mu g \cdot kg^{-1}$-dw		
Sediment	$\mu g \cdot kg^{-1}$-dw	<0.1 - 30	0.5 (20)
Aquatic invertebrates	$\mu g \cdot kg^{-1}$-lw		
Benthic invertebrates	$\mu g \cdot kg^{-1}$-lw	0.5 [a] - 100	30 (5)
Fish - lower TL	$\mu g \cdot kg^{-1}$-lw	5 [a] - 25	10 (3)
Fish – upper TL	$\mu g \cdot kg^{-1}$-lw	10 - 300	30 (3)
Birds (muscle tissue)	$\mu g \cdot kg^{-1}$-lw	25 - 150	65 (3)
Marine mammals	$\mu g \cdot kg^{-1}$-lw	30 - 750	150 (5)
Terrestrial mammals	$\mu g \cdot kg^{-1}$-lw		
Humans	$\mu g \cdot kg^{-1}$-lw	<DL - 190	

[a] includes food basket survey data

Table 3.5. Summary of monitoring data collected in "remote" regional environments including the range of reported values and selected median values.

Compartment	**Units**	**Range (*n*)**	**Median (*Cf*)**
Air	$pg \cdot m^{-3}$	1 – 6.1	4 (3)
Water	$\mu g \cdot L^{-1}$		
Soil	$\mu g \cdot kg^{-1}$-dw		
Sediment	$\mu g \cdot kg^{-1}$-dw	<DL – 4.3	0.16 (25)
Aquatic invertebrates	$\mu g \cdot kg^{-1}$-lw	<DL – 1.9	0.4 (5)
Benthic invertebrates	$\mu g \cdot kg^{-1}$-lw	1.4	1.4 (5)
Fish - lower TL	$\mu g \cdot kg^{-1}$-lw		
Fish – upper TL	$\mu g \cdot kg^{-1}$-lw	0.4 – 2.0	1 (4)
Birds	$\mu g \cdot kg^{-1}$-lw	20 - 175	60 (5)
Marine mammals	$\mu g \cdot kg^{-1}$-lw	<0.1 - 45	2 (20)
Terrestrial mammals	$\mu g \cdot kg^{-1}$-lw		
Humans	$\mu g \cdot kg^{-1}$-lw		

3.4 TIME TRENDS OF ENVIRONMENTAL CONCENTRATIONS

3.4.1 Abiotic Compartments

Sediment cores from depositional zones within study areas can be used to estimate historical levels of contamination. Typically, sediments are very slow to respond to changes in emission patterns and act as a repository and can even be a source for certain chemicals after emissions have been restricted. The ages of the subsections of the sediment core are determined using ^{210}Pb dating. Several studies have determined historical profiles of HBCD contamination in Europe and in Japan (Christensen et al. 2004; Remberger et al. 2004; Minh et al. 2007; Kohler et al. 2008).

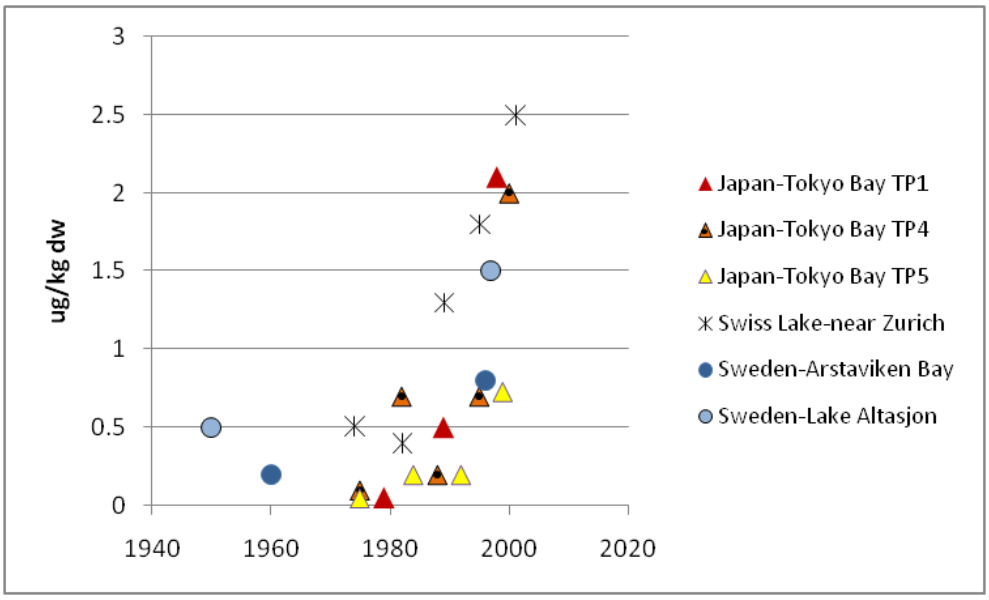


Figure 3.10. Time trend for HBCD in urban sediments

The Bear Island (Bjørnøya, NO) sediment core data were not included in this analysis as only one sample (1973-1982) was above the limit of detection (Christensen et al. 2004).

Of the sediment cores included in this analysis, the maximum concentration is 2.5 $\mu g \cdot kg^{-1}$ dw from 2001 in Lake Greifensee (CH) (Kohler et al., 2008). Figure 3.10 shows a clear increase in concentrations with time. Using simple linear regression, doubling times were calculated and are presented in Table 3.6. Although the absolute concentrations are near background levels, the Swiss and Japanese sediments show a dramatic increase, with doubling times of approximately 8 years for Greifensee and < 3 years for Tokyo Bay. It is recognized that a complication in the analysis of sediment core time trends is the uncertain degradation processes that may be occurring in deeper, older and possibly more anaerobic sediments.

Table 3.6. Calculated doubling times (years) from sediment cores in Europe and Japan.

Location	Slope	Intercept	Doubling time, years
Tokyo Bay, JP	0.06	-113.61	2.9
Greifensee, CH	0.08	-155.52	8.3
Stockholm, SE	0.02	-32.97	156.4

Atmospheric data for HBCD are sparse, particularly for remote regions. There are some data from remote regions in Sweden; however, they were not sampled at the same time or in a consistent manner.

3.4.2 Biota: Source Regions

The temporal trend in total HBCD concentrations in blubber of harbour porpoises stranded or bycaught in UK coastal waters over the period 1994–2006 was presented in (Law et al. 2006c; Law et al. 2008). These data are presented in Figure 3.11 as geometric mean HBCD concentrations (95% CI also shown).

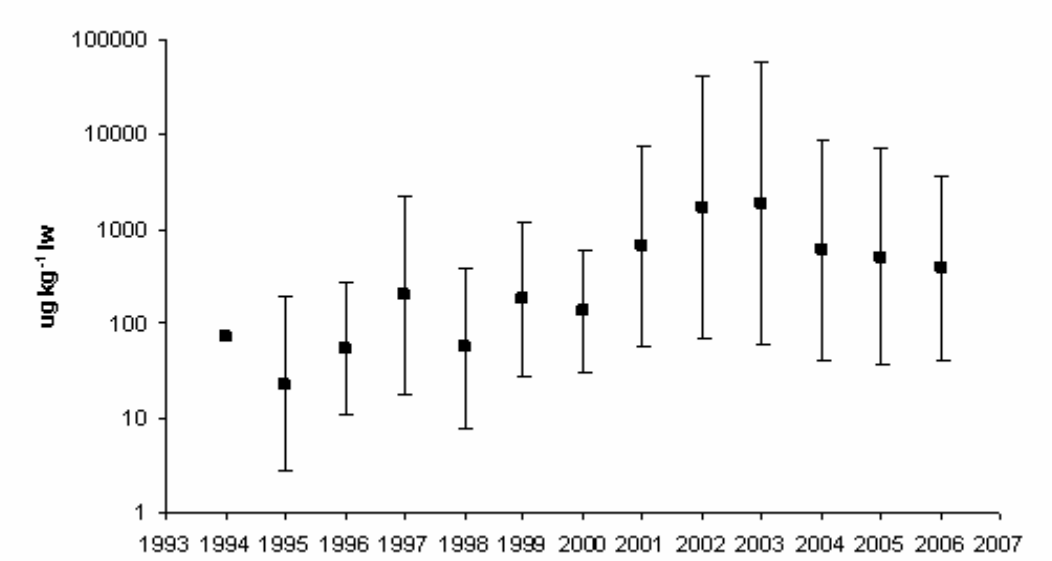


Figure 3.11. Temporal trends in geometric mean total HBCD concentrations (μg·kg^{-1} lw) in blubber of harbour porpoises stranded or bycaught in UK coastal waters for the period 1994–2006 (Law et al. 2006c; Law et al. 2008).

Law et al. (2006) included data from 1994–2003 and interpreted the data as indicating, "evidence for an increase in recent years". Additional data from animals sampled over the period 2003–2006 is presented in Law et al. (2008) and was interpreted as indicating, "a significant downturn in levels of hexabromocyclododecane in the blubber of harbour porpoises stranded or bycaught in the UK". According to Law et al. (2008), "investigation of time trends confirmed a statistically significant increase between 2000 and 2001 (p <0.01) and a statistically significant decrease between 2003 and 2004 (p < 0.05). Neither trend was confounded by age, sex, nutritional status, or location". The

significance of the apparent decreases in measured concentrations (all samples) for 2005 and 2006 could not be confirmed by the statistical methods applied. When examining the temporal trends by gender however, Law et al. (2008) report that "the downward trend for adult females from 2003 was not statistically significant (p = 0.072) but was from 2002 (p = 0.019)" and that in adult males, "there is a decrease from 2003 to 2004 (although it is *not quite* statistically significant at the 5% level) but after 2004 levels increase again".

The main reason for the high level of interest in these data is that a major HBCD production site, located in NE England, ceased production at the end of 2003 (EC 2008). According to (EC 2008), this facility used to be the largest point source of HBCD emissions in Europe with releases to air in the range of 0.6 to 3.4 $t{\cdot}yr^{-1}$, 1.3 $t{\cdot}yr^{-1}$to surface water and 2 $t{\cdot}yr^{-1}$ to wastewater from the onsite sewage treatment plant in 2000/2001. Unfortunately, emission estimates and/or production volumes over the period 1994–2003 are not available. This information would be quite valuable since the statistically significant increase in measured concentrations observed in harbour porpoises over the period 2000–2001 is as interesting as the apparent decline of HBCD over the period 2003–2004 with respect to understanding response times of the environment and food webs to changes in emissions of HBCD. Considering the outcomes of all trend analyses presented in Law et al. (2008), the limited number of data points for the post-emissions phase for these organisms in addition to the paucity of monitoring data for any other relevant environmental media in the same region, it is difficult to conclude that the decline in HBCD concentrations in harbour porpoises is representative of the post-2003 period. Consequently, no comparisons of the response times of biota to emission reductions between HBCD and other chemicals are attempted using these data.

Roosens et al. (2008) reported a 3.5-fold decrease in mean concentrations of total HBCD in eel muscle sampled in 2006 (10 $mg{\cdot}kg^{-1}$ lw) for locations in the River Scheldt compared to 2000 (35.5 $mg{\cdot}kg^{-1}$ lw). In the same study, mean concentrations of PBDEs declined by a factor of nearly 35, possibly reflecting the restriction of penta-PBDE technical mixture in 2004. While it is possible that the apparently declining HBCD concentrations in eel also reflect reduced emission/exposure, no corroborative monitoring data of ambient environmental concentrations or food items was included in this study. Therefore, it is not possible to use these data to estimate e.g. relative response times in these organisms or arrive at meaningful conclusions regarding the overall persistence of HBCD in the environment.

Monitoring data for herring muscle (1999–2007), guillemot eggs (1969–2007) and human breast milk (1980–2004) are available for locations in the Baltic Sea region. The temporal trends of these data are presented in Figures 3.12, 3.13 and 3.14 respectively.

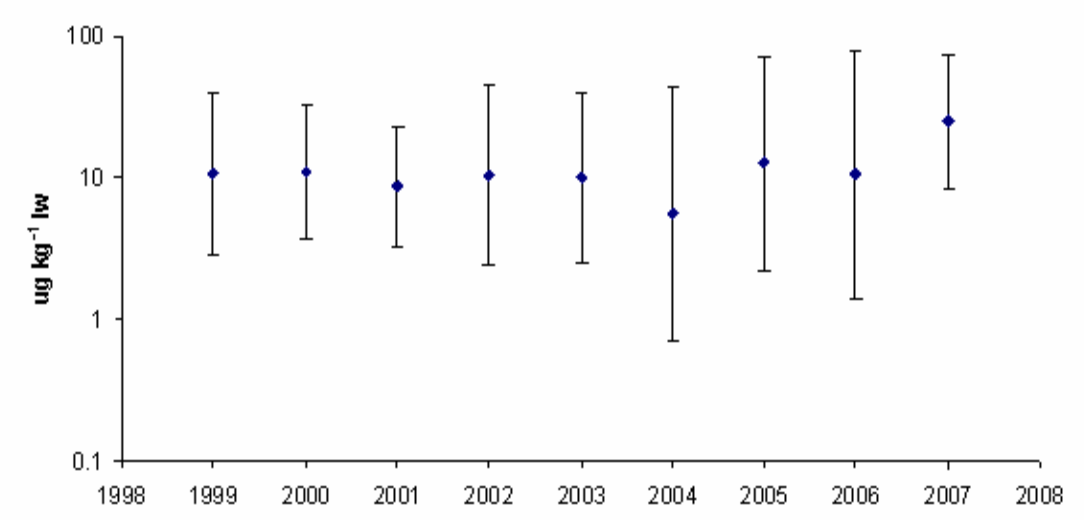


Figure 3.12. Geometric mean total HBCD concentrations (µg·kg^{-1} lw) in herring muscle sampled at various locations in the Baltic Sea (Naturhistoriska riksmuseet 2008).

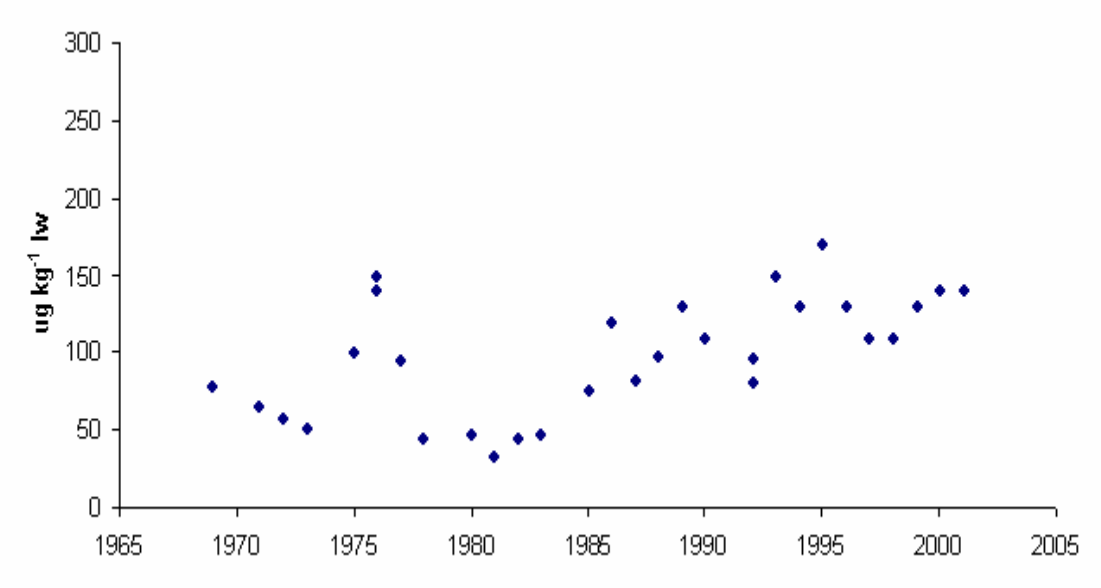


Figure 3.13. Mean total HBCD concentration (µg·kg^{-1} lw) in guillemot eggs (Sellström et al. 2003). Data from (Naturhistoriska riksmuseet 2008) not included here.

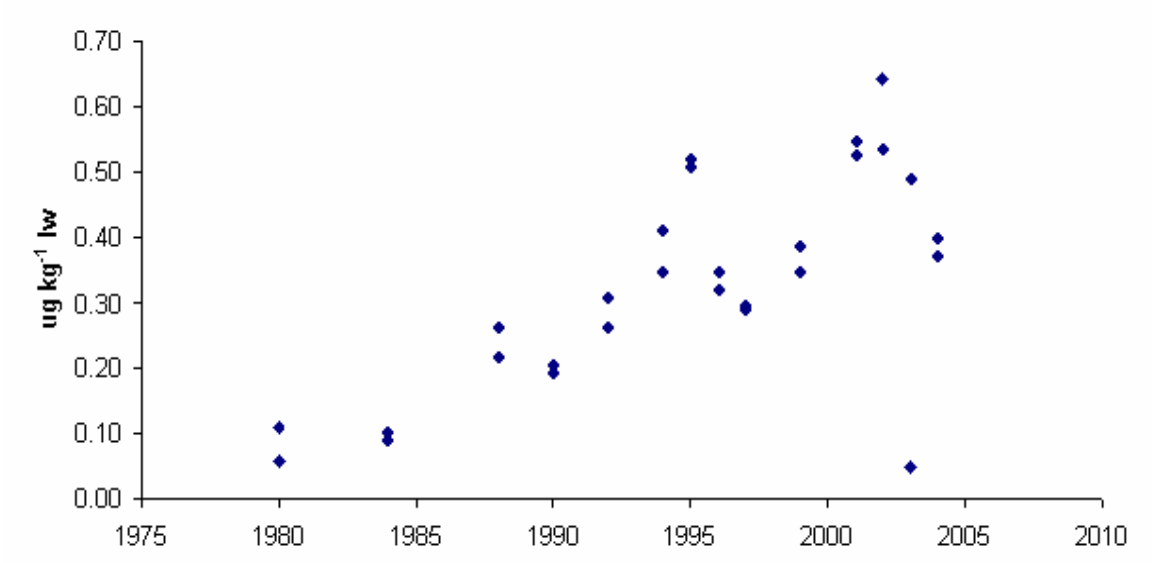


Figure 3.14. Mean total HBCD concentration (µg·kg^{-1} lw) in human breast milk samples (Fangstrom et al. 2008).

Reported concentrations in guillemot eggs (Sellström et al. 2003) and human breast milk (Fangstrom et al. 2008) show slight increases over the period 1980–1995. The overall increasing trends in reported concentrations over the time series are statistically significant in both cases as well. Interestingly, reported concentrations of total HBCD in herring muscle appear fairly constant over the period 1999–2007. The fact that concentrations in Baltic herring have been relatively stable while concentrations in higher trophic level consumers may still be increasing could reflect differences in uptake and elimination kinetics. Hendriks et al. (2001) demonstrated that uptake and elimination kinetics are inversely proportional to body size. This fact implies that the time to reach near-steady state body burdens (assuming constant exposure levels) is also inversely proportional to body size. Whether emissions to the Baltic region have actually stabilized cannot be confirmed however due to a lack of data on historical production, use and releases of HBCD as well as a lack of temporal data for relevant exposure media (e.g. air, water column).

Ismail et al. (2009) reported fairly constant concentrations in lake trout from Lake Ontario over the period 1979–2004. The highest mean concentrations in muscle tissue were observed for 1979. Not surprisingly then, when linear regression techniques were applied, a decreasing trend (statistically significant) in concentrations was observed. The fact that concentrations in lake trout were highest in 1979 is somewhat counterintuitive given the expectation that production/use (and hence emissions) has increased even in North America over the past two decades. Further information on production/use and emissions from this region could help clarify the interpretation of these data.

3.4.3 Biota: Remote Regions

Temporal trend data for biota inhabiting remote regions are available for several bird species (herring gull, Atlantic Puffin, Kittwake) inhabiting remote islands north of Norway (1983–2003) and Peregrine falcons inhabiting Greenland (1986–2003). These data are presented in Figure 3.15 and Figure 3.16.

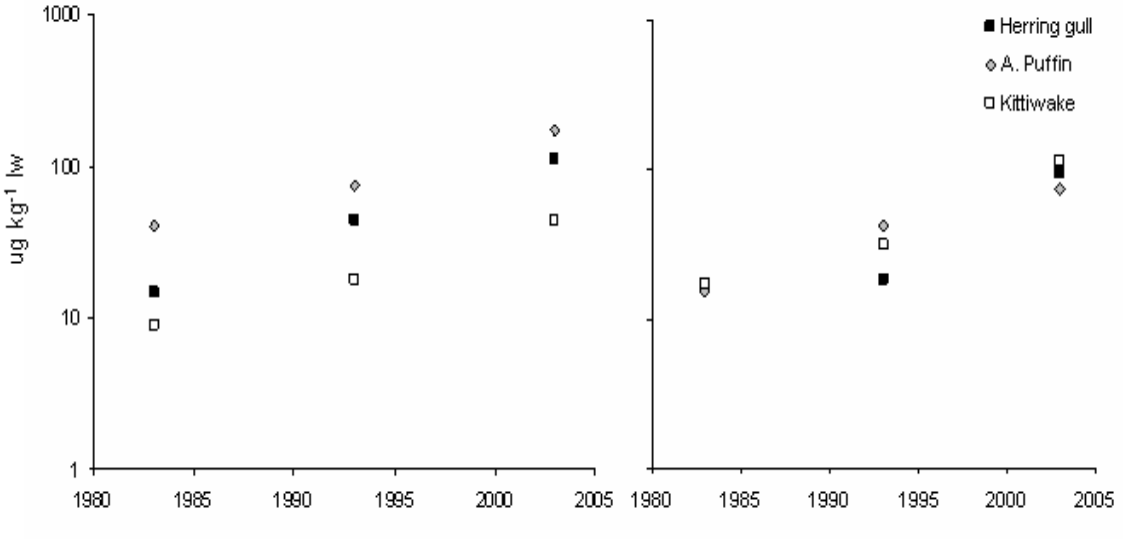


Figure 3.15. Mean total HBCD concentrations (µg·kg^{-1} lw) in eggs of herring gull, Atlantic Puffin and Kittiwake from two remote islands in the north of Norway, Røst (left panel) and Hornøya (right panel) (Knudsen et al. 2005).

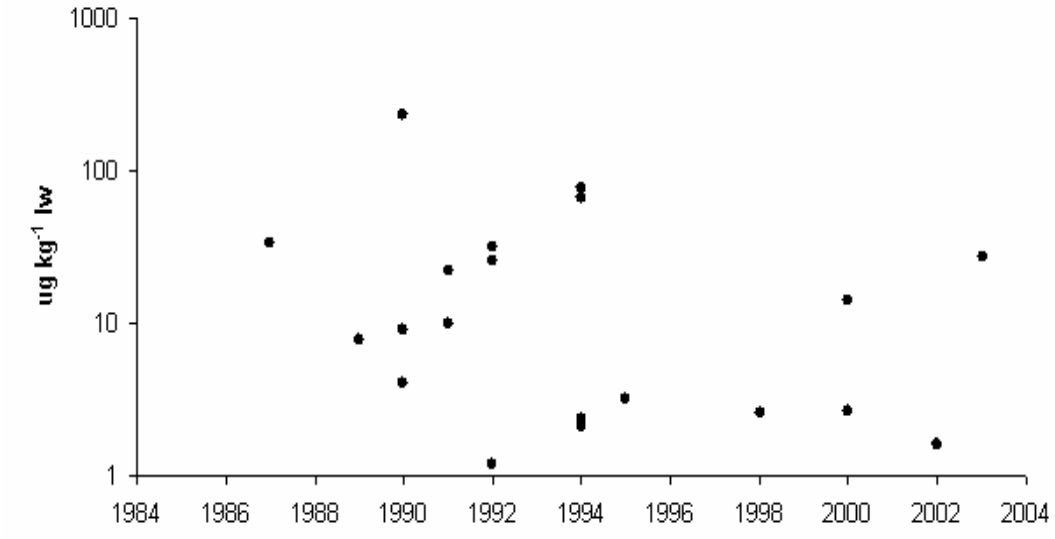


Figure 3.16. Reported concentrations of total HBCD in peregrine falcon eggs (µg·kg^{-1} lw) inhabiting Greenland (Vorkamp et al. 2005).

As shown in Figures 3.15 and 3.16, the temporal trends from these two studies are not similar; a statistically significant increase is seen in herring gull, Atlantic Puffin and Kittiwake eggs whereas a non-statistically significant decrease is observed for Peregrine falcon eggs. (Braune et al. 2007) also documented declining concentrations of total HBCD in the eggs of ivory gulls (*Pagophila eburnea*) inhabiting Seymour Island (Canadian Arctic) based on levels in samples from 1976, 1987 and 2004. These results are consistent with the temporal trend of HBCD concentrations in Lake Ontario lake trout (Ismail et al. 2009). It is possible that these diverging temporal trends reflect different exposure sources (e.g. North America vs Europe) and their relative importance in various regions of the Arctic but there is no additional information to corroborate this assertion at this time.

3.4.4 Key Considerations

Temporal trends in reported HBCD concentration in organisms from different locations do not show a uniform pattern. In some species, concentrations of HBCD may have stabilized over the past decade or even begun to decrease whereas there are indications from other studies that concentrations are still increasing in other species, including humans.

Analysis of temporal trend data in biota would be greatly facilitated by i) information on historical and current production/use of HBCD as well as the corresponding environment releases and ii) data characterizing the temporal trends in relevant exposure media (e.g. air, water, sediment cores). These data gaps limit the ability to interpret the temporal evolution of HBCD concentrations in the environment and compare them to the temporal evolution of other hydrophobic compounds which have been subject to restrictions or bans on use.

3.5 FATE AND EXPOSURE MODELLING

Basic mass balance modelling concepts are described in the Appendix (Section 9.4.1). The models were used to (i) characterize environmental distributions and long range transport potential of HBCD using available emissions and monitoring data, (ii) corroborate concentration measurements with current emission estimates and predict response to reductions in emissions estimates, and (iii) compare HBCD degradation and long range transport properties with those of known POPs (e.g. PCBs), candidate POPs (e.g. PBDE 99), and recognized non-POPs (e.g. *p*-cresol) (Klasmeier et al. 2006).

<u>HBCD Partitioning in the Atmosphere</u>

Before embarking on detailed modelling studies, it is useful to first explore the partitioning behaviour of HBCD in the atmosphere. This important partitioning equilibrium determines the rate of atmospheric deposition and degradation of semi-volatile organic chemicals (Lei et al. 2004). The gas-particle partitioning of HBCD as a function of temperature can be estimated using a simple K_{OA} based equation and applying the log K_{OA} of 10.46 for t-HBCD from Table 2.2 and a default energy of air-particle phase transfer of -80 kJ/mol. HBCD is predicted to change from a gas phase compound to a particle-associated chemical within the global environmental temperature range (+ 35 to

-35 °C) (Figure 3.17). Depending on the concentration and composition of the particles, that transition can occur within a slightly different temperature range. In a highly polluted atmosphere, a larger fraction of HBCD is expected to be particle bound than in the remote atmosphere at the same temperature. This may explain the high particle-bound fraction measured in Guangzhou (see Section 3.3.1).

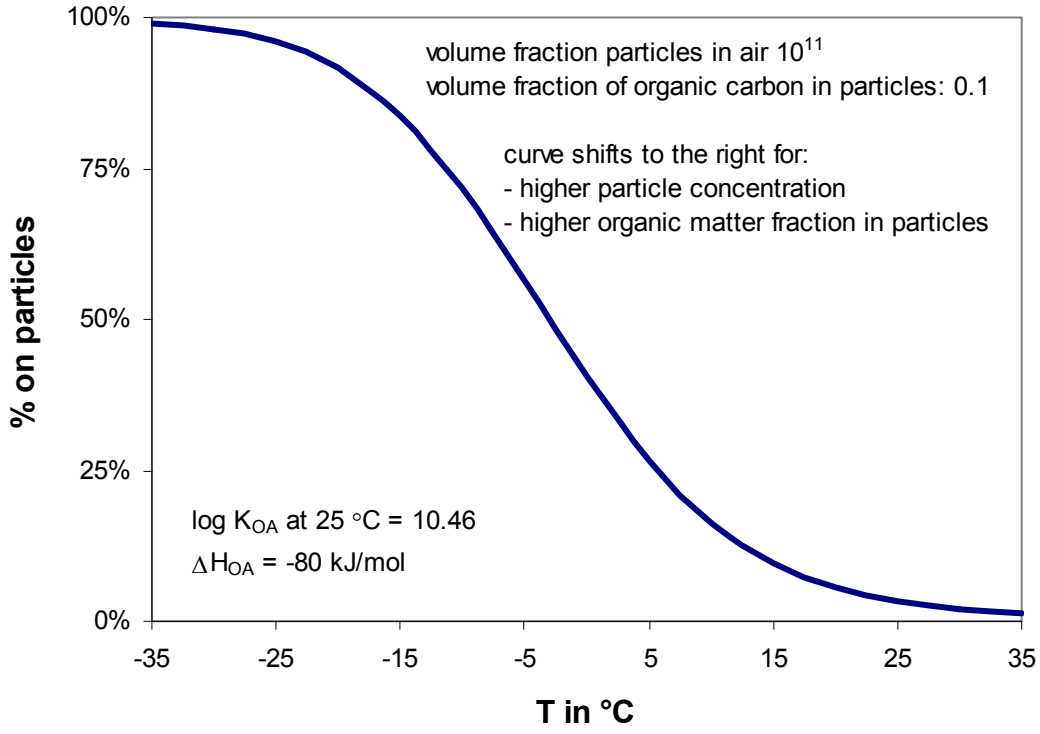


Figure 3.17. Estimated gas-particle partitioning behaviour of HBCD as a function of atmospheric temperature.

This suggests that HBCD will be subject to different atmospheric processes at different temperatures. At higher temperatures, a significant fraction of HBCD is present in the vapour phase, and may thus undergo gas phase reaction with OH radicals and gaseous deposition. At lower temperatures, the association with particles may prevent the oxidation of HBCD, but also increase the rate of dry deposition.

Interestingly, the air-water equilibrium partition coefficient (K_{AW}) of HBCD also places it in a transition area with respect to the partitioning between the atmospheric gas phase and water droplets in the atmosphere (Lei and Wania 2004). HBCD is not very efficiently scavenged by warm rain, but could be quite efficiently scavenged by fairly cold precipitation. In this regard it does not matter whether cold temperatures drive the gaseous HBCD onto particles or water droplets, because particles tend to be washed out efficiently by precipitation.

The long range transport of HBCD would presumably be most pronounced during cold, dry periods. Low temperature would cause HBCD to sorb to particles, where it is likely

shielded from OH radical attack. In the absence of precipitation, small particles can remain airborne for extended periods of time and can therefore be transported long distances. Most simple long range transport assessment models (e.g. the Tool) would fail to recognize the potential for such transport, because they either only consider relatively high temperature conditions and/or rely on the assumption of continuous precipitation.

In summary, whereas in a warm (model) environment, precipitation scavenging is unlikely to be an important deposition pathway for HBCD, the situation may be quite a bit different in a cold environment. By extension, the continuous rain assumption of RAIDAR and the Tool is unproblematic as long as we only model warm environments.

3.5.1 Benchmarking

Models were used to compare HBCD properties with those of selected benchmark chemicals. The physical-chemical properties and half-lives are listed in Tables 2.2, 2.3, 2.5, and 2.9.

3.5.1.1 The Tool

The Tool V2.0 (Wegmann et al. 2009) is an OECD sanctioned evaluative model for estimates of long range transport potential and overall persistence in the environment. The Tool was used to calculate the characteristic travel distance (CTD, km), transfer efficiency (TE; %), and overall persistence (P_{OV}, d) for t-HBCD and the benchmark chemicals. CTD is an estimate of the distance at which the concentration of a chemical in a moving parcel of air that interacts with the surface falls to (1/e) or 37% of its initial value (Bennett et al. 1999). TE is the percentage of emission flux that is deposited to the soil and water of a hypothetical region adjacent to the region receiving the emissions (Wegmann et al. 2009). P_{OV} is a metric of the overall reaction time in the defined environment and provides relative comparisons for chemical persistence as a result of degradation processes and intermedia partitioning. It includes half-life information for air, water, soil and sediment and the quantity of chemical in each compartment. It has been advocated as a more representative and integrated metric of chemical persistence rather than comparing estimated half-lives in individual environmental compartments to media specific criteria (Webster et al. 1998).

It is noted that a sediment compartment is not included in The Tool predictions and that this screening level model may not accurately capture gas-particle partitioning and related processes for all substances under all environmental conditions (see above).

Three calculations were made for t-HBCD. The first assumes selected median HLs for degradation in air, water and soil, the second assumes the lower bound degradation HLs in air, water and soil (t-HBCD, low), and the third assumes the upper bound degradation HLs in air, water and soil (t-HBCD, high) (Table 2.5).

For each chemical, i.e., “different HBCD scenarios”, and the benchmark chemicals, The Tool makes three sets of calculations, one assuming 100% emissions to air, one assuming 100% emissions to water, and one assuming 100% emissions to soil. Results for each scenario are available; however, the default output for the model is the highest (most

conservative, or 'worst case') value for each parameter (CTD, TE, and P_{OV}). Unless otherwise noted below, only the results for the 'worst case' calculations are presented.

The Tool CTD Benchmarking

Figure 3.18 compares the long range transport predictions of CTD and TE as well as P_{OV} for the t-HBCD scenarios and the benchmark chemicals. The CTD predictions are approximately 200, 600 and 1,500 km for the three different HBCD degradation scenarios (lower bound, median, and upper bound HL estimates, respectively). These 'worst case' results are for 100% emissions to air. When 100% of the emissions are assumed to water, the CTD predictions for HBCD are approximately 20, 200 and 1,300 km for the lower bound, median, and upper bound HL estimates, respectively. CTD is not calculated when 100% emissions are to soil.

The 'worst case' CTD estimates for HBCD (100% emissions to air, upper bound degradation HLs) are near the middle of the values for the benchmark chemicals. The lowest CTD value for the benchmark chemicals is for a non-POP: atrazine (125 km); however, the CTD for aldrin (a POP) is only about twice as high (230 km). The highest CTD values are for a non-POP: tetrachloromethane (330,000 km), and a POP: hexachlorobenzene (200,000 km). Figure 3.18 shows that CTDs for POPs and non-POPs span about 3.5 orders of magnitude. The 'worst case" CTD values for the three HBCD scenarios are closer to most of the non-POP CTD values than most of the POP CTD values. All three HBCD scenarios show that these CTD values are lower than CTD predictions for candidate POPs. HBCD CTD predictions are generally closer to values observed for "traditional non-POPs", regardless of degradation HL assumption. Clearly these CTD predictions for HBCD are much lower than values observed for "traditional POPs" such as hexachlorobenzene and PCBs.

The Tool TE Benchmarking

The TE predictions are approximately 0.05, 0.42 and 2.7% for the three different HBCD degradation scenarios (lower bound, median, and upper bound HL estimates, respectively). These 'worst case' results are for 100% emissions to air. When 100% of the emissions are assumed to water, the TE predictions for HBCD are approximately <0.0001, 0.0007 and 0.16 % for the lower bound, median, and upper bound HL estimates, respectively. When 100% of the emissions are assumed to soil, the TE predictions for HBCD are approximately <<0.0001, <<0.0001 and 0.0001 % for the lower bound, median, and upper bound HL estimates, respectively. The TE predictions for the lower bound and median HL assumptions are in the range of the TE predictions for most of the non-POPs. The TE prediction for the upper bound HL assumption is about 1 order of magnitude lower than TE predictions for the candidate POPs and half of the benchmark POPs. The highest TE predictions are for hexachlorobenzene (a POP; 1,450%) and tetrachloromethane (a non-POP; 140%). The lowest TE values are for a POP: aldrin (0.001%), and a non-POP: *p*-cresol (0.02%).

The Tool P_{OV} Benchmarking

The Tool 'worst case' P_{OV} predictions range from about 10 days for *p*-cresol and the HBCD lower bound HL scenario to 4,700 days for PCB 180. The P_{OV} predictions are

approximately 12, 120 and 1,200 days for the three different HBCD degradation scenarios (lower bound, median, and upper bound HL estimates, respectively). These 'worst case' predictions occur when 100% of the HBCD emissions are assumed to soil. When 100% of the emissions are assumed to air, the P_{OV} predictions for HBCD are approximately 1.3, 20 and 320 days for the lower bound, median, and upper bound HL estimates, respectively. When 100% of the emissions are assumed to water, the P_{OV} predictions for HBCD are approximately 12, 120 and 970 days for the lower bound, median, and upper bound HL estimates, respectively. The 'worst case' P_{OV} predictions for HBCD (median and lower bound HLs) are similar to most of the 'worst case' P_{OV} predictions for non-POPs (≤120 d) and separate from most POPs and candidate POPs (≥600 d). The upper bound degradation HL scenario results in P_{OV} predictions that are comparable to many POP and candidate POP benchmarks. The results highlight the uncertainty in environmental degradation half-life estimates on predictions of P_{OV} for HBCD. The benchmark simulations also shows that there is a POP (heptachlor; 70 d) with a 'worst case' P_{OV} comparable to most non-POPs and that there is a non-POP (tetrachloromethane; 1,270 d) with a 'worst case' P_{OV} comparable to many POPs and candidate POPs.

The mode-of-entry to the environment and the rates of degradation are shown to have significant impacts on LRT potential and P_{OV} predictions for HBCD. In particular, the mode-of-entry assumption influences LRT potential to a greater extent than the degradation HLs. The degradation HL assumptions have a greater influence on the predictions for P_{OV}. Due to the uncertainties in these two key parameters, there are uncertainties in comparisons with the benchmark chemicals. In general, however, HBCD shows a trend of having LRT and P_{OV} predictions that are more consistent with values for most of the benchmark non-POPs than values for most of the benchmark POPs and candidate POPs.

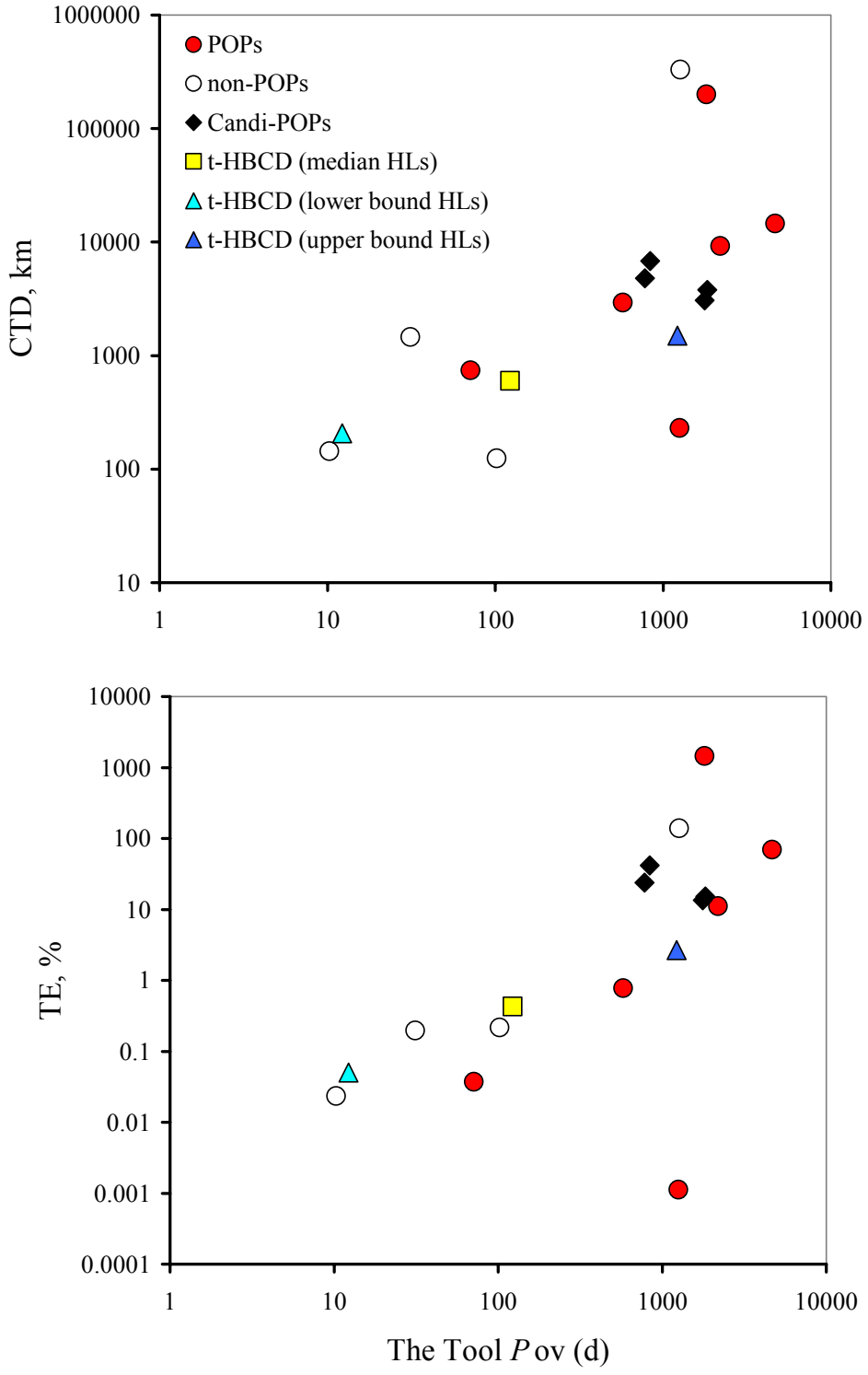


Figure 3.18. Long range transport (LRT) estimates (characteristic travel distance, CTD; transfer efficiency, TE) and overall persistence (P_{OV}) estimates for the selected substances using The Tool V2.0 model.

3.5.1.2 RAIDAR

Figure 3.19 provides a conceptual overview of the Risk Assessment IDentification And Ranking (RAIDAR) model (V2.0). Details are provided elsewhere (Arnot et al. 2006; Arnot and Mackay 2008). Briefly, RAIDAR is a screening level steady state evaluative model that combines information on chemical partitioning, degradation, environmental fate and transport, food web bioaccumulation, exposure, effect endpoint and emission rate in a coherent mass balance framework. RAIDAR includes a regional scale environment with an area of 100,000 km^2 (~90% land, ~10% water with underlying sediment) and a variety of representative plant, invertebrate and vertebrate species including fish, wildlife, agricultural crops, livestock, and humans. Primary producers and invertebrates bioconcentrate chemical from their ambient environment of air, water, soil, or sediment while all other species bioaccumulate chemical from their ambient environment and from their diet. The mass balance bioaccumulation models include major routes of chemical uptake and elimination. Only farfield exposures are currently considered for humans. The model does not include occupational or industrial exposures or indoor air and consumer product uses (e.g. personal care or household products) for exposures to humans. It is assumed that there are no losses or additions of chemical to food as a result of processing (e.g. animal husbandry) and preparation (e.g. cooking, washing).

RAIDAR "active" soil depth is 20 cm and sediment depth is 5 cm. These compartments are assumed to be aerobic. Default calculations include chemical degradation on aerosols in the bulk air compartment and suspended particles in the bulk water compartment. RAIDAR calculations are at an assumed constant temperature approximating temperatures associated with the physical-chemical property estimates. Since substances such as HBCD can be transported between regions greater than the RAIDAR evaluative environmental scale (i.e. neighbouring source regions), advective losses in air and water were considered negligible for all model calculations and benchmarking exercises in the present assessment.

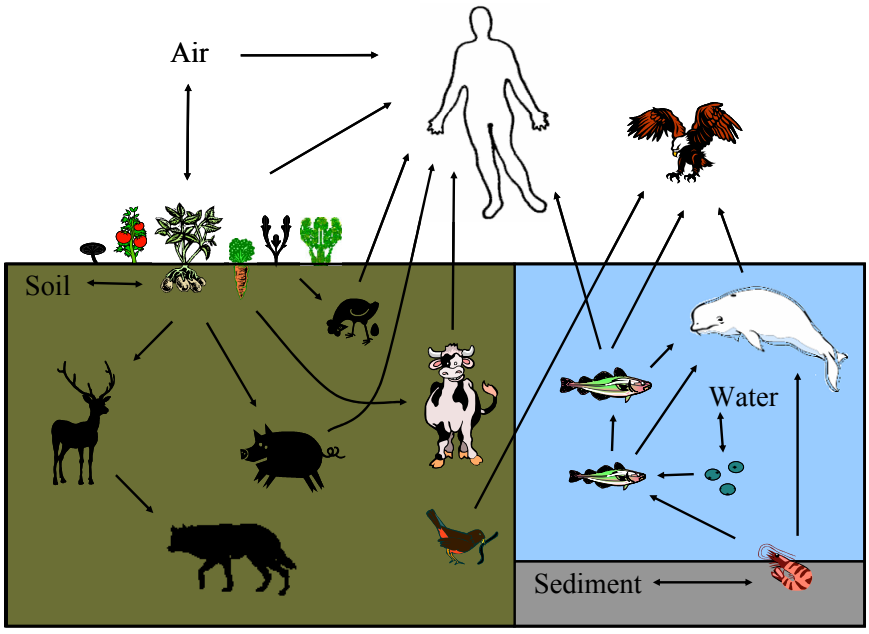


Figure 3.19. A conceptual overview of the RAIDAR model (modified from Arnot et al. 2006).

Fundamentally, risk is a function of the rate, or quantity (Q), of chemical released to the environment; fate, transport and persistence (P) in the physical environment (air, water, soil, sediment); bioaccumulation (B) in plants and animals; and toxicity (T) (Arnot and Mackay 2008). Elements of P and B determine the potential for chemical exposure while QPB determine actual exposures. RAIDAR combines P and B information into an exposure assessment factor (EAF) thus providing single values for transparent chemical comparisons and rankings based on exposure assessment objectives. The RAIDAR model is used to predict regional scale chemical concentrations in the environment and representative species. The model is not parameterized to a particular regional environment. The model uses an assumed unit emission rate for comparing HBCD with benchmark chemicals. Estimates of actual emissions can be used to scale unit emissions for comparisons of RAIDAR predictions with monitoring data.

RAIDAR EAF Benchmarking

RAIDAR V2.0 was used to calculate maximum exposure assessment factors (EAF) in the regional environment for HBCD and the benchmark chemicals. Essentially this is a hazard-based exposure metric identifying the representative organism in the regional environment that is expected to have the highest level of chemical exposure. The EAF combines information on chemical emission, fate and transport, degradation, food web bioaccumulation for all representative species in the RAIDAR environment. EAF is similar to the human intake fraction; however, it also includes absorption efficiency. It is calculated using a unit emission rate (E_U) of 1 $kg \cdot h^{-1}$ for each chemical; therefore, the

maximum exposure potentials predicted for a set of chemicals can be compared and ranked.

The individual diastereomers and t-HBCD were modelled for comparison. In addition, the lower and upper bound HLs were also considered as t-HBCD (low) and t-HBCD (high), respectively, to assess the uncertainty of these parameters on the EAF and RAIDAR P_{OV} estimates. Level II and Level III fate model calculations were considered. For the Level II fate calculations mode-of-entry information is not required because the model assumes equilibrium between the physical compartments of the environment (air, water, soil and sediment). The Level III fate calculations require mode-of-entry information because intermedia transport processes (e.g. precipitation) are considered, which can result in non-equilibrium conditions between the physical environmental compartments. For the Level III fate calculations, three scenarios were considered for mode-of-entry. The first assumes 100% of the unit emissions are to air, the second assumes that 100% of the emissions are to water and the third assumes that 50% of the emissions are to air and 50% are to water.

Table 3.7 lists the organisms identified as having the highest potential for exposure, the associated EAF estimates, i.e., maximum exposures, and the relative rankings of the chemicals based on the Level II fate calculations. The relative exposure potential for t-HBCD and the three diastereomers (median HLs) are in the middle of the rankings (10-13). t-HBCD (low bound HLs) shows a ranking equivalent to the non-POPs and t-HBCD (high bound HLs) shows a ranking equivalent to the POPs and the candidate POPs. Aquatic (marine) mammals are identified as the organisms with the highest exposure potential for all HBCD scenarios. This is a result of high lipid contents for these organisms and high dietary intake of HBCD through the consumption of fish. HBCD is not highly biotransformed in fish, resulting in high dietary sources for the aquatic/marine mammals. Piscivorous birds are the representative organisms that show the second highest exposure potential (data not shown).

Table 3.7. RAIDAR V2.0 Level II maximum exposure assessment factors (EAF), relative rankings and the organism of concern for HBCD and benchmark chemicals. EAF values listed are base 10 logarithms.

Status	Abbrev	Rank	Log EAF	Organism of concern
POP	HCB	2	-2.08	Aquatic mammal
POP	PCB28	14	-4.45	Aquatic mammal
POP	PCB101	5	-2.71	Aquatic mammal
POP	PCB180	1	-1.48	Aquatic mammal
POP	Aldrin	16	-4.92	Terrestrial carnivore
POP	Heptachlor	15	-4.84	Terrestrial carnivore
NOP	Biphenyl	19	-7.26	Benthic invertebrate
NOP	*p*-cresol	21	-8.33	Upper trophic level fish
NOP	Atrazine	18	-6.64	Benthic invertebrate
NOP	CCl4	20	-7.45	Aquatic mammal
Candi-POP	α-HCH	9	-3.88	Aquatic mammal
Candi-POP	β-HCH	6	-3.29	Aquatic mammal
Candi-POP	γ-HCH	8	-3.57	Terrestrial carnivore
Candi-POP	PBDE99	4	-2.66	Aquatic mammal
	t-HBCD	11	-4.38	Aquatic mammal
	α-HBCD	13	-4.40	Aquatic mammal
	β-HBCD	12	-4.39	Aquatic mammal
	γ-HBCD	10	-4.38	Aquatic mammal
	t-HBCD (low)	17	-5.37	Aquatic mammal
	t-HBCD (high)	7	-3.41	Aquatic mammal

Table 3.8 lists the organisms identified as having the highest potential for exposure, the associated EAF estimates (i.e. maximum EAFs), and the relative rankings of the chemicals based on the Level III (100% emissions to air) fate calculations. The relative exposure potential for t-HBCD and the three diastereomers (median HLs) are in the middle of the rankings (10-13). t-HBCD (low bound HLs) shows a ranking equivalent to the non-POPs and t-HBCD (high bound HLs) shows a ranking equivalent to the POPs and candidate POPs. These relative results are similar to the Level II results except that now the terrestrial carnivore (e.g. wolf) is identified as the organism with the highest exposure potential for all HBCD scenarios. This is a result of the higher trophic level of this animal and the fate and distribution of HBCD under this release scenario, i.e., HBCD is predominantly distributed to soil.

Table 3.8. RAIDAR V2.0 Level III (100% emissions to air) maximum exposure assessment factors (EAF), relative rankings and the organism of concern for HBCD and benchmark chemicals. Values listed are base 10 logarithms.

Status	Abbrev	Rank	Log EAF	Organism of concern
POP	HCB	2	-1.15	Aquatic mammal
POP	PCB28	14	-4.08	Aquatic mammal
POP	PCB101	4	-2.15	Aquatic mammal
POP	PCB180	1	-0.72	Aquatic mammal
POP	Aldrin	17	-4.90	Terrestrial carnivore
POP	Heptachlor	15	-4.30	Terrestrial carnivore
NOP	Biphenyl	20	-7.71	Foliage vegetation
NOP	*p*-cresol	21	-8.34	Foliage vegetation
NOP	Atrazine	18	-6.23	Foliage vegetation
NOP	CCl4	19	-7.45	Aquatic mammal
Candi-POP	α-HCH	9	-3.60	Terrestrial carnivore
Candi-POP	β-HCH	7	-3.21	Aquatic mammal
Candi-POP	γ-HCH	6	-3.12	Terrestrial carnivore
Candi-POP	PBDE99	3	-1.90	Aquatic mammal
	t-HBCD	11	-3.86	Terrestrial carnivore
	α-HBCD	12	-3.87	Terrestrial carnivore
	β-HBCD	13	-3.92	Terrestrial carnivore
	γ-HBCD	10	-3.86	Terrestrial carnivore
	t-HBCD (low)	16	-4.33	Terrestrial carnivore
	t-HBCD (high)	8	-3.47	Terrestrial carnivore

Table 3.9 lists the organisms identified as having the highest potential for exposure, the associated EAF estimates (i.e. maximum EAFs), and the relative rankings of the chemicals based on the Level III (100% emissions to water) fate calculations. These relative results are similar to the Level II results.

Table 3.9. RAIDAR V2.0 Level III (100% emissions to water) maximum exposure assessment factors (EAF), relative rankings and the organism of concern for HBCD and benchmark chemicals. Values listed are base 10 logarithms.

Status	Abbrev	Rank	Log EAF	Organism of concern
POP	HCB	2	-0.97	Aquatic mammal
POP	PCB28	9	-2.74	Aquatic mammal
POP	PCB101	4	-1.35	Aquatic mammal
POP	PCB180	1	-0.31	Aquatic mammal
POP	Aldrin	7	-2.38	Aquatic mammal
POP	Heptachlor	15	-3.09	Aquatic mammal
NOP	Biphenyl	18	-6.15	Benthic invertebrate
NOP	*p*-cresol	21	-8.15	Upper trophic level fish
NOP	Atrazine	19	-6.51	Benthic invertebrate
NOP	CCl4	20	-6.99	Benthic invertebrate
Candi-POP	α-HCH	16	-3.28	Aquatic mammal
Candi-POP	β-HCH	8	-2.63	Aquatic mammal
Candi-POP	γ-HCH	14	-3.02	Aquatic mammal
Candi-POP	PBDE99	3	-1.14	Aquatic mammal
	t-HBCD	11	-2.76	Aquatic mammal
	α-HBCD	13	-2.79	Aquatic mammal
	β-HBCD	12	-2.78	Aquatic mammal
	γ-HBCD	10	-2.76	Aquatic mammal
	t-HBCD (low)	17	-3.32	Aquatic mammal
	t-HBCD (high)	6	-2.36	Aquatic mammal

Table 3.10 lists the organisms identified as having the highest potential for exposure, the associated EAF estimates (i.e. maximum EAFs), and the relative rankings of the chemicals based on the Level III (50% emissions to air and 50% emissions to water) fate calculations. These results are similar to the Level II results and the Level III results assuming 100% emissions to water. This highlights the low air-water partition coefficient (K_{AW}) of HBCD and the moderately high K_{OW} (i.e. significant transport to sediments when entering the water).

Table 3.10. RAIDAR V2.0 Level III (50% emissions to air and 50% emissions to water) maximum exposure assessment factors (EAF), relative rankings and the organism of concern for HBCD and benchmark chemicals. Values listed are base 10 logarithms.

Status	Abbrev	Rank	Log EAF	Organism of concern
POP	HCB	2	-1.05	Aquatic mammal
POP	PCB28	9	-3.02	Aquatic mammal
POP	PCB101	4	-1.58	Aquatic mammal
POP	PCB180	1	-0.47	Aquatic mammal
POP	Aldrin	7	-2.68	Aquatic mammal
POP	Heptachlor	15	-3.38	Aquatic mammal
NOP	Biphenyl	18	-6.44	Benthic invertebrate
NOP	*p*-cresol	21	-8.45	Upper trophic level fish
NOP	Atrazine	19	-6.53	Foliage vegetation
NOP	CCl4	20	-7.25	Benthic invertebrate
Candi-POP	α-HCH	16	-3.44	Aquatic mammal
Candi-POP	β-HCH	8	-2.83	Aquatic mammal
Candi-POP	γ-HCH	14	-3.19	Aquatic mammal
Candi-POP	PBDE99	3	-1.37	Aquatic mammal
	t-HBCD	11	-3.04	Aquatic mammal
	α-HBCD	13	-3.08	Aquatic mammal
	β-HBCD	12	-3.06	Aquatic mammal
	γ-HBCD	10	-3.04	Aquatic mammal
	t-HBCD (low)	17	-3.62	Aquatic mammal
	t-HBCD (high)	6	-2.63	Aquatic mammal

γ-HBCD has a consistently higher EAF ranking than α-HBCD; however, the differences between isomers are relatively insignificant in the context of the current study. The general similarities in the results for t-HBCD and the diastereomers suggests that the representative properties for t-HBCD are likely sufficient to characterize HBCD for the modelling exercises in the context of the present study. Notably all diastereomers and t-HBCD currently share the same environmental degradation and biotransformation half-lives as a result of substance specific data limitations and the available estimates for the partitioning properties are comparable. In general, the differences between the EAFs for t-HBCD (median HLs for degradation), t-HBCD (lower bound or fast HLs for degradation), and t-HBCD (upper bound or slow HLs for degradation), are slight; however, the rankings relative to the benchmark chemicals can be affected significantly. Clearly, these preliminary results show that assumptions concerning mode-of-entry and environmental HLs are critically important for the benchmark assessment of HBCD.

RAIDAR P_{OV} Bencmarking

Table 3.11 compares the predictions for P_{OV} based on the different assumptions used in the RAIDAR model simulations. For HBCD, Level III simulations show lower P_{OV} predictions than the Level II calculations. There are also slight differences for the different diastereomers, with α- and β-HBCD showing the longest overall environmental persistence. This is a reflection of differences in the distribution of the diastereomers in the environment as a result of the differences in partitioning properties and not a result of degradation half-lives, which are assumed the same for all forms of HBCD in these simulations. In most cases the P_{OV} predictions for HBCD are more similar to P_{OV} values for the non-POP benchmark chemicals than to P_{OV} values for the POP and candidate POP benchmark chemicals. These general comparisons are most notable when the median or lower bound HL estimates are assumed; however, even when the upper bound HL estimates are assumed P_{OV} predictions of HBCD typically below, or well below, P_{OV} predictions for benchmark POP and candidate POP chemicals. The P_{OV} predictions for t-HBCD are higher using The Tool than using RAIDAR, particularly for emissions to water, because The Tool does not consider degradation in sediment.

The Level III simulations show that mode-of-entry is important for P_{OV} predictions for most, if not all, substances. For HBCD, P_{OV} predictions are lower when emissions are to air than when emissions are to water assuming either the median or lower bound HL estimates. However, when the upper bound HL estimates are selected there is little difference between the P_{OV} predictions based on the mode-of-entry assumption.

Table 3.11. RAIDAR V2.0 overall persistence (P_{OV}; d) estimates using Level II and Level III fate calculations.

Status	Abbrev	Level II	Level III (Air)	Level III (Water)	Level III (A&W)
POP	HCB	2028	1467	1542	1505
POP	PCB28	296	34	491	263
POP	PCB101	2149	317	1455	886
POP	PCB180	4588	2097	1954	2025
POP	Aldrin	77	2	861	432
POP	Heptachlor	53	3	90	47
NOP	Biphenyl	10	5	16	10
NOP	*p*-cresol	5	1	7	4
NOP	Atrazine	80	26	73	49
NOP	CCl4	1287	1288	1211	1249
Candi-POP	α-HCH	739	489	620	554
Candi-POP	β-HCH	1776	1677	1414	1546
Candi-POP	γ-HCH	726	564	623	594
Candi-POP	PBDE99	1834	1159	1740	1449
	t-HBCD	118	15	66	41
	α-HBCD	118	16	77	46
	β-HBCD	119	30	74	52
	γ-HBCD	118	14	65	40
	t-HBCD (low)	12	1	11	6
	t-HBCD (high)	1124	314	295	304

RAIDAR Sensitivity and Uncertainty Analysis

A sensitivity analysis was done for t-HBCD using the Level III 50% emissions to water, 50% emissions to air mode-of-entry scenario. The endpoint selected for the sensitivity analysis was the total body burden based on the assumed unit emission rate (TBB_U; ng) for the organism with the highest potential for exposure, i.e., the aquatic (marine) mammal. The justification for the selection of this representative organism is supported by the monitoring data. The sensitivity analysis indicates the influence of model input parameters (physical chemical properties and primary transformation half-lives) on the model output. Based on the sensitivity of the parameter (*Sens*) and the uncertainty of the parameter (e.g., *Cf* listed in Tables 2.2 and 2.3), the relative contribution of variance (*CV*) for each parameter on t-HBCD exposure to the representative marine mammal can be

determined. For example, the sensitivity (*Sens*) of an input parameter (P_i) on TBB calculations (model output) can be approximately quantified as (MacLeod et al. 2002)

$$Sens = (\Delta TBB/TBB)/(\Delta P/P_i) \quad (3.1)$$

where ΔTBB is the change in the TBB quantity and Δ*P* is a fixed change to a selected input parameter (e.g., 0.1%). The contribution to variance (uncertainty) of the substance input parameters (CV_i) on the TBB calculations are evaluated as a function of the variance (uncertainty) σ^2_i and sensitivity ($Sens_i$) of the individual input parameters as described by (MacLeod et al. 2002)

$$CV_i = \frac{\sigma^2_{P_i} S^2_{P_i}}{\sum_{k=1}^{n} \sigma^2_{P_k} S^2_{P_k}} \quad (3.2)$$

Table 3.12 shows that the most sensitive parameters to the TBB_U calculations are the primary biotransformation HLs in the marine mammal and in fish (its primary dietary item). The degradation HLs in water and sediment are also sensitive input parameters, but to a lesser extent than the biotransformation HLs. The biotransformation rates obviously influence the level in the marine mammal because it largely determines the residence time in the body. Biotransformation is particularly important for chemicals with high bioaccumulation potential in air breathing organisms (high K_{OW} and high K_{OA}). Due to these partitioning properties and the high lipid content of the organism, other routes of chemical elimination (respiration, urination) are very slow. Thus an increase in biotransformation half-life results in an increase in the TBB. The uncertainty in the biotransformation half-life is less than the assumed uncertainties in water and sediment; however, because of the sensitivity of the model to this parameter it has the highest contribution to variance in the prediction. Approximately 94% of the uncertainty associated with TBB_U for t-HBCD is a function of the biotransformation HLs in fish and the marine mammal.

It is emphasized that this analysis is dependent on the structure of the model (assumed feeding relationships, organic carbon content in sediment) and the selected endpoint. The selection of other endpoints (e.g. exposure to fish or invertebrates, or concentrations in different compartments) will result in different sensitivities on the output. Since the marine mammal is identified as the organism with the highest EAF according to LIII fate calculations that include emissions to water and marine mammals show some of the highest concentrations in the environment this selected endpoint for the sensitivity analysis is justifiable.

Table 3.12. Summary of RAIDAR model sensitivity for input parameters on estimated total body burdens in the aquatic/marine mammal based on a unit emission rate (TBB_U; ng/g) assuming equal emissions of HBCD to air and water.

Parameter	Sensitivity	Contribution to Variance
Vapor Pressure	-0.01	0.00
Water Solubility	0.01	0.00
K_{OW}	0.03	0.00
HL - Air	0.02	0.00
HL - Water	0.23	0.05
HL - Soil	0.00	0.00
HL - Sediment	0.21	0.03
Biotrans HL - Fish	0.82	0.50
Biotrans HL - Av/Mam	0.91	0.42

3.5.1.3 The Tool and RAIDAR

Two models were combined to compare HBCD against the benchmark chemicals by estimating screening level exposure potential in a "source region" and a "remote region". Details of this method are provided elsewhere (Cowan-Ellsberry et al. 2009). RAIDAR V2.0 is used to assess fate, partitioning, degradation and food web bioaccumulation to predict unit total body burdens (TBB_U; mmol) in a "representative" marine mammal based on a unit emission rate of 1 $kg \cdot h^{-1}$ (Arnot et al. 2006; Arnot and Mackay 2008). The OECD Tool (Wegmann et al. 2009) is used to calculate the characteristic travel distance (CTD, km) of each substance *in air* and scale estimated body burdens in the RAIDAR "source region" environment to estimates of body burdens in a "remote region" environment. The potential for exposure in a "remote region" is assessed using the CTD to estimate an effective emission rate into the remote region as a result of long range transport *in air* from a source region 2,500 km away.

For the present simulations only atmospheric transport to the remote region is considered and not transport in oceans or river water. The estimated body burden in the "remote region" is calculated by scaling the body burden in the "source region" by the fraction of the chemical that is transported to the "remote region". For this screening assessment identical environmental conditions (e.g. temperature, surface coverage of water, soil and vegetation) and exposure pathways (e.g. dietary selection) describe both the source and remote regions. This screening level approach also may not accurately capture gas-particle partitioning and related processes for all substances under all environmental conditions (see above). Level III RAIDAR fate calculations were assumed 100% emissions to air and no advective losses from the source region were assumed for calculating exposures in the source region. Median environmental HLs were selected.

Figure 3.20 compares the TBB_U predictions for the various substances in source and remote evaluative environments. For the source region simulation, the TBB_U predictions span 9 orders of magnitude from a POP (PCB 180) to a non-POP (*p*-cresol). HBCD "source region" TBB_U prediction is lower than most POPs and candidate POPs; however, it is comparable to some existing POPs (heptachlor, aldrin). For the "remote" region benchmarking scenario, the TBB_U predictions span >20 orders of magnitude from a POP (PCB 180) to a non-POP (atrazine); however, certain non-POPs have higher TBB_U predictions in remote regions than listed POPs. The HBCD TBB_U prediction is acomparable to certain POPs (heptahclor), but several orders of magnitude lower than other POPs (HCB, PCB 180). The HBCD TBB_U prediction is also about 3 orders of magnitude lower than the TBB_U predictions for the candidate POPs. It is noteworthy that The Tool long range transport predictions assume 100% of the emissions in the source region are to air. As shown above, the actual mode-of-entry to the source region environment can greatly influence the CTD calculation for most substances, including HBCD.

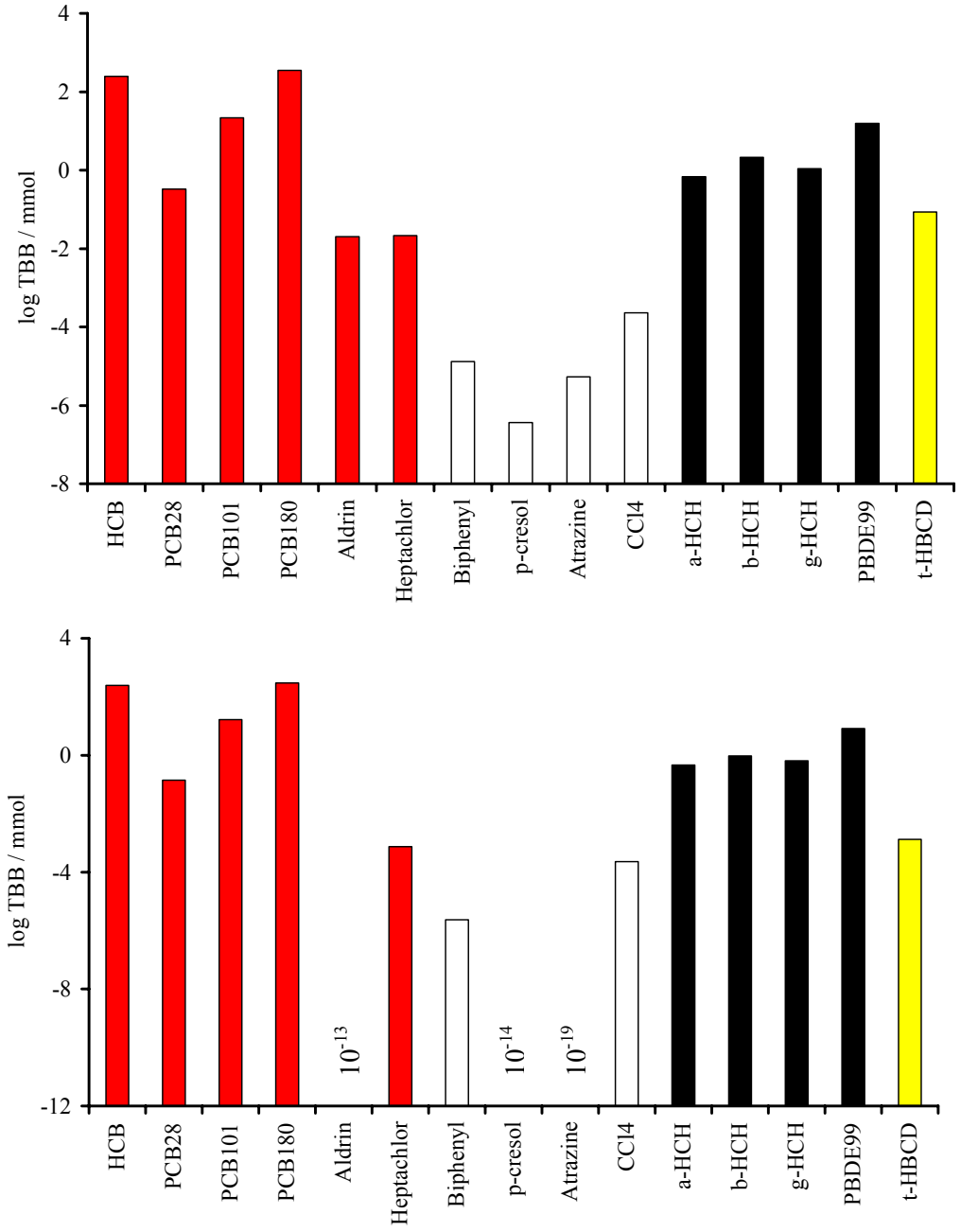


Figure 3.20. Total body burdens (TBB; mmol) estimated in "source" (upper figure) and "remote" (lower figure) regional environments based on an assumed unit emission rate (1 $kg \cdot h^{-1}$). Red bars – POPs; White bars – non-POPs; Black bars – Candi-POPs.

3.5.1.4 GloboPOP: Arctic Contamination Potential ($eACP_{10}$)

Wania (2003b; 2006) introduced the Arctic Contamination Potential (ACP) as a metric of long range transport efficiency, a model-calculated value which is independent of emission rate. There are two different formulations of the ACP metric, mACP and eACP, both of which are calculated using the GloboPOP model (Wania and Mackay 1995). mACP represents the relative enrichment of contaminants in the northernmost climatic zone (N-polar, see Figure 3.21) and is calculated as

$$mACP = \frac{M_{T1} - M_{A1}}{M_{TG}}(100\%) \tag{3.3}$$

where M_{T1} is the total mass of contaminant in the N-polar zone (all compartments), M_{A1} is the total mass of contaminant in the atmosphere of the N-polar zone and M_{TG} is the total mass of contaminant in the global environment (all compartments). eACP represents the potential for absolute contamination of the northernmost climatic zone and is calculated as

$$eACP = \frac{M_{T1} - M_{A1}}{E_{TG}}(100\%) \tag{3.4}$$

where E_{TG} is the total mass of contaminant emitted into global environment over the model simulation period (default is 10 years). mACP and eACP tend to be similar for perfectly persistent compounds (Wania 2006) but can vary substantially for degradable substances such as those considered here. We use eACP here because it is a target-oriented indicator of long range transport (LRT) potential that has been compared to other model-derived indicators of LRT potential in the past (Fenner et al. 2005). Both ACPs are sensitive to the assumed emission mode of entry (i.e. compartment that chemical is emitted into) with lower values expected as the fraction of contaminant released to the atmosphere is reduced (Wania 2003b). The geographical distribution of emissions can also influence the results. The typical approach applied in the past for ranking exercises was to assume that emissions occur 100% to the atmosphere for all chemicals, all of which are distributed according to the proportion of the global population living in each climatic zone. To be comparable with previous studies and also consistent with the publicly available version of the model, the default distribution of emissions was assumed here (based on population in each zone). The length of the simulation was also left at 10 years, hence the metric we present is $eACP_{10}$ (note that eACP values increase as a function of time (Gouin and Wania 2007) but this is not relevant for this benchmarking exercise).

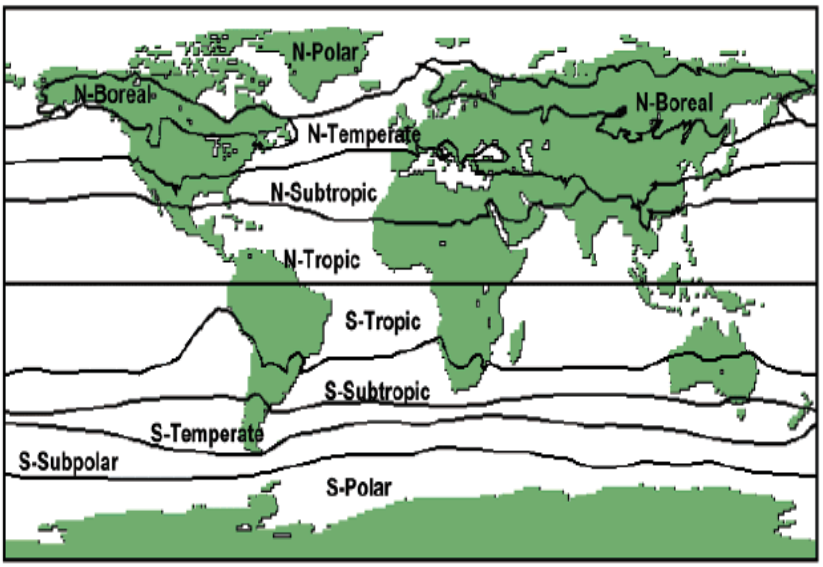


Figure 3.21. Zonal subdivision of the GloboPOP model, based on latitudinal climatic bands.

Note that GloboPOP differs from some of the other model tools applied in this assessment with regards to the treatment of atmospheric degradation. Here, k_{OH} is used as model input and the degradation half-life is calculated as a function of latitudinally-specific OH radical concentration and temperature.

We first conducted $eACP_{10}$ simulations using physical-chemical property values for α-, β-, and γ-HBCD as well as values characterizing the technical mixture (t-HBCD). The results of these preliminary simulations are presented in the Appendix (Figure 9.1). Since no substantial differences in $eACP_{10}$ were observed, results presented below are for t-HBCD only. As with the other benchmarking exercises, a series of simulations were conducted to generate results across the range of degradation half-lives presented in Table 2.5 (lower bound, median and upper bound estimates).

The calculated $eACP_{10}$ for the chemicals included in this benchmarking exercise are presented in Figure 3.22 as a function of the reactivity with OH radicals in the atmosphere (k_{OH}, $cm^3 \cdot molecule^{-1} \cdot s^{-1}$). Note that this figure should not be interpreted to mean that OH radical reactivity is the sole determinant of long range transport (LRT) potential. Atmospheric deposition and air-surface exchange are also important processes influencing environmental fate, hence degradation half-lives in other media also affect model output. As a general trend however, substances which are more recalcitrant to OH radical reactions have longer degradation half-lives in other media. Hence k_{OH} can broadly serve as a proxy for overall persistence. The k_{OH} and $eACP_{10}$ values are also presented in Table 3.13.

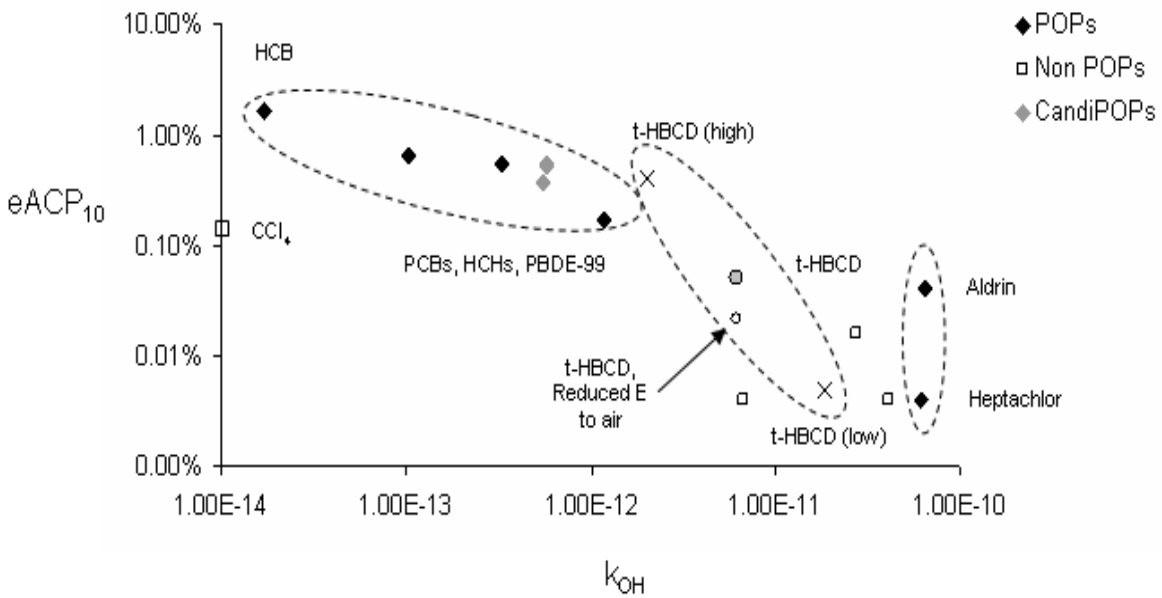


Figure 3.22. Arctic Contamination Potential after 10 years of constant emissions ($eACP_{10}$) of POP, non-POP, Candi-POPs and t-HBCD (under different assumptions) as a function of reactivity with OH radicals (k_{OH}, $cm^3 \cdot molecule^{-1} \cdot s^{-1}$). Unless noted otherwise, emissions assumed to be 100% to air and reactions with OH radicals assumed to occur only in the gas phase. Note: The $eACP_{10}$ of CCl_4 is shown on the y-axis because it was assumed to have a negligible k_{OH}.

The $eACP_{10}$ values of the POP compounds range from 0.004–1.6%. Aldrin and heptachlor had the lowest $eACP_{10}$ values (0.04 and 0.004% respectively), substantially lower than the other POPs. This model output is related to the elevated reactivity towards OH radicals and lower half-life values in other environmental media of Aldrin and particularly Heptachlor in comparison to the other POPs. For example, according to AOPWIN v1.92 (US EPA 2009), Heptachlor and Aldrin are roughly 1 to 3 orders of magnitude more susceptible to OH radical attack compared to the other POPs. HCB had the highest $eACP_{10}$ (1.61%) while the PCBs ranged from 0.17% (PCB28) to 0.65% (PCB180).

The $eACP_{10}$ values for the non-POPs included in the benchmarking exercise ranged from 0.004–0.18%. The lowest $eACP_{10}$ values were calculated for biphenyl and p-cresol (0.004%) followed by atrazine (0.016%). These three compounds are characterized by low degradation half-lives in all bulk environmental compartments. In contrast, CCl_4 had a relatively high $eACP_{10}$ due to its persistence in the atmosphere and longer half-lives in water, soil and sediments compared to the other non-POPs.

The $eACP_{10}$ values for the candidate POPs included in the benchmarking exercise ranged from 0.37–0.55%. $eACP_{10}$ values of the HCH isomers were similar (0.50–0.55%) while a lower value was calculated for PBDE99 (0.37%). These calculated $eACP_{10}$ values fall

within the same range as the PCBs indicating that these candidate POPs are broadly similar to other POPs with respect to long range transport potential.

$eACP_{10}$ output for t-HBCD is presented for four different scenarios i) lower bound HL estimates (i.e. lowest degradation half-lives in all compartments ii) t-HBCD (median half-life values, all compartments) iii) upper bound HL estimates and iv) Reduced emissions to Air (median half-life values, 30% of emissions to air, 70% to surface water). Assuming 100% emissions to air i.e. Scenarios i) – iii), the $eACP_{10}$ of t-HBCD ranges from 0.005% (lower bound HLs) to 0.4% (upper bound HLs); the $eACP_{10}$ assuming the default scenario values is 0.05%. With reduced emissions to air (and median HLs), the $eACP_{10}$ is reduced to 0.02% demonstrating the influence of mode of entry on this metric of long range transport potential. The fact that the range of estimated $eACP_{10}$ for t-HBCD spans two-orders of magnitude indicates the sensitivity of the model output to assumptions regarding degradation half-lives. Interestingly, $eACP_{10}$ is least sensitive to the assumed half-life in freshwater sediments, largely due to the fact that this compartment is only a small fraction of the global environment. Assumed half-lives in soil and water (includes ocean surface water) have a greater influence since these compartments have comparatively large volumes. Therefore, if experimental studies demonstrate the degradation half-lives in these compartments are substantially longer than the maximum values assumed here, it is no longer reasonable to assume that the maximum $eACP_{10}$ calculated here is representative.

The overall interpretation of these results is complicated by the range of $eACP_{10}$ values exhibited by the POP compounds. Assuming median parameter values, the $eACP_{10}$ of t-HBCD is lower than PCBs, HCB and the other candidate POPs but higher than Aldrin and Heptachlor. In comparison to t-HBCD (median k_{OH} value), Heptachlor and Aldrin are roughly 1 order of magnitude more susceptible to OH radical attack. The $eACP_{10}$ of Aldrin is roughly the same as t-HBCD however because it is more persistent in all other compartments. Heptachlor, on the other hand, has lower $eACP_{10}$ primarily because it is degraded in other compartments with similar HL as t-HBCD (median values). Partitioning properties (i.e. distribution) also influence overall persistence and $eACP_{10}$. For example, Aldrin and Heptachlor have log K_{AW} values 2 orders of magnitude higher. Strictly speaking, this benchmarking exercise could be interpreted to mean that t-HBCD has a long range transport potential similar to other POPs. However, the long range transport potential of Aldrin and Heptachlor is clearly different from other POPs (and Candidate POPs) included in the benchmarking exercise. For example, the results imply that the presence of Aldrin and Heptaclor in remote regions necessarily requires higher absolute emissions in comparison to other POPs in order for a similar mass of chemical to reach this environment. Under default assumptions regarding degradation half-life, the same argument could be made for t-HBCD based on its $eACP_{10}$. It is also worth noting that the $eACP_{10}$ of all non-POPs falls within the overall range of POPs as well (0.004 – 1.61%). In conclusion, while these simulations provide some insight into the relative long range transport potential of t-HBCD, absolute emissions and mode of entry of these emissions must also be considered.

Two additional $eACP_{10}$ simulations were conducted to further elucidate the fate and transport of t-HBCD in the global environment, one assuming perfect persistence in all compartments (i.e. negligible degradation) and one assuming no atmospheric degradation (default degradation half-lives in all other compartments). The model output is also included in Table 3.13. The $eACP_{10}$ assuming perfect persistence is 0.93%, approximately 20 times higher than the default value. In this simulation, the main processes limiting long range transport are atmospheric deposition and other sink processes (e.g. sediment burial, transport to deep ocean via particle settling). The $eACP_{10}$ value assuming no degradation in the atmosphere (and default half-lives in all other compartments) is 0.11%. This model output confirms the fact the i) degradation in other compartments has an influence on the long range transport potential of t-HBCD and ii) air-surface exchange processes (e.g. wet/dry deposition) transfer significant quantities of t-HBCD from the atmosphere to other compartments in source regions.

Table 3.13. Selected compounds, OH radical reactivity and $eACP_{10}$ calculated using GloboPOP model.

Status	Chemical	Scenario	k_{OH}	$eACP_{10}$
POP	HCB	Default HLs	1.69e-14	1.61%
POP	PCB28	Default HLs	1.19e-12	0.17%
POP	PCB101	Default HLs	3.35e-13	0.53%
POP	PCB180	Default HLs	1.05e-13	0.65%
POP	Aldrin	Default HLs	6.46e-11	0.04%
POP	Heptachlor	Default HLs	6.11e-11	0.004%
Non-POP	Biphenyl	Default HLs	6.77e-12	0.004%
Non-POP	*p*-cresol	Default HLs	4.11e-11	0.004%
Non-POP	Atrazine	Default HLs	2.73e-11	0.016%
Non-POP	CCl_4	Default HLs	-	0.18%
Candi-POP	α-HCH	Default HLs	5.73e-13	0.55%
Candi-POP	β-HCH	Default HLs	5.73e-13	0.53%
Candi-POP	γ-HCH	Default HLs	5.73e-13	0.50%
Candi-POP	PBDE99	Default HLs	5.50e-13	0.37%
-	t-HBCD (low)	Lower bound HLs	1.84e-11	0.005%
-	t-HBCD (median)	Default HLs	6.12e-12	0.05%
-	t-HBCD (high)	Higher bound HLs	2.04e-12	0.40%
-	t-HBCD (median)	Reduced E to Air	6.12e-12	0.02%
-	t-HBCD (median)	Perfect Persistence	-	0.93%
-	t-HBCD (median)	No Atmospheric Deg	-	0.11%

3.5.1.5 AC-BAP

The chemical screening methodology for POPs introduced by Brown and Wania (2008) will be used for chemical comparisons in this section. This method aims to identify the domain of physical-chemical properties that result in LRT to the physical Arctic environment and accumulation in the Arctic human food chain. For example, properties of HBCD will be compared with the chemical space maps for Arctic Contamination-Bioaccumulation Potential (AC-BAP) without the actual application of the combined Globo-POP and ACC-Human models (Czub et al. 2008).

The Arctic contamination and bioaccumulation potential (AC-BAP) provides a metric relating global chemical emission to the body burden and thus the internal exposure of a human living in the remote Arctic (Czub et al. 2008). The AC-BAP combines the zonally averaged global transport model Globo-POP, which has been used in previous ACP calculations (Wania 2003b; Wania 2006), with the human food-chain bioaccumulation model ACC-HUMAN (Czub and McLachlan 2004), which has been used to estimate bioaccumulation in Inuit. The AC-BAP is defined as the ratio of human body burden m_H (g·person^{-1}) and the cumulative emissions of a chemical to the global environment e_{TG} (g):

$$AC-BAP=\frac{m_H}{e_{TG}} \tag{3.3}$$

AC-BAP has a unit of person^{-1} and represents the fraction of the cumulative global emissions stored in a single Arctic resident. A high value stands for a high potential of a chemical to reach the remote Arctic and to bioaccumulate in humans based on the assumed food chain and evaluative environments.

In an effort to understand the influence of partitioning on the AC-BAP, Czub et al. (2008) calculated AC-BAP for a range of hypothetical chemicals that were assumed to be perfectly persistent and to be emitted at a constant rate into the global atmosphere for a period of 10 or 70 years[1]. The resulting AC-BAP-values were plotted as a function of the octanol-air and air-water partition coefficient, yielding a chemical space diagram which identifies partitioning property combinations that favour accumulation of a globally emitted, persistent chemical in Arctic residents (Czub et al. 2008).

The partitioning properties of HBCD (FAVs) in Table 2.2 were compared with the "chemical space" diagrams for AC-BAP developed by Czub et al. (2008). Figure 3.23 shows the chemical space diagram for AC-BAP developed by Czub et al. (2008) and the estimated location of HBCD. Assuming complete persistence of HBCD in the environment and in food webs ("persistent HBCD") and using the results of the AC-BAP model predictions for the chemical space the following inferences can be made. "Persistent HBCD" is expected to have approximately 20-30% of the maximum AC-BAP

[1] The zonal distribution of global emissions is assumed to follow the latitudinal distribution of the global population.

estimate assuming 10 years of emissions for all chemicals. "Persistent HBCD" is expected to have approximately 15-25% of the maximum AC-BAP estimate assuming 70 years of emissions for all chemicals. In contrast to the study by Brown and Wania (2008) relying on EPI Suite™ predictions (U.S. EPA 2009), the partitioning properties recommended here, place "persistent HBCD" into the part of the chemical space with elevated AC-BAP (>10 % of the maximum). This suggests that HBCD does have partitioning properties that favour transport to the Arctic environment and bioaccumulation in the Arctic human food chain.

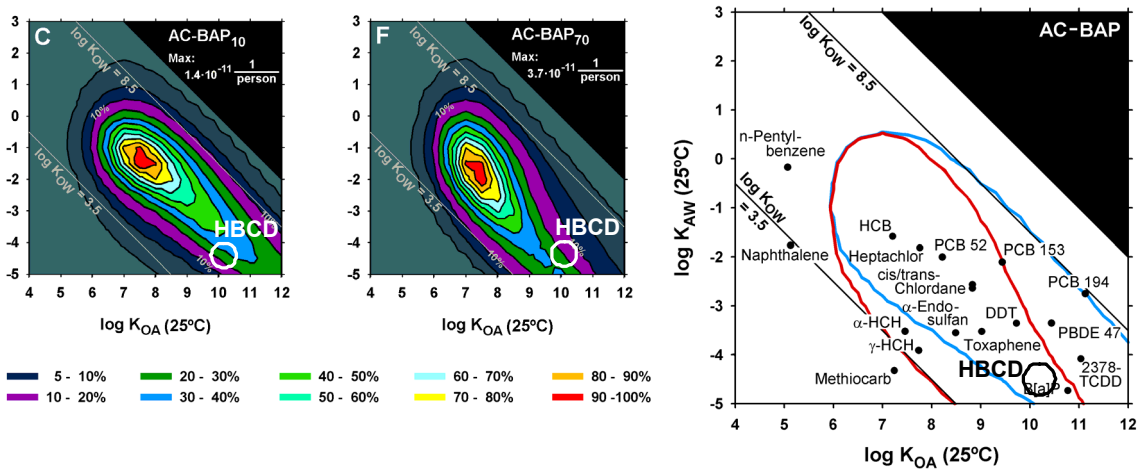


Figure 3.23. Chemical space diagrams identifying partitioning properties favoring accumulation of a globally emitted, persistent chemical in an Inuit woman as quantified with the Arctic Contamination and Bioaccumulation Potential (AC-BAP) (Figures from Czub et al., 2008). The circle in the lower right of each panel represents the approximate partitioning properties of HBCD (mixture). In the panel to the right, the blue line encircles areas of elevated AC-BAP_{10} (10 years of emissions) whereas the red line represents an elevated AC-BAP_{70} (70 years of emissions). HBCD, when persistent, has an elevated ACP (> 10 % of the maximum). The partitioning properties of other organic chemicals are indicated for comparison.

The AC-BAP of "persistent HBCD" is somewhat higher than that of persistent PBDE 47 and persistent 2,3,7,8-tetrachlorodibenzo-p-dioxin. This suggests that if degradation and biotranformation of HBCD are similar to these two chemicals and emissions patterns are similar (mode of entry, quantity, and spatial-temporal aspects) than exposure potential in the Arctic environment may also be comparable.

It should be mentioned that both 1,3,5,7,9,11-hexabromocyclododecane (CAS 25637-99-4) and 1,2,5,6,9,10-hexabromocyclododecane (CAS 3194-55-6) had been ear-marked as potential Arctic contaminants in the study by Brown and Wania (see Table 1 in Brown and Wania, 2008). In both cases this was a result of a so-called high POP Score, indicating high structural resemblance with a selection of known POPs[2], rather than partitioning properties that would yield an elevated AC-BAP, if HBCD were perfectly

[2] Structural attributes of known POPs were a high degree of halogenation and internal connectivity, and an intermediate molecular size.

persistent. Brown and Wania (2008) used partitioning properties supplied by EPI Suite™ predictions (U.S. EPA 2009), so a revisiting of this analysis using the partitioning properties recommended here is appropriate.

3.5.2. Model Comparisons with Monitoring Data

"Realistic" simulations of HBCD using emissions estimates allow for comparisons between model predictions and available monitoring data. These comparisons can be used to corroborate emission estimates and selected properties for HBCD.

3.5.2.1 RAIDAR and Real World Regional Scale Monitoring Data

The RAIDAR model was used to predict concentrations in general source regions of the European environment (e.g. regional areas in Sweden) and these estimates were compared to available monitoring data representative of regional non-point sources (Table 3.4). This comparison seeks to corroborate actual emission rate estimates and uncertainties in persistence estimates (e.g. degradation half-lives in water). This is done by considering various assumptions for emissions in a regional area (100,000 km^2) and different mode-of-entry scenarios and comparing model predictions with the monitoring data. The various estimates for actual emissions can be used to scale the results based on the unit emission rate to the environment (E_U) of 1 $kg \cdot h^{-1}$. For example, if the actual estimated emission rate (E_A) is 0.5 $kg \cdot h^{-1}$ the unit output from the model is scaled lower by a factor of 2. Therefore if the unit concentration in a marine mammal, C_U ($ng \cdot g^{-1}$ lw) is 100, based on the actual emission rate estimate the corresponding actual concentration predicted by the model C_A ($ng \cdot g^{-1}$ lw) is 50. This approach exploits the linear calculations of the model.

It is emphasized that the model predicts steady state chemical concentrations in the environment. The application of a steady state model for comparisons with monitoring data is not appropriate if chemical concentrations in the environment have not approached steady state. Whether or not a chemical can achieve steady state (or nearness to steady state) depends on the relationship between the rate at which a chemical can respond to changes in emissions (i.e. response time; see section 3.5.3.1) and the extent to which emissions have changed with time. There is insufficient evidence to determine if HBCD emissions have remained constant in recent years; temporally-resolved emission data are not available and time-trend data, while showing a slight long-term increase over 20-30 years (Section 3.4), are inconclusive for recent years. The less persistent a chemical is the less important it is that emissions remain constant over time when undertaking steady state modelling because the time response of concentrations to changing emissions can be relatively fast. Levels of HBCD in the environment appear to be near steady state as a result of long term production and use in regional environments in Europe. The apparent pseudo-steady state conditions are further supported by predicted response times and the time required to steady state (see Section 3.5.3.1 below). This evidence supports the applicability of a steady state model for comparisons to monitoring data.

For model comparisons with monitoring data the general model structure must be reasonably representative of the regional environments in which the monitoring data were

obtained. This approach may work well in spite of changing emissions as long as the emission estimates and monitoring data used for comparison are from the same general time period. Similar comparisons of RAIDAR predictions for PBDE-99 with monitoring data have shown that this relatively simple approach can reasonably reproduce environmental observations (Cowan-Ellsberry et al. 2009). For simplicity in this analysis, and due to data limitations, only t-HBCD predictions will be considered for comparisons with ΣHBCD monitoring data.

The degree of fit between model predictions and observations can be quantified providing an indication of Model Bias ($MB = C_A / C_O$). If the predicted concentration C_A is greater than the observed concentration C_O then MB is greater than 1. If the predicted concentration C_A is lower than the observed concentration C_O then MB is less than 1. Model Bias can be calculated as the average MB for different compartments and provides insights into model fit with the observed data. Assuming the structure of the model is representative of the environment and the input parameters are reasonable and that the system is near steady state, the estimates for the actual emission rate, mode-of-entry, and half-lives can be explored by minimizing MB.

An initial estimate for the actual emission rate was determined as follows. According to the RAR ~9.942 tonnes of HBCD are released to the European Union (EU) environment annually (See Table 3.1) (EC 2008). The area of the EU member states is ~4,500,000 km^2. The EU region is approximately 45 times greater than the RAIDAR regional environment. The annual unit emission rate in RAIDAR is 1 $kg \cdot h^{-1}$ or 8.76 $t \cdot yr^{-1}$. The default RAIDAR unit emission rate is about 40 times greater than the actual estimated emission rate after adjusting for differences in scale (1/45). The unit emission rate in RAIDAR was therefore scaled as 1/40 to approximate the actual emissions to a representative 100,000 km^2 regional scale European environment. A confidence factor of 5 was assigned to the actual emissions estimate. According to the RAR, HBCD is released to the environment as follows: 6% to air, 72% to wastewater and 22% to surface water. Therefore, the initial assumption was that approximately 95% of the emissions are to water and 5% are to air. The available regional scale monitoring data (Table 3.4) and food basket studies from Norway (Knutsen et al. 2008) and Sweden were selected to represent European regions for model comparisons.

Figure 3.24 illustrates the initial comparison of RAIDAR predictions and monitoring data based on the RAR emissions and mode-of-entry assumptions and the median half-life values in the environment for HBCD (Table 2.5). There is reasonable agreement between model predictions and observed data for most compartments, particularly considering the uncertainty in the monitoring and modelling estimates. Many of the estimates for biota are within a factor of 3 and the overall MB for biota is 0.97 (underestimating by ~3%). Average MB estimates can cancel each other out, so they must be interpreted cautiously. There is a tendency toward a systemic under prediction for certain compartments. Measured regional scale abiotic concentrations are particularly lacking, except for air. The monitoring data for air reflect available regional scale measurements in Sweden and the model underestimates these measurements according to the current scenario. Data for sediment is for a regional non-point source (upstream estimates). There are no

representative regional monitoring data for soil. All soil measurements have been near production facilities. Estimates for RAIDAR regional scale water and soil concentrations were compared with estimates from BETR-World (see below).

The data for aquatic biota are reasonably good; however, predicted concentrations in food sources originating in the terrestrial environment are too low. The underestimation of measured levels in air and terrestrial organisms (eating mostly crop materials) suggests that emissions to air may be higher than presently estimated. This is further supported by monitoring data for dust in indoor environments and regional air concentrations from other global regions with lower use patterns than Europe. Assuming the model is representative of the conditions, this slight underestimation suggests that either the half-life estimates are too short, the emissions estimates are too low, the mode-of-entry proportions are different, or some combination of these three factors.

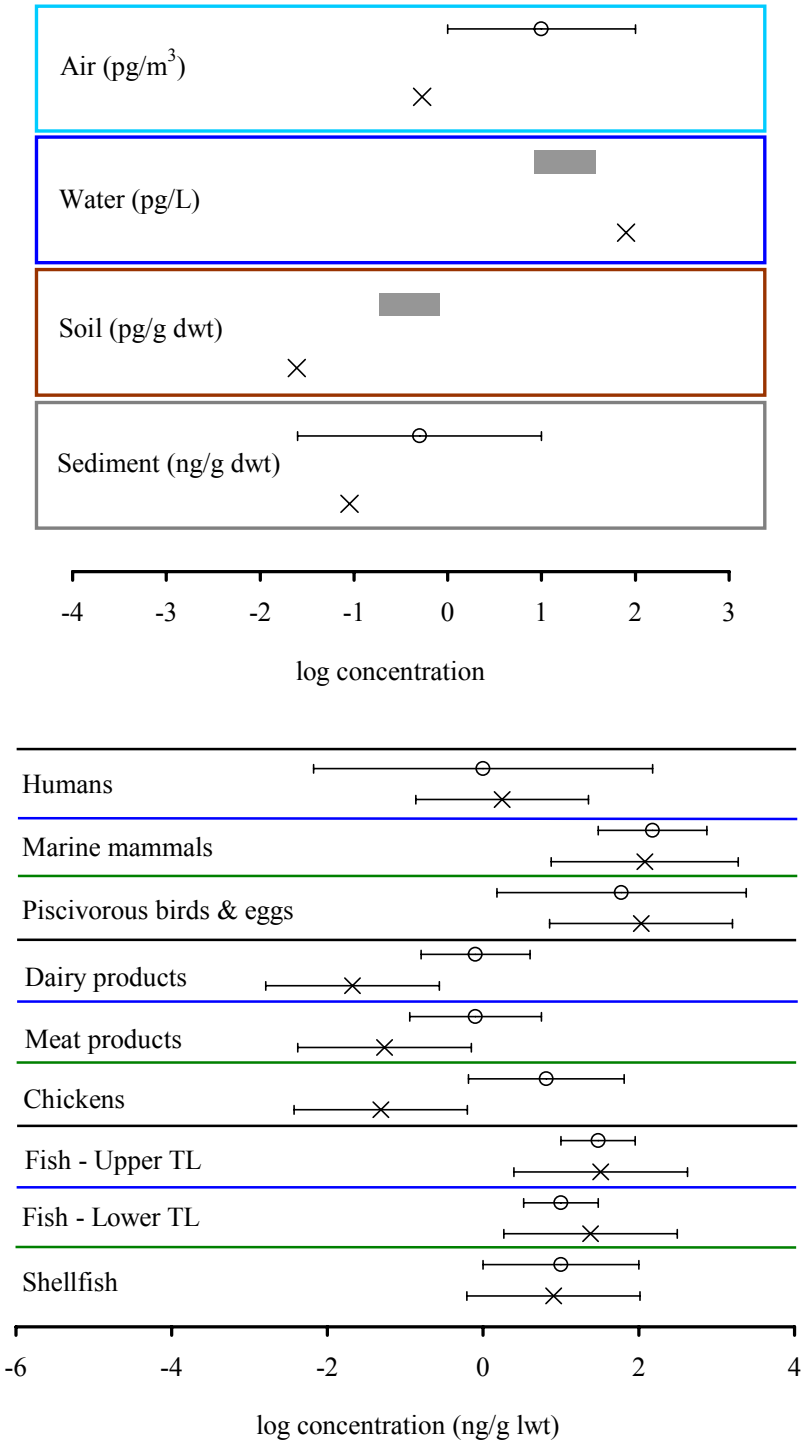


Figure 3.24. RAIDAR model predictions (×) and monitoring data (o) for HBCD based on an assumed actual emission rate of 9.942 $t \cdot yr^{-1}$ (EU region) x 1/40 (regional scale and unit emission differences), 95% of which is released to water and 5% to air and using median environmental half-lives. The median value for birds is from muscle and the range reflects data for eggs. The gray bars are BETR-World model estimates for the European region.

RAIDAR does not consider wastewater treatment. It is estimated that approximately 20% of emissions to treated wastewater are prevented from entering the natural environment (EC 2008). This would suggest that the proportional mode-of-entry for total emissions is therefore higher to air than 5% and total emissions may be lower than the initial scaled estimate.

As an alternative to the initial assumption, the proportion of emissions to air were increased from 5% to 30% and the emissions to water were decreased accordingly (70%). Figure 3.25 illustrates the results of these adjustments showing better agreement in the air and terrestrial organisms with some minimal "loss" in predictive accuracy for other compartments. Model Bias for the biotic compartments is lower under this scenario at 0.79 (average underprediction of 21%).

There are a number of different scenarios that could be explored, in particular with regards to the uncertain degradation half-lives. Another alternative scenario considered was to use the original emissions scaling factor, the 30:70 ratio for air : water mode-of-entry and the upper bound estimates for environmental degradation half-lives (Table 2.5). Figure 3.26 illustrates the results of these assumptions in comparison to the source regional scale monitoring data. There is still reasonable agreement in many compartments; however, there is now a greater MB in predictions for biota with an overall average MB for biological compartments of 2.23.

Figure 3.26 also includes model predictions for humans, marine mammals and birds assuming the biotransformation half-lives for these organisms are equivalent to the estimates for fish on a body weight basis. When this conservative assumption is applied the model predictions are much greater than the available monitoring data. This suggests that the median biotransformation half-lives selected in the present study are reasonable and that HBCD (or at least some of the diastereomers) are biotransformed at faster rates in birds and mammals than in fish.

In summary, the steady state model predictions are in good agreement with monitoring data for a wide range of media. Levels of HBCD in the environment appear to be near steady state in all environmental media as a result of long term production and use in regional environments in Europe. Steady state model predictions for persistent chemicals have not provided as good predictions for slow responding compartments such as soils and sediments based on comparisons with monitoring data for certain PAHs and hexachlorobenzene (Armitage et al. 2007). This evidence suggests that HBCD is not as persistent as the PAHs and hexachlorobenzene.

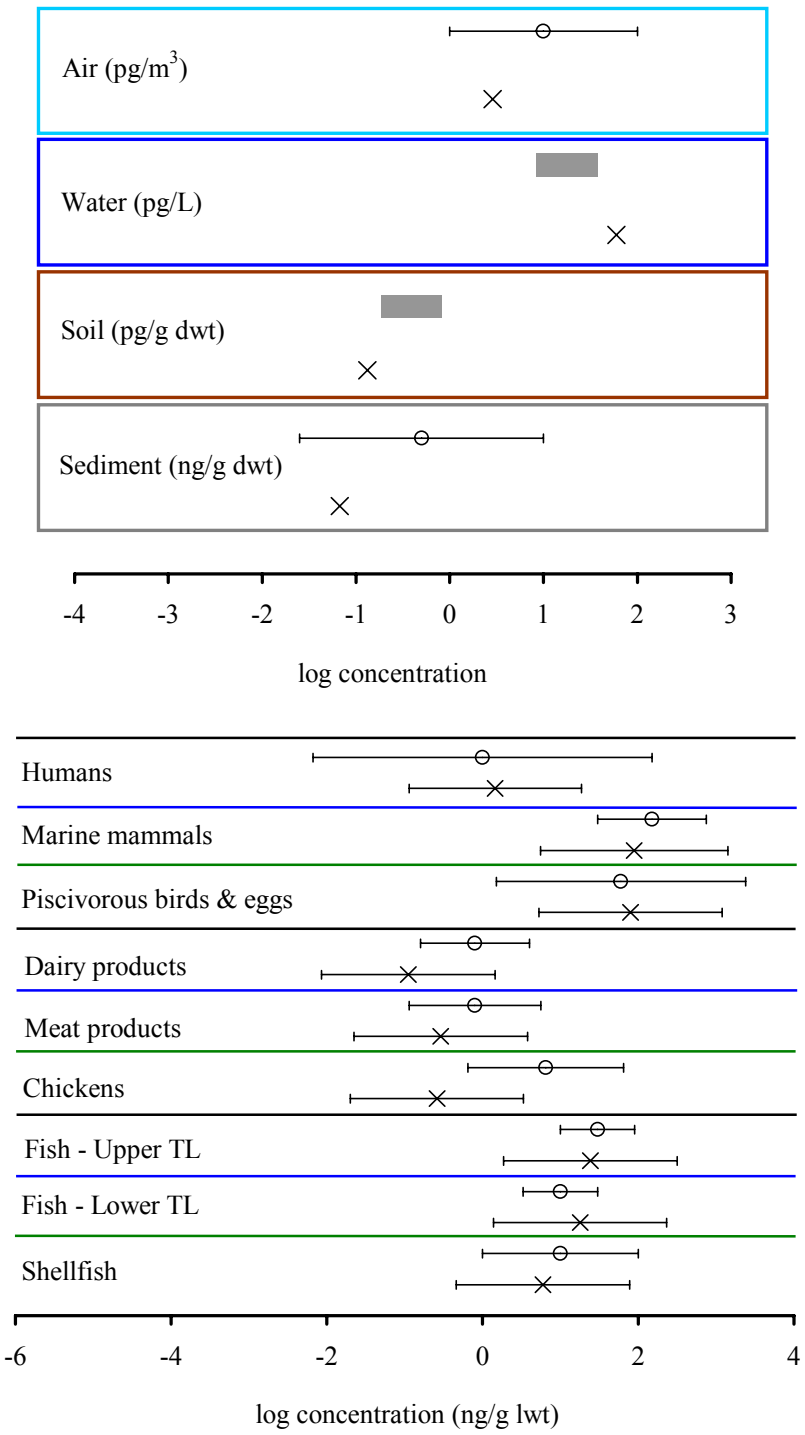


Figure 3.25. RAIDAR model predictions (×) and monitoring data (o) for HBCD based on an assumed actual emission rate of 9.942 $t \cdot yr^{-1}$ (EU region) x 1/40 (regional scale and unit emission differences), 70% of which is released to water and 30% to air and median environmental half-lives. The median value for birds is from muscle and the range reflects data for eggs. The gray bars are BETR-World model estimates for the European region.

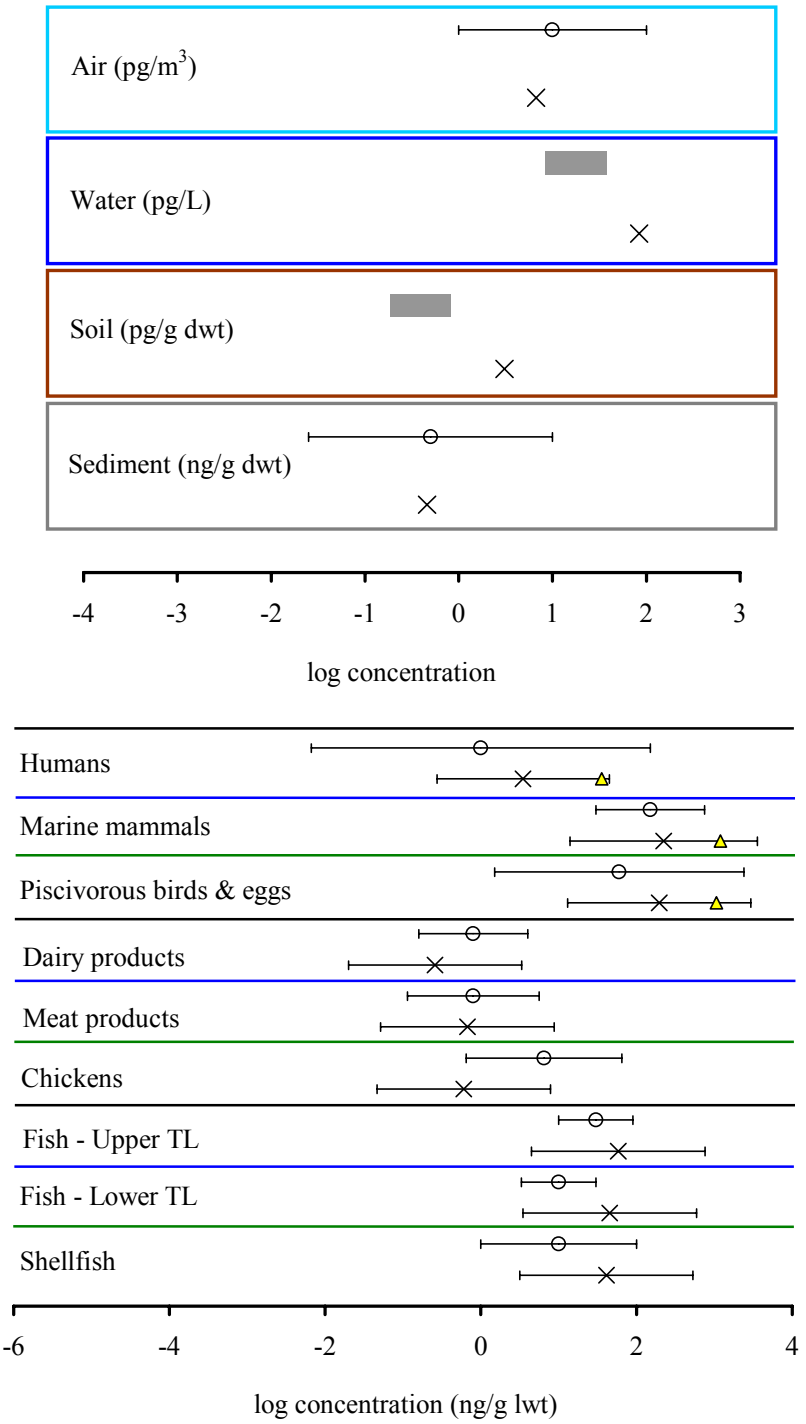


Figure 3.26. RAIDAR model predictions (×) and monitoring data (o) for HBCD based on an assumed actual emission rate of 9.942 $t \cdot yr^{-1}$ (EU region) x 1/40 (regional scale and unit emission differences), 70% of which is released to water and 30% to air and 95th percentile values for environmental half-lives. The median value for birds is from muscle and the range reflects data for eggs. The gray bars are BETR-World model estimates for the European region. The yellow triangles are estimates assuming metabolic biotransformation rates in birds and mammals are equal to the estimate for fish.

The predicted concentrations in humans are somewhat higher than the median of the measured value; however, many uncertainties affect the comparisons of human predictions with monitoring data for humans. RAIDAR calculates concentrations for adult male humans; whereas the available monitoring data are predominantly for nursing mothers. In general, reproductive and lactational losses are recognized as lowering lipid normalized concentrations of hydrophobic chemicals in reproductive females compared to males. Thus, predictions for males in RAIDAR may over predict monitoring data for females. Humans are included in this analysis because the general human diet is composed of a range of items including meat and dairy products, fish, vegetables and fruits which represent a range of possible sources of exposure. In addition, due to the limited monitoring data presently available the human data provide a valuable source of reference. On the other hand, due to differences in diet (omnivorous, vegetarian), and the import of foods from various regions, dietary levels can vary widely for the sample human population. This generally results in a high degree of variance in human data. Finally, the model does not consider potential near-field exposures to humans such as the indoor environment.

The model-measurement comparison can be affected by detection limits. Typically, when summary statistics are calculated from monitoring data with a significant number of non-detects (e.g. soils, water, humans), those non-detects are often repleaced with 0.5 the detection limit value or some other correction. This results in uncertain values for the lower bound estimates (because they cannot be detected) and a general bias towards overestimated medians. Datasets with few non-detects, such as for marine mammals, upper trophic level fish, piscivorous birds and their eggs, are considered to be more accurate compared to samples with frequent non-detects.

There are a number of different scenarios that can be considered; however, since there are uncertainties in the actual emissions, degradation half-lives, physical-cemical properties, mode-of-entry and the model, it is difficult to ascertain all parameters at the same time. In general, the model – monitoring data comparison suggests that current parameterization estimates are reasonable. If current emission and scaling estimates are accurate, than this would suggest that a ratio of 30:70 (air:water) for mode-of-entry and half-lives that are between the median and upper bound estimates are appropriate starting points for subsequent modelling. It must be recognized that there is an inverse relationship between emissions estimates and degradation half-lives.

3.5.2.2 BETR-World

The BETR-World model is a global scale, geographically explicit model that allows the user to both assess the transport potential of chemicals from source regions and create a mass-balance of these chemicals through the definition of historical or estimated regional loadings. The model consists of 25 predominantly terrestrial or oceanic regions that are linked through quantified atmospheric, fluvial and oceanic advective pathways. Each region consists of a maximum of seven environmental media, upper atmosphere, lower atmosphere, freshwater, freshwater sediments, soil, vegetation and coastal and/or oceanic water. The terrestrial regions are delineated to incorporate latitudinal climatic changes as

well as political boundaries. This marriage of environmental and political consideration effectively captures both the conditions affecting long range atmospheric transport of chemicals as well as providing a framework to support regulatory decisions (Toose et al. 2004). The model accommodates both dynamic and steady state conditions and the efficiency of transport of chemical emitted from each region to a target region, such as the Arctic, can be determined (Reid and Mackay 2008).

For the BETR-World model calculations emission estimates (ca. 2001) were derived from various sources; but mainly those described in the TemaNord (2008) document (Table 3.1) and those sourced from BSEF as cited from the Arctic Monitoring and Assessment Program (AMAP 2005) (see Table 9.9 in the Appendix). Based on Tables 3.1 and 9.7 emissions were scaled for the proportion releases per unit demand in Europe. Australia has regulated the use of HBCD and the annual use for 2001 was about 40 metric tonnes (NICNAS Australian Government Department of Health and Ageing 2005). Emissions were assigned at low levels to regions for which there were no direct data as monitoring data show that HBCD is found in locations around the world including South Africa (Polder et al. 2008b). The assumed emissions for the BETR-World regions based on these data are summarized in the Appendix (Table 9.9). For mode-of-entry it was assumed that emissions were 70% to the lower air compartment and 30% to freshwater for every region. The FAVs for t-HBCD were used (Table 2.2). The model was run twice; once for the "default" environmental half-lives and a second time for "upper 95th percentile" environmental half-lives as described in Section 2 (Table 2.5). Half-lives in vegetation and coastal waters were assumed equal to the half-lives for water.

Figure 3.27 shows that the model predicts European concentrations, except the lone freshwater measurement, to within a factor of 4.2. The lone "Arctic" measurement from freshwater sediments on Bear Island, NO (3.87 ng·g^{-1}) is underpredicted by 5 and 3 orders of magnitude for the median (4.8×10^{-5} ng·g^{-1}) and the upper bound HL (1.1×10^{-3} ng·g^{-1}) simulations. The literature indicates that this monitoring location is a relative "hotspot" for contaminants. This remote lake is located in a very barren region; however, it is highly productive due to the inputs of guano from seabirds (Christensen et al. 2004). In addition to the "biotransport" of contaminants to the lake via migratory seabirds, it is a location with unusually high precipitation for the high arctic due to the topography of the region, creating yet another unusual vector by which contamination of this remote lake can occur. At the scale of the BETR-World model, where the smallest regions are still several hundred thousand square kilometers, hotspots such as these cannot be adequately captured. The other very low value shown on Figure 3.27 is from monitoring of the Detroit River suspended sediments; also a highly industrial and urbanized area that is not well predicted by this large global-scale low-resolution model. The BETR-World model predicts background concentrations in the global environment and is not of sufficiently high spatial resolution to predict concentration "hot-spots". Predictions using the upper-boundary half-lives underpredict the average concentrations by 3x in air and 10x in freshwater sediments. The predictions approach lower values of measured sediment concentrations in Tokyo Bay (0.056 to1.3 ng·g^{-1}in the middle to outer bay, respectively (Minh et al. 2007)).

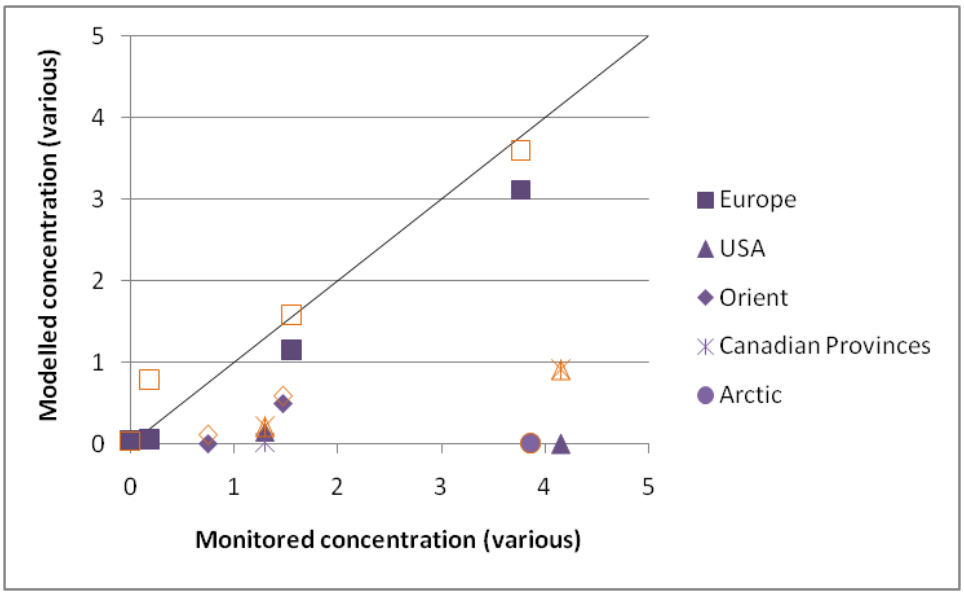


Figure 3.27. Comparison of BETR-World model results and with available monitoring data. Solid points compare monitoring data to results using the default half-lives physical-chemical parameters and open points compare to results using the 95th percentile half-lives.

Figures 3.28 and 3.29 show the air and freshwater sediment concentrations predicted by the model based on the upper bound half-lives. The model aims to predict average concentrations over large regions. Figure 3.28 shows that lower air (0 to 1 km) concentrations are less than detectable limits in the southern hemisphere, over the North American Pacific Ocean and in the middle Atlantic Ocean. Other locations, notably the Arctic and the Orient, are predicted to be above detectable levels at about 0.5 pg·m^{-3}. Levels reported in the air in China are between 0.08 to 3 pg·m^{-3} (Yu et al, 2008). Levels in the United States are predicted to be between 0.5 and 1 pg·m^{-3} which is supported by the data of remote monitoring stations there (0.6 to 8 pg·m^{-3}, Hoh and Hites 2005).

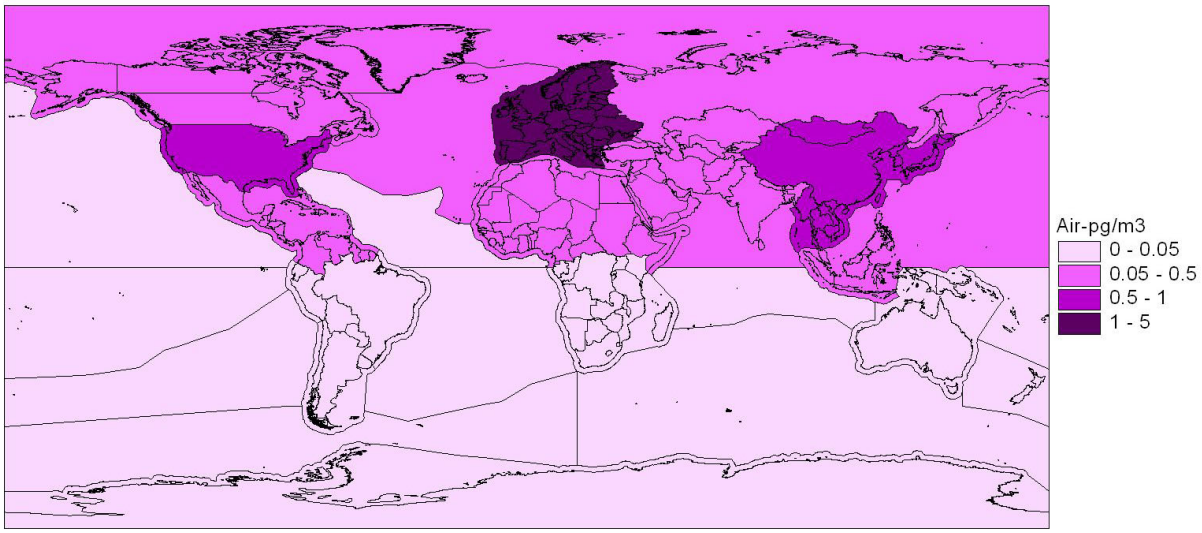


Figure 3.28. Atmospheric concentrations predicted by BETR-World model using the upper boundary half-lives.

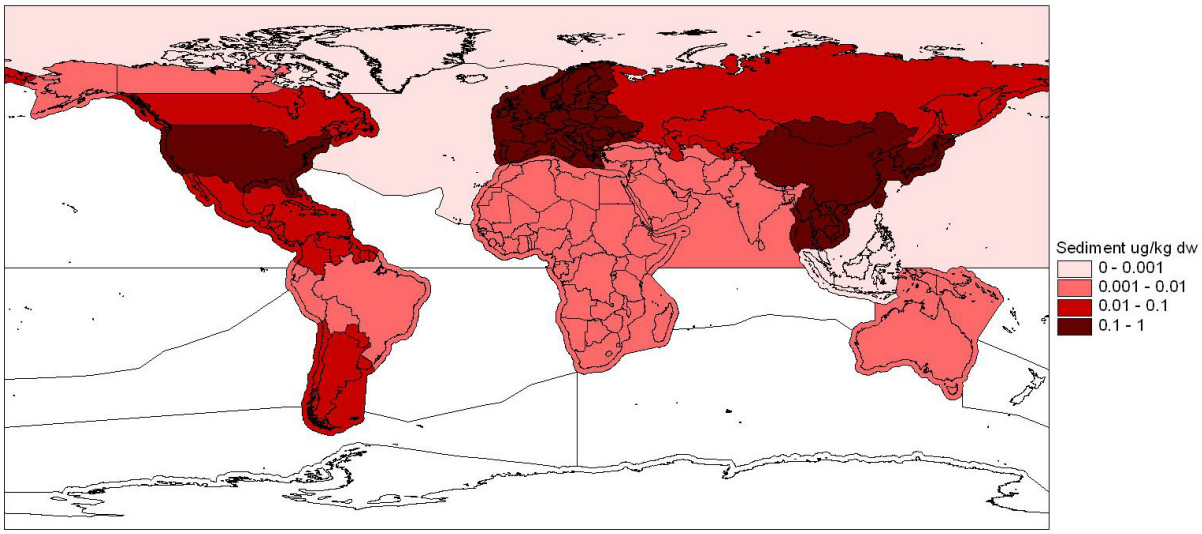


Figure 3.29. Freshwater sediment concentrations predicted by BETR-World model using the upper boundary half-lives.

Figure 3.29 shows the predicted levels of HBCD in freshwater sediment. Regions without local emissions are predicted to have less than 1 pg·g^{-1} dw (0.001 µg·kg^{-1} dw). Northern Canada (and Alaska), central South America, Africa, the Middle East, India and Australia are predicted to have between 1-10 pg·g^{-1} dw. Reported detection limits for HBCD in sediments vary between 0.01 to 1.2 µg·kg^{-1} dw; the top predicted level is in Europe (0.04 to 0.78 µg·kg^{-1} dw), which may be below some studies' detection limits. This estimated range for regional scale, non-point source sediment concentrations agrees well with measured background (upstream) concentrations (River Cinca, ES: 0.1 µg·kg^{-1} dw (Eljarrat et al. 2004) Lake Marsjon, SE: 0.279 µg·kg^{-1} dw (Sellström et al. 1998). Urban measurements of freshwater sediments in Stockholm, SE range between 0.8 to 1.5 µg·kg^{-}

[1] dw (Remberger et al. 2004) and ones from a lake near Zurich, CH range between 1.8 to 2.5 in near-surface and surface sediments (Kohler et al. 2008).

BETR-World estimates for transport pathways of HBCD to the Arctic using the upper bound environmental half-lives are summarized in Table 9.9. Seventy-two per cent of the chemical arriving in the Arctic is being transported there via the air (60% in the upper atmosphere and 12% in the lower atmosphere); 0.2% arrives from riverine outflows to Arctic water; the remaining 27.5% enters via marine water inflows, including 15.66% from European Marine water and 9% from the North Atlantic. Total advective inflow of chemical is 1655 kg·yr^{-1}. Total advective outflow is 1155 kg·yr^{-1}; 330 kg·yr^{-1} advection permanently from the system (primarily to deep oceans) and 169 kg·yr^{-1} chemical degradation losses.

3.5.3 Time Trends and Response Times

3.5.3.1 CoZMoMAN

The CoZMoMAN model (Breivik et al.) is an amalgam of the contaminant fate model CoZMo-POP (Wania et al. 2006) and the human food chain bioaccumulation model ACC-Human (Czub and McLachlan 2004). As such, it calculates - based on a mechanistic description of the underlying processes - the entire chain of events linking a chemical's emissions to the environment to the residue levels it may establish in the human body. In particular, it does so dynamically, i.e. it allows for temporal changes in emissions and in environmental parameters (such as temperature or the OH radical concentration in the atmosphere). One of the key pieces of information that such a model can provide is the time scale of concentration changes in response to changes in emissions. Specifically, it can estimate the time it takes for a chemical to reach a steady state situation, during which concentrations in a medium stay constant because the inputs and outputs to that medium are balancing each other. If that time is short, it can lend credibility to the use of steady state models such as RAIDAR. CoZMoMAN can also assess the time it takes for concentrations to decline, after emission have been reduced or eliminated altogether.

CoZMoMAN was used here to estimate the time to steady state and the extent of contamination reversibility for HBCD (i.e. how long will it take the environment to clean up if emissions were reduced to zero?). For this purpose, no realistic emission scenario was used but instead a hypothetical scenario that consists of 20 years of continuous emissions at a rate of 1 ton per 1 million km^2 and year (1 *t*·yr^{-1} per 1Mio km^2), followed by 20 years without any emissions. In the default scenario, it was assumed that 30 % of the emissions take place to the atmosphere, and 70 % to the fresh water environment. Additional calculations were performed assuming release either entirely to the atmosphere or entirely to water. The default parameterization for the environment and human food chain of the Western Baltic Proper drainage basin was used (Breivik et al.). The FAVs for the partitioning properties of t-HBCD from Table 2.2 and the degradation half-lives from Table 2.5 were used in these simulations. Additionally, a second set of simulations with the half-lives in water, sediment and soil compartments at the upper

bound (HL_{soil} = HL_{water} = 850 days, $HL_{sediment}$ = 210 days) was conducted to assess the impact of the considerable uncertainty in the degradation rates on the model results.

Figure 3.30 displays the calculated concentrations in air, soil, water, sediment, herring and humans. The water concentrations are for the estuarine compartment, which does not receive direct emissions of HBCD, but is downstream from the fresh water compartment which does receive the emissions. The herring concentrations are the average of 10 different age classes, although the variability in concentrations between age classes is relatively limited. The human concentration is for a female 20 years of age at the onset of the simulation.

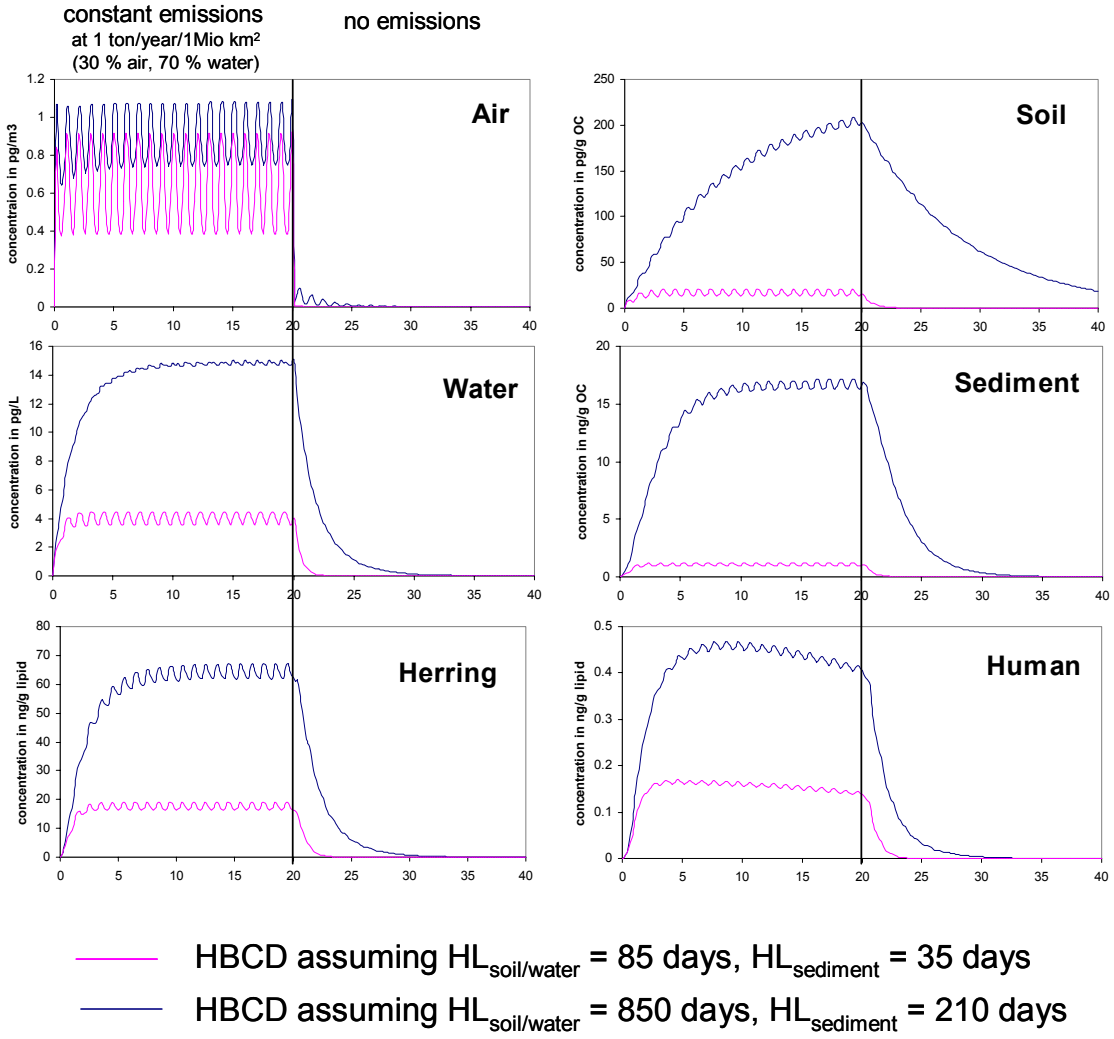


Figure 3.30. Time variant concentrations of HBCD in several media as calculated by CoZMoMAN assuming 20 years of steady emissions to air (30%) and water (70%), followed by 20 years of no emissions.

Time to Steady State

Using the default degradation half-lives HBCD rapidly reaches a steady state distribution in all model compartments (pink line in Figure 3.30), i.e., concentrations remain constant from year to year after only a few years of continuous emissions. With the longer degradation half lives (upper bound estimates for soil, water and sediment), the time to steady state is longer, yet still less than 10 years in most compartments (blue line in Figure 3.30). The exception is the soil compartment, which is still accumulating HBCD after 20 years of continuous emissions (upper right panel in Figure 3.30). We may infer that a steady state modelling approach is generally acceptable for HBCD, as long as emission rates remain fairly constant on the time scale of a decade and as long as soil is not the primary compartment of interest.

Contamination Reversibility

The situation is mirrored in the time it takes for contaminant levels to drop after emissions ceased. If the default half-lives are realistic, most of the HBCD will disappear from the environment and from organisms within a time frame of a few years (pink line in Figure 3.30). If the longer half-lives are more suitable, the time for recovery is on the time scale of a decade (blue line in Figure 3.30), again with the exception of soil concentrations, which will require multiple decades to decline.

Seasonal Fluctuations

Figure 3.30 and Table 3.14 reveals that the extent of air concentration variability during a year depends on the degradation half-lives and the mode of emission. It is larger for shorter half-lives and with a decreasing fraction of HBCD being emitted to air. It is particularly pronounced if there are no emissions to air. The model results for seasonal air fluctuations are supported by the limited seasonal monitoring data for air.

Absolute Concentration Values

Figure 3.30 also reveals that the absolute concentration levels in most media would be considerably higher if HBCD is more persistent in the surface media than the default degradation half-lives suggest. The difference is media dependent, ranging from less than a factor of two for air concentrations to an order of magnitude for soil and sediment concentrations.

In addition to the degradation half lives, the absolute concentration values are further highly dependent on the mode of emission. Table 3.14 lists the average concentration in various media during years 10 to 20 of the simulation, when HBCD reaches steady state in most compartments. Either default or slow degradation half-lives were used in these simulations.

Table 3.14. Average concentrations in air, herring and humans during the second decade of simulation in CoZMoMAN using continuous emissions at a rate of 1 $t \cdot yr^{-1}$ per 1Mio km^2 for different assumption concerning the mode of emission and the degradation half-lives in water, soil and sediment.

	100 % to air		**30 % air, 70 % water**		**100 % to water**	
Half lives	**Default**	**Long**	**Default**	**Long**	**Default**	**Long**
			Air in pg/m^3			
Average	2.01	2.79	0.63	0.91	0.04	0.10
Max	3.03	3.54	0.91	1.08	0.08	0.19
Min	1.09	2.04	0.38	0.743	0.004	0.02
Max/min	2.78	1.74	2.39	1.45	19.95	11.07
			Herring in ng/g lipid			
Average	28	110	18	64	13	45
Max	31	115	19	67	14	46
Min	26	105	16	61	12	43
			Human in ng/g lipid			
Average	0.19	0.53	0.09	0.27	0.05	0.16
Max	0.22	0.62	0.10	0.32	0.06	0.17
Min	0.17	0.47	0.08	0.24	0.04	0.14

Although the focus of the simulations here is on the time response and not on the absolute concentration values, it should be noted that the CoZMoMAN model is linear with respect to the emissions. This implies that the results can be scaled for emissions rates other than 1 $t \cdot yr^{-1} \cdot$per 1Mio km^2 by simple multiplication. For example, if the real emission rate is 5 $t \cdot yr^{-1} \cdot$per 1Mio km^2 all of the concentrations in Figure 3.30 and Table 3.14 have to be multiplied by a factor of 5. Following the data in the RAR and the same approach described above for RAIDAR for estimates of actual (i.e. 9.942 $t \cdot yr^{-1} \cdot$per 4.5Mio km^2), the unit emissions would be multiplied by a factor of about 2.2.

Whereas it is not surprising that air concentrations are much higher when emission occur exclusively into the atmosphere, it is less intuitive, why also herring and human concentrations should be higher, if a higher fraction of HBCD is emitted to the atmosphere rather than the water compartment. The explanation lies in the fact that the concentrations are for fish being exposed to the concentrations in the estuarine water compartment downstream from the fresh water compartment receiving the emissions. Apparently, the atmosphere is more efficient in delivering contaminant to the estuarine

water compartment than water advection – because most of the emitted HBCD is retained in the sediments of the receiving water compartment.

Overall, it is clear that reproducing absolute concentration values with the models will only be feasible, if the rate and mode of emission is well established and if the degradation half-lives in the surface media are better constrained. The model in fact can give some indication of how this may be achieved:

- The simulated air concentrations are fairly insensitive to the degradation half-lives in surface media (Figure 3.30), yet highly dependent on the assumed mode and rate of emission (Table 3.14). This implies that the emission rate to the atmosphere may be estimated fairly accurately, if reliable atmospheric background concentrations of HBCD become available.
- Furthermore the extent of seasonal air concentration variability may provide some clues as to the likely mode of emission. Table 3.14 suggests that much higher summer-winter differences in air concentrations can be expected, if direct releases to the atmosphere are very limited and emission occur mostly to the water compartment.
- Once the rate and mode of emission is established, the likely magnitude of degradation rates may be estimated by choosing values for the half-lives that reconcile modelled and measured concentrations and time trends in soils and sediments.

To place into context the time to steady state and the extent of reversibility of contamination of HBCD, we performed additional CoZMoMAN calculations for three benchmark POPs (polychlorinated biphenyl congeners 101 and 180, and hexachlorobenzene). The emission scenario for these substances was assumed to be identical to the one used in the HBCD simulations, namely 20 years of steady emissions into air (30 %) and fresh water (70%), followed by 20 years without emissions. The time trends in Figure 3.31 are normalized to the average over the 40 year simulation period to focus on the differences in trends rather than the absolute concentration levels being predicted. The time trend for HBCD in Figure 3.31 refers to the simulations using the upper bound half-lives in surface media (water and soil). In all media, HBCD displays a shorter time to steady state than the three benchmark POPs. It also shows faster concentration declines in all media than the benchmark POPs. In summary, we would expect HBCD concentrations in the physical environment and in the organisms making up the human food chain to respond much faster to changes in emissions than categorized POPs.

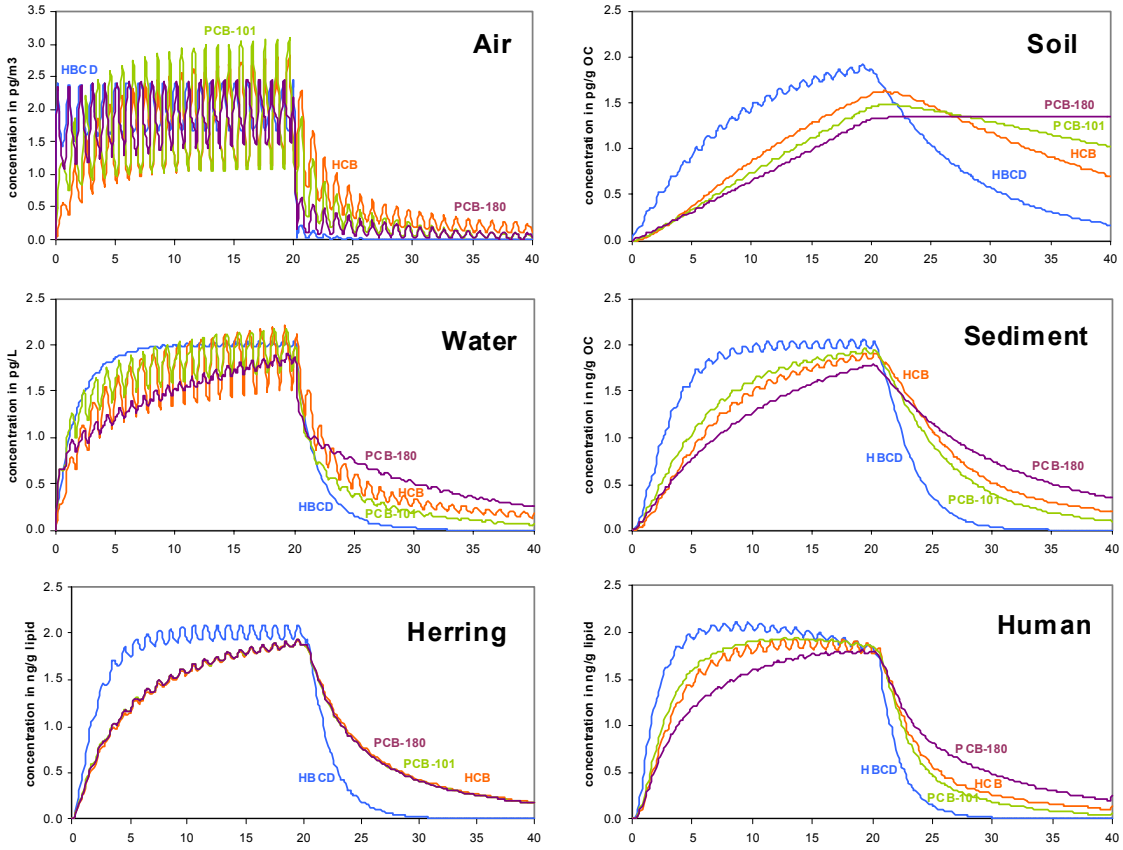


Figure 3.31. Comparison of simulated time trends of HBCD (long half-lives in surface media and three benchmark POPs, normalized to the average over the entire simulation period.

Table 3.15 compares the maximum predicted concentration during the 40 year simulation period for HBCD and the three benchmark POPs. The lower persistence of HBCD assures that the maximum concentrations it can achieve in air, soil, and sediment are lower than those of POPs, if they were emitted in an identical fashion. On the other hand, if the longer surface half-lives are used, CoZMoMAN predicts higher maximum concentrations for HBCD in water and herring compared to PCB-101 and PCB-180. Also, the predicted maximum concentration of HBCD in humans is very similar to that predicted for HCB. This highlights that a chemical that is generally less persistent than categorized POPs, could potentially bioaccumulate to higher concentrations in organisms than those POPs, if they were emitted in similar fashion and in comparable amounts.

Table 3.15. Maximum concentration of HBCD (using either median or upper bound estimates for half-lives in water and soil) and three benchmark POPs predicted during the hypothetical 40 year simulations displayed in Figure 3.31.

	HBCD (median HL)	**HBCD (upper bound HL)**	**HCB**	**PCB-101**	**PCB-180**
Air in pg/m^3	0.9	1.1	194	14	2.5
Soil in pg/g OC	21	208	1700	500	261
Water in ng/m^3	4.4	15	24	13	7.1
Sediment in ng/g OC	1.2	17	21	48	95
Herring in ng/g lipid	19	67	34	34	38
Human in ng/g lipid	0.2	0.5	0.6	2.0	4.1

4.0 EFFECTS CHARACTERIZATION

Often bright line exposure based criteria are used to screen "chemicals of concern" for more comprehensive evaluations (e.g. PBT screening assessments). Simply because a substance does not "pass" a screening level bright line criterion for toxicity, does not mean it necessarily causes adverse effects in the environment (Arnot and Mackay 2008). Estimating the likelihood of significant adverse effects or risk, which is a function of magnitude and duration of exposure that induces a given adverse effect response, is the issue regulatory programs ultimately seek to address. The objective of this section is to compile and critically review the available ecological and mammalian toxicity data for the selection of adverse effects threshold values for comparison with measured and modelled exposure levels in the environment.

Two key issues need to be considered for the effects characterization of HBCD. The first is whether or not available ecotoxicity test data can be used for screening (PBT) and/or more comprehensive risk (POP) assessment. Most ecotoxicity endpoints are based on concentrations in exposure media (water and soil) assuming that this surrogate measurement can be directly compared to criteria for effects or no effects. This assumption can be confounded for many reasons, particularly for chemicals with low water solubility. The second issue relates to how best to use available laboratory toxicity testing information to evaluate the potential for significant adverse effects in real world environments. Both issues are discussed by including a review of the current scientific literature.

4.1 DATA COMPILATION AND REVIEW

The data available for the toxicity of HBCD has been complied and is well presented in the European Commission RAR (EC 2008). Unless noted otherwise the material reported below is cited as reported in that document. This information is presented to illustrate the

data that was used to form the basis of the toxicity component of the PBT evaluation for HBCD by the European Commission (EC 2008) and a proposed POPs categorization by TemaNord (2008). This information was also used by the European Commission to develop risk-based environmental regulatory evaluation targets - predicted no effect concentrations (PNECs). From the European Commission (2008) report, it can be seen that there are other valid toxicity data that were reviewed but do not appear in the tables below as they demonstrate cases where HBCD showed less, or much less, toxicity.

4.1.1 Terrestrial Data

Table 4.1 summarizes the key terrestrial toxicity data selected for review in the present study from the European Commission RAR (EC 2008). Other available laboratory studies with mammals indicate that HBCD is not carcinogenic, mutagenic, or toxic to reproduction.

The soil micro-organism and vascular plant data are from good quality tests; however, the results are of limited use for establishing HBCD toxicity as they are reported as greater than a no observed effect concentration (> NOEC). These results provide no indication of an exposure where toxic effects might be expected and can therefore only mean that HBCD *does not* appear to be toxic to these organisms under these conditions. The soil invertebrate test produced both a NOEC and a lowest observed effect concentration (LOEC). These two observations are about a factor of 2 apart; therefore, it is reasonable to use the NOEC as a conservative estimate of soil invertebrate chronic effect.

The rat testing data from van der Ven et al. (2006) is based on a modelled dose-effect interpretation where the Benchmark Dose Lower-confidence Limit (BMD-L) is considered equivalent to a chronic no observed adverse effect level (NOAEL). The corresponding critical effect dose (CED) for this effect endpoint of liver weight increase in females is 22.9 $mg\cdot(kg\text{-}bw\cdot d)^{-1}$. The 2-generation rat reproduction study of Ema et al. (2008) used relatively widely spaced exposure levels with the lower exposure of 10 to 14 $mg\cdot(kg\text{-}bw\cdot d)^{-1}$ considered as a very conservative NOAEL and the next dose level of 101 to 141 $mg\cdot(kg\text{-}bw\cdot d)^{-1}$ considered as an effect level. The toxicity to mice (Erickson et al. 2006) is based on a single oral gavage dose 10 days after birth. Some behavioral effects were reported at 0.9 $mg\cdot kg^{-1}$ and many behavioral effects were reported at the higher dose of 13.5 $mg\cdot kg^{-1}$. However, it is difficult to accurately convert this value to a comparable continuous daily dosage, and the proposal that it be used to assess risk in bird eggs is an assumption not supported by a detailed justification in the RAR (EC 2008).

Table 4.1. Summary of key data on the terrestrial environmental effects of HBCD: Studies considered valid in previous EU assessments[1].

Compartment & Species	Method	Results	Remark and reference
TERRESTRIAL COMPARTMENT			
BIRDS & MAMMALS			
Rat		28-day NOAEL/MBD-L of 22.9 mg/kg bw/day.	(van der Ven et al. 2006)
Rat		2-generation NOAEC/NOAEL of 150 ppm dry weight in food (10 mg/kg bw/day)	(Ema et al. 2008)
Mouse		Single dose oral gavage of 0.9 mg/kg body weight produced behaviourial disturbances.	(Erickson et al. 2006). Internal dose estimate of 0.9 mg/kg wet weight calculated by assuming 100% absorption of oral dose. Mouse data was suggested to be used for risk evaluation with comparisons to bird egg monitoring data.
INVERTEBRATES			
Earthworms *(Eisenia fetida/andrei)*	OECD 207, OECD Proposed Earthworm reproduction test, USEPA 850-6200, GLP.	No effect on 28-day survival with exposure up to a mean measured concentration as 4,190 mg/kg dry soil. EC50 and NOEC are both greater than 4,190 mg/kg dry soil. LOEC of 235 mg/kg dry soil and a NOEC of 128 mg/kg dry soil. in 56-day reproductive study.	(Aufderheide et al. 2003). Adjusted NOEC of 59 mg/kg dry soil based on a organic matter correction from 7.4% to the 3.4% content of standard soils.
PLANTS			
Corn, cucumber, onion, ryegrass, soybean, and tomato	OECD 308 (proposed revision), USEPA 850-4100, 850-4225 (public drafts), GLP.	No statistically significant changes to mass or growth were observed after a 12 day exposure to a 3-isomer mixture of HBCD. NOEC > the highest measured exposure level of 6,200 mg/kg dry soil.	(Porch et al. 2002)
MICRO-ORGANISMS			
Soil micro-organisms	Nitrogen transformation, OECD 216	NOEC ≥ 750 mg/kg dry weight	(Förster 2007)

[1] Extracted from Tables 3-173 and Sections 3.3.4.1 to 3.3.4.3 in European Commission (2008)

4.1.2 Aquatic Data

4.1.2.1 Data Quality Considerations

Available aquatic toxicity data have been used for PBT (EC 2008) and POP (TemaNord 2008) assessments. HBCD is poorly soluble in water (hydrophobic). Chemicals with low water solubility can be notoriously difficult to assess for toxicity using external exposure metrics (e.g., Maeder et al. 2004; Mayer and Reichenberg 2006; Meador 2006; McCarty and Arnot 2008). Therefore, before reviewing the aquatic toxicity data, key issues related to the quality of these data and their applicability for PBT and POP assessments are first presented.

Most of the available aquatic toxicity data are of uncertain reliability for toxicity assessment and effects characterization for risk assessment and POP categorization because either (i) test organisms were exposed to water concentrations that exceed the water solubility limits for γ-HBCD (effectively t-HBCD for toxicity testing purposes when t-HBCD is tested), (ii) bioavailability was not quantified (the truly dissolved, bioavailable chemical concentration was not measured), (iii) cosolvents were used without quantifying the influence of the cosolvent on bioavailability and partitioning between the bulk water compartment and the test organism, (iv) organisms were exposed to a mixture of isomers simultaneouly, or (v) some combination of the aforementioned issues. Further details of these issues and how they relate to previous assessments of HBCD are presented in Section 9.5 and briefly summarized below. Despite the uncertainties described below in the available aquatic toxicity data some general conclusions are also presented.

The water solubility of α-, β-, and γ-HBCD are 41, 15, and 2.5 $\mu g \cdot L^{-1}$, respectively (see Section 2.3.2 and Appendix 9.1). A water solubility estimate for t-HBCD (the sum of all isomers) is 58 $\mu g \cdot L^{-1}$; however, the γ-HBCD isomer makes up about 80% of t-HBCD. Thus, when the solubility limits for all components of the mixture are considered (i.e. 58 $\mu g \cdot L^{-1}$), there are undissolved precipitates of the β- and γ-HBCD isomers in the water column as they are substantially above their water solubility limits. The issue of β- and γ-HBCD precipitates forming at the water solubility limit of t-HBCD (i.e. sum of water solubilities for each isomer) is recognized in the European Commission RAR in the section for chemical properties, but seemingly ignored in the evaluation of the aquatic toxicity data (EC 2008). Precipitates in the water column may exert effects on the test organism as a result of physical interactions with the precipitates and not as a result of the actual toxicity of the chemical. Only by keeping the t-HBCD concentration near the water solubility limit for γ-HBCD is it possible for all of the HBCD present during the toxicity test to remain dissolved in the water. Thus a water solubility of about 2 to 3 $\mu g \cdot L^{-1}$ (the approximate water solubility for γ-HBCD) represents a concentration at which all isomers of HBCD are truly dissolved and no significant precipates are expected.

For substances with low water solubility (i.e. hydrophobic), such as HBCD, some fraction of the total water column concentration will not be bioavailable to the test organism because it partitions into or onto organic phases in the bulk water compartment. Typical measurements of the bulk exposure water concentration use solvent extraction

methods that measure both the truly dissolved and the bound, or sorbed, non-bioavailable fraction of chemical. This confounds the quantification of the concentration in the water that is actually being absorbed by the organism during the toxicity test.

There can be complications interpreting toxicity data for hydrophobic substances when cosolvents are used. The use of cosolvents affects the bioavailability of a substance in aqueous exposure media. The degree to which the altered bioavailability occurs is speculative without a greater quantitative understanding of the influence of the cosolvent on toxicity endpoints in comparison to "pure" water phase exposures. Contrary to guidance from the OECD for testing with the use of cosolvents (OECD 2000), key quantitative parameters necessary to make these corrections and interpretations were not reported for the HBCD studies. The standard practice of including a solvent control in the design does not address this issue. The solvent control establishes whether the solvent itself is causing any adverse effects. It does not provide any information about how the cosolvent may be affecting the test substance or test organisms (e.g. bulk phase water solubility, bioavailablity) and thereby altering toxicity.

Evaluating the toxicity of mixtures is a challenge in terms of a theoretical foundation and in practical application (McCarty and Borgert 2006b; McCarty and Borgert 2006a). The general challenge is identifying which substance is responsible for any effect and if that substance acts in isolation or if there are possible additive, synergistic or antagonistic interactions. In addition to the broad general challenge, the OECD guidance document on testing of difficult substances and mixtures (2000) specifically advises against the use of cosolvents in cases such as t-HBCD (mixture of isomers). Similarly, the use of generator systems such as generator columns is specifically contraindicated for toxicity testing of difficult substances and mixtures such as HBCD. Generator systems are not considered appropriate for substances which differ in their water solubility since these differences will result in selective depletion of the more water soluble components from the column or disk matrix and thus influence their relative concentrations in the water phase (OECD 2000).

4.1.2.2 Aquatic Toxicity Data Review

Table 4.2 summarizes the key aquatic toxicity data selected for review in the present study from the European Commission RAR (EC 2008). The available sediment toxicity is not addressed because these data are not readily translated to a water-based exposure and there were no body/tissue residue-effect data that could be used in the present risk assessment.

<u>Plants, Algae and Microorganisms</u>

There are no toxicity data for freshwater or marine vascular plants but there are some results for freshwater and marine algae. In addition to the usual challenges of interpreting algae test data (e.g. the use of growth media with various and variable levels of mineral, vitamins, and nutrients, rather than plain water, and measuring effects at in a specific exponential growth phase) almost all of the reported results were obtained with cosolvents. As different cosolvents were used at different concentrations, effects on bioavailability and toxicity are unquantified. However, since many of these tests were

near or in excess of water solubility levels, the results when cosolvents were used must be considered uncertain and likely overestimates of the actual aquatic toxicity. In other words, if the test had been conducted without cosolvent any toxicity that may have been reported would have occurred at a higher exposure concentration. Although the microorganism test with activated sludge did not use a cosolvent, the reported value is greatly in excess of HBCD water solubility and is thus of little utility for PBT/POPs classification or general risk assessment.

Invertebrates

There are acute and chronic toxicity data for the aquatic invertebrate Daphnia. The acute test is of little utility as no effects were attributed to the HBCD exposure and the results indicate that the 48-hr EC50 is greater than the highest measured exposure level of 3.2 $\mu g \cdot L^{-1}$. The 21-day chronic test indicated a NOEC of 3.1 $\mu g \cdot L^{-1}$ and a LOEC of 5.6 $\mu g \cdot L^{-1}$ based on reduced mean length. A cosolvent was used in both cases. Therefore these data are considered unreliable since they are most likely overestimates of the actual aquatic toxicity under these conditions.

Fish

There are acute and chronic toxicity data for the rainbow trout. The 96-hour acute test shows no effects at all at the highest measured concentration of 2.5 $\mu g \cdot L^{-1}$. Similarly, the 88-day chronic study reported that no effects were observed at all for several endpoints (hatching, survival, growth) and that the NOECs was greater than the highest exposure level of 3.7 $\mu g \cdot L^{-1}$. Neither of these studies found any effects due to HBCD exposure and the aquatic exposure levels associated with adverse effects is at some unknown higher level. Despite the lack of effects, due to the use of cosolvent, these data are likely overestimates of the actual aquatic toxicity under these conditions.

Table 4.2. Summary of key data on the aquatic environmental effects of HBCD: Studies considered valid in previous EU assessments[1].

Compartment & Species	Method	Results	Remark and Reference	Cosolvent Used
AQUATIC COMPARTMENT				
FISH				
Acute toxicity				
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)	dimethylformamide
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 & fry survival ≥3.7 Growth ≥3.7	(Drottar et al. 2001)	acetone
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)	dimethylformamide
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)	dimethylformamide
ALGAE				
Selenastrum capricornutum	OECD 201 and TSCA40/797/1050	72 h EC_{50} and LOEC >2.5 µg/l. NOEC could not be estimated.	Study 2 (Roberts and Swigert 1997)	dimethylformamide
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. 1987a) Not according to guidelines, results only used as supportive	acetone
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 µg/l	Study 4 (Desjardins et al. 2004)	No cosolvent, generator column used for single exposure concentration
Skeletonema costatum	OECD 201	NOEC >10 µg/l EC_{50} 52 µg/l	Study 5 (Desjardins et al. 2005)	NOEC: dimethylformamide EC50: no cosolvent, generator column used for single exposure concentration

Compartment & Species	Method	Results	Remark and Reference	Cosolvent Used
SEWAGE TREATMENT PLANT				
MICRO-ORGANISMS				
Activated sludge	Respiration inhibition OECD 209	EC_{50} 15 mg/l	Limit test with one test concentration, EC_{50} is estimated. (Schaefer and Siddiqui 2003)	No cosolvent

[1] Extracted from Table 3-173 in European Commission (2008). Cosolvent information extracted from the associated text and added.

4.2 EFFECT VALUES FOR PBT OR POPS CLASSIFICATION AND RISK ASSESSMENT

The European Commission RAR (2008) employs toxicity data in two distinct ways. The first is the PBT classification process - does HBCD meet the criteria for classification as a PBT - while the second is the generation of predicted no-effect concentrations (PNEC) for use in risk assessment. TemaNord (2008) used the same toxicity data in their screening level POPs classification process. In the present assessment we first critically evaluate the values selected in the aforementioned assessments for screening HBCD as a potential PBT chemical (hazard) and for assessing the potential of significant adverse effects from exposure to HBCD in the environment (risk). Then, based on our analysis of the available data, alternative toxicity endpoints are proposed for assessing the potential of significant adverse effects from exposure to HBCD in the environment (risk).

4.2.1 PBT and POPs "T" Classification

The key toxicity data considered valid and diagnostic in the European Commission RAR (2008) and TemaNord POP proposal (2008) are the aquatic toxicity data. These data included the reproduction NOEC for Daphnia of 3.1 $\mu g \cdot L^{-1}$ (Drottar and Krueger 1998), the chronic ELS NOEC for rainbow trout of 3.7 $\mu g \cdot L^{-1}$ (Drottar et al. 2001) and the growth EC50 marine algae test for Skeletonema of 52 $\mu g \cdot L^{-1}$ (Walsh et al. 1987b).

Figure 4.1 illustrates the aquatic toxicity data listed in Table 4.2 and the water solubility limit for γ-HBCD (about 2 to 3 $\mu g \cdot L^{-1}$). This is effectively the water solubility limit for t-HBCD for aquatic toxicity testing purposes since γ-HBCD will precipitate at concentrations greater than its water solubility limit. The testing data appear to demonstrate some adverse effects (e.g. LOEC) above the water solubility of γ-HBCD; however, these "effect data" are highly uncertain due to the unquantified influence of undissolved HBCD (precipitate) and the presence of cosolvent.

Given the low water solubility of HBCD, it is quite likely that a "toxicity cutoff" is being observed (Veith et al. 1983). Mayer and Reichenberg (2006) also discuss a melting point cut-off for baseline toxicity. The melting point value they propose is ~220 °C which is near the melting point for γ-HBCD. In essence, this cutoff occurs because it is not possible to sustain a concentration in the water at a level high enough to cause a toxic effect during the test exposure. Since HBCD is not showing any significant adverse toxic

effects at the water solubility limit, this and other observations suggests that it may possess only a baseline narcotic mode of toxic action (the least potent mode of action).

For the majority of the key data points used in the previous HBCD evaluations no effects of any kind at any of the exposure levels were observed (i.e. NOEC). Most of these "NOEC values" have been derived when there are no effects observed at the highest exposure concentration. Although reporting a NOEC value in this manner is correct it can be misleading, particularly if there are no reported concentrations of an effect concentration (i.e. LOEC). A NOEC clearly indicates that no effects were found at the specified exposure; however, for the majority of the reported NOEC values there were no indications of an exposure concentration where some effects might be expected (i.e. LOEC). Thus, an actual toxic exposure concentration might be close to the NOEC level, or perhaps 3 to 5 times higher, or perhaps 10 to 1000 times higher. As detailed in Section 9.5.3, without an LOEC value there is no way of knowing where the transition threshold from "no effect" to "effect" occurs. This issue (i.e. NOEC without LOEC) limits the applicability of the NOEC data for chemical assessments. Importantly, as can be seen in Figure 4.1 all of the available NOECs at or below the effective water solubility limit for t-HBCD (2 to 3 $\mu g \cdot L^{-1}$), indicate the same basic information: no adverse toxic effects were observed. In such circumstances the use of "no effects" data to indicate possible "toxic effects" for chemical assessments is deemed to be of poor scientific defensibility, particularly when there are other viable alternatives to assess the likelihood for significant adverse effects in the environment. Remaining mindful of these issues, the available aquatic toxicity data are examined in further detail below.

Fish

The key fish data from Table 4.2 and Figure 4.1 indicate no effects for either acute or chronic exposure to rainbow trout at 2.5 and 3.7 $\mu g \cdot L^{-1}$, respectively, in tests employing a cosolvent. In particular, the chronic ELS NOEC for rainbow trout (Drottar et al. 2001) is actually reported as > 3.7 $\mu g \cdot L^{-1}$ as no effects for several endpoints were observed at any exposure concentration tested. In both of these fish studies the measured exposure levels were only about 1/3 to 1/2 of the nominal levels, suggesting substantial water solubility/bioavailability problems, despite the use of cosolvent. Other fish toxicity data reviewed in the European Commission RAR (2008) indicate acute toxicity test results at approximately 100 and 10,000 $mg \cdot L^{-1}$, where cosolvent was used, and no effects were reported. Based on this complete lack of observed adverse effects in exposed fish it is clear that HBCD does not appear to be toxic to fish via water-borne exposure, even with the enhanced bioavailability due to the use of cosolvent.

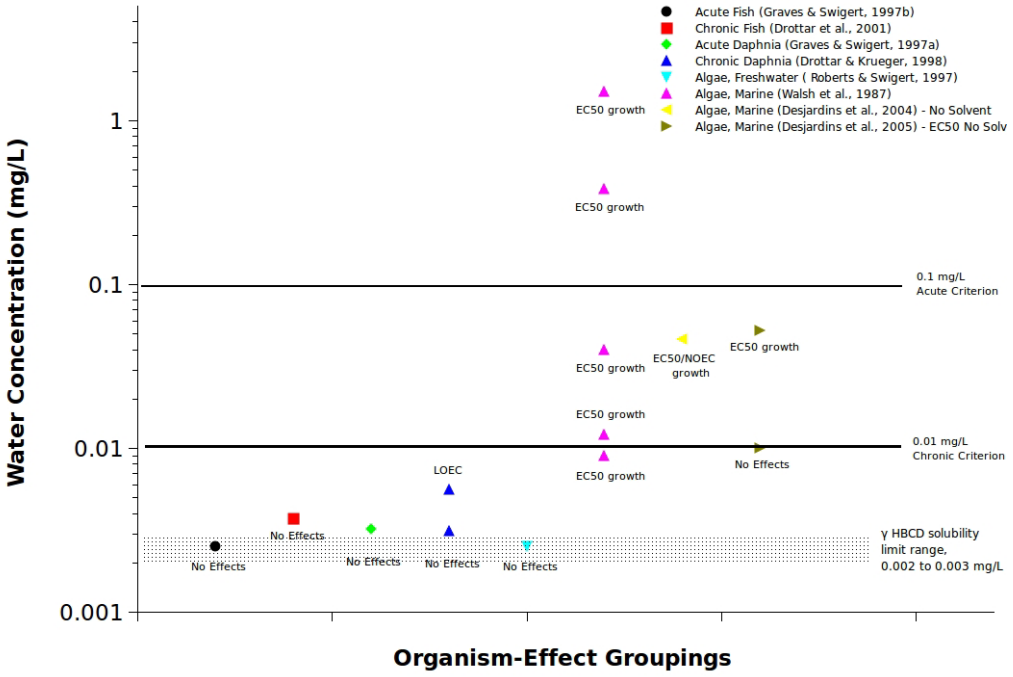


Figure 4.1. Key aquatic toxicity exposure data for PBT or POPs assessment.

Invertebrates

The key aquatic invertebrate data from Table 4.2 and Figure 4.1 indicate no effects in a 48-hour exposure of Daphnia at the highest exposure level of 3.2 $\mu g \cdot L^{-1}$ and 21-day exposure of Daphnia producing a NOEC of 3.1 $\mu g \cdot L^{-1}$ and a growth LOEC of 5.6 $\mu g \cdot L^{-1}$. Since cosolvent was used in both studies, the reported values are most likely confounded and overestimate toxicity such that studies carried out without cosolvent would likely generate toxicity estimate with a higher numerical value showing HBCD to be less toxic. Furthermore, the LOEC is above the water solubility limit for γ-HBCD (effectively t-HBCD for toxicity testing purposes since γ-HBCD will precipitate at concentrations greater than its water solubility limit).

In addition to the cosolvent and water solubility issues, a detailed examination of the results from the 21-day studies (Drottar and Krueger 1998) indicates some problems in the latter part of this test, as can be seen in Table 3-157 in European Commission (2008). Mortality began to be observed in the negative control exposure with 1 death at 14 days and 2 at 21 days. Additionally, there was 1 death in the solvent control at 21 days. Also, non-monotonic distribution of deaths began to be seen at 14 days with 1, 1 and 2 deaths in the control, 1.6, and 11 $\mu g \cdot L^{-1}$ exposure levels, respectively, but not at the 0.87, 3.1 and 5.6 $\mu g \cdot L^{-1}$ exposures. Although some additional deaths occur at other exposure levels at 21 days, the distribution is still non-monotonic as no mortality occurred at the 3.1 $\mu g \cdot L^{-1}$ level. The non-monotonic distribution of deaths and the total of 3 deaths in the controls versus only 5 deaths in the highest exposure level of 11 $\mu g \cdot L^{-1}$ at 21 days suggest that some other factor other than HBCD was involved in causing the mortality. This also

raises uncertainty about the validity of the growth and reproductive effects reported, since a general increase in some other stressor sufficient to cause general increases in mortality will also affect non-lethal endpoints.

The available Daphnia data do not clearly and unequivocally establish that HBCD causes adverse effects to Daphnia at exposure levels at or below than the water solubility limit for γ-HBCD (or effectively, t-HBCD). As well, available data do not unequivocally establish that the chronic toxicity of HBCD to aquatic invertebrates occurs below the PBT "T" criterion of 10 $\mu g \cdot L^{-1}$.

Algae

The key algae data from Table 4.2 and Figure 4.1 indicate a combination of no-effect and effects data and most obtained with the use of cosolvents. In the first study for a freshwater alga (Roberts and Swigert 1997) little information is reported on effects. An NOEC could not be estimated and the EC50 and LOEC were both reported as greater than 2.5 $\mu g \cdot L^{-1}$. A cosolvent was employed in this test. In the second study with three marine algae (Walsh et al. 1987b) several growth EC50s were obtained that varied both within and between species and no NOEC values were calculated. A cosolvent was also employed in this study. Since this work deviated from standard testing methodology the European Commission (2008) judged that this information should only be used in support of other work obtained with standard methods. Thus, it should not be used as primary evidence in either PBT or POPs "T" evaluation.

The third study with marine alga (Desjardins et al. 2004) did not use a co-solvent but was complicated by a non-standard methodology in which only 1 exposure concentration was employed. A growth inhibition of approximately 10% was observed at 72-hours at an exposure concentration of 40.6 $\mu g \cdot L^{-1}$, obtained with the use of an HBCD-saturated generator column. The 72-hour EC50 for growth is reported as greater than 40.6 $\mu g \cdot L^{-1}$ and the NOEC is reported as less than 40.6 $\mu g \cdot L^{-1}$. These data were obtained in a study where a generator column was used to produce the exposure media. Problems with this methodology are discussed elsewhere (Section 9.5), and the OECD (2000) testing guidance specifically recommends against this practice. Thus, these data cannot be considered as reliable for comparisons with the PBT acute "T" criterion of 100 $\mu g \cdot L^{-1}$, the PBT chronic "T" criterion of 10 $\mu g \cdot L^{-1}$, or in a POPs screening assessment.

The fourth study with marine alga (Desjardins et al. 2005) contains information from two studies, one with co-solvent and one without. The study without cosolvent was complicated by by a non-standard methodology in which only 1 exposure concentration was employed. A growth inhibition of 51% was observed at 72-hours at an exposure concentration of 52.6 $\mu g \cdot L^{-1}$, obtained with the use of an HBCD-saturated generator column. Thus, the 72-hour EC50 for growth is reported as 52 $\mu g \cdot L^{-1}$. A NOEC could not be estimated. These data were also obtained using a generator column to produce the exposure media (see Section 9.5 and (OECD 2000)) The study with cosolvent reported no adverse effects after 72 -hours of exposure in any of the test concentrations employed. The NOEC was reported as being greater than 10 $\mu g \cdot L^{-1}$, which was the highest exposure

concentration used (i.e. there was no LOEC reported) and at a concentration above the water solubility limit for γ-HBCD.

Given the various issues discussed above, the available algae data cannot be considered to clearly and unequivocally establish that the chronic toxicity of HBCD to algae is below the European Commission PBT "T" criterion of 10 $\mu g \cdot L^{-1}$. In fact, adverse effects to algae are only reported at exposure levels in excess of the water solubility for the main γ-isomer. Perhaps the best algae data set (Desjardins et al. 2004; Desjardins et al. 2005), indicates that, despite the confounding issues, the chronic toxicity of HBCD to algae is above the "T" criterion of 10 $\mu g \cdot L^{-1}$ and therefore it should not be considered a PBT based on these studies. Specifically, both the solvent and no-solvent growth EC50 estimates of 40.6 and 52 $\mu g \cdot L^{-1}$ demonstrate chronic toxicity effects above the NOEC criterion of 10 $\mu g \cdot L^{-1}$ and the NOEC estimate of >10 $\mu g \cdot L^{-1}$ indicates that the NOEC is somewhere between the criterion value of 10 $\mu g \cdot L^{-1}$ and the EC50 estimates.

Conclusions

The screening level PBT risk evaluation presented in the European Commission RAR (2008) and the TemaNord POPs classification proposal (2008) identified and selected key environmental toxicity data for HBCD. The present review of the available data does not support the conclusion that HBCD meets the PBT "T" criterion nor the suggestion that these data are directly applicable for a POP classification. Much of the HBCD data considered valid and applicable in previous PBT and POP assessments is shown to contravene published international guidance on aquatic testing protocols (e.g. OECD 2000). Assessments must fully consider the implications of uncertainty that exist when using data that are in violation of standardized testing protocols.

The water solubility of γ-HBCD (or effectively t-HBCD for toxicity testing purposes), is about 2 to 3 $\mu g \cdot L^{-1}$, which is below the PBT NOEC criterion of 10 $\mu g \cdot L^{-1}$. Therefore, it is not possible to have a sufficient amount of dissolved t-HBCD in the water column to reach the NOEC criterion value to demonstrate that HBCD is "not T" for the purposes of screening level PBT classification. Clearly, due to water solubility limitations, it is impossible to have a valid aquatic toxicity test result for HBCD that can be adequately assessed using the existing PBT NOEC criterion. The simplest and most informative statement on the aquatic toxicity testing data for HBCD is that no effects have been reported for exposure concentrations at or below the estimated water solubility limit for γ-HBCD.

In summary, using toxicity information based on water-based exposure to aquatic organisms does not appear to be a reliable or appropriate methodology to establish whether HBCD should be classified as a PBT or a POP on the basis of toxicity. A detailed interpretive review of available data does not support a conclusion that HBCD meets the "T" criterion for PBT or POPs classification, as presented in European Commission (2008) and TemaNord (2008), respectively.

4.2.2 Previous Approaches for Effect Characterization

Table 4.3 contains a summary of the critical toxicity data and the recommended assessment factors considered for PNEC estimation by the European Commission RAR (2008). The TemaNord (2008) report did not present a general environmental risk assessment, but rather focused only on Stockholm Convention and UN-ECE "POPs" classification screening criteria. In general, these data are demonstrated to be confounded by various technical issues and are not recommended for comprehensive risk assessment. The Technical Guidance Document (EC 2003) used for the European Commission RAR (2008) also suggests that the lowest NOEC should be used in the PNEC estimation. Both a NOEC and the lowest effect exposure level (LOEC) are considered more appropriate for developing a PNEC since the threshold for the occurrence of adverse effects is somewhere between these two estimated endpoints. Without some reliable indication of exposure levels associated with an observed effect (i.e. LOEC), a NOEC is not considered appropriate for the estimation of a PNEC since in isolation it contains no reliable information on possible effect levels. Thus, the TGD is considered to represent a simplistic approach, acceptable perhaps for a screening evaluation, but not recommended for more comprehensive risk assessments. Rather than force a process beyond its domain of applicability, two alternative methodologies are proposed that are considered more appropriate for the assessment of potential significant adverse effects. The body/tissue-residue and the Total Daily Intake (TDI) approaches are viable alternatives examined in the next section.

Table 4.3. Summary of critical toxicity data and PNEC estimates for environmental effects of HBCD from European Commission RAR (2008) [1].

Compartment & Target	EC Section Reference	Critical Toxicity Data	Predicted No-Effect Concentration
TERRESTRIAL COMPARTMENT			
Plants	3.3.2.1.1	No appropriate data	NA
Invertebrates (soil)	3.3.2.2	59 mg/kg dry soil (Earthworm NOEC) Assessment factor = 10	5.9 mg/kg dry soil
Mammals	3.3.4.2	150 ppm dry weight in food (10 mg/kg/d dose equivalent) (Rat reproduction NOAEL) Assessment factor = 30	5 mg/kg in food
Bird egg	3.3.4.3	0.9 mg/kg wet weight (Mouse behavior LOAEL)	NA
AQUATIC COMPARTMENT			
Freshwater micro-organisms	3.3.1.5	15 mg/L (Activated sludge respiration inihibition) Assesment factor = 100	150 µg/L
Freshwater organisms	3.3.2.1	3.1 µg/L (*Daphnia magna* NOEC) Assessment factor = 10	0.31 µg/L
Freshwater sediment organisms	3.3.1.7	8.6 mg/kg sediment dry weight (Worm NOEC) Assessment factor = 10	0.86 mg/kg sediiment, dry weight
Saltwater organisms	3.3.5.2	3.1 µg/L (*Daphnia magna* NOEC) Assessment factor = 100	0.031 µg/L
Saltwater sediment organisms	3.3.5.4	8.6 mg/kg sediment dry weight (Worm NOEC) Assessment factor = 50	0.17 mg/kg sediment, dry weight

[1] Extracted from European Commission, 2008, for continuous exposures only.

4.2.3 Current Approaches for Effect Characterization

This section outlines the development of two general and related alternative approaches for deriving threshold values to characterize effects levels. These approaches are later used to assess HBCD for its potential to cause significant adverse effects in different regions of the global environment. First the body/tissue-residue approach is discussed followed by the Total Daily Intake (TDI) approach.

Body/tissue-residue Approach

McCarty and Mackay (1993) examined some disparate trends in environmental fate and environmental toxicity and suggested a consolidated approach. It was suggested that routinely providing more detailed information for toxicity data, particularly some quantitative indication of the amount of substance in the body and/or tissues of exposed organisms that was associated with various types and degrees of adverse effects (the critical body residue approach), would provide both an improved understanding of the toxicity of the substance and facilitate regulatory use of that enhanced toxicity information. The facilitated regulatory use would occur in two areas. The first is by more

direct comparison of laboratory-derived body/tissue residue dose-effect data with measured body/tissue concentrations of substances obtained from monitoring. The second is by enhancing both retrospective and prospective use of increasingly sophisticated environmental distribution, fate, and exposure models to explain and predict toxicity. For hydrophobic substances, such as HBCD, a further advantage is that an organism-based dose metric is largely free of the aforementioned issues that plague aquatic exposure-based metrics (bioavailability, cosolvents). Toxicity information developed based on body/tissue-residues is more directly comparable with the monitoring data and model predictions for various organisms at various trophic levels in the environment. A recent study provides a good example of using this approach to evaluate potential risks of POPs to top predators (Leonards et al. 2008) and assessments of this type have also been advocated elsewhere (e.g. Wu et al. 2008).

A guide to developing residue-based regulatory guidance for risk assessment has been published (Meador 2006). The Society of Environmental Toxicology and Chemistry (SETAC) held a workshop on the tissue-residue approach in 2007 (Meador et al. 2008) and another on POPs and PBTs in 2008 (Solomon et al. 2009). In these workshops and published proceedings the use of residue-based toxicity data was recommended. The U.S. EPA has also incorporated a residue-based approach in the proposed revisions to the Guidelines for Deriving Ambient Water Quality Criteria for the Protection of Aquatic and Aquatic-Dependent Wildlife document (U.S. EPA 2005). The U.S. EPA's Office of Pesticide Programs has adopted a residue-based approach for pesticide registration and has published a white paper on the registration process for candidate pesticides with PBT-like properties (Anderson et al. 2008a). Residue-based risk assessments have been prepared for a candidate pesticide by the U.S. EPA (Anderson et al. 2008b) and on behalf of the proponent (McCarty and Arnot 2008).

The scientific and regulatory communities are in the process of either evaluating or formally adopting a critical body/tissue residue-based approach to better address the potential for significant adverse effects in the environment. This approach is not a panacea and many challenges exist, including how best to adequately address the target/nontarget lipid issue and the lack of a comprehensive, widely-applicable, generally accepted mode of toxic action classification scheme (McCarty and Borgert 2006a,b). In this latter regard for organic chemicals the extremes of the range of toxic potency appear to be defined by baseline narcosis and TCDD-like toxicity.

For HBCD a comprehensive review of all available residue-based toxicity information would take some time as the focus has been on exposure-based data and would require a careful review of all of the original publications. Some residue-based data can be gleaned from the reviews provided in the European Commission risk assessment (2008). This, along with some other pertinent information, is presented in Figure 4.2.

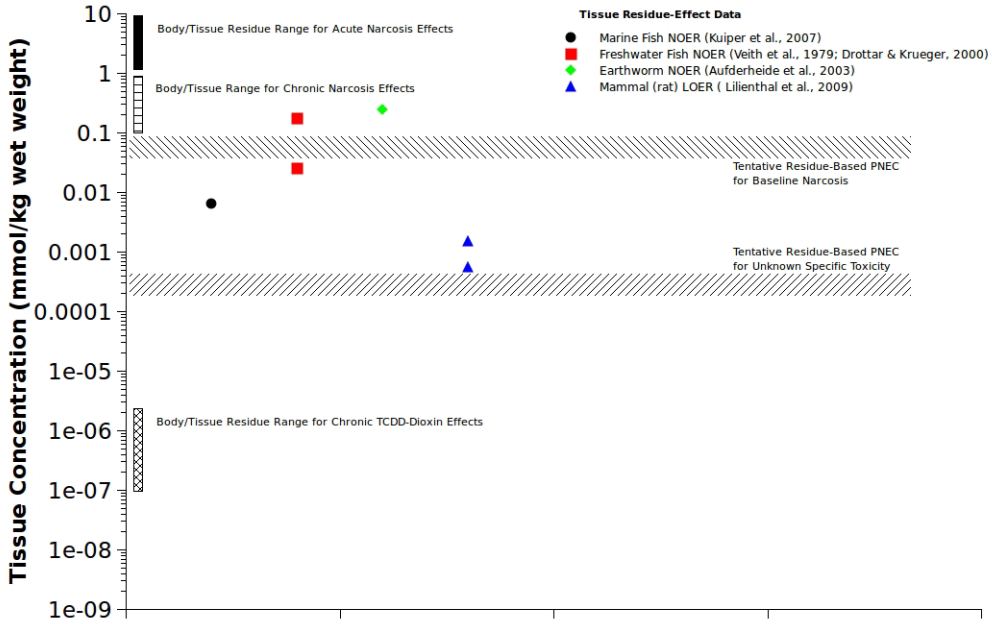


Figure 4.2. Residue-based organism toxicity and residue-based PNECs.

On the left side of Figure 4.2 the approximate whole-body residue-effect ranges for the least toxic (baseline narcosis) and most toxic modes of action (TCDD-like toxicity) for small aquatic organisms of the order of 5% lipid (McCarty and Mackay 1993). This provides perspective on the range of possible residue-based toxicity dose-response relationships.

The following aquatic and terrestrial toxicity data on HBCD were used to set the tentative residue-based PNECs that are developed below and depicted in Figure 4.2. A marine fish no-observed effect residue (NOER) is based on a 78-day sediment/diet exposure endocrine/reproductive study by (Kuiper et al. 2007). The highest average muscle concentration resulting from the various exposures was 274.2 $\mu g \cdot g^{-1}$ lipid with a reported maximum of 446 $\mu g \cdot g^{-1}$ lipid weight. It was reported that this residue "... did not affect any investigated parameters." The lipid content of the muscle tissue was reported as 0.9%. The residue-based NOER was estimated as (446 $\mu g \cdot g^{-1}$ lw×0.009 = 4 $mg \cdot kg^{-1}$ ww/641.7 $g \cdot mol^{-1}$ =) 0.0063 $mmol \cdot kg^{-1}$ ww.

In a 32-day BCF test with freshwater fish (Veith et al. 1979) no adverse effects were observed. The mean exposure concentration was 6.2 $\mu g \cdot L^{-1}$ with an estimated BCF of 18,100 based on measured fish levels. Lipid levels for these fish are of the order of 5%. The residue-based NOEC was estimated as (6.2 $\mu g \cdot L^{-1}$×18100 = 112.22 $mg \cdot kg^{-1}$ ww/641.7 $g \cdot mol^{-1}$ =) 0.175 $mmol \cdot kg^{-1}$ ww.

In a 35-day BCF exposure with freshwater fish no adverse effects were observed (Drottar and Krueger 2000). The highest measured whole-body fish concentration level was 16,154 $\mu g \cdot kg^{-1}$ ww and the lipid level was about 5-10%. This residue-based NOER is (16.2 $mg \cdot kg^{-1}$ ww/641.7 $g \cdot mol^{-1}$ =) 0.025 $mmol \cdot kg^{-1}$ ww.

In a 56-day soil exposure survival and reproduction study with earthworm (Aufderheide et al. 2003) at 28-days the highest exposure level of 4,190 $mg \cdot kg^{-1}$ dry soil resulted in a maximum tissue concentration of 150 $mg \cdot kg^{-1}$ ww. This corresponds to a NOER of (150 $mg \cdot kg^{-1}$ ww/641.7 $g \cdot mol^{-1}$ =) 0.24 $mmol \cdot kg^{-1}$ ww in earthworms based on no-effects for survival. Effects on reproduction were found after 56 days at intermediate exposure levels but no BCF or tissue residue information was available. Earthworms have been shown to exhibit residue-effect relationships similar to fish and aquatic invertebrates for a number of organic chemicals (Lanno and McCarty 1997).

There are three mammalian toxicity studies with t-HBCD where some residue data were collected. The 90-d rat study by Chengelis (2001) reported HBCD levels in adipose tissue which is largely a storage compartment for hydrophobic substances and is unlikely to be a site where toxic action occurs. This information was not used as it cannot be converted back to general tissue wet weight. Another group of researchers conducted both a 28-d (van der Ven et al. 2006) and a 1 generation reproduction study (Lilienthal et al. 2009) where some liver residue data was collected. In the 28-d study the lowest effect levels were reported for female rats which exhibited thyroid and liver weight changes at BMD-L levels of 1.6 and 22.9 $mg \cdot (kg\text{-}bw \cdot d)^{-1}$, respectively. The corresponding liver HBCD residue levels were 43 and 192 $\mu g \cdot g^{-1}$ lw, respectively. Corrected to wet weight from lipid weight, using a lipid content of 5%, corresponds to residues of 0.0034 and 0.015 $mmol \cdot kg^{-1}$ ww, respectively. The European Commission (2008) considered the BMD-L based on liver weight change to be the most robust. van der Ven et al. (2009) found a variety of effects due to HBCD exposure over one generation. The most sensitive response they noted was modest behavior changes in rats (BMD-L based on changes in auditory thresholds and effects of catalepsy) at HBCD liver levels of 7 and 16-20 $\mu g \cdot g^{-1}$ lipid weight, respectively (Lilienthal et al. 2009). The lipid content of the rat livers was approximately 5%. This corresponds to NOERs of (7 and 20 $\mu g \cdot g^{-1}$ lw/20 = 0.35 and 1 $mg \cdot kg^{-1}$ wet weight/641.7 $g \cdot mol^{-1}$ =) 0.00055 and 0.0015 $mmol \cdot kg^{-1}$ ww, respectively. A full regulatory assessment of the relevance of the findings of the 1 generation study has yet to be made, and the ecological significance of some of the detected changes is uncertain. However, in the interests of presenting an alternative to the baseline narcosis scenario, the lowest data for the modest behaviorial changes (more than an order of magnitude below the robust BMD-L for liver weight change) will be used as a "worst case" exercise to develop a tentative residue-based specific toxicity PNEC.

The following is a preliminary interpretation based on available data, as presented in Figure 4.2, and should be considered as illustrative rather than definitive for development and use of a residue-based PNEC approach for HBCD. The NOER data for fish and the earthworm suggest that HBCD acts by the least toxic mode of action for organic chemicals, baseline narcosis, as the NOER data range up into the lower part of the chronic baseline narcosis residue range established for fish and some invertebrates.

Although there are no observed residue-based effect data for HBCD, the acute and chronic mode of action information serve as the LOER and indicate where adverse effects are expected. As a baseline narcotic toxicant adverse effects will be largely determined by bioconcentration/bioaccumulation potential. However, the very low water solubility of HBCD limits the amount of exposure that any organism will encounter. The exposure-based toxicity cutoff that is encountered in aquatic exposures with highly hydrophobic, very low water solubility substances is operational here and this suggests that other exposure routes - primarily dietary exposure - will be most important. Although there are no residue-based toxicity data for aquatic invertebrates or algae, the available exposure-based information suggests a toxicity cutoff due to water solubility limitations. Some residue information for these organism groups could provide confirmation of this view.

The only bird or mammal residue-effect data available was that reported above for rats. However, as noted above, for the sake of generating specific-toxicity PNEC, it is assumed that the lowest residue-based dose-response data represents the lower end of a range of toxic effects caused by an unknown specific toxicity mode of action (i.e. something between baseline narcosis and TCDD-like toxicity). Also, for the sake of argument, it is assumed that this is also applicable to birds and bird eggs. Such assumptions, although not necessarily true in this case, are not unreasonable since organic chemicals may have different key modes of action in different organisms. Fish and invertebrates may exhibit only baseline narcosis since they do not have the biochemical pathway/receptor for the specific mode of action. On the other hand, higher organisms such as mammal and/or birds that may have the appropriate pathway/receptor, exhibit effects due to the specific mode of action at a lower residue level than baseline narcosis, thus preempting effects due to baseline narcosis.

Two residue-based PNEC development scenarios are plausible. In the first, HBCD is assumed to be a narcotic to all organisms. For this scenario a tentative residue-based baseline narcosis PNEC is established starting just below the lower end of the observed chronic toxicity residue range. In this case the rat behavior data can be viewed as the extreme lower tail of chronic narcotic effects that are not routinely evaluated and are of uncertain and questionable relevance to the key ecological response categories of population survival, growth, and reproduction employed in environmental risk assessment.

In the second residue-based PNEC development scenario it is assumed that the rat residue data suggest that HBCD operates by a specific but unknown mode of toxic action that is further assumed to be operational for all organisms. The rat data, which are essentially threshold for modest behavioral responses, are assumed to be at the low end of the residue-based dose-effect relationship for this unknown specific mode of action. Thus, a tentative residue-based specific toxicity PNEC is established just below the lowest rat data.

Total Daily Intake (TDI) Approach

The other general alternative approach to characterize the potential for adverse effects in the environment is to utilize available dietary exposure data as estimates for allowable Total Daily Intake (TDI). Dietary exposure tests of HBCD to laboratory models (rats) have been conducted to determine NOAEL and LOAEL based on exposure to t-HBCD dosed in food for a range of toxic endpoints. The European Commission RAR includes a detailed summary of the test data available ca. 2007 – 2008 (EC 2008). These data are primarily from 28 day exposure periods.

From studies by Chengelis (Chengelis 2001) a LOAEL of 125 mg·(kg-bw·d)$^{-1}$ for liver weight gain was considered reliable (EC 2008). From data reported by van der Ven et al. (2006), a NOAEL/BMD-L of 22.9 mg·(kg-bw·d)$^{-1}$ was selected for the risk characterization (EC 2008). For many of the endpoints tested in the van der Ven et al. study (2006), there were no significant effects reported at the highest exposure level (200 mg·(kg-bw·d)$^{-1}$), particularly in males. These endpoints with no significant effects in males include thyroid weight increase, liver weight increase, pituitary weight increase, and bone density. Using the van der Ven et al. (2006) test data, Germer et al. (Germer et al. 2006) investigated enzyme induction. The only significantly induced enzymatic activities were detected for CYP 2B in males and CYP 3A4 in females from 10 mg·(kg-bw·d)$^{-1}$ (EC 2008). Other reported "very conservative" NOAEL values in the European risk assessment (i.e., Ema et al. 2008) were approximately 10 mg·(kg-bw·d)$^{-1}$ (EC 2008). A NOAEL of 10 mg·(kg-bw·d)$^{-1}$ was also deduced based on a two-generation reproductive toxicity study in rats (EC 2008). Thus, for the purpose of risk characterization in the present study a NOAEL of 10 mg·(kg-bw·d)$^{-1}$ was selected.

5.0 HAZARD CHARACTERIZATION (PBT/POP)

5.1 SCREENING CRITERIA

Table 5.1 presents the PBT and vPvB screening criteria proposed in the European Commission Technical Guidance Document (TGD) (EC 2003). Table 5.2 summarizes the criteria adopted in the United Nations Stockholm Convention for identifying potential POPs (UNEP 2001). Table 5.3 summarizes the screening criteria in the United Nations Economic Commission for Europe (UN-ECE) Protocol on POPs (UN-ECE 1998). The POP screening criteria are generally similar with a few exceptions. In the UN-ECE Protocol a vapour pressure less than 1000 Pa is also included as a screening criterion for potential long range transboundary transport.

The UN-ECE criteria also include: Paragraph 2(a); monitoring or equivalent scientific information suggesting long range transboundary atmospheric transport, and Paragraph 2(b); whether sufficient information exists to suggest that the substance is likely to have significant adverse human health and/or environmental effects as a result of its long range transboundary atmospheric transport (UN-ECE 1998). The Stockholm Convention also requests (Annex D, Paragraph 2), where possible, a comparison of toxicity or ecotoxicity data with detected or predicted levels of a chemical resulting or anticipated from its long range environmental transport (UNEP 2001).

Table 5.1. PBT and vPvB screening criteria according to European Commission TGD (EC 2003).

Category	PBT-criteria	vPvB-criteria
Persistence (P)	Half-life > 60 d in marine water Or > 40 d in freshwater Or > 180 d in marine sediment Or > 120 d in freshwater sediment	Half-life > 60 d in marine or freshwater Or half-life > 180 d in marine or freshwater sediment
Bioaccumulation (B)	BCF [a] > 2000	BCF > 5000
Toxicity (T)	Chronic NOEC < 0.01 mg/l Or CMR[b] Or Endocrine disrupting effects	-

[a] Bioconcentration Factor
[b] Carcinogenic, Mutagenic and Toxic to Reproduction

Table 5.2. POP screening criteria according to the Stockholm Convention (UNEP 2001).

Category	Criteria
1b. Persistence (P)	Half-life in water > 2 months Half-life in sediments > 6 months Half-life in soils > 6 months[a]
1c. Bioaccumulation (B)	Bioconcentration factor (BCF) > 5000 Bioaccumulaiton factor (BAF) > 5000[b] log K_{OW} > 5
1e. Adverse Effects ("T")	(i) Evidence of adverse effects to human health or to the environment that justifies consideration of the chemical within the scope of the Convention; or (ii) Toxicity or ecotoxicity data that indicate the potential for damage to human health or to the environment.
1d. Long Range Transport Potential (LRT)	(i) Measured levels of the chemical in locations distant from the sources of its release that are of potential concern; (ii) Monitoring data showing that long range environmental transport of the chemical, with the potential for transfer to a receiving environment, may have occurred via air, water or migratory species; or iii) Atmospheric half-life > 2 days for a chemical that migrates significantly through air.

[a] Or evidence that the substance is otherwise sufficiently persistent to be of concern within the scope of the Convention

[b] Or evidence that a chemical presents other reasons for concern, such as the high bioaccumulation in other species, high toxicity or ecotoxicity; or monitoring data in biota indicating that the bioaccumulation potential of the chemical is sufficient to justify its consideration with the scope of the Convention

Table 5.3. POP screening criteria according to the UN-ECE Protocol (UN-ECE 1998).

Category	Criteria
1c. Persistence (P)	Half-life in water > 2 months Half-life in sediments > 6 months Half-life in soils > 6 months[a]
1d. Bioaccumulation (B)	Bioconcentration factor (BCF) > 5000 Bioaccumulaiton factor (BAF) > 5000[b] log K_{OW} > 5
1b. Toxicity (T)	Potential to adversely affect human health and/or the environment
1a. Potential Long range Atmospheric Transport (LRT)	Vapour pressure < 1000 Pa Atmospheric half-life > 2 days[c]

[a] Or evidence that the substance is otherwise sufficiently persistent to be of concern within the scope of the protocol

[b] Or if the bioaccumulation potential is significantly lower than these criteria, other factors, such as the high toxicity of the substance, that make it of concern within the scope of the protocol

[c] Or monitoring data showing that the substance is found in remote regions

5.2 Assessment of Persistence and Long Range Transport

This section discusses the categorization of P and LRT. A caveat of the P categorization methodology is that a substance can be categorized as P even if it does not cross bright-line thresholds for degradation in specific environmental media if it is detected remote regions, as is the case for HBCD. We critically evaluate the selection of single compartment half-life P categorization recommendations in the EU RAR and compare model predictions of overall persistence (P_{OV}) for HBCD with benchmark chemicals.

5.2.1 Persistence

The selected degradation half-lives (HLs) for HBCD were presented in Table 2.5. The median HLs in sediments and soils, 35 and 85 days respectively, do not exceed any of the "P" criteria presented in Tables 5.1–5.3 whereas the median HL in water (85 days) does. The upper bound HL estimates for sediments and soils also exceed the "P" criteria. It should be noted that median HL in water was derived from model estimates due to the lack of reliable data. Although there are uncertainties associated with the degradation studies showing elevated persistence in sediments and soils (e.g. (Davis et al. 2004)), there is no definitive evidence demonstrating that these data should be completely disregarded. Overall, based on available data, it can be concluded that HBCD likely fulfills some but not all of the bright-line cut-off "P" criteria presented above

However, it has been argued that overall persistence (P_{OV}) in the environment is a better indicator of chemical persistence rather than comparing multiple half-life estimates for air, water, soil and sediment against bright-line cut-off criteria in these individual compartments (Webster et al. 1998). Indeed models are necessary to assess "P" because it is not possible to trace a chemical in the actual environment. There is no screening "P" criterion for the P_{OV} metric; therefore, P_{OV} predictions for HBCD were compared to P_{OV} predictions for POPs, non-POPs and candidate POPs (Figure 3.18 and Table 3.11). When default (median) HLs for HBCD were selected the P_{OV} is usually at least 1 order of magnitude, or more, lower than five of the six benchmark POPs. Notable exceptions are for benchmark comparisons with aldrin and heptachlor when emissions are assumed to air. HBCD P_{OV} predictions (median HLs) are also about 1 order of magnitude lower than the four candidate POPs. Certain non-POP benchmarks have P_{OV} predictions greater than HBCD. When upper bound HL estimates were selected for HBCD and 100% emissions are to air, the P_{OV} predictions are in a similar range as many benchmark POPs and the candidate POPs. When upper bound HL estimates were selected for HBCD and at least 50% of emissions are to water, the P_{OV} predictions are generally much lower than many benchmark POPs and the candidate POPs (Table 3.11). When lower bound HL estimates for HBCD were selected the P_{OV} predictions are much lower than benchmark POPs (except for heptachlor) and the candidate POPs and more similar with most non-POPs. Without reduced uncertainty for the HL estimates it is difficult to arrive at an unambiguous conclusion regarding P_{OV} benchmarking for HBCD.

Recommendations/Conclusions

It is not possible to definitively conclude whether or not HBCD fulfills the "P" degradation half-life criteria based on the available experimental data. Model assessments of P_{OV} against benchmark chemicals do not provide any definitive categorization for persistence, largely due to a wide range of P_{OV} values for listed POPs. Additional studies on aerobic degradation in water, sediments and soils may help to reduce the uncertainty in the estimated half-lives for HBCD and the "P" criteria comparisons. Further data on atmospheric chemistry of this compound would also be highly valuable for assessments of P_{OV}. Regardless, in the context of POP assessments, HBCD detected in remote regions can always be cited as evidence that HBCD is a substance of concern, i.e., the substance is otherwise sufficiently persistent to be of concern within the scope of the POPs criteria.

5.2.2 Long Range Transport Potential

The estimated sub-cooled liquid vapour pressures of HBCD (t-, α-, β-, γ-) were presented in Table 2.2. All values are far below the vapour pressure criterion presented in Table 5.3. The degradation half-life of HBCD in the atmosphere due to reactions with OH radicals was estimated to range from 0.4 to 4 days, considering the expected uncertainties associated with reaction rates and radical concentrations. While the median value (1.3 d) is below the criterion value of 2 days, the uncertainty associated with the estimate includes values that exceed the criterion.

The CTD, TE and $eACP_{10}$ of HBCD compared to POPs, non-POPs and candidate POPs are shown in Figures 3.18 and 3.22, respectively. Similar to P_{OV}, the CTD, TE and

$eACP_{10}$ predictions for HBCD are lower than some POPs (e.g. HCB, PCBs) included as benchmarks but higher than others (e.g. aldrin, heptachlor). The non-POPs included in the study also exhibit a range of CTD, TE and $eACP_{10}$ values, some higher than HBCD and some lower. Hence, this element of the benchmarking exercise is also ambiguous. Overall, the mass balance modelling suggests that HBCD is subject to a low to moderate level of long range transport potential (compared with existing POPs). Long range transport potential benchmark estimates also show that the potential is lower when emissions are greater to the water compartment than to air.

Available monitoring data from remote regions indicate that HBCD has the potential to reach remote regions. While it is true that concentrations in biota appear to be much lower than in industrialized source regions, the studies documenting HBCD in various organisms in the Arctic provide sufficient evidence of LRT potential according to criteria presented in Tables 5.2 and 5.3. It should be recognized that some sources of emissions are expected in "remote" regions as a result of historical and current human activities. For example, people in remote areas have been using products that are expected to contain HBCD (e.g. insulation, textiles). These "source" emissions in "remote" regions may be exacerbated by the common practice of burning garbage (products containing HBCD) in Northern communities. The potential emissions related to use and disposal of HBCD-containing products in remote areas is unclear. Thus, without improved knowledge of relative contributions of quantities released in remote regions compared with quantities released in source regions that are measured in remote regions it is difficult to ascertain the actual quantity in remote regions that is explicitly the result of long range transport.

HBCD seems to have a "low" to "moderately-low" LRT potential based on model estimates; however, detectable levels in remote regions suggest that emissions have been sustained at high enough rates to allow for these findings. Use patterns, monitoring data and emissions estimates suggest that HBCD has been used for at least a few decades. Thus, continuous emissions combined low transport efficiency can still result in detectable concentrations in remote regions.

Recommendations/Conclusions

The uncertainty in the estimated long range transport potential could be reduced if additional studies characterizing the degradation half-life of HBCD in air, water, soil and sediment were available. In particular, empirical data on atmospheric chemistry of this compound (e.g. OH radical reactivity) would be highly valuable. However, available monitoring data indicate that HBCD is present in remote regions. This is considered sufficient evidence that HBCD has long range transport potential as identified by the screening criteria.

5.3 Assessment of Bioaccumulation

The bioconcentration studies cited in (EC 2008) and (Veith et al. 1979; Drottar and Kruger 2000; TemaNord 2008) had BCFs > 5,000 (i.e. "B" or "vB"). These studies used co-solvents to conduct the experiments, which introduces some uncertainty into the interpretation of the BCFs since guidelines recommend against the use of solvents for

bioconcentration tests (OECD 1996). A recent publication suggests that co-solvents and dispersants (surfactants) may not drastically change BCF estimates (Yakata et al. 2006), particularly in the context of the large uncertainty generally associated with estimating BCFs for hydrophobic substances (Arnot and Gobas 2006). However, Yakata et al. (2006) tested six single compounds individually and only one was above its water solubility limit. Thus, it is difficult to establish the relevance of the information in Yakata et al. (2006) to the HBCD situation – a mixture of isomers with differing water solubilities. Overall, the BCF values derived from these studies are believed to be generally representative and exceed the "B" and "vB" screening criteria outlined in Tables 5.1, 5.2 and 5.3. Furthermore, the estimated log K_{OW} values (Final Adjusted Value, see Table 2.2) for the three major isomers are all > 5, in excess of the "B" and "vB" criteria outlined in Tables 5.1, 5.2 and 5.3.

The majority of monitoring data also support the bioaccumulation categorization using BCF data such that HBCD (or at least certain isomers) are found to biomagnify (i.e. biomagnification factors, BMF, are > 1) and exceed BAF criteria in the environment (e.g. (Tomy et al. 2004; Law et al. 2006a; Sormo et al. 2006; Tomy et al. 2008)). Interpretations of the monitoring data are complicated by the observations that HBCD undergoes bioisomerization in biota and perhaps other compartments of the environment. Hence, it remains uncertain if all diastereomers are bioaccumulative in the field. For example, Tomy et al. (2008) reported trophic magnification factors (TMF) of 2.1 and 0.5 for α- and γ-HBCD respectively in a study of an eastern Arctic food web. There are other examples where different conclusions could be reached regarding bioaccumulation potential depending on what data are considered. Trophic level and lipid-normalized biomagnification factors (BMF_{TL}s) for α-HBCD were greater than 1 for many predator-prey comparisons (e.g. narwhal **:** polar cod, beluga **:** polar cod) but not in all cases (e.g. beluga : redfish $BMF_{TL} < 1$). Data from a food web study in the Western Canadian Arctic (Ismail et al. 2009) seem to contradict the patterns seen in Tomy et al. (2008). For example, the calculated BMF_{TL} of α-HBCD for beluga **:** polar cod is 0.4 (although BMF_{TL}s greater than one were observed in other cases, e.g., beluga **:** herring and beluga **:** Arctic cisco). There also appear to be discrepancies in field-derived BMFs for ringed seal **:** polar cod feeding relationships among different studies. For example, Sormo et al. (Sormo et al. 2006) reported a lipid-normalized BMF of approximately 11 (based on mean values, total HBCD) for ringed seals and polar cod sampled from Svalbard whereas data reported in (Tomy et al. 2008) yield a lipid-normalized BMF of 0.1 (mean values, total-HBCD), i.e., two orders of magnitude lower.

Recommendations/Conclusions

Although there are uncertainties associated with the laboratory BCF data for fish and field data characterizing bioaccumulation potential, the weight of evidence suggests that HBCD is "B" according to the screening criteria presented in Tables 5.1, 5.2 and 5.3. It remains uncertain if all diastereomers are bioaccumulative in all species based on the uncertainty of the bioisomerization and biotransformation rates for each isomer.

5.4 ASSESSMENT OF TOXICITY (HAZARD)

The toxicity hazard evaluation of HBCD was carried out with respect to classification as a PBT based on European Commission "T" criterion and as a POP with respect to the descriptive hazard based criteria (Tables 5.2 and 5.3). There is no available evidence to suggest that HBCD is carcinogenic, mutagenic, a reproductive toxicant or an endocrine disrupting chemical. The exposure-based aquatic ecotoxicity data available for HBCD are of limited value for screening categorizations as all exposure data are confounded by water solubility/bioavailability issues including the use of a cosolvent or use of a generator column. In OECD testing guidance for difficult substances and mixtures (HBCD is both) there is a specific caution concerning the use of these practices. Notwithstanding thest problems, the ecotoxicity data show that no aquatic exposure-based toxicity effects occur at or below the water solubility limit for γ-HBCD (effectively, t-HBCD for toxicity testing purposes). The water solubility limit of γ-HBCD is about 2 to 3 $\mu g \cdot L^{-1}$, which happens to be about 3 to 5 times lower than the chronic NOEC PBT criterion of <10 $\mu g \cdot L^{-1}$. Since reliable aquatic exposure-based toxicity data cannot be obtained at concentrations that exceed a chemical's water solubility limit, it is impossible for HBCD to be above the EU PBT criterion. For the majority of chemicals with water solubility limits below "T" critieria (i.e. <10 $\mu g \cdot L^{-1}$), such criteria are generally not applicable. The concerns with using aquatic-based exposure criteria for screening assessments are well documented in the scientific literature (McCarty and Mackay 1993; Gobas et al. 2001; Maeder et al. 2004). The available evidence for HBCD suggests it possesses a baseline narcotic mode of toxic action, the least toxic mode of action for organic chemicals. Based on the available ecotoxicity data, HBCD is considered to have "low toxicity".

The POP assessment programs have no quantitative criteria for "T". It has been suggested that criteria from the Dutch "PTB project" can be used as an assessment for "T" in UN-ECE POP assessments (Lerche et al. 2002). Following this approach, available mammalian and ecotoxicity data for HBCD suggest that HBCD is a Category 1 chemical (the least "toxic" category). The majority of the qualitative statements for screening potential toxicity and adverse effects in the UN Stockholm Convention (Table 5.2) and the UN-ECE POP Protocol (Table 5.3), respectively, seek to identify substances with the potential for toxic effects. All substances have the "potential for adverse effects", based on a function of the dose and the duration of exposure. Thus, strictly speaking, these qualifying statements are ineffective for screening potential POPs for "T". More appropriately, both the UN-ECE Protocol and the UN Stockholm Convention recognize that substances should be evaluated for their potential adverse effects as a result of environmental exposures due to long range environmental (or atmospheric) tranport, i.e., the risk of toxicity occurring in remote regions. These more comprehensive assessments are beyond the hazard based screening criteria, but are arguably more relevant for chemical management objectives. The following section provides comprehensive risk characterizations based on the likelihood of toxic effects from HBCD in "local/near point source", "source" region and "remote" regional scales.

Recommendations/Conclusions

A detailed evaluation of key exposure-based aquatic toxicity studies for HBCD indicates that there are a number of uncertainties. The data are largely unusable for a valid screening assessment of the toxicity of HBCD for PBT/POPs categorization. The PBT and POP screening criteria are not effective for classifying very hydrophobic substances, especially mixtures such as HBCD, for their toxicity potential. It is recommended that future toxicity testing focus on improving data requirements for evaluating the potential for adverse effects in the environment. These data endpoints should focus on body/tissue-residues.

6.0 RISK CHARACTERIZATION

6.1 ECOLOGICAL RISK ASSESSMENT

It is critical to assess whether HBCD concentrations measured or modelled in different environmental compartments of remote areas and elsewhere are likely to cause adverse effects. Adverse effects of HBCD are necessarily a function of actual dose estimates, i.e., exposure, and estimates for effects or no effects levels. In this section the critically evaluated and selected metrics for effect and no effects levels based on the two alternative approaches - body/tissue-residue and TDI - are compared with estimates of actual exposure in the environment. The primary focus is the likelihood of significant adverse effects in remote regions (i.e. the Arctic); however, evaluations for the likelihood of adverse effects in source regions and near recognized point sources (impacted areas near industrial sites) of HBCD are also presented.

6.1.1 Body/Tissue Residue Based Risk Assessment

Figure 6.1 contains the tentative residue-based PNECs for the two proposed scenarios that were developed in Section 4.2.3 and presented in Figure 4.2. The ranges of European monitoring data for invertebrates, freshwater and marine fish, marine bird eggs, and marine mammals as summarized by the European Commission RAR (2008) are also included. The limited data for freshwater invertebrates are combined with the more abundant and broader range of marine invertebrate data in Figure 4.2. The lower section of each monitoring data range represents "remote" regions, including Arctic sites, while the upper section represents mostly sampling data near known industrial sites ("local/near point source"), as evaluated in the European Commission RAR (2008).

In the first risk assessment scenario HBCD is assumed to be a baseline narcotic to all organisms. All of the monitoring data are below this residue-based PNEC indicating that no adverse effects are expected due to exposures of organisms in the field, either in "remote" areas or in "source" regions" and "local/near point source" areas close to production and industrial locations.

The conclusions based on the second risk assessment scenario, where HBCD is assumed to operate by a specific mode of toxic action for all organisms, are more ambiguous. Most of the invertebrate monitoring data are below the specific-toxicity residue-based PNEC. The upper part of the invertebrate distribution at this PNEC is for mussels

sampled in a contaminated Norwegian Fjord. The remainder of the invertebrate distribution starts more than an order of magnitude below the upper end of the range. Available monitoring data for marine fish are well below the specific-toxicity residue-based PNEC and hence no adverse effects are expected. However, for freshwater fish the upper third of the monitoring data exceeds the specific-toxcity residue-based PNEC. Similarly, for marine mammals, about 1/3 of the data are above the specific-toxicity residue-based PNEC range. The upper limit of the the bird data also enters this range. These results suggest that freshwater fish and marine mammals in contaminated regions near local point source emissions may exhibit some adverse effects from HBCD exposure but that those organisms in remote regions would not. The reported levels in birds from contaminated industrial regions/local sites are also near the threshold of where adverse effects from HBCD may occur whereas levels from remote regions are not. A more detailed discussion of the potential environmental risks associated with the field monitoring data and the PNEC values is presented below.

The invertebrate monitoring data are not persued further since the data are below the residue-based PNEC, except for mussels from a known contaminated site ("near/local point source). Rather than use the summary ranges presented in the European Commission (2008) report, the various monitoring data reviewed there were examined and specific information judged to be representative of "local" (near known point sources of HBCD release), "source" (in the general region where point sources are found), and "remote" (largely Arctic) observations were selected. This is appropriate as the initial risk assessment analysis, presented above, indicated that HCBD levels observed fish, mammals, and birds in remote areas, such as the Arctic and near-Arctic, did not appear to have concentrations that are associated with possible adverse effects. As the likelihood of adverse effects in biota in remote areas is a key issue in POPs classification, a more detailed evaluation is necessary to ensure the validity of this conclusion. Figures 6.2, 6.3 and 6.4 contain the risk characterization results using monitoring data from local, source, and remote regions, respectively (Tables 3.3 to 3.5). For Figures 6.2, 6.3 and 6.4 both bird egg levels and body tissue levels are included. Discussions on the suitability of using some of the bird monitoring data were presented in Section 3.3.7.

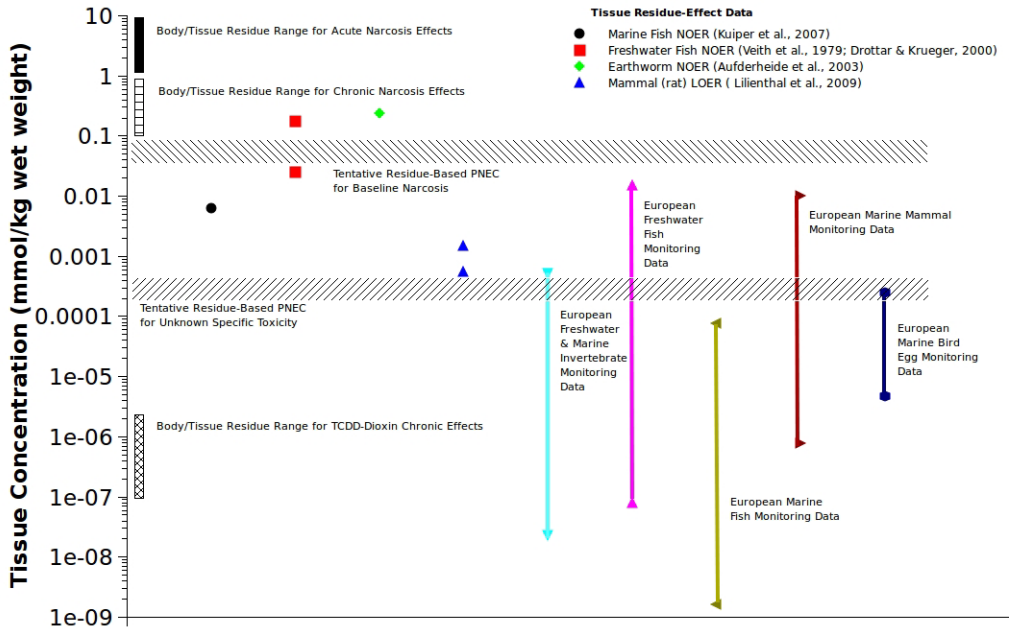


Figure 6.1. Residue-based risk assessment with summary monitoring data from the EU RAR (EC 2008).

Inspection of Figure 6.2 (monitoring data from "remote" areas) indicates that adverse effects in fish, birds and marine mammals are unlikely due to the low measured tissue levels of HBCD. This is true for both mode of toxic action assumptions, including the unidentified specific mode of toxic action that has a residue-based PNEC approximately 100 times lower than the baseline narcosis criterion. The marine mammal data has the largest range of the three organism groups. The upper end of this range is also the closest of the three ranges to the unidentified residue-based specific mode of toxic action PNEC, but does not enter it. Both of these observations for marine mammals are likely due to the wide ranging habits of some of these organisms and their very high lipid content (related to the very substantial blubber content in their bodies). Lipid concentrations represent a challenge for interpretation due to a lack of knowledge of target-to-storage lipid ratios; therefore, only wet weight concentrations are considered in the present assessment.

In terms of the potential for significant adverse effects from exposures to HBCD at different geographic scales, the information presented in Figures 6.1 to 6.4 suggest that there is potential for adverse effects occurring in organisms in areas near a known point source emission (Figure 6.4). However, adverse effects as a result of the long range transboundary atmospheric transport are unlikely to occur in any organisms in remote regions including those occupying higher trophic levels (i.e. marine mammals). This brief and simple residue-based risk assessment exercise, despite its uncertainties and assumptions, provides a clear risk conclusion for HBCD.

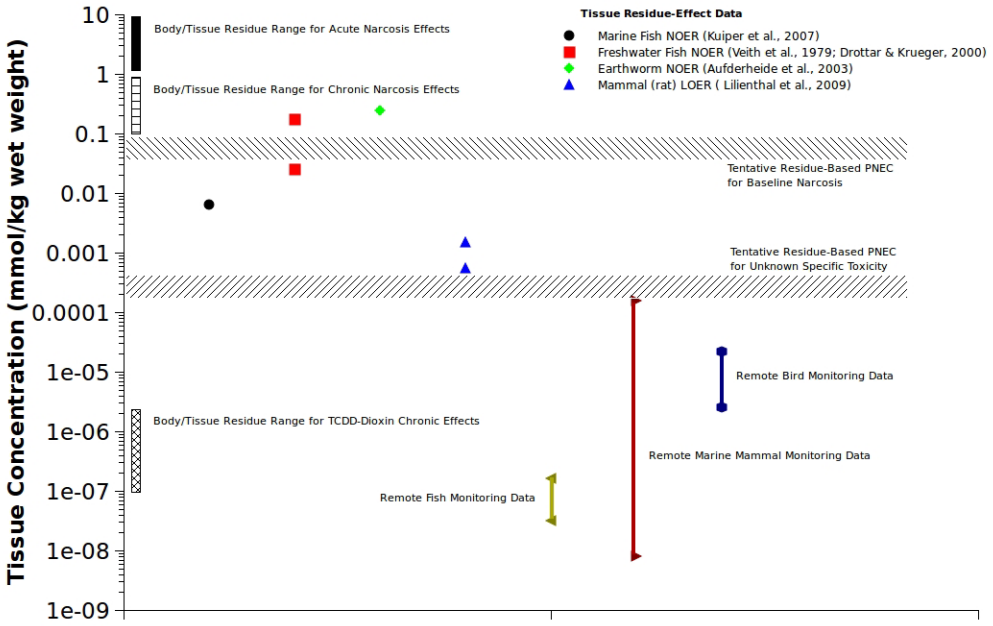


Figure 6.2. Residue-based risk assessment with available "remote" region monitoring data.

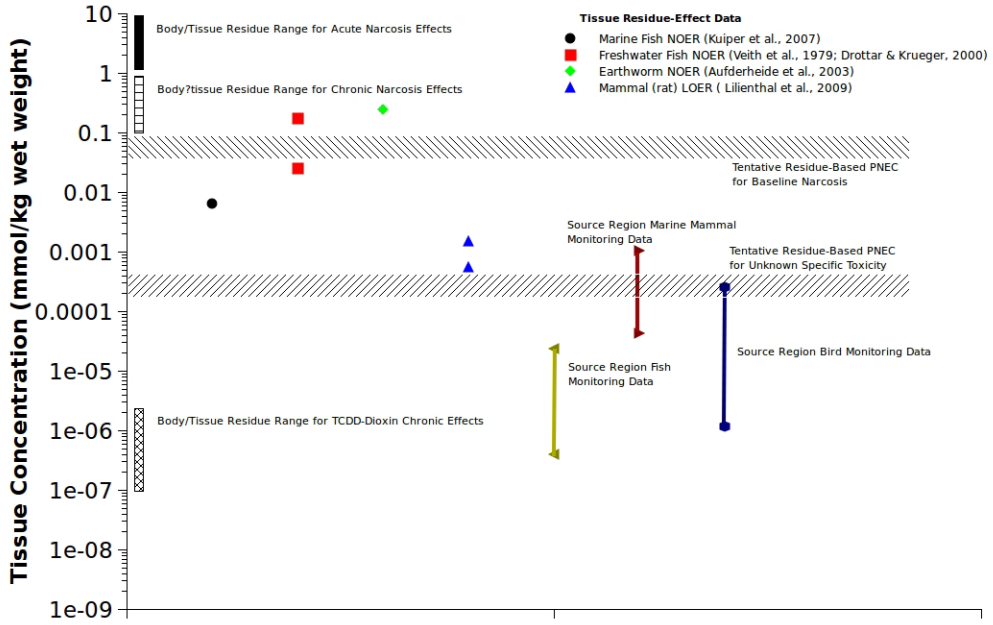


Figure 6.3. Residue-based risk assessment with available "source" region monitoring data.

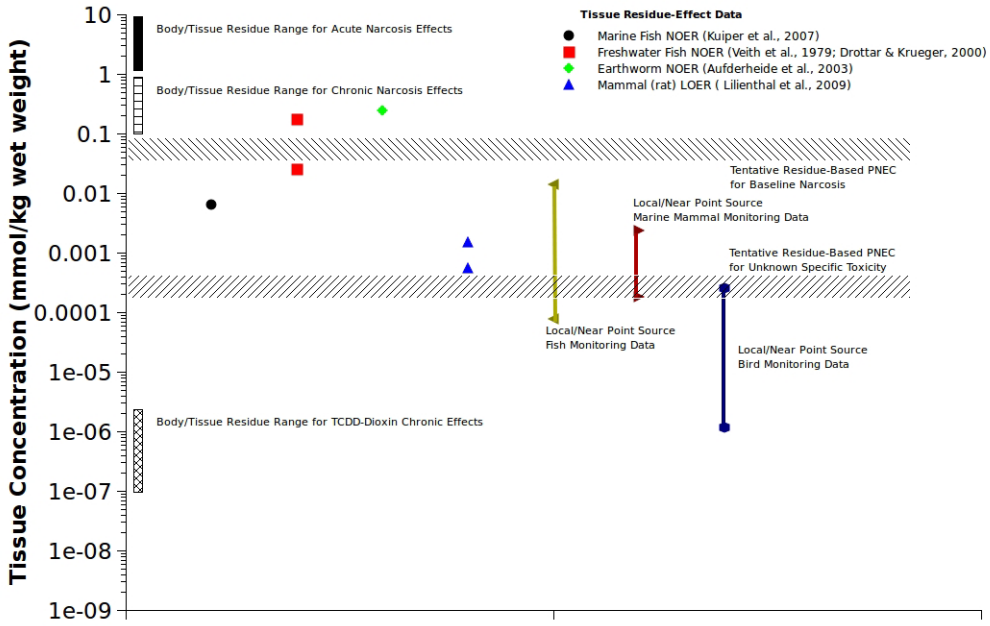


Figure 6.4. Residue-based risk assessment with available "local/near point source" monitoring data.

6.1.2 Total Daily Intake Based Risk Assessment

A second alternative approach for assessing the potential for adverse effects in the environment compares estimates of total daily intake rates for organisms in the environment with estimates of total daily intakes from laboratory tests associated with NOAEL (10 mg·(kg-bw·d)$^{-1}$). The highest reported t-HBCD concentrations in piscivorous marine mammals in remote regions (Svalbard) are for adult male ringed seals (8 to 25 yrs old) (Sormo et al 2006). The average, median, and upper bound values are 19.6, 17.0 and 34.5 ng·g-lipid^{-1}, respectively. The unit emission rate in the RAIDAR model was calibrated to obtain a concentration of 34.5 ng·g-lipid^{-1} in the representative piscivorous marine mammal. The corresponding total daily dietary intake of t-HBCD to obtain a steady state concentration of 34.5 ng·g-lipid^{-1} in the marine mammal is about 8.1×10^{-5} mg·(kg-bw·d)$^{-1}$. This suggests that current estimates of total daily dietary intake of t-HBCD in piscivorous marine mammals in remote regions are approximately 5 orders of magnitude below available NOAEL data. There are some recognized uncertainties with this approach, in particular with regards to the actual steady state conditions in the environment (mammal and diet) and from the laboratory studies; however, a similar general approach for characterizing potential risks to Arctic marine mammals using mammalian reference dose metrics from the laboratory for PFOS has been suggested (Kelly et al. 2009).

Estimates for the 95th percentile of concentrations in piscivorous marine mammals from source region and local (near point source) scale monitoring data were also selected for comparisons with the NOAEL TDI estimate of 10 mg·(kg-bw·d)$^{-1}$ using the approach described above. The upper bound concentration estimates for regional source and local/near point source piscivorous marine mammals are about 750 and 6,300 ng·g-lipid^{-1}, respectively. These values correspond to RAIDAR total daily dietary intake rate estimates of about 1.8×10^{-3} and 1.5×10^{-2} mg·(kg-bw·d)$^{-1}$ for source region and local/near point source piscivorous marine mammals, respectively. These estimated TDI rates are also well below the dietary consumption NOAEL.

The approach illustrated in the present evaluation could be further refined using a bioaccumulation model specific for the organisms in which the data are reported (i.e. ringed seal) rather than the general parameters used to calculate bioaccumulation for the representative marine mammal in RAIDAR. A more comprehensive assessment should also include TDI estimates for pups and calves of marine mammals and an uncertainty analysis. For initial screening level purposes the present estimation is deemed sufficient in the context of the difference between the estimated intake rates and the NOAEL. More comprehensive analysis should also be considered for other monitoring data in remote regions, most notably for polar bears and for certain bird species. There are presently no effects data available for risk characterization from bird species; therefore, the mammalian data would likely have to be considered.

7.0 CONCLUSIONS

7.1 ECOLOGICAL RISK ASSESSMENT

Two versions of a body/tissue residue-based risk assessment were conducted. The first was based on baseline narcosis and the other was based on an unidentified and assumed mode of toxic action associated with PNEC body/tissue residues 100 times lower than baseline narcosis. Neither supported classification of HBCD as a POP on the basis of toxicity as neither assessment indicated that adverse effects are expected in upper trophic level organisms living in remote areas distant from known point-source emissions of HBCD.

The likelihood of adverse effects to piscivorous marine mammals (recognized trophic position associated with high exposure levels) was also evaluated by comparing estimates of total daily intakes of t-HBCD in the environment with estimates of total daily intakes associated with NOAELs from laboratory studies. The corresponding total daily dietary intake of t-HBCD to obtain a steady state concentration corresponding with the highest measured concentration in a piscivorous marine mammal in a remote region is about 5 orders of magnitude below the NOAEL. The corresponding total daily dietary intake of t-HBCD to obtain a steady state concentration corresponding with the upper bound of measured concentrations in piscivorous marine mammals in "source" and local scale (near point source) regional environments ranges from about 2 to 3 orders of magnitude below the NOAEL.

Simply because a substance meets screening level hazard categorization criteria and can be detected in remote environments does not constitute a potential for adverse effects or indicate there is a likelihood that adverse effects will occur. A more scientifically justifiable approach is to estimate the actual risk for adverse effects by evaluating measured and/or modelled exposure levels in the environment with effects and/or no-effects levels. Following this standard risk-based approach current exposure data for HBCD in upper trophic level species in remote regions show that exposure levels are below current estimates associated with NOAELs. The analysis does show some cause for concern for HBCD in "local/near point source" regions. It is recommended that these instances be considered for more comprehensive site-specific risk assessments conducted within regulatory frameworks typically employed for non-POP substances. These recommendations are further supported by the modelled and measured spatial trends in the environment.

7.2 POP ASSESSMENT

7.2.1 Persistence and Long Range Transport Potential

Based on the assessment of degradation studies and model output considered in this report, it cannot be definitively concluded that HBCD does or does not meet the criteria for Persistence (P) and Long Range Transport (LRT) potential. Since there do not appear to be any analytical or methodological issues related to the majority of monitoring studies from remote regions, these data must be considered reliable. Thus, according to the POP

screening criteria (see Tables 5.2 and 5.3) the presence of HBCD in remote regions can be used to arrive at the conclusion that HBCD is persistent and exhibits sufficient LRT potential to warrant consideration as a potential POP. The model benchmarking results demonstrate no definitive conclusions regarding P and LRT assessments for HBCD.

Monitoring data support the previous assessment decisions for "P" and "LRT potential" as defined by the POP criteria, i.e., a substance does not have to unequivocally fulfill the P- and LRT-criteria if it is present in wildlife distant from areas of its use.

7.2.2 Bioaccumulation

Although there are uncertainties associated with the laboratory BCF data for fish and assessments of bioaccumulation potential based on field biomonitoring data, the weight of evidence suggests that HBCD is bioaccumulative according to existing POPs screening criteria (BCF > 5000). All isomers have estimated log K_{OW} > 5 (See Table 2.2) which is also sufficient to fulfill this particular screening criteria. However, it remains uncertain if all diastereomers are bioaccumulative in the field, based on the uncertainty of the bioisomerization and biotransformation rates for each isomer.

Available laboratory and field monitoring data and physical-chemical property data generally support the classification of HBCD as "B".

7.2.3 Toxicity and Potential for Adverse Effects

There are no quantitative screening criteria for toxicity and adverse effects according to the UN-ECE POP Protocol and UN Stockholm Convention (Tables 5.2 and 5.3). Available laboratory data show that HBCD is a substance of low toxicity, i.e., likely a baseline narcotic mode of toxic action for aquatic organisms. Substances nominated for POP designation should be evaluated to determine if the substance is likely to have significant adverse human health and/or environmental effects as a result of its long range transboundary atmospheric transport (UN-ECE 1998) including, where possible, a comparison of toxicity or ecotoxicity data with detected or predicted levels of a chemical resulting or anticipated from its long range environmental transport (UNEP 2001). This approach is generally considered a risk characterization or risk assessment. Modelling and monitoring data show that upper trophic level organisms that consume fish have the highest exposure levels to HBCD. The presented residue-based and TDI-based risk assessments indicate that no adverse effects are expected in upper trophic level organisms in regions remote from locations associated with the release of HBCD to the environment. Thus, HBCD does not meet the general and vigorous component of the POPs "T" criterion relating to the likelihood of adverse effects in remote regions.

Neither a detailed interpretation of available laboratory toxicity information, nor two separate risk assessment approaches employing available monitoring data for upper foodchain organisms in remote regions, supports the classification of HBCD as "T" according to POPs classification criteria.

7.3 COMMENTARY ON PREVIOUS ASSESSMENTS

Previous Assessments

HBCD has been subject to a PBT assessment according to criteria presented in the European Commission Technical Guidance Document (TGD) (EC 2003; EC 2008). According to the screening criteria of the UN Stockholm Convention (UNEP 2001) and the UN-ECE Protocol, the TemaNord assessment proposes a POP categorization for HBCD (TemaNord 2008). The major findings of these previous assessments are briefly reviewed below with a focus on the proposed POP categorization.

It was concluded in the European RAR (EC 2008) that HBCD fulfills the "B" (and "vB") criterion as well as the "T" criterion (Chronic NOEC *Daphnia* survival, reproduction growth 3.1 $\mu g \cdot L^{-1}$; Chronic LOEC *Daphnia* reduced length 5.6 $\mu g \cdot L^{-1}$). According to the European Commission, HBCD does not unequivocally fulfill the specific P-criterion, with some reliable studies indicating biodegradation can occur (EC 2008). For example, when adjusted to 12 ^{o}C, some experimentally-derived degradation half-lives in aerobic sediments are > 180 d but not others. The scientific validity of this temperature adjustment was not questioned (EC 2008). Additional evidence cited in favour of fulfillment of the P-criterion include the presence of HBCD in wildlife and abiotic samples remote from areas of use and the presence of HBCD in deep sediment layers. The final decision, that HBCD fulfills the PBT-criteria overall, was also justified based on the fact that the TGD specifically advises that, "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" (EC 2003).

Similar data and arguments were presented in the POP proposal (TemaNord 2008) to arrive at a decision that HBCD fulfills the screening criteria presented in Tables 5.2 and 5.3. HBCD was concluded to fulfill the "P" criteria primarily based on i) temperature adjusted degradation half-lives in aerobic sediments and ii) a study showing "no degradation observed" for soil. The presence of HBCD in remote regions and deep sediment layers was also discussed in the context of "P" screening criteria. The long range transport potential of HBCD was assessed through estimates of atmospheric half-life, application of fate and transport models and consideration of available monitoring data in remote regions. (TemaNord 2008) concluded that HBCD fulfilled the numerical criteria for atmospheric half-life and vapour pressure and showed similar LRT potential as mid-sized PCBs and PBDEs (based on model output). The strongest evidence of LRT potential presented in (TemaNord 2008) is the summary of monitoring data from remote regions which demonstrate the HBCD is present in these areas. HBCD was classified as "B" in (TemaNord 2008) because it fulfills the numerical criteria for "B" based on experimental BCF data (range 8973 – 21940) and experimental log K_{OW} (5.62).

The POP proposal (TemaNord 2008) concluded that HBCD has "high aquatic ecotoxicity" based on tests with *Daphnia magna* showing a 28d-NOEC of 3.1 $\mu g \cdot L^{-1}$ (Drottar and Krueger 1998) and on a growth inhibition test with *Skeletonema costatum* (72h-EC50 of 52 $\mu g \cdot L^{-1}$; (Desjardins et al. 2005)). No criteria or justifications were presented to support the "high aquatic ecotoxicity" proposal. Further, the confounding factors associated with these tests (e.g. use of cosolvent) were not thoroughly discussed.

While available laboratory studies with mammals do not indicate that HBCD is carcinogenic, mutagenic or toxic to reproduction, effects in liver, thyroid gland and thyroid homeostasis were cited as additional reasons for concern. There was no clear assessment for the potential of HBCD to adversely affect human health and/or the environment. Appendix 4 of the TemaNord POP proposal includes a summary table of listed sources concluded to cause potential risks as conducted in the EU RAR ("conclusion iii") (TemaNord 2008); however, it is noteworthy that all of the listed sources are specific or generic industrial sites and not remote locations.

Critical Appraisal of Previous Assessments

Substances identified in category-based assessments may not always prove to require regulatory action when subjected to a more rigorous examination (Arnot and Mackay 2008; McCarty and Arnot 2008; Wu et al. 2008). In this section we critically evaluate the previous assessments, with a focus on the POP proposal.

The model – monitoring corroboration in the present study suggests that estimates for HBCD emissions to the environment proposed in the RAR (EC 2008) are, to a first approximation, reasonable. The modelling analysis suggests that the proportion of emissions to air may be greater than previously assumed and "actual" emissions may be different. Ultimately more reliable monitoring and half-life data can be used to further evaluate HBCD emission rates and mode-of-entry to the environment. The findings in the present study also support conclusions from the previous assessments that there are concerns for HBCD released to the environment at local scales near industrial sites.

HBCD was classified as "P" in (TemaNord 2008) based primarily on temperature-adjusted degradation half-lives of α- and γ-HBCD in aerobic sediments (12 ^{o}C) and negligible degradation in soil from one particular study. There is no consensus on the validity of applying a temperature-correction to adjust biodegradation rate constants. In fact, Boethling et al. (2009) specifically recommend against such adjustments for screening level persistence categorization using half-life criteria. However, according to both Annex D of the Stockholm Convention (UNEP 2001) and the UN-ECE POPs protocol (UN-ECE 1998), a substance can be classified as "P" if there is evidence that the substance is, "otherwise sufficiently persistent to be of concern within the scope of the convention/protocol". Since the presence of HBCD in remote regions can be interpreted as evidence that a compound is sufficiently persistent and the quality and representativeness of the monitoring data are not in dispute, the overall screening conclusions in TemaNord (2008) regarding "P" are deemed reasonable. The monitoring data from remote regions also provide evidence of sufficient long range transport (LRT) potential to be considered under the POP protocol. Similarly, based on the evaluation of available data, the screening assessment of bioaccumulation potential, "B", in TemaNord (2008) is also deemed reasonable.

The previous conclusion that all key aquatic toxicity data are reliable for PBT assessment and can also be considered for the POP classification (TemaNord 2008) is not reasonable. As detailed in the present study, there are significant reliability issues with regards to most of the previously selected aquatic-exposure based "T" endpoints. For the PBT

assessment, HBCD is essentially categorized as "T" because the substance is hydrophobic. There are no indications of toxic effects at or below the water solubility limit; however, the water solubility limit (~ 2 to 3 $\mu g \cdot L^{-1}$) is below the PBT "T" screening level criterion (i.e. "T" < 10 $\mu g \cdot L^{-1}$). Following this approach, all substances with water solubility limits < 10 $\mu g \cdot L^{-1}$ must be considered "T" for the EU PBT assessments. Clearly this policy needs further consideration for scientifically defensible assessments of commercial chemicals.

With respect to POP categorization, the TemaNord (2008) justification for toxicity and potential adverse effects is also considered to be unsatisfactory. Key concerns regarding the applicability of available toxicity data are not thoroughly discussed. The POPs proposal relies heavily on the RAR ecotoxicity data and the "T" classification in the RAR, a conclusion which is not supported by our findings. Most importantly, the proposal considers the toxicity data in isolation without quantifying the potential for adverse effects as a result of actual exposures in the environment. Further deliberations on this topic can be found in Section 9.5.4.

In summary, there were no comparisons of toxicity or ecotoxicity data with detected or predicted levels of HBCD in the POP proposal (TemaNord 2008) for either industrialized or remote regions despite the abundance of available data. The present risk assessment shows that based on available data there do not appear to be significant adverse health and/or environmental effects as a result of long range transport.

The "P" and "B" classifications in previous assessments are deemed reasonable despite the discussed uncertainties; however, as discussed in the present study there are concerns regarding the previous assessments of toxicity and potential for adverse effects. The ecological risk assessment summarized in 7.1 provides an example of the type of analysis that should be considered.

7.4 RECOMMENDATIONS

The risk characterization in the present study is considered to be a more scientifically defensible approach for assessing the potential adverse effets for HBCD and its isomers on human health and the environment than previous assessments. Evaluations for remote regions show low (residue-based approaches) to very low (total daily-intake-based approach) risk based on available monitoring and toxicity data. Based on the current estimates of low likelihood for adverse effects in remote regions, the relatively rapid response times of HBCD in many environmental compartments to reduced emissions compared to categorized POPs, and spatial monitoring trends, it is recommended that efforts to reduce emissions at point sources (production and use facilities) continue. Certain "local/near point source" areas may require additional site-specific risk assessment work to establish appropriate regulatory activites.

The proposed methods and current results can be considered a starting point for any further assessments for HBCD, and more appropriately, the individual diastereomers. The present data limitations prevent a more comprehensive quantification of associated

uncertainties with the risk assessment results. There are a number of recommendations throughout the document that should be considered to reduce uncertainties in future assessments of HBCD. In particular, the model sensitivity analysis shows that metabolic biotransformation rates are the chemical properties that contribute the greatest variance to estimated exposure levels in higher trophic level organisms. Better information is needed to characterize diastereoisomer conversion in the environment and food webs. Recent data suggest that isomerization may be occurring in many compartments of the environment and perhaps during sample storage and analysis. Standardized methods for measuring HBCD and its isomers are necessary to reduce uncertainties in the monitoring data. The focus of future toxicity assessments should be for obtaining body/tissue residue levels associated with effect or no effect endpoints.

Most monitoring data presently available are for "site-specific" areas near production sites. Monitoring data are needed for regional areas, in particular for receiving environments (air and water) to reduce uncertainty in the present assessment. Monitoring studies should be continued and expanded in representative regional environments such as the Sweden/Baltic area. Other key environmental compartments need to be measured to have a more complete characterization of time trends and chemical distribution. Environmental compartments in remote regions also need to be monitored more thoroughly to test the hypotheses proposed in the present evaluation based on the models and available measurements. As more monitoring and test data become available, more modelling scenarios can also be considered.

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9.0 APPENDIX

9.1 PHYSICAL CHEMICAL PROPERTIES

The general objectives in this section are to obtain and review measured and predicted estimates of physical-chemical properties relevant for modelling the fate and bioaccumulation of HBCD in the environment. Based on this review, values were then selected to derive thermodynamically consistent estimates, final adjusted values (FAVs), which were then used for modelling and data analysis in the report. For the physical-chemical property consistency procedure, relative variance values were selected for the property estimates (Schenker et al. 2005). Discrepancies between the values in the present study and in the Draft EU Risk Assessment Report (RAR) are discussed.

Molar mass

The molar mass (molecular weight) of HBCD is 641.7 $g.mol^{-1}$ (U.S. EPA 2009).

Melting point

The RAR lists and discusses a variety of melting point ranges for HBCD (EC 2008). The melting intervals for two commercial products are 175 to 183 °C and 187 to195 °C. EPI Suite estimates a melting point of 180 °C (453 K), which is a weighted value from three melting point submodels and is not specific for the diastereoisomers or commercial (technical) mixtures (U.S. EPA 2009). A melting point of 190 °C (463 K) was selected as an average value as input data for EUSES (EC 2008). For consistency with the RAR the same melting point T_M (K) is selected for technical HBCD for the present study; however, it is recognized that mixed isomer ranges lead to some uncertainty in the selection of this value. The RAR lists ranges of the melting point for the HBCD diastereoisomers as 179 to 181 °C, 170 to 172 °C and 207 to 209 °C for α-, β-, and γ-HBCD, respectively (EC 2008).

Fugacity ratio

The fugacity ratio F (unitless) was not included in the RAR and is necessary for estimating the properties of the subcooled liquid which are used as a reference state for fugacity and activity calculations and for the thermodynamic consistency calculations. The fugacity ratio can be estimated as

$$F = \exp(6.79(1 - T_M/T)) \quad (9.1)$$

where T is the system temperature (K) and the assumption that the entropy of fusion is 56.5 $J \cdot K^{-1} \cdot mol^{-1}$ (Mackay 2001). Thus, for technical HBCD (T_M = 463 K), F is 0.023. The diastereoisomer specific F values are 0.029, 0.036, and 0.015 for α-, β-, and γ-HBCD, respectively. Considering there is greater uncertainty for the entropy of fusion at the melting point, a value of 0.023 was used for F for all diastereoisomer specific calculations.

It is recommended that the entropy of fusion be measured.

Water solubility

Table 9.1 lists the solid state water solubilities S_S for HBCD from three studies using the generator column method, which is considered reliable in the EU RAR (EC 2008). The first two studies were reported to be in general accordance with OECD guidelines and GLP compliant. The test substance in the first was a composite sample consisting of 8 % α-, 5.37 % β- and 86.63 % γ-HBCD. The test substance in the second study was a composite sample consisting of 6.0 % of α-, 8.5 % of β-, and 79.1 % γ-HBCD. The RAR reports that in the second study only γ-HBCD could be quantified; however, a review of the test report indicates no diastereoisomer-specific analysis (Stenzel and Markley 1997). The third study reports solubilities of the three diastereoisomers in a salt-water medium used for an algal growth inhibition test. The solubilities in salt-water are lower than those in fresh water and are generally consistent with the "salting out" effect (Xie et al. 1997).

The measurements considered reliable in the EU RAR are also considered reliable in the present study. The first study had fewer impurities and unknowns then the second study and all isomers could be quantified; therefore, these data are considered the most reliable for the present study. The estimate from the second study provides an approximation for the commercial mixture. Differences in temperature between some of the measured values at 20°C and the analyses in the present study (e.g. 25°C) are considered negligible in consideration of the estimation uncertainty.

When technical HBCD is added to water using a generator a column all three diastereoisomers are assumed to dissolve as discussed and illustrated in Figure 1-2 of (EC 2008). That discussion states that based on the data from Study 1:

> "γ-HBCD will reach its maximum solubility at 2.1 μg/l (with a total HBCD dissolved concentration of 2.4 μg/l), β-HBCD at 14.7 μg/l (with a total HBCD dissolved concentration of 39 μg/l) and α-HBCD at 48.8 μg/l (with a total dissolved HBCD concentration of 65.6 μg/l). At this level, 65.6 μg/l dissolved HBCD, the total content of HBCD in the water is 610 μg/l, with 544.4 μg/l representing non-dissolved γ- and β-HBCD."

When using mass balance models and interpreting test data it must be recognized that if a chemical concentration in the "pure" water phase exceeds its water solubility it will precipitate out of solution. This is discussed in the EU RAR; however, a value of 66 $\mu g.L^{-1}$ was selected as a "worst case" estimate for the technical product for input in EUSES. When a phase is "super-saturated" in this manner, then the mass balance equations must consider a "compartment" for the residual pool of precipitated chemical. The residual pool can then supplement the water phase as concentrations decrease in a manner analogous to the generator column. Most models, including EUSES, are not programmed to address "super-saturation" in the pure water phase. A concern with the EUSES calculations is that the selection of a water solubility value about 20 times greater than the water solubility limit of the predominant γ-diastereoisomer in technical HBCD may result in some errors for calculations of intermedia transport (air-water partitioning, bioaccumulation) and for reactions in the water. As discussed in the EU RAR and above,

when a value of 66 μg.L^{-1} is assumed for the water solubility, the concentration of total HBCD in the water (dissolved and precipitate) is approximately 300 times greater than the value for γ-HBCD. It is recommended that, if possible, diastereoisomer-specific model simulations be conducted using data for each isomer. If HBCD is modelled as the technical mixture the selected water solubility value should be near that of the lowest diastereoisomer, i.e., γ-HBCD.

Table 9.1. Summary of water solubility data for HBCD considered reliable in the EU RAR (reproduced from (EC 2008)).

Test substance	**Medium**	**Water solubility (μg.L^{-1})****	**Reference**
α-HBCD	Water	48.8±1.9 (CV = 2.8%)	Study 1
β-HBCD		14.7±0.5 (CV = 3.4%)	
γ-HBCD		2.1±0.2 (CV = 10.5%)	
Σ HBCD (technical product)		65.6	
α-HBCD	Salt-water	34.3	Study 3
β-HBCD		10.2	
γ-HBCD		1.76	
Σ HBCD (technical product)		46.3	
HBCD (technical product)*	Water	3.4±2.3***	Study 2

* γ-HBCD was assumed in the RAR. **20 °C, ***25 °C; CV – Coefficient of Variation

Model estimates for the water solubility were also obtained and reviewed. These estimates do not differentiate between diastereoisomers. When a log K_{OW} value of 5.63 is used as input, EPI Suite Ver. 4.0 estimates S_S values of 1.3 μg/L and 3.0 μg/L from the WSKOWWIN Ver. 1.41 and WATERNT Ver. 1.01 submodels, respectively (U.S. EPA 2009). These S_S estimates are generally consistent with the measured values for γ-HBCD.

The SPARC model Ver. 4.2 (http://ibmlc2.chem.uga.edu/sparc/) generates two slightly different sets of physical-chemical properties depending on the SMILES notation used. The SMILES notation obtained for CAS# 25637-99-4 shows a substitution pattern of 1,3,5,7,9,11-HBCD, which is inconsistent with the diastereoisomer configurations. The SMILES notation obtained for CAS# 3194-55-6 shows a substitution pattern of 1,2,5,6,9,10-HBCD, which is consistent with the diastereoisomer configurations (vicinal Bromine substitution. Of interest, the SMILES in the EINECS database for CAS# 25637-99-4 shows a substitution pattern that is also inconsistent with the recognized diastereoisomer structures. Thus, the SMILES notation for CAS# 3194-55-6 was used for SPARC calculations. SPARC estimates an S_S of 0.31 μg/L and an activity coefficient in water of $10^{9.58}$ (T_M=190°C). The S_S estimate is about 1 order of magnitude lower than the measured values for γ-HBCD.

There is general consistency between the different estimates for water solubility. For the present study the measured S_S from Study 1 were the selected values for the individual HBCD diastereoisomers. The S_S (mol.m^{-3}) estimates are 7.60×10^{-5}, 2.29×10^{-5}, 3.27×10^{-6} mol.m^{-3} for α-, β-, γ-HBCD diastereoisomers, respectively. These estimates correspond to subcooled liquid water solubility values S_L (mol.m^{-3}) of 3.27×10^{-3}, 9.86×10^{-4}, 1.41×10^{-4} mol.m^{-3} for α-, β-, γ-HBCD diastereoisomers, respectively. A value of 3.0 µg/L (S_S = 4.67×10^{-6} mol.m^{-3}, S_L = 2.01×10^{-4} mol.m^{-3}) is selected for technical (commercial) mixture HBCD for the thermodynamic consistency calculations (see below). The coefficient of variation of 10.5% reported for γ-HBCD in Study 1 (EC 2008) is used as a general predictor of uncertainty in the water solubility estimates for the thermodynamic consistency calculations. A relative variance value of 1 was assigned to the water solubility estimates for the diastereoisomers and a relative variance value of 2 was assigned for the selected commercial mixture value.

Vapor pressure

The RAR discusses the selection of a solid state vapor pressure P_S for HBCD from two measurements (EC 2008). Measured values for P_S of HBCD include 1.6×10^{-5} Pa at 20°C using the effusion method and 6.27×10^{-5} Pa at 21°C using the spinning rotor method. The OECD vapor pressure guidelines include three different effusion methods; however, only the isothermal thermogravimetry effusion method is recommended by the OECD for substances with vapor pressures $<10^{-3}$ Pa (OECD 2002). Based on the evaluation in the RAR, one of the other two effusion methods was used. The spinning rotor method is not recommended for substances with vapor pressures $<10^{-4}$ Pa (OECD 2002). The spinning rotor gauge test was conducted according to Good Laboratory Practice (GLP) guidelines (EC 2008). The composition of HBCD in the effusion method is undetermined and the following percentages of diastereoisomers were reported for the spinning rotor method: α-HBCD 6.0; β-HBCD 8.5; and γ-HBCD 79.1. According to OECD guidelines, both of these methods are not recommended for substances with vapor pressures as low as the values measured for HBCD; however, both methods produced similar values. A value of 6.3×10^{-5} Pa at 21°C for P_S was used in the EU risk assessment.

Wania (2003a) reported a liquid state vapor pressure P_L (subcooled) of 2.41×10^{-5} Pa at 25°C for t-HBCD. The value was obtained using the gas chromatographic-retention time method, which is suitable for substances with vapor pressures as low as 10^{-5} Pa. This value is selected for the FAV calculations.

EPI Suite Ver. 4.0 estimates a P_S of 2.25×10^{-6} Pa (U.S. EPA 2009) and SPARC Ver. 4.2 estimates a P_S of 2.63×10^{-9} Pa (http://ibmlc2.chem.uga.edu/sparc/). The EPI Suite estimate is about 3 times higher than the measured estimate by Wania whereas the SPARC estimate is 275 times lower.

At present there are no isomer-specific estimates for vapor pressure. Goss et al. (2008) used the COSMOtherm model to calculate octanol-water (K_{OW}) and air-water (K_{AW}) partitioning of α-, β-, γ-HBCD diastereoisomers at 25°C (see details below). From the isomer-specific quantum chemical software estimates of K_{AW} and from isomer-specific estimates of water solubility S_S it is possible to estimate P_S for the three isomers as

$$P_S = K_{AW}.RT.S_S \quad (9.2)$$

where R is the gas constant (8.314 $Pa.m^3(mol.K)^{-1}$) and T is the temperature (298 K). This approach assumes that temperature differences (20 to 25°C) are minimal. The resulting isomer-specific estimates for P_S are 2.72×10^{-10} Pa, 3.58×10^{-11} Pa, and 1.86×10^{-11} Pa for α-, β-, γ-HBCD, respectively. These model estimates are about 10^4 times lower than the measured values measured by Wania. Goss et al. (2008) note that "the absolute values of the calculated air-water partition coefficients may be considerably off the real values". COSMOtherm also underestimates K_{AW} for hexachlorocyclohexane (HCH) (Goss et al. 2008). Since the K_{OW} estimates for HCH and HBCD are similar to measured estimates, this suggests that errors in COSMOtherm K_{AW} estimates relate to errors in P. Thus, the COSMOtherm estimates for K_{AW} and P_S are not considered reliable; however, in relative terms the P_S results are similar to the measured values for S_S such that values for γ-HBCD are about 1 order of magnitude less than values for α-HBCD.

There are limited data and conflicting results for estimates of the vapor pressure. For the present study the measured P_S estimate 7.23×10^{-7} Pa ($P_L = 3.11\times10^{-5}$ Pa using an F of 0.023) using the gas saturation method is considered the most reliable estimate for the commercial HBCD mixture and is selected as an input for thermodynamic consistency analyses (see below). Estimates for the diastereoisomers were derived by assuming P_L for γ-HBCD is approximately 2.0×10^{-5} Pa and that the relative differences in values estimated by COSMOtherm are applicable such that P_L for α- and β-HBCD are approximately 2.93×10^{-4} Pa and 3.86×10^{-5} Pa, respectively. Due to the limited data and uncertainty in estimating vapor pressure, greater uncertainty is considered for these values compared to the water solubility estimates. A relative variance value of 3 was assigned to the P_L of the technical mixture and a value of 5 was assigned to the diastereoisomers.

It is suggested that measured estimates for isomer-specific vapor pressures be obtained using OECD recommended methods for low vapor pressure substances.

Solubility in Octanol

There are no known reported measurements for the solubility of commercial HBCD or the diastereoisomers in octanol. Estimates are provided below in the section on the octanol-water partition coefficient.

Henry's law constant (Air-water partition coefficient)

To our knowledge there are no measured estimates for Henry's law constant H ($Pa.m^3.mol^{-1}$) currently available. The dimensionless air-water partition coefficient K_{AW} is H/RT. EPI Suite Ver. 4.0 estimates (HENRYWIN Ver. 3.2) for Henry's law constants are 0.174 and 6.52×10^{-6} $Pa.m^3.mol^{-1}$ using the bond and group methods, respectively (U.S. EPA 2009). The group method estimate is considered erroneous. The log K_{AW} estimate corresponding to the bond method is -4.15. H can also be estimated from vapor pressure and water solubility as P_S/S_S or P_L/S_L. In this manner the selected values of 2.41×10^{-5} Pa and 2.01×10^{-4} $mol.m^{-3}$ correspond to an estimate for H of 0.120 $Pa.m^3.mol^{-1}$

(log K_{AW} = -4.32). SPARC Ver. 4.2 estimates a value for H of 0.0055 $Pa.m^3.mol^{-1}$ (log K_{AW} = -5.66) using the same approach, i.e., P_L/S_L (http://ibmlc2.chem.uga.edu/sparc/). The estimates from EPI Suite bond method and the relative solubilities of the selected values for vapor pressure and water solubility are consistent, independent estimates. The EPI Suite bond method estimate is selected for thermodynamic consistency calculations for t-HBCD with an assigned relative variance value of 4.

Diastereoisomer estimates for K_{AW} have been reported by Goss et al. using the COSMOtherm model (Goss et al. 2008). The log K_{AW} values are -8.84, -9.20, and -8.64 for α-, β-, γ-HBCD, respectively. These values correspond to H values of $3.6x10^{-6}$, $1.6x10^{-6}$, $5.7x10^{-6}$ $Pa.m^3.mol^{-1}$ for α-, β-, γ-HBCD, respectively. As discussed above and by the authors the absolute estimates are considered to be erroneous, but the relative values are considered reasonable. Diastereoisomer estimates of log K_{AW} from selected values for P_L/S_L are -4.27, -4.63, and -4.07 for α-, β-, γ-HBCD, respectively. These estimates are dependent on the values for P_L/S_L, which have already been selected for thermodynamic consistency calculations. Independent estimates for the diastereoisomers were derived by assuming log K_{AW} for γ-HBCD is equal to the EPI Suite estimate (-4.15) and that the relative differences in values estimated by COSMOtherm are applicable such that log K_{AW} for α- and β-HBCD are -4.35 and -4.71, respectively.

It is recommended that isomer-specific Henry's law constants be measured.

Octanol-water partition coefficient

The EU RAR reports a measured log K_{OW} of HBCD of 5.625 at 25±0.05°C using the generator column method, which is also in accordance with GLP practices (EC 2008). The test sample was a composite containing 8.5 % β-, 6.0 % α- and 79.1 % γ-HBCD (total HBCD 93.6 %). The study was generally carried out according to the guidelines, although the concentration of the stock solution was approximately 0.2 % instead of 1 %. As noted above in the discussion on water solubility and in the RAR (EC 2008), the generator column method for mixtures may result in excess, non-dissolved quantities of diastereoisomers in the water column. MacGregor and Nixon (2004) report a mean water concentration of 3.97 μg-HBCD.L^{-1} (standard deviation 1.53 μg-HBCD.L^{-1}). The water concentration is near the water solubility limit for γ-HBCD.

The RAR also summarizes the diastereoisomer-specific log K_{OW} values estimated for α-, β- and γ-HBCD using reversed-phase high-performance liquid chromatography (RP-HPLC) methods (Hayward et al. 2006). The estimated log K_{OW} values for α-, β- and γ-HBCD were 5.07±0.09, 5.12±0.09 and 5.47±0.10, respectively. The calibration compounds were chlorobenzenes and polychlorinated biphenyls, which as discussed by the authors, are not ideal because of the structural dissimilarities between the organochlorines and HBCD (Hayward et al. 2006). Assuming the solubility in octanol is similar for the diastereoisomers, the K_{OW} estimates are relatively consistent with measurements of water solubility such that K_{OW} increases with decreasing water solubility. The HPLC derived K_{OW} values for the diastereoisomers are comparable but lower than the measured K_{OW} estimate for commercial HBCD using the generator column method.

The RAR includes a few other measured and modelled estimates for log K_{OW} that were considered unreliable. The RAR selected a log K_{OW} of 5.625 for the commercial HBCD mixture.

Table 9.2 lists 11 model estimates including EPI Suite Ver. 4.0, SPARC Ver 4.2 and COSMOfrag model estimates. The mean value of the model estimates for log K_{OW} is 6.73 with a standard deviation of 0.76. These model estimates do not differentiate between K_{OW} values for the diastereoisomers. Goss et al. (2008) reported diastereoisomer log K_{OW} estimates of 5.59, 5.44 and 5.53, for α-, β- and γ-HBCD, respectively, using the COSMOtherm model. The COSMOtherm model estimates are of the same general magnitude as the generator column K_{OW} measurement for commercial HBCD and the diastereoisomers determined from calibration using RP-HPLC. A notable difference is the relative ranking of hydrophobicity between the RP-HPLC and COSMOtherm estimates.

There are uncertainties in the measured and estimated values for K_{OW} for the commercial mixture and the diastereoisomers. The solubilities of the diastereoisomers in octanol are expected to be similar and differences in K_{OW} are expected to be the result of differences in water solubility. SPARC Ver. 4.2 calculates an activity coefficient in octanol γ_O of 12.88 (unitless) and a solid state solubility in octanol S_{OS} of 16.21 $mol \cdot m^{-3}$ (liquid state solubility in octanol S_{OL} of 698 $mol \cdot m^{-3}$) for HBCD. The S_{OL} value was selected as input for the thermodynamic consistency calculations for the commercial HBCD mixture and the diastereoisomers. A relative variance value of 4 was assigned to this property estimate.

The measured log K_{OW} value of 5.63 was selected as input for the consistency calculations for t-HBCD and a relative variance value of 3 was assigned. For the diastereoisomers the estimates by Goss et al. were selected as input for the consistency calculations and relative variance values of 4 were assigned. Although these estimates were derived in silico they are close to measured estimates and considering the potential underestimation of HBCD using the generator column method and the lack of ideal calibration substances for the RP-HPLC method, the selected values are considered reasonable for the thermodynamic consistency calculations.

It is recommended that isomer-specific octanol-water partition coefficients and solubility in octanol be measured.

Table 9.2. Model estimates for the octanol-water partition coefficient (K_{OW}).

log K_{OW}	Model	Reference
7.74	EPI Suite Ver. 4.0	(U.S. EPA 2009)
6.45	ALPOGPs	(Tetko et al. 2005)
5.23	AC logP	(Tetko et al. 2005)
6.75	AB/logP	(Tetko et al. 2005)
6.14	miLogP	(Tetko et al. 2005)
6.66	CosmoFrag	(Tetko et al. 2005)
6.69	SPARC Ver. 4.2	http://ibmlc2.chem.uga.edu/sparc/
6.96	ALOGP	(Tetko et al. 2005)
6.30	MLOGP	(Tetko et al. 2005)
8.01	XLOGP2	(Tetko et al. 2005)
7.10	XLOGP3	(Tetko et al. 2005)
6.73	**Average log K_{OW}**	
0.76	**Standard Deviation**	

Octanol-air partition coefficient

There are no reported measurements for the octanol-air partition coefficient (K_{OA}, dimensionless); however, there are a few methods for estimation. EPI Suite Ver. 4.0 estimates a log K_{OA} of 10.47 with the KOAWIN program and a log K_{OA} of 11.89 (based on EPI estimates for K_{OW} and K_{AW} as log K_{OW} - log K_{AW}). K_{OA} can also be estimated from the relative fugacity capacities of octanol and air, Z_O and Z_A ($mol \cdot m^{-3} \cdot Pa^{-1}$), respectively as Z_O / Z_A. Z_A is 1/RT and Z_O can be estimated as $1/(\nu_O \gamma_O P_L)$, where ν_O is the molar volume of "dry" octanol (1.57×10^{-4} $m^3 \cdot mol^{-1}$). The predicted γ_O of 12.88 (SPARC Ver. 4.2) and the selected P_L values of 2.41×10^{-5}, 2.93×10^{-4}, 3.86×10^{-5}, 2.00×10^{-5} for t-, α-, β-, and γ-HBCD, respectively were used to estimate K_{OA}, i.e., Z_O / Z_A. At 25 ^oC, log K_{OA} values are 10.71, 9.62, 10.50, and 10.79 for the t-HBCD and α-, β- and γ-HBCD diastereoisomers, respectively. Log K_{OA} can also be estimated from the COSMOtherm K_{AW} and K_{OW}; however, as discussed above the estimates for K_{AW} are considered erroneous.

No K_{OA} values were selected as inputs for the thermodynamic consistency calculations because the values for the diastereoisomers are dependent on the estimates for the extrapolated estimates for P_L; however, the estimates provided above for K_{OA} can be used for comparison with the final adjusted values (FAVs).

It is recommended that measured estimates for isomer-specific octanol-air partition coefficients be obtained.

Thermodynamic consistency for model input parameters

The methods of Schenker et al. (Schenker et al. 2005) were used to obtain thermodynamically consistent estimates for the solubility and partitioning properties of t-HBCD and the three diastereoisomers. The method was applied to t-HBCD in the current study; however, the thermodynamic consistency calculation should generally not be used for mixtures, but rather for discrete substances with distinct properties. The method was applied for consistency with diastereomer methods and for comparing model estimates for t-HBCD and individual diastereomers with monitoring data for t-HBCD. Considering the uncertainty in the FAVs for t-HBCD in the context of the greater uncertainties associated with actual emissions, mode-of-entry, isomerization and degradation in the environment, the errors in calculating "representative" FAVs for t-HBCD are considered to be comparatively low. These methods are described in Section 2.3.2. The selected input and output values for the FAV calculations are summarized in Tables 9.3 and 9.4, respectively.

Table 9.3. Selected values for physical-chemical properties and relative variance values used as inputs for thermodynamic consistency calculations.

Substance	**P_L, Pa**	**S_L, mol·m^{-3}**	**S_{OL}, mol·m^{-3}**	**Log K_{AW}**	**Log K_{OW}** [a]	**Log K_{OA}**
t-HBCD	2.41×10^{-5}	2.01×10^{-4}	698	-4.15	5.63	10.71 [b]
α-HBCD	2.93×10^{-4}	3.27×10^{-3}	698	-4.35	5.59	9.62 [b]
β-HBCD	3.86×10^{-5}	9.86×10^{-4}	698	-4.71	5.44	10.50 [b]
γ-HBCD	2.00×10^{-5}	1.41×10^{-4}	698	-4.15	5.53	10.79 [b]
			Relative variance			
t-HBCD	3	2	4	4	3	
α-HBCD	5	1	4	5	4	
β-HBCD	5	1	4	5	4	
γ-HBCD	5	1	4	5	4	

[a] log K_{OW} values corresponding with log (Co/Cw) values of 6.02, 5.97, 5.76, and 5.89 for t-, α-, β-, and γ-HBCD, respectively

[b] values not used as inputs for calculation of FAVs because they are not "independent" values, i.e., they were derived from vapor pressure and octanol solubility estimates (see text). The values are included for comparisons with FAVs

Table 9.4. Final adjusted values (FAVs at 25°C), relative variance values, percent adjustments, and assigned confidence factors used in the present study for mass balance modelling and data analyses obtained from the thermodynamic consistency calculations (Schenker et al. 2005).

Substance	P_L, Pa	S_L, mol·m^{-3}	S_{OL}, mol·m^{-3}	Log K_{AW}	Log K_{OW} [a]	Log K_{OA}
t-HBCD	3.03×10^{-5}	2.33×10^{-4}	380	-4.28	5.77	10.46
α-HBCD	3.00×10^{-4}	2.77×10^{-3}	1340	-4.36	5.38	9.96
β-HBCD	4.29×10^{-5}	9.89×10^{-4}	630	-4.76	5.47	10.47
γ-HBCD	2.42×10^{-5}	1.68×10^{-4}	300	-4.23	5.80	10.40
			Relative variance			
t-HBCD	1.95	1.27	2.13	2.13	1.95	3.45
α-HBCD	2.70	0.82	2.20	2.70	2.20	4.50
β-HBCD	2.70	0.82	2.20	2.70	2.20	4.50
γ-HBCD	2.70	0.82	2.20	2.70	2.20	4.50
			Percent adjusted			
t-HBCD	26%	16%	-45%	-26%	39%	26%
α-HBCD	2%	-15%	92%	-2%	-38%	2%
β-HBCD	11%	0%	-9%	-10%	7%	11%
γ-HBCD	21%	19%	-57%	-18%	87%	21%
			Confidence factor (*Cf*)			
t-HBCD	2.5	2.3	3	3	3	3
α-HBCD	4	2	4	4	4	4
β-HBCD	4	2	4	4	4	4
γ-HBCD	4	2	4	4	4	4

[a] log K_{OW} values corresponding with log (Co/Cw) values of 6.22, 5.68, 5.81, 6.25 for t-, α-, β-, and γ-HBCD, respectively

9.2 DEGRADATION, BIOTRANSFORMATION AND HALF-LIVES

Table 9.5. BIOWIN™ v4.10 model output for HBCD (not isomer specific) (US EPA 2009).

Model	Output	Interpretation
BIOWIN1 (linear)	0.1650	Does NOT biodegrade fast
BIOWIN2 (non-linear)	0.0000	Does NOT biodegrade fast
BIOWIN3 (Ultimate deg)	1.9548	Time frame : Months
BIOWIN4 (Primary deg)	3.1342	Time frame: Weeks
BIOWIN5 (MITI linear)	-0.4234	NOT Readily Degradable
BIOWIN6 (MITI non-linear)	0.0000	NOT Readily Degradable
BIOWIN7 (anaerobic linear)	2.5072	Biodegrades FAST

Table 9.6. General equations used to estimate aerobic environmental biodegradation half-lives from BIOWIN™ model output and corresponding values calculated for HBCD (Arnot et al. 2005; US EPA 2009).

Model	Regression Equation	r^2	Half-life (d)
BIOWIN1 (linear)	$\log_{10}(t_{1/2}) = -1.32x + 2.24$	0.72	105.2
BIOWIN3 (Ultimate deg)	$\log_{10}(t_{1/2}) = -1.07x + 4.20$	0.77	128.3
BIOWIN4 (Primary deg)	$\log_{10}(t_{1/2}) = -1.46x + 6.51$	0.78	85.9

where x is the numerical output generated for each respective BIOWIN™ model (presented above in Table 9.5)

9.3 MONITORING DATA

This section includes summaries of monitoring data that were discussed in the report that may not have been recognized in previous assessments. The abiotic data summarize all available data, whereas the biotic data are for studies published in the last 3 to 4 years.

Table 9.7. Summary of abiotic monitoring data.

Year	Location	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	LOD	Other	Median	Min	Max	n	Reference
Air (pg/m^3)	***gaseous***											
2004	China-Guangzhou-Industrial-1	0.08	0.04	0.02	0.02		Point					(Yu et al. 2008)
2004	China-Guangzhou-Industrial-2	0.09	0.05	0.02	0.02		Point					(Yu et al. 2008)
2004	China-Guangzhou-Urban-1	0.11	0.06	0.03	0.02		Urban					(Yu et al. 2008)
2004	China-Guangzhou-Background-1	0.12	0.07	0.03	0.02		Urban					(Yu et al. 2008)
	particulate											
2003	USA-Indiana	1				<0.07	Urban	0.75	0.2	3.6	37	(Hoh et al. 2005)
2003	USA-Arkansas	1.6				<0.13	Semi-remote	0.4	0.2	11	30	(Hoh et al. 2005)
2003	USA-Lousiana	0.6				<0.13	Remote	<dl	0.16	6.2	25	(Hoh et al. 2005)
2003	USA- Indiana	3.6	2.808	0.432	0.3492	<0.07	Urban	0.75	0.2	3.6	1	(Hoh et al. 2005)
2003	USA- Indiana	2.9	1.334	0.2117	1.363	<0.07	Urban	0.75	0.2	3.6	1	(Hoh et al. 2005)
2003	USA- Indiana	2.1	0.672	0.1281	1.281	<0.07	Urban	0.75	0.2	3.6	1	(Hoh et al. 2005)
2003	USA-Arkansas	11	6.82	1.1	3.08	<0.13	Semi-remote	0.4	0.2	11	1	(Hoh et al. 2005)
2003	USA-Louisiana	2.4	0.408	0.408	1.584	<0.13	Remote	<dl	0.16	6.2	1	(Hoh et al. 2005)
2003	Midwest USA	5.55				< 0.1	Semi-remote		0.1	11	156	(Covaci et al. 2006)
2003	USA-Northern Michigan	1.2				<0.07	Remote	0.5	0.2	8	35	(Hoh et al. 2005)
2003	USA-Chicago, IL	4.5				<0.13	Urban	4.2	0.9	9.6	37	(Hoh et al. 2005)
2003	USA-Northern Michigan	8	6.48	0.696	0.8	<0.07	Remote	0.5	0.2	8	1	(Hoh et al. 2005)
2003	USA-Chicago, IL	9.6	1.056	0.6144	7.968	<0.13	Urban	4.2	0.9	9.6	1	(Hoh et al. 2005)
2004	China-Guangzhou-Industrial-1	0.6	0.36	0.08	0.16		Point					(Yu et al. 2008)

Year	Location	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	LOD	Other	Median	Min	Max	n	Reference
2004	China-Guangzhou-Industrial-2	0.79	0.54	0.1	0.15		Point					(Yu et al. 2008)
2004	China-Guangzhou-Urban-1	2.98	2.01	0.33	0.64		Urban					(Yu et al. 2008)
2004	China-Guangzhou-Background-1 ***total***	1.52	0.92	0.18	0.42		Urban					(Yu et al. 2008)
1990.5	Hoburgen and Ammarnas, Sweden	5.5					Remote		5	6	2	(Covaci et al. 2006)
1990.5	Sweden-Ammarnas	6.1					Remote				2	(deWit 2002)
2000	Sweden-80kmSW of Aspvreten	1070000					Point					(Remberger et al. 2004)
2000	Aspvreten, Sweden	280					Remote				1	(Remberger et al. 2004)
2000	Rörvik, Sweden	5				1	Remote				1	(Remberger et al. 2004)
2000	Pallas, Finland	3					Remote				1	(Remberger et al. 2004)
2000.5	Remote locations in Sweden and Finland	141					Remote		2	280	6	(Covaci et al. 2006)
2001	Aspvreten, Sweden	25					Remote				1	(Remberger et al. 2004)
2001	Rörvik, Sweden	1				1	Remote				1	(Remberger et al. 2004)
2001	Pallas, Finland	2					Remote				1	(Remberger et al. 2004)
2004	City Background-1 Guangzhou, China	1.7					Urban		1.1	2.3		(Yu et al. 2008)
2004	Industrial-1 Guangzhou, China	0.7					Point		0.3	1.2		(Yu et al. 2008)
2004	Industrial-2 Guangzhou, China	0.9					Point		0.4	1.8		(Yu et al. 2008)
2004	Urban-1 Guangzhou China	3.1					Urban		2.2	3.9		(Yu et al. 2008)
2005	Japan-outside suburban homes	14					Urban		13	15		(Takigami et al. 2008)
2000	Sweden-30km N of Stockholm	13						Point-Landfill				(Remberger et al. 2004)
2000	Sweden-Boras Textile Industry	19					Point					(Remberger et al. 2004)
2000	Sweden-Stockholm-schoolyard	76					Urban					(Remberger et al. 2004)
2000	Sweden-Stockholm	610					Urban					(Remberger et al. 2004)

Year	Location	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	LOD	Other	Median	Min	Max	n	Reference
2001	Sweden-30km N of Stockholm	180					Point-Landfill					(Remberger et al. 2004)
2001	Sweden-Boras Textile Industry	740					Point					(Remberger et al. 2004)
Precipitation (ng/L)												
2005	Great Lakes Basin	17.5					Semi-remote		0	35		(Backus et al. 2005)
Sediment (ng/g dw)	***core***											
1940	Norway, Bear Island	0.14	0.06	0.03	0.05	all <	Remote				1	(Christensen et al. 2004)
1950	Sweden-Lake Altasjon	0.5					Urban					(Remberger et al. 2004)
1952.5	Norway, Bear Island	0.22	0.09	0.05	0.08	all <	Remote				1	(Christensen et al. 2004)
1960	Sweden-Arstaviken Bay	0.2					Urban					(Remberger et al. 2004)
1966	Norway, Bear Island	0.19	0.08	0.04	0.07	all <	Remote				1	(Christensen et al. 2004)
1974	Swiss Lake-near Zurich	0.51					Urban				1	(Kohler et al. 2008)
1975	Japan-Tokyo Bay TP4	0.1				0.01	Urban					(Minh et al. 2007)
1975	Japan-Tokyo Bay TP5	0.05				0.01	Urban					(Minh et al. 2007)
1979	Japan-Tokyo Bay TP1	0.05				0.01	Urban					(Minh et al. 2007)
1980	Norway, Bear Island	4.37	0.43	0.06	3.88	< 0.06	Remote				1	(Christensen et al. 2004)
1982	Japan-Tokyo Bay TP4	0.7				0.01	Urban					(Minh et al. 2007)
1982	Swiss Lake-near Zurich	0.4					Urban				1	(Kohler et al. 2008)
1984	Japan-Tokyo Bay TP5	0.2				0.01	Urban					(Minh et al. 2007)
1988	Japan-Tokyo Bay TP4	0.2				0.01	Urban					(Minh et al. 2007)
1989	Japan-Tokyo Bay TP1	0.5				0.01	Urban					(Minh et al. 2007)
1989	Swiss Lake-near Zurich	1.3					Urban				1	(Kohler et al. 2008)
1992	Japan-Tokyo Bay TP5	0.2				0.01	Urban					(Minh et al. 2007)
1994	Norway, Bear Island	0.31	0.14	0.06	0.11	all <	Remote				1	(Christensen et al. 2004)
1995	Japan-Tokyo Bay TP4	0.7				0.01	Urban					(Minh et al. 2007)
1995	Swiss Lake-near Zurich	1.8					Urban				1	(Kohler et al. 2008)

Year	Location	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	LOD	Other	Median	Min	Max	n	Reference
1996	Sweden-Arstaviken Bay	0.8					Urban					(Remberger et al. 2004)
1996	Sweden-Fjaderholmmarna islands	0.9					Urban					(Remberger et al. 2004)
1997	Sweden-Lake Altasjon	1.5					Urban					(Remberger et al. 2004)
2001	Swiss Lake-near Zurich	2.5					Urban				1	(Kohler et al. 2008)
	estuarine											
1998	Japan-Tokyo Bay TP1	2.1	0.336	0.01	1.764	0.01	Remote					(Minh et al. 2007)
1999	Japan-Tokyo Bay TP5	0.73	0.01	0.01	0.72	0.01	Remote					(Minh et al. 2007)
2000	2000/58-Middelgat (Hoedekenskerke)	7.7	<0.2	<0.5	7.7	1.2	Unknown				10	(Morris et al. 2004)
2000	2000/64-Zandvlietsluis	99	2.6	<1.8	96	1.2	Unknown				10	(Morris et al. 2004)
2000	2000/67-Terneuzen	59	2.1	0.3	57	1.2	Unknown				10	(Morris et al. 2004)
2000	2000/0073-Hoofdplaat (reg. station)	0.7	<0.2	<0.2	0.7	1.2	Unknown				10	(Morris et al. 2004)
2000	2000/2758-Humber (E England)	6	<2.4	<2.4	6	1.2	Unknown				1	(Morris et al. 2004)
2000	Japan-Tokyo Bay TP4	2	0.94	0.01	1.06	0.01	Remote					(Minh et al. 2007)
2001	2001/1466-Leie, St.-Martens-Leerne	38	7.4	<0.1	31	1.2	Unknown				1	(Morris et al. 2004)
2001	2001/1458-Scheldt, Dutch border	83	<0.2	<0.2	83	1.2	Unknown				10	(Morris et al. 2004)
2001	2001/1465-Scheldt, Oudenaarde	950	180	60	710	1.2	Point-downstream				10	(Morris et al. 2004)
2001	Western Scheldt-Netherlands	38					Unknown		14	71	3	(Verslycke et al. 2005)
2002	Japan-Tokyo Bay T2	0.056				0.01	Semi-remote				1	(Minh et al. 2007)
2002	Japan-Tokyo Bay T3	0.18	0.036	0.01	0.144	0.01	Semi-remote				1	(Minh et al. 2007)
2002	Japan-Tokyo Bay T4	0.11	0.0187	0.01	0.0913	0.01	Semi-remote				1	(Minh et al. 2007)
2002	Japan-Tokyo Bay T5	0.13	0.0403	0.01	0.0897	0.01	Remote				1	(Minh et al. 2007)
2002	Japan-Tokyo Bay TP7	1.2	0.252	0.084	0.864	0.01	Semi-remote				1	(Minh et al. 2007)
2002	Japan-Tokyo Bay TP8	0.33	0.0495	0.01	0.2805	0.01	Semi-remote				1	(Minh et al. 2007)
2003	DRE-1 Jernbanebrua	1.1	0.78	0.2	0.32	.3, .2, .4	Unknown					(Schlabach et al. 2004a)
2003	DRF-1 Hovedbasseng	0.4	0.3	0.2	0.4	.3, .2, .4	Unknown					(Schlabach et al. 2004a)

Year	Location	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	LOD	Other	Median	Min	Max	n	Reference
2003	DRF-2 Lierterminalen	14.14	10.15	0.65	3.34	.3, .2, .4	Point					(Schlabach et al. 2004a)
2003	DRF-3 Teigenkaianlegg	0.4	0.3	0.2	0.4	.3, .2, .4	Unknown					(Schlabach et al. 2004a)
2003	DRF-4 Tangenflytedokk	0.4	0.3	0.2	0.4	.3, .2, .4	Unknown					(Schlabach et al. 2004a)
	river/lake											
1980	Lake Ellasjøen, Bjørnøya, Norwegian Arctic	3.87	0.43	0.06	3.38		Remote				4	(Evenset et al. 2007)
1995	Sweden-Lake Marsjon 1	0.279				0.279	Remote		0.279	0.279	2	(Sellström et al. 1998)
1995	Sweden-Lake Oresjo 2	1.54				0.28	Point-downstream		0.28	1.54	2	(Sellström et al. 1998)
1995	Sweden - River Viskan-downstream from industry/urban 3	73.6					Point-downstream		73.6	76.8	2	(Sellström et al. 1998)
1995	Sweden-River Viskan at Moga 4	1591					Point-downstream		1591	1591	2	(Sellström et al. 1998)
1995	Sweden-River Viskan upstream from Skene 5	54					Point-downstream		54	54	2	(Sellström et al. 1998)
1995	Sweden-River Viskan, downstream from Skene 6	210					Point-downstream		192	228	2	(Sellström et al. 1998)
1995	Sweden-River Haggan upstream from Fritsla 7	2.728					Remote		0.186	2.728	2	(Sellström et al. 1998)
1995	Sweden-River Haggan downstream from Fritsla 8	0.468					Point-downstream		0.096	0.468	2	(Sellström et al. 1998)
1995	Sweden, Lake Skaresjon 9	0.37					Remote		0.37	0.37	2	(Sellström et al. 1998)
2000	2000/2345-Tees (NE England)	363	59	37	267	1.2	Point-downstream				1	(Morris et al. 2004)
2000	2000/2352-Tees (NE England)	295	60	23	212	1.2	Point-downstream				1	(Morris et al. 2004)
2000	2000/2359-Tees (NE England)	511	106	111	294	1.2	Point-downstream				1	(Morris et al. 2004)
2000	2000/2589-Tyne (NE England)	322	32	16	274	1.2	Unknown				1	(Morris et al. 2004)
2000	2000/2586-Skerne (NE England)	1678	745	396	537	1.2	Unknown				1	(Morris et al. 2004)
2000	2000/2584-Skerne (NE England)	174	40	16	118	1.2	Point				1	(Morris et al. 2004)
2000	2000/3989-Mersey (NW England)	22	<2.4	22	<2.4	1.2	Point-downstream				1	(Morris et al. 2004)
2000	2002/989-Clyde (Scotland)	187	64	31	92	1.2	Unknown				1	(Morris et al. 2004)
2000	2000/1979-Rhine, Lobith	8.1	<0.6	3.9	4.2	1.2	Point-downstream				1	(Morris et al. 2004)

Year	Location	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	LOD	Other	Median	Min	Max	n	Reference
2000	2000/1981-Hollands Diep	9.9	<1.4	<1.4	9.9	1.2	Unknown				1	(Morris et al. 2004)
2000	2000/35617-Meuse, Eijsden	<0.2	<0.2	<0.4	<0.2	1.2	Remote				1	(Morris et al. 2004)
2000	2000/35618-Kiezersveer	2.8	2.8	<0.4	<0.2	1.2	Unknown				1	(Morris et al. 2004)
2000	Sweden-River Viskan-downstream	9.6					Point-downstream		1	25	4	(Remberger et al. 2004)
2000	Sweden-River Viskan-upstream	0.2				< 0.2	Remote				2	(Remberger et al. 2004)
2000	Sweden-30km N of Stockholm	0.1				0.1	Point-Landfill					(Remberger et al. 2004)
2000	Sweden-30km N of Stockholm	0.1				0.1	Point-Landfill					(Remberger et al. 2004)
2000	Sweden-Boras Textile Industry River Viskan 1	0.1				0.1	Remote					(Remberger et al. 2004)
2000	Sweden-Boras Textile Industry river Viskan 2	0.2					Remote					(Remberger et al. 2004)
2000	Sweden-Boras Textile Industry River Viskan 3	25					Point-downstream					(Remberger et al. 2004)
2000	Sweden-Boras Textile Industry River Viskan 4	1					Point-downstream					(Remberger et al. 2004)
2000	Sweden-Boras Textile Industry River Viskan 5	4.6					Point-downstream					(Remberger et al. 2004)
2000	Sweden-Boras Textile Industry River Viskan 6	7.8					Point-downstream					(Remberger et al. 2004)
2002	Norway-landfil leachate	22	2	0.8	19	< 0.1	Unknown		0.1	84	11	(Schlabach 2002)
2002	Spain-River Cinca-upstream of source	0.1				< 0.1	Remote				2	(Eljarrat et al. 2004)
2002	Spain-River Cinca-downstream of source	302					Point-downstream		90	514	2	(Eljarrat et al. 2004)
2003	Norway-Lake Mjosa area	2.7	0.2	2.2	0.3	<0.9, <0.2, <0.3	Unknown		0.9	7.9	4	(Schlabach et al. 2004b)
2003	Norway-Drammens River/Drammensfjord offshore sites	2.5	1.4	0.2	0.9	< 0.2	Unknown		0.9	14	11	(Schlabach et al. 2004b)
2003	DRE-7 Vikersund	0.12	0.1	0.08	0.12	.1, .08, .12	Remote					(Schlabach et al. 2004a)
2003	DRE-6 Loselva	0.6	0.33	0.08	0.27	.1, .08, .12	Unknown					(Schlabach et al. 2004a)
2003	DRE-5 Hokksund	0.45	0.09	0.08	0.36	.1, .08, .12	Unknown					(Schlabach et al. 2004a)
2003	DRE-4 Mjøndalen	4.14	0.99	0.07	3.08	.1, .08, .12	Unknown					(Schlabach et al. 2004a)

Year	Location	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	LOD	Other	Median	Min	Max	n	Reference
2003	DRE-3 Langesøya	0.4	0.15	0.08	0.25	.1, .08, .12	Unknown					(Schlabach et al. 2004a)
2003	DRE-2 Politihuset	2.4	1.55	0.2	0.85	.3, .2, .4	Unknown					(Schlabach et al. 2004a)
	sea											
2000	Netherlands-North Sea 1	4.4				< 0.2	Unknown				1	(Klamer et al. 2005)
2000	Netherlands-North Sea 10	0.2				< 0.2	Remote				1	(Klamer et al. 2005)
2000	Netherlands-North Sea 2	3.4				< 0.2	Unknown				1	(Klamer et al. 2005)
2000	Netherlands-North Sea 3	6.9				< 0.2	Unknown				1	(Klamer et al. 2005)
2000	Netherlands-North Sea 4	5.4				< 0.2	Unknown				1	(Klamer et al. 2005)
2000	Netherlands-North Sea 5	5				< 0.2	Unknown				1	(Klamer et al. 2005)
2000	Netherlands-North Sea 6	6.2				< 0.2	Unknown				1	(Klamer et al. 2005)
2000	Netherlands-North Sea 7	0.76				< 0.2	Remote				1	(Klamer et al. 2005)
2000	Netherlands-North Sea 8	0.83				< 0.2	Remote				1	(Klamer et al. 2005)
2000	Netherlands-North Sea 9	1				< 0.2	Remote				1	(Klamer et al. 2005)
2004	German Bight	3.4					Remote		0.3	6.5	12	(Lepom et al. 2007)
2005	Scheldt Basin-Netherlands, Belgium	42.5				< 0.2	Unknown		14	71	3	(Verslycke et al. 2005)
Soil (ng/g dw)	***solids***											
	S1-Europe-1	597					Point				10	(Petersen et al. 2004)
	S2-Europe-1	171					Point				10	(Petersen et al. 2004)
	S3-Europe-1	344					Point				10	(Petersen et al. 2004)
	S4-Europe-1	23200					Point				10	(Petersen et al. 2004)
	S5-Europe-1	111					Point				10	(Petersen et al. 2004)
	S1-Europe-2	569	80	56	433		Point				10	(Petersen et al. 2004)
	S2-Europe-2	135	33	18	84		Point				10	(Petersen et al. 2004)
	S3-Europe-2	137	28	0	109		Point				10	(Petersen et al. 2004)
	S4-Europe-2	20600	2930	1520	16100		Point				10	(Petersen et al. 2004)
	S5-Europe-2	59	18	0	41		Point				10	(Petersen et al. 2004)

Year	Location	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	LOD	Other	Median	Min	Max	n	Reference
2000	Sweden-80kmSW of Aspvreten	140					Point					(Remberger et al. 2004)
2000	Sweden-80kmSW of Aspvreten	1300					Point					(Remberger et al. 2004)
2000	Sweden-80kmSW of Aspvreten	1000					Point					(Remberger et al. 2004)
2000	Sweden-near XPS producing plant	567					Point		140	1300	3	(Remberger et al. 2004)
Water	***dissolved (µg/L)***											
2000	Sweden-30km N of Stockholm	0.003					Point-Landfill					(Remberger et al. 2004)
2000	Sweden-30km N of Stockholm	0.009					Point-Landfill					(Remberger et al. 2004)
	suspended solids (ng/g dw)											
1999	Sweden-Lake Malaren, Riddarfjarden	1.6					Urban					(Remberger et al. 2004)
1999	Sweden-Klubben	1				1	Urban					(Remberger et al. 2004)
1999	Sweden-Slussen	2.1					Urban					(Remberger et al. 2004)
2003	Canada/USA-Detroit River	1.3				<0.01	Urban		0.1	3.7	8	(Marvin et al. 2006)
	total (µg/L)											
	W1-Europe-1	20300					Point				1	(Petersen et al. 2004)
	W2-Europe-1	30.4					Point				1	(Petersen et al. 2004)
	W3-Europe-1	318					Point				1	(Petersen et al. 2004)
	W4-Europe-1	9.1					Point				1	(Petersen et al. 2004)
	W5-Europe-1	206					Point				1	(Petersen et al. 2004)
	W1-Europe-2	19000	7580	3230	8220		Point				1	(Petersen et al. 2004)
	W2-Europe-2	36	3.8	3.5	28.7		Point				1	(Petersen et al. 2004)
	W3-Europe-2	361	76	17	268		Point				1	(Petersen et al. 2004)
	W4-Europe-2	11	1.6	0.6	8.8		Point				1	(Petersen et al. 2004)
	W5-Europe-2	205	51	39	115		Point				1	(Petersen et al. 2004)

Table 9.8. Summary of biotic monitoring data.

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
1994	East	Harbour porpoise	blubber	0.88	ug kg-1 ww	64	64	< 5	< 5	Law RJ et al. (2006)
1995	Scotland	Harbour porpoise	blubber	0.93	ug kg-1 ww	10	10	< 5	< 5	Law RJ et al. (2006)
1995	Scotland	Harbour porpoise	blubber	0.86	ug kg-1 ww	42	34	8	< 5	Law RJ et al. (2006)
1996	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	29	29	< 5	< 5	Law RJ et al. (2006)
1996	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	24	16	8	< 5	Law RJ et al. (2006)
1996	Scotland	Harbour porpoise	blubber	0.90	ug kg-1 ww	19	12	7	< 5	Law RJ et al. (2006)
1996	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	106	87	9	10	Law RJ et al. (2006)
1996	East	Harbour porpoise	blubber	0.86	ug kg-1 ww	125	125	< 5	< 5	Law RJ et al. (2006)
1996	East	Harbour porpoise	blubber	0.92	ug kg-1 ww	103	103	< 5	< 5	Law RJ et al. (2006)
1996	West	Harbour porpoise	blubber	0.86	ug kg-1 ww	34	34	< 5	< 5	Law RJ et al. (2006)
1997	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	75	64	11	< 5	Law RJ et al. (2006)
1997	Scotland	Harbour porpoise	blubber	0.90	ug kg-1 ww	434	434	< 12	< 5	Law RJ et al. (2006)
1998	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	221	204	7	10	Law RJ et al. (2006)
1998	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	22	22	< 5	< 5	Law RJ et al. (2006)
1998	Scotland	Harbour porpoise	blubber	0.93	ug kg-1 ww	13	13	< 5	< 5	Law RJ et al. (2006)
1998	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	62	62	< 5	< 5	Law RJ et al. (2006)
1998	East	Harbour porpoise	blubber	0.85	ug kg-1 ww	82	59	23	< 5	Law RJ et al. (2006)
1998	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	48	41	7	< 5	Law RJ et al. (2006)
1999	East	Harbour porpoise	blubber	0.76	ug kg-1 ww	69	69	< 4	< 4	Law RJ et al. (2006)
1999	Scotland	Harbour porpoise	blubber	0.88	ug kg-1 ww	216	209	7	< 5	Law RJ et al. (2006)
1999	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	468	458	< 5	10	Law RJ et al. (2006)
1999	West	Harbour porpoise	blubber	0.88	ug kg-1 ww	44	44	< 5	< 5	Law RJ et al. (2006)
1999	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	287	267	11	9	Law RJ et al. (2006)
2000	West	Harbour porpoise	blubber	0.91	ug kg-1 ww	56	56	< 5	< 5	Law RJ et al. (2006)
2000	West	Harbour porpoise	blubber	0.85	ug kg-1 ww	240	218	12	< 5	Law RJ et al. (2006)
2000	West	Harbour porpoise	blubber	0.86	ug kg-1 ww	42	42	< 5	< 5	Law RJ et al. (2006)
2000	East	Harbour porpoise	blubber	0.83	ug kg-1 ww	39	39	< 4	< 4	Law RJ et al. (2006)
2000	East	Harbour porpoise	blubber	0.84	ug kg-1 ww	41	41	< 5	< 5	Law RJ et al. (2006)
2000	East	Harbour porpoise	blubber	0.84	ug kg-1 ww	103	103	< 5	< 5	Law RJ et al. (2006)
2000	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	201	192	< 5	9	Law RJ et al. (2006)
2000	West	Harbour porpoise	blubber	0.87	ug kg-1 ww	238	227	11	< 5	Law RJ et al. (2006)
2000	West	Harbour porpoise	blubber	0.83	ug kg-1 ww	216	203	13	< 4	Law RJ et al. (2006)
2000	East	Harbour porpoise	blubber	0.86	ug kg-1 ww	250	221	16	13	Law RJ et al. (2006)
2000	East	Harbour porpoise	blubber	0.86	ug kg-1 ww	262	233	18	11	Law RJ et al. (2006)
2000	East	Harbour porpoise	blubber	0.56	ug kg-1 ww	131	125	< 3	6	Law RJ et al. (2006)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
2000	West	Harbour porpoise	blubber	0.88	ug kg-1 ww	104	93	< 5	11	Law RJ et al. (2006)
2001	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	297	279	9	9	Law RJ et al. (2006)
2001	Scotland	Harbour porpoise	blubber	0.90	ug kg-1 ww	368	346	13	9	Law RJ et al. (2006)
2001	West	Harbour porpoise	blubber	0.88	ug kg-1 ww	912	875	24	13	Law RJ et al. (2006)
2001	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	125	103	14	8	Law RJ et al. (2006)
2001	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	622	603	8	11	Law RJ et al. (2006)
2001	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	185	176	< 5	9	Law RJ et al. (2006)
2001	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	149	141	8	< 5	Law RJ et al. (2006)
2001	Scotland	Harbour porpoise	blubber	0.85	ug kg-1 ww	10958	10900	37	21	Law RJ et al. (2006)
2001	West	Harbour porpoise	blubber	0.76	ug kg-1 ww	707	707	< 4	< 4	Law RJ et al. (2006)
2001	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	576	543	22	11	Law RJ et al. (2006)
2001	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	2689	2660	13	17	Law RJ et al. (2006)
2001	Scotland	Harbour porpoise	blubber	0.88	ug kg-1 ww	201	201	< 5	< 5	Law RJ et al. (2006)
2001	West	Harbour porpoise	blubber	0.90	ug kg-1 ww	1610	1610	< 5	< 5	Law RJ et al. (2006)
2001	West	Harbour porpoise	blubber	0.89	ug kg-1 ww	1505	1490	15	< 5	Law RJ et al. (2006)
2001	West	Harbour porpoise	blubber	0.89	ug kg-1 ww	222	222	< 5	< 5	Law RJ et al. (2006)
2002	West	Harbour porpoise	blubber	0.88	ug kg-1 ww	1089	1060	12	17	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	496	496	< 5	< 5	Law RJ et al. (2006)
2002	West	Harbour porpoise	blubber	0.90	ug kg-1 ww	2702	2690	12	< 5	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	841	830	< 5	11	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.90	ug kg-1 ww	326	311	6	9	Law RJ et al. (2006)
2002	West	Harbour porpoise	blubber	0.88	ug kg-1 ww	17609	17600	9	< 5	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.90	ug kg-1 ww	191	172	11	9	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.90	ug kg-1 ww	93	84	< 5	9	Law RJ et al. (2006)
2002	West	Harbour porpoise	blubber	0.89	ug kg-1 ww	15316	15300	8	8	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	18437	18400	18	9	Law RJ et al. (2006)
2002	East	Harbour porpoise	blubber	0.91	ug kg-1 ww	855	855	< 5	< 5	Law RJ et al. (2006)
2002	East	Harbour porpoise	blubber	0.91	ug kg-1 ww	3433	3420	< 5	13	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	10442	10400	29	13	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.90	ug kg-1 ww	356	330	26	< 20	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.85	ug kg-1 ww	463	448	15	< 10	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.82	ug kg-1 ww	7223	7180	37	6	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.88	ug kg-1 ww	1354	1340	< 5	14	Law RJ et al. (2006)
2002	Scotland	Harbour porpoise	blubber	0.88	ug kg-1 ww	1054	1030	15	9	Law RJ et al. (2006)
2003	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	17411	17400	11	< 5	Law RJ et al. (2006)
2003	West	Harbour porpoise	blubber	0.87	ug kg-1 ww	155	143	< 5	12	Law RJ et al. (2006)
2003	East	Harbour porpoise	blubber	0.91	ug kg-1 ww	11440	11440	< 5	< 5	Law RJ et al. (2006)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
2003	East	Harbour porpoise	blubber	0.85	ug kg-1 ww	7740	7740	< 5	< 5	Law RJ et al. (2006)
2003	East	Harbour porpoise	blubber	0.83	ug kg-1 ww	8223	8210	< 4	13	Law RJ et al. (2006)
2003	East	Harbour porpoise	blubber	0.90	ug kg-1 ww	19208	19200	< 5	8	Law RJ et al. (2006)
2003	East	Harbour porpoise	blubber	0.88	ug kg-1 ww	15939	15900	20	18	Law RJ et al. (2006)
2003	East	Harbour porpoise	blubber	0.91	ug kg-1 ww	9460	9460	< 5	< 5	Law RJ et al. (2006)
2003	West	Harbour porpoise	blubber	0.89	ug kg-1 ww	132	132	< 5	< 5	Law RJ et al. (2006)
2003	East	Harbour porpoise	blubber	0.87	ug kg-1 ww	1078	1070	< 5	8	Law RJ et al. (2006)
2003	East	Harbour porpoise	blubber	0.85	ug kg-1 ww	6645	6570	54	21	Law RJ et al. (2006)
2003	East	Harbour porpoise	blubber	0.85	ug kg-1 ww	2200	2200	< 5	< 5	Law RJ et al. (2006)
2003	West	Harbour porpoise	blubber	0.88	ug kg-1 ww	1030	1030	< 5	< 5	Law RJ et al. (2006)
2003	Scotland	Harbour porpoise	blubber	0.85	ug kg-1 ww	453	453	< 5	< 5	Law RJ et al. (2006)
2003	West	Harbour porpoise	blubber	0.88	ug kg-1 ww	9186	9170	16	< 5	Law RJ et al. (2006)
2003	East	Harbour porpoise	blubber	0.88	ug kg-1 ww	8460	8410	50	< 5	Law RJ et al (2006)
2003	Scotland	Harbour porpoise	blubber	0.90	ug kg-1 ww	310	310	< 10	< 5	Law RJ et al. (2008)
2003	Scotland	Harbour porpoise	blubber	0.90	ug kg-1 ww	2790	2790	< 10	< 5	Law RJ et al. (2008)
2003	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	8490	8490	< 10	< 5	Law RJ et al. (2008)
2003	West England	Harbour porpoise	blubber	0.92	ug kg-1 ww	1190	1190	< 10	< 5	Law RJ et al. (2008)
2003	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	200	200	< 10	< 5	Law RJ et al. (2008)
2003	Scotland	Harbour porpoise	blubber	0.86	ug kg-1 ww	710	710	< 10	< 5	Law RJ et al. (2008)
2003	West England	Harbour porpoise	blubber	0.91	ug kg-1 ww	280	280	< 10	< 5	Law RJ et al. (2008)
2003	West England	Harbour porpoise	blubber	0.91	ug kg-1 ww	530	530	< 10	< 5	Law RJ et al. (2008)
2003	Scotland	Harbour porpoise	blubber	0.86	ug kg-1 ww	6230	6230	< 10	< 5	Law RJ et al. (2008)
2003	Scotland	Harbour porpoise	blubber	0.90	ug kg-1 ww	11500	11500	< 10	< 5	Law RJ et al. (2008)
2003	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	220	220	< 10	< 5	Law RJ et al. (2008)
2003	West England	Harbour porpoise	blubber	0.93	ug kg-1 ww	600	600	< 10	< 5	Law RJ et al. (2008)
2003	West England	Harbour porpoise	blubber	0.92	ug kg-1 ww	93	93	< 10	< 5	Law RJ et al. (2008)
2003	West England	Harbour porpoise	blubber	0.94	ug kg-1 ww	72	72	< 10	< 5	Law RJ et al. (2008)
2003	Scotland	Harbour porpoise	blubber	0.88	ug kg-1 ww	560	560	< 10	< 5	Law RJ et al. (2008)
2003	West England	Harbour porpoise	blubber	0.88	ug kg-1 ww	2140	2140	< 10	< 5	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.76	ug kg-1 ww	370	370	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	440	440	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	3870	3870	< 10	< 5	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.90	ug kg-1 ww	600	600	< 10	< 5	Law RJ et al. (2008)
2004	East England	Harbour porpoise	blubber	0.91	ug kg-1 ww	620	620	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	3410	3410	< 10	< 5	Law RJ et al. (2008)
2004	East England	Harbour porpoise	blubber	0.91	ug kg-1 ww	390	390	< 10	< 5	Law RJ et al. (2008)
2004	East England	Harbour porpoise	blubber	0.93	ug kg-1 ww	200	200	< 10	< 5	Law RJ et al. (2008)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
2004	West England	Harbour porpoise	blubber	0.94	ug kg-1 ww	1650	1650	< 10	< 5	Law RJ et al. (2008)
2004	South England	Harbour porpoise	blubber	0.95	ug kg-1 ww	120	120	< 10	< 5	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.88	ug kg-1 ww	280	280	< 10	< 5	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.95	ug kg-1 ww	920	920	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	230	230	< 10	< 5	Law RJ et al. (2008)
2004	South England	Harbour porpoise	blubber	0.94	ug kg-1 ww	170	170	< 10	< 5	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.87	ug kg-1 ww	3450	3450	< 10	< 5	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.82	ug kg-1 ww	4150	4150	< 10	< 5	Law RJ et al. (2008)
2004	East England	Harbour porpoise	blubber	0.89	ug kg-1 ww	34	34	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.86	ug kg-1 ww	4060	4060	< 10	< 5	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.89	ug kg-1 ww	87	87	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.85	ug kg-1 ww	240	240	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.85	ug kg-1 ww	3110	3110	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.84	ug kg-1 ww	260	260	< 10	< 5	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.89	ug kg-1 ww	1120	1120	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	170	170	< 10	< 10	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.92	ug kg-1 ww	780	780	< 10	< 5	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.92	ug kg-1 ww	190	190	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.77	ug kg-1 ww	710	710	< 9	< 9	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.91	ug kg-1 ww	1170	1170	< 10	< 5	Law RJ et al. (2008)
2004	South England	Harbour porpoise	blubber	0.93	ug kg-1 ww	94	94	< 10	< 5	Law RJ et al. (2008)
2004	West England	Harbour porpoise	blubber	0.86	ug kg-1 ww	3720	3720	< 10	< 5	Law RJ et al. (2008)
2004	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	100	100	< 10	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.93	ug kg-1 ww	167	167	< 5	< 5	Law RJ et al. (2008)
2005	East England	Harbour porpoise	blubber	0.9	ug kg-1 ww	1160	1160	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	8470	8470	< 10	< 5	Law RJ et al. (2008)
2005	East England	Harbour porpoise	blubber	0.89	ug kg-1 ww	616	616	< 5	< 5	Law RJ et al. (2008)
2005	East England	Harbour porpoise	blubber	0.92	ug kg-1 ww	55	55	< 5	< 5	Law RJ et al. (2008)
2005	West England	Harbour porpoise	blubber	0.95	ug kg-1 ww	1620	1620	< 6	< 6	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.9	ug kg-1 ww	514	508	< 5	6	Law RJ et al. (2008)
2005	South England	Harbour porpoise	blubber	0.79	ug kg-1 ww	440	440	< 4	< 4	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.85	ug kg-1 ww	550	550	< 10	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.88	ug kg-1 ww	2105	2100	< 5	5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.84	ug kg-1 ww	922	922	< 5	5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.83	ug kg-1 ww	2398	2398	< 5	< 5	Law RJ et al. (2008)
2005	East England	Harbour porpoise	blubber	0.91	ug kg-1 ww	51	51	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	239	239	< 5	< 5	Law RJ et al. (2008)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
2005	East England	Harbour porpoise	blubber	0.91	ug kg-1 ww	158	158	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.9	ug kg-1 ww	114	114	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.94	ug kg-1 ww	42	42	< 5	< 5	Law RJ et al. (2008)
2005	South England	Harbour porpoise	blubber	0.91	ug kg-1 ww	1590	1590	< 5	< 5	Law RJ et al. (2008)
2005	South England	Harbour porpoise	blubber	0.91	ug kg-1 ww	1012	1012	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	4128	4122	< 5	6	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.88	ug kg-1 ww	2720	2720	< 10	< 5	Law RJ et al. (2008)
2005	East England	Harbour porpoise	blubber	0.91	ug kg-1 ww	233	233	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.9	ug kg-1 ww	1153	1148	< 5	5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.9	ug kg-1 ww	861	861	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	740	740	< 10	< 5	Law RJ et al. (2008)
2005	East England	Harbour porpoise	blubber	0.9	ug kg-1 ww	63	63	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	245	245	< 5	< 5	Law RJ et al. (2008)
2005	East England	Harbour porpoise	blubber	0.81	ug kg-1 ww	189	183	6	< 4	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.9	ug kg-1 ww	95	95	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	224	224	< 5	< 5	Law RJ et al. (2008)
2005	East England	Harbour porpoise	blubber	0.9	ug kg-1 ww	106	99	7	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.88	ug kg-1 ww	1580	1580	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.95	ug kg-1 ww	433	433	< 6	< 6	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	69	69	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.9	ug kg-1 ww	190	190	< 10	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	193	193	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	420	420	< 10	< 5	Law RJ et al. (2008)
2005	West England	Harbour porpoise	blubber	0.89	ug kg-1 ww	150	150	< 5	< 5	Law RJ et al. (2008)
2005	West England	Harbour porpoise	blubber	0.92	ug kg-1 ww	348	348	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	84	84	< 10	< 5	Law RJ et al. (2008)
2005	West England	Harbour porpoise	blubber	0.92	ug kg-1 ww		< 5	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	888	888	< 5	< 5	Law RJ et al. (2008)
2005	West England	Harbour porpoise	blubber	0.93	ug kg-1 ww	166	166	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	69	69	< 10	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.81	ug kg-1 ww	226	226	< 5	< 5	Law RJ et al. (2008)
2005	West England	Harbour porpoise	blubber	0.94	ug kg-1 ww	490	490	< 6	< 6	Law RJ et al. (2008)
2005	West England	Harbour porpoise	blubber	0.79	ug kg-1 ww	49	49	< 4	< 4	Law RJ et al. (2008)
2005	West England	Harbour porpoise	blubber	0.7	ug kg-1 ww	492	492	< 4	< 4	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	8310	8310	< 10	< 5	Law RJ et al. (2008)
2005	West England	Harbour porpoise	blubber	0.78	ug kg-1 ww	1201	1201	< 4	< 4	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.8	ug kg-1 ww	280	280	< 10	< 5	Law RJ et al. (2008)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
2005	Scotland	Harbour porpoise	blubber	0.84	ug kg-1 ww	201	201	< 5	< 5	Law RJ et al. (2008)
2005	South England	Harbour porpoise	blubber	0.85	ug kg-1 ww	1031	1031	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.9	ug kg-1 ww	193	193	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.9	ug kg-1 ww	869	869	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.93	ug kg-1 ww	110	110	< 4	< 2	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	390	390	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.9	ug kg-1 ww	4225	4225	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	2689	2689	< 5	< 5	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.92	ug kg-1 ww	4113	4113	< 5	< 5	Law RJ et al. (2008)
2005	West England	Harbour porpoise	blubber	0.93	ug kg-1 ww	2363	2357	< 5	6	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.97	ug kg-1 ww	1038	1038	< 6	< 6	Law RJ et al. (2008)
2005	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	635	631	4	< 5	Law RJ et al. (2008)
2006	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	278	278	< 5	< 5	Law RJ et al. (2008)
2006	Scotland	Harbour porpoise	blubber	0.93	ug kg-1 ww	548	548	< 5	< 5	Law RJ et al. (2008)
2006	East England	Harbour porpoise	blubber	0.72	ug kg-1 ww	344	200	99	45	Law RJ et al. (2008)
2006	Scotland	Harbour porpoise	blubber	0.87	ug kg-1 ww	311	311	< 5	< 5	Law RJ et al. (2008)
2006	West England	Harbour porpoise	blubber	0.9	ug kg-1 ww	6358	6358	< 5	< 5	Law RJ et al. (2008)
2006	Scotland	Harbour porpoise	blubber	0.93	ug kg-1 ww	2415	2415	< 5	< 5	Law RJ et al. (2008)
2006	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	218	218	< 5	< 5	Law RJ et al. (2008)
2006	Scotland	Harbour porpoise	blubber	0.93	ug kg-1 ww	81	81	< 5	< 5	Law RJ et al. (2008)
2006	Scotland	Harbour porpoise	blubber	0.89	ug kg-1 ww	132	132	< 5	< 5	Law RJ et al. (2008)
2006	Scotland	Harbour porpoise	blubber	0.91	ug kg-1 ww	652	652	< 5	< 5	Law RJ et al. (2008)
2006	Scotland	Harbour porpoise	blubber	0.94	ug kg-1 ww	88	88	< 6	< 6	Law RJ et al. (2008)
2006	West England	Harbour porpoise	blubber	0.93	ug kg-1 ww	517	508	< 5	9	Law RJ et al. (2008)
2006	East England	Harbour porpoise	blubber	0.89	ug kg-1 ww	115	115	< 5	< 5	Law RJ et al. (2008)
2006	Scotland	Harbour porpoise	blubber	0.88	ug kg-1 ww	64	64	< 5	< 5	Law RJ et al. (2008)
2006	East England	Harbour porpoise	blubber	0.86	ug kg-1 ww	115	85	12	18	Law RJ et al. (2008)
2006	West England	Harbour porpoise	blubber	0.9	ug kg-1 ww	267	267	< 5	< 5	Law RJ et al. (2008)
2006	East England	Harbour porpoise	blubber	0.91	ug kg-1 ww	524	524	< 5	< 5	Law RJ et al. (2008)
2006	East England	Harbour porpoise	blubber	0.83	ug kg-1 ww	697	677	12	8	Law RJ et al. (2008)
2006	East England	Harbour porpoise	blubber	0.89	ug kg-1 ww	1139	1139	< 5	< 5	Law RJ et al. (2008)
2006	West England	Harbour porpoise	blubber	0.9	ug kg-1 ww	120	120	< 5	< 5	Law RJ et al. (2008)
2006	West England	Harbour porpoise	blubber	0.9	ug kg-1 ww	1554	1554	< 5	< 5	Law RJ et al. (2008)
2006	West England	Harbour porpoise	blubber	0.92	ug kg-1 ww	130	130	< 5	< 5	Law RJ et al. (2008)
2006	West England	Harbour porpoise	blubber	0.82	ug kg-1 ww	415	415	< 4	< 4	Law RJ et al. (2008)
2006	East England	Harbour porpoise	blubber	0.6	ug kg-1 ww	1535	1535	< 3	< 3	Law RJ et al. (2008)
2006	East England	Harbour porpoise	blubber	0.86	ug kg-1 ww	489	365	80	44	Law RJ et al. (2008)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
2006	South England	Harbour porpoise	blubber	0.69	ug kg-1 ww	277	277	< 4	< 4	Law RJ et al. (2008)
2006	South England	Harbour porpoise	blubber	0.86	ug kg-1 ww	112	105	7	< 5	Law RJ et al. (2008)
2006	West England	Harbour porpoise	blubber	0.9	ug kg-1 ww	122	122	< 5	< 5	Law RJ et al. (2008)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	448.8				Zegers et al. (2005)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	781.3				Zegers et al. (2005)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	2593.2				Zegers et al. (2005)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	770.2				Zegers et al. (2005)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	497.0				Zegers et al. (2005)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	1054.5				Zegers et al. (2005)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	1564.3				Zegers et al. (2005)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	1068.2				Zegers et al. (2005)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	744.3				Zegers et al. (2005)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	393.3				Zegers et al. (2005)
2001 - 2003	North, North Sea	Harbour porpoise	blubber		ug kg-1 lw	637.5				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	678.9				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	1218.5				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	1232.8				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	537.9				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	666.0				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	1676.0				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	1160.2				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	815.9				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	1596.5				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	1064.7				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	1143.8				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	2311.8				Zegers et al. (2005)
2000 - 2003	South, North Sea	Harbour porpoise	blubber		ug kg-1 lw	754.0				Zegers et al. (2005)
2001 - 2003	NW Scotland	Harbour porpoise	blubber		ug kg-1 lw	1502.6				Zegers et al. (2005)
2001 - 2003	NW Scotland	Harbour porpoise	blubber		ug kg-1 lw	9590.5				Zegers et al. (2005)
2001 - 2003	NW Scotland	Harbour porpoise	blubber		ug kg-1 lw	6324.8				Zegers et al. (2005)
2001 - 2003	NW Scotland	Harbour porpoise	blubber		ug kg-1 lw	1009.0				Zegers et al. (2005)
2001 - 2003	NW Scotland	Harbour porpoise	blubber		ug kg-1 lw	5056.3				Zegers et al. (2005)
2002 - 2003	Irish Sea	Harbour porpoise	blubber		ug kg-1 lw	930.8				Zegers et al. (2005)
2002 - 2003	Irish Sea	Harbour porpoise	blubber		ug kg-1 lw	6301.3				Zegers et al. (2005)
2002 - 2003	Irish Sea	Harbour porpoise	blubber		ug kg-1 lw	5343.2				Zegers et al. (2005)
2002 - 2003	Irish Sea	Harbour porpoise	blubber		ug kg-1 lw	466.3				Zegers et al. (2005)
2002 - 2003	Irish Sea	Harbour porpoise	blubber		ug kg-1 lw	3440.8				Zegers et al. (2005)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
2002 - 2003	Irish Sea	Harbour porpoise	blubber		ug kg-1 lw	1981.2				Zegers et al. (2005)
2002 - 2003	Irish Sea	Harbour porpoise	blubber		ug kg-1 lw	8786.4				Zegers et al. (2005)
2002 - 2003	Irish Sea	Harbour porpoise	blubber		ug kg-1 lw	2383.0				Zegers et al. (2005)
2001 - 2003	South Ireland	Harbour porpoise	blubber		ug kg-1 lw	2269.0				Zegers et al. (2005)
2001 - 2003	South Ireland	Harbour porpoise	blubber		ug kg-1 lw	1158.4				Zegers et al. (2005)
2001 - 2003	South Ireland	Harbour porpoise	blubber		ug kg-1 lw	710.0				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Harbour porpoise	blubber		ug kg-1 lw	78.8				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Harbour porpoise	blubber		ug kg-1 lw	142.6				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Harbour porpoise	blubber		ug kg-1 lw	142.3				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	174.9				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	630.5				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	279.1				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	97.6				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	180.2				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	418.4				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	344.0				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	199.1				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	388.8				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	898.6				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	460.6				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	717.1				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	188.2				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	197.3				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	215.0				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	253.4				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	235.7				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	442.7				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	619.3				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	472.2				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	438.5				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	597.3				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	417.9				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	365.7				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	568.9				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	468.5				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	760.6				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	702.7				Zegers et al. (2005)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
2002	NW France	Common dolphin	blubber		ug kg-1 lw	820.6				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	503.6				Zegers et al. (2005)
2002	NW France	Common dolphin	blubber		ug kg-1 lw	377.7				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	64.0				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	280.6				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	251.4				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	183.6				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	259.3				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	290.8				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	90.5				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	368.4				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	205.0				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	454.5				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	109.4				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	159.8				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	90.7				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	170.6				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	51.5				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	123.9				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	216.2				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	162.3				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	163.4				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	115.8				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	246.6				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	93.6				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	89.9				Zegers et al. (2005)
2001 - 2003	Galicia, Spain	Common dolphin	blubber		ug kg-1 lw	182.2				Zegers et al. (2005)
2001 - 2002	West Ireland	Common dolphin	blubber		ug kg-1 lw	727.3				Zegers et al. (2005)
2001 - 2002	West Ireland	Common dolphin	blubber		ug kg-1 lw	411.1				Zegers et al. (2005)
2001 - 2002	West Ireland	Common dolphin	blubber		ug kg-1 lw	193.2				Zegers et al. (2005)
2001 - 2002	West Ireland	Common dolphin	blubber		ug kg-1 lw	3416.3				Zegers et al. (2005)
2001 - 2002	West Ireland	Common dolphin	blubber		ug kg-1 lw	1591.0				Zegers et al. (2005)
2001 - 2002	West Ireland	Common dolphin	blubber		ug kg-1 lw	998.0				Zegers et al. (2005)
1997	US East Coast	white-sided dolphin	liver	0.031	ug kg-1 lw		68			Peck et al. (2008)
1993	US East Coast	white-sided dolphin	liver	0.0186	ug kg-1 lw		36			Peck et al. (2008)
1997	US East Coast	white-sided dolphin	liver	0.0275	ug kg-1 lw		29			Peck et al. (2008)
1993	US East Coast	white-sided dolphin	liver	0.0216	ug kg-1 lw		23			Peck et al. (2008)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
1993	US East Coast	white-sided dolphin	liver	0.0254	ug kg-1 lw		5.8			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	liver	0.0265	ug kg-1 lw		140			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	liver	0.0224	ug kg-1 lw		4.5			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	liver	0.0193	ug kg-1 lw		4.4			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	liver	0.0268	ug kg-1 lw		130			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	liver	0.0338	ug kg-1 lw		7.7			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	liver	0.0208	ug kg-1 lw		11			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	liver	0.0275	ug kg-1 lw		25			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	liver	0.032	ug kg-1 lw		81			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	liver	0.0334	ug kg-1 lw		13			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	liver	0.0174	ug kg-1 lw		3			Peck et al. (2008)
2000	US East Coast	white-sided dolphin	liver	0.0217	ug kg-1 lw		26			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.744	ug kg-1 lw		118			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.814	ug kg-1 lw		89.4			Peck et al. (2008)
1993	US East Coast	white-sided dolphin	blubber	0.848	ug kg-1 lw		214			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.695	ug kg-1 lw		79.4			Peck et al. (2008)
1997	US East Coast	white-sided dolphin	blubber	0.725	ug kg-1 lw		245			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.803	ug kg-1 lw		136			Peck et al. (2008)
1993	US East Coast	white-sided dolphin	blubber	0.86	ug kg-1 lw		164			Peck et al. (2008)
1993	US East Coast	white-sided dolphin	blubber	0.748	ug kg-1 lw		360			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.789	ug kg-1 lw		100			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.759	ug kg-1 lw		41			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.699	ug kg-1 lw		55			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.702	ug kg-1 lw		73			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.725	ug kg-1 lw		79			Peck et al. (2008)
2000	US East Coast	white-sided dolphin	blubber	0.744	ug kg-1 lw		80			Peck et al. (2008)
2001	US East Coast	white-sided dolphin	blubber	0.797	ug kg-1 lw		73			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.668	ug kg-1 lw		190			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.792	ug kg-1 lw		22			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.779	ug kg-1 lw		32			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.789	ug kg-1 lw		62			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.669	ug kg-1 lw		52			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.745	ug kg-1 lw		84			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.776	ug kg-1 lw		19			Peck et al. (2008)
1997	US East Coast	white-sided dolphin	blubber	0.435	ug kg-1 lw		260			Peck et al. (2008)
2000	US East Coast	white-sided dolphin	blubber	0.59	ug kg-1 lw		78			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.719	ug kg-1 lw		200			Peck et al. (2008)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
1997	US East Coast	white-sided dolphin	blubber	0.771	ug kg-1 lw		140			Peck et al. (2008)
1997	US East Coast	white-sided dolphin	blubber	0.545	ug kg-1 lw		190			Peck et al. (2008)
1994	US East Coast	white-sided dolphin	blubber	0.758	ug kg-1 lw		110			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.695	ug kg-1 lw		110			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.793	ug kg-1 lw		86			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.725	ug kg-1 lw		150			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.778	ug kg-1 lw		68			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.746	ug kg-1 lw		118			Peck et al. (2008)
2002	US East Coast	white-sided dolphin	blubber	0.822	ug kg-1 lw		55			Peck et al. (2008)
2002	US East Coast	white-sided dolphin	blubber	0.825	ug kg-1 lw		41			Peck et al. (2008)
2002	US East Coast	white-sided dolphin	blubber	0.789	ug kg-1 lw		47			Peck et al. (2008)
2003	US East Coast	white-sided dolphin	blubber	0.923	ug kg-1 lw		65			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.645	ug kg-1 lw		210			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.557	ug kg-1 lw		150			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.671	ug kg-1 lw		170			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.601	ug kg-1 lw		140			Peck et al. (2008)
2000	US East Coast	white-sided dolphin	blubber	0.685	ug kg-1 lw		76			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.702	ug kg-1 lw		68			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.577	ug kg-1 lw		350			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.627	ug kg-1 lw		100			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.66	ug kg-1 lw		86			Peck et al. (2008)
1998	US East Coast	white-sided dolphin	blubber	0.744	ug kg-1 lw		120			Peck et al. (2008)
2000	US East Coast	white-sided dolphin	blubber	0.523	ug kg-1 lw		250			Peck et al. (2008)
2000	US East Coast	white-sided dolphin	blubber	0.352	ug kg-1 lw		330			Peck et al. (2008)
2000	US East Coast	white-sided dolphin	blubber	0.451	ug kg-1 lw		170			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.685	ug kg-1 lw		93			Peck et al. (2008)
1999	US East Coast	white-sided dolphin	blubber	0.582	ug kg-1 lw		190			Peck et al. (2008)
2001	US East Coast	white-sided dolphin	blubber	0.705	ug kg-1 lw		34			Peck et al. (2008)
2001	US East Coast	white-sided dolphin	blubber	0.669	ug kg-1 lw		82			Peck et al. (2008)
2003	US East Coast	white-sided dolphin	blubber	0.671	ug kg-1 lw		160			Peck et al. (2008)
2003	US East Coast	white-sided dolphin	blubber	0.598	ug kg-1 lw		170			Peck et al. (2008)
2004	US East Coast	white-sided dolphin	blubber	0.399	ug kg-1 lw		220			Peck et al. (2008)
1993	California coast	California sea lion	blubber	0.1325	ug kg-1 ww	0.71	0.71	< 0.3	< 0.3	Stapleton et al. (2006)
1996	California coast	California sea lion	blubber	0.7917	ug kg-1 ww		< 0.3	< 0.3	< 0.3	Stapleton et al. (2006)
1996	California coast	California sea lion	blubber	0.6054	ug kg-1 ww	1.33	1.33	< 0.3	< 0.3	Stapleton et al. (2006)
1996	California coast	California sea lion	blubber	0.0816	ug kg-1 ww	1.12	1.12	< 0.3	< 0.3	Stapleton et al. (2006)
1996	California coast	California sea lion	blubber	0.5538	ug kg-1 ww		< 0.3	< 0.3	< 0.3	Stapleton et al. (2006)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
1996	California coast	California sea lion	blubber	0.1893	ug kg-1 ww		< 0.3	< 0.3	< 0.3	Stapleton et al. (2006)
1996	California coast	California sea lion	blubber	0.3598	ug kg-1 ww	0.31	0.31	< 0.3	< 0.3	Stapleton et al. (2006)
1997	California coast	California sea lion	blubber	0.3027	ug kg-1 ww	1.26	1.26	< 0.3	< 0.3	Stapleton et al. (2006)
1997	California coast	California sea lion	blubber	0.5492	ug kg-1 ww	2.38	2.38	< 0.3	< 0.3	Stapleton et al. (2006)
1997	California coast	California sea lion	blubber	0.6396	ug kg-1 ww	1.17	1.17	< 0.3	< 0.3	Stapleton et al. (2006)
1997	California coast	California sea lion	blubber	0.0632	ug kg-1 ww		< 0.3	< 0.3	< 0.3	Stapleton et al. (2006)
1997	California coast	California sea lion	blubber	0.2017	ug kg-1 ww	1.51	0.94	0.58	< 0.3	Stapleton et al. (2006)
1997	California coast	California sea lion	blubber	0.5359	ug kg-1 ww	2.67	1.79	0.87	< 0.3	Stapleton et al. (2006)
1997	California coast	California sea lion	blubber	0.4429	ug kg-1 ww	1.54	1.54	< 0.3	< 0.3	Stapleton et al. (2006)
1997	California coast	California sea lion	blubber	0.5356	ug kg-1 ww		< 0.3	< 0.3	< 0.3	Stapleton et al. (2006)
1997	California coast	California sea lion	blubber	0.4236	ug kg-1 ww	6.56	6.26	0.3	< 0.3	Stapleton et al. (2006)
1998	California coast	California sea lion	blubber	0.5831	ug kg-1 ww	0.63	0.63	< 0.3	< 0.3	Stapleton et al. (2006)
1999	California coast	California sea lion	blubber	0.4436	ug kg-1 ww	2.15	2.15	< 0.3	< 0.3	Stapleton et al. (2006)
1999	California coast	California sea lion	blubber	0.6635	ug kg-1 ww	3.18	3.18	< 0.3	< 0.3	Stapleton et al. (2006)
2000	California coast	California sea lion	blubber	0.0703	ug kg-1 ww	6.75	6.75	< 0.3	< 0.3	Stapleton et al. (2006)
2000	California coast	California sea lion	blubber	0.495	ug kg-1 ww	4.71	4.71	< 0.3	< 0.3	Stapleton et al. (2006)
2000	California coast	California sea lion	blubber	0.5099	ug kg-1 ww	5.91	5.91	< 0.3	< 0.3	Stapleton et al. (2006)
2000	California coast	California sea lion	blubber	0.6097	ug kg-1 ww	2.31	2.31	< 0.3	< 0.3	Stapleton et al. (2006)
2002	California coast	California sea lion	blubber	0.6268	ug kg-1 ww	5.6	5.6	< 0.3	< 0.3	Stapleton et al. (2006)
2002	California coast	California sea lion	blubber	0.6783	ug kg-1 ww	8.63	8.63	< 0.3	< 0.3	Stapleton et al. (2006)
2003	California coast	California sea lion	blubber	0.6904	ug kg-1 ww	11.85	11.85	< 0.3	< 0.3	Stapleton et al. (2006)
1979	Lake Ontario (east)	lake trout	whole body	0.129	ug kg-1 lw	33	25	0.94	6.5	(Ismail et al. 2009)
1983	Lake Ontario (east)	lake trout	whole body	0.141	ug kg-1 lw	28	25	0.4	2.9	Ismail et al. (in press)
1988	Lake Ontario (east)	lake trout	whole body	0.114	ug kg-1 lw	18	15	0.26	2.5	Ismail et al. (in press)
1993	Lake Ontario (east)	lake trout	whole body	0.104	ug kg-1 lw	32	27	0.38	4.6	Ismail et al. (in press)
1998	Lake Ontario (east)	lake trout	whole body	0.121	ug kg-1 lw	25	22	0.28	2.3	Ismail et al. (in press)
2004	Lake Ontario (east)	lake trout	whole body	0.163	ug kg-1 lw	16	15	0.16	1.4	Ismail et al. (in press)
1972	Offshore Japan	Northern fur seal	blubber	0.64 - 0.69	ug kg-1 lw	< 0.1				(Kajiwara et al. 2006)
1976	Offshore Japan	Northern fur seal	blubber	0.027 - 0.60	ug kg-1 lw	2.4				Kajiwara et al. (2006)
1980	Offshore Japan	Northern fur seal	blubber	0.71 - 0.79	ug kg-1 lw	0.77				Kajiwara et al. (2006)
1982	Offshore Japan	Northern fur seal	blubber	0.23 - 0.38	ug kg-1 lw	6.5				Kajiwara et al. (2006)
1985	Offshore Japan	Northern fur seal	blubber	0.48 - 0.60	ug kg-1 lw	1.6				Kajiwara et al. (2006)
1988	Offshore Japan	Northern fur seal	blubber	0.70 - 0.82	ug kg-1 lw	3.6				Kajiwara et al. (2006)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
1991	Offshore Japan	Northern fur seal	blubber	0.64 - 0.80	ug kg-1 lw	15				Kajiwara et al. (2006)
1994	Offshore Japan	Northern fur seal	blubber	0.72 - 0.80	ug kg-1 lw	33				Kajiwara et al. (2006)
1997	Offshore Japan	Northern fur seal	blubber	0.77 - 1	ug kg-1 lw	33				Kajiwara et al. (2006)
1998	Offshore Japan	Northern fur seal	blubber	0.72 - 0.88	ug kg-1 lw	24				Kajiwara et al. (2006)
1982	Pacific Coast, Japan	Melon-headed whale	blubber	0.46 - 0.80	ug kg-1 lw	7				Kajiwara et al. (2006)
2001	Pacific Coast, Japan	Melon-headed whale	blubber	0.63 - 0.74	ug kg-1 lw	390				Kajiwara et al. (2006)
1990	S.China Sea	Finless porpoise	blubber	0.87	ug kg-1 lw	5.6	4.8	0.34	0.51	(Isobe et al. 2007)
1990	S.China Sea	Finless porpoise	blubber	0.73	ug kg-1 lw	37	12	4	21	Isobe et al. (2007)
1990	S.China Sea	Finless porpoise	blubber	0.92	ug kg-1 lw	23	10	1.5	11	Isobe et al. (2007)
1990	S.China Sea	Finless porpoise	blubber	0.78	ug kg-1 lw	9.4	9.4			Isobe et al. (2007)
1990	S.China Sea	Finless porpoise	blubber	0.83	ug kg-1 lw	30	15	1.3	14	Isobe et al. (2007)
1990	S.China Sea	Finless porpoise	blubber	0.91	ug kg-1 lw	4.7	4.4		0.33	Isobe et al. (2007)
1990	S.China Sea	Finless porpoise	blubber	0.71	ug kg-1 lw	14	11	0.71	2.4	Isobe et al. (2007)
2000	Hong Kong	Finless porpoise	blubber	0.4	ug kg-1 lw	55	55			Isobe et al. (2007)
2000	Hong Kong	Finless porpoise	blubber	0.34	ug kg-1 lw	30	29		0.21	Isobe et al. (2007)
2001	Hong Kong	Finless porpoise	blubber	0.63	ug kg-1 lw	21	21			Isobe et al. (2007)
2001	Hong Kong	Finless porpoise	blubber	0.65	ug kg-1 lw	28	28			Isobe et al. (2007)
2001	Hong Kong	Finless porpoise	blubber	0.32	ug kg-1 lw	43	43			Isobe et al. (2007)
1997	Hong Kong	Humpback dolphin	blubber	0.53	ug kg-1 lw	83	83			Isobe et al. (2007)
1998	Hong Kong	Humpback dolphin	blubber	0.13	ug kg-1 lw	31	31		0.16	Isobe et al. (2007)
2000	Hong Kong	Humpback dolphin	blubber	0.34	ug kg-1 lw	120	120			Isobe et al. (2007)
2001	Hong Kong	Humpback dolphin	blubber	0.29	ug kg-1 lw	51	50		0.64	Isobe et al. (2007)
2001	Hong Kong	Humpback dolphin	blubber	0.32	ug kg-1 lw	64	64			Isobe et al. (2007)
2001	Hong Kong	Humpback dolphin	blubber	0.17	ug kg-1 lw	380	370		4.6	Isobe et al. (2007)
2001	Hong Kong	Humpback dolphin	blubber	0.18	ug kg-1 lw	47	46	0.59	0.55	Isobe et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.0078	ug kg-1 lw	12				(Ramu et al. 2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.017	ug kg-1 lw	6				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.0093	ug kg-1 lw					Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.016	ug kg-1 lw	66				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.018	ug kg-1 lw	45				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.01	ug kg-1 lw					Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.021	ug kg-1 lw	39				Ramu et al. (2007)

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2005	South Korea, coast	blue mussel	soft-flesh	0.0081	ug kg-1 lw					Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.018	ug kg-1 lw	500				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.021	ug kg-1 lw	21				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.023	ug kg-1 lw	73				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.019	ug kg-1 lw	53				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.014	ug kg-1 lw					Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.012	ug kg-1 lw					Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.0075	ug kg-1 lw	140				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.012	ug kg-1 lw	30				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.0086	ug kg-1 lw	49				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.013	ug kg-1 lw	350				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.0072	ug kg-1 lw	42				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.013	ug kg-1 lw	52				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.012	ug kg-1 lw	18				Ramu et al. (2007)
2005	South Korea, coast	blue mussel	soft-flesh	0.0095	ug kg-1 lw	38				Ramu et al. (2007)
1998	N-Pacific-1	skipjack tuna	muscle	0.037	ug kg-1 lw	25	22	0.22	1.6	(Ueno et al. 2006)
1997	N-Pacific-2	skipjack tuna	muscle	0.018	ug kg-1 lw	29	24	0.63	4.2	Ueno et al. (2006)
1998	N-Pacific-3	skipjack tuna	muscle	0.008	ug kg-1 lw	1.1	0.86	0.25	< 0.5	Ueno et al. (2006)
1997	off-Japan-1	skipjack tuna	muscle	0.049	ug kg-1 lw	32	30	0.27	1.9	Ueno et al. (2006)
1997	off-Japan-2	skipjack tuna	muscle	0.048	ug kg-1 lw	45	40	0.3	4.2	Ueno et al. (2006)
1997	Japan Sea	skipjack tuna	muscle	0.074	ug kg-1 lw	6.5	5	0.66	1.4	Ueno et al. (2006)
1997	E-China Sea-1	skipjack tuna	muscle	0.026	ug kg-1 lw	44	29	0.75	14	Ueno et al. (2006)
1997	E-China Sea-2	skipjack tuna	muscle	0.008	ug kg-1 lw	28	21	0.41	6.5	Ueno et al. (2006)
1998	off-Taiwan	skipjack tuna	muscle	0.004	ug kg-1 lw	27	24	0.38	2.3	Ueno et al. (2006)
2001	S-China Sea	skipjack tuna	muscle	0.012	ug kg-1 lw	3.2	2.5	0.1	0.65	Ueno et al. (2006)
1997	off-Philippines	skipjack tuna	muscle	0.006	ug kg-1 lw	0.86	0.86	< 0.1	< 0.4	Ueno et al. (2006)
1998	Bay of Bengal	skipjack tuna	muscle	0.008	ug kg-1 lw	0.27	0.27	< 0.1	< 0.4	Ueno et al. (2006)
1999	off-Indonesia	skipjack tuna	muscle	0.008	ug kg-1 lw	0.41	0.41	< 0.1	< 0.4	Ueno et al. (2006)
1999	off-Seychelles	skipjack tuna	muscle	0.007	ug kg-1 lw		< 0.1	< 0.1	< 0.4	Ueno et al. (2006)
2000	off-Brazil	skipjack tuna	muscle	0.031	ug kg-1 lw	0.28	0.28	< 0.03	< 0.1	Ueno et al. (2006)
Unknown	Unknown	Human	Milk	N/R	ug kg-1 lw	0.7				Geyer et al. 2004
1969	Baltic Sea	Guillemot	Egg	0.111	ug kg-1 lw	78				Sellström et al., 2003
1971	Baltic Sea	Guillemot	Egg	0.105	ug kg-1 lw	66				Sellström et al., 2003
1972	Baltic Sea	Guillemot	Egg	0.121	ug kg-1 lw	58				Sellström et al., 2003
1973	Baltic Sea	Guillemot	Egg	0.117	ug kg-1 lw	52				Sellström et al., 2003
1975	Baltic Sea	Guillemot	Egg	0.114	ug kg-1 lw	100				Sellström et al., 2003
1976	Baltic Sea	Guillemot	Egg	0.153	ug kg-1 lw	140				Sellström et al., 2003

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
1976	Baltic Sea	Guillemot	Egg	0.153	ug kg-1 lw	150				Sellström et al., 2003
1977	Baltic Sea	Guillemot	Egg	0.122	ug kg-1 lw	95				Sellström et al., 2003
1978	Baltic Sea	Guillemot	Egg	0.114	ug kg-1 lw	45				Sellström et al., 2003
1980	Baltic Sea	Guillemot	Egg	0.159	ug kg-1 lw	47				Sellström et al., 2003
1981	Baltic Sea	Guillemot	Egg	0.178	ug kg-1 lw	34				Sellström et al., 2003
1982	Baltic Sea	Guillemot	Egg	0.119	ug kg-1 lw	45				Sellström et al., 2003
1983	Baltic Sea	Guillemot	Egg	0.124	ug kg-1 lw	48				Sellström et al., 2003
1985	Baltic Sea	Guillemot	Egg	0.13	ug kg-1 lw	76				Sellström et al., 2003
1986	Baltic Sea	Guillemot	Egg	0.125	ug kg-1 lw	120				Sellström et al., 2003
1987	Baltic Sea	Guillemot	Egg	0.125	ug kg-1 lw	83				Sellström et al., 2003
1988	Baltic Sea	Guillemot	Egg	0.128	ug kg-1 lw	98				Sellström et al., 2003
1989	Baltic Sea	Guillemot	Egg	0.123	ug kg-1 lw	130				Sellström et al., 2003
1990	Baltic Sea	Guillemot	Egg	0.128	ug kg-1 lw	110				Sellström et al., 2003
1992	Baltic Sea	Guillemot	Egg	0.122	ug kg-1 lw	81				Sellström et al., 2003
1992	Baltic Sea	Guillemot	Egg	0.109	ug kg-1 lw	97				Sellström et al., 2003
1993	Baltic Sea	Guillemot	Egg	0.111	ug kg-1 lw	150				Sellström et al., 2003
1994	Baltic Sea	Guillemot	Egg	0.106	ug kg-1 lw	130				Sellström et al., 2003
1995	Baltic Sea	Guillemot	Egg	0.113	ug kg-1 lw	170				Sellström et al., 2003
1996	Baltic Sea	Guillemot	Egg	0.112	ug kg-1 lw	130				Sellström et al., 2003
1997	Baltic Sea	Guillemot	Egg	0.122	ug kg-1 lw	110				Sellström et al., 2003
1998	Baltic Sea	Guillemot	Egg	0.11	ug kg-1 lw	110				Sellström et al., 2003
1999	Baltic Sea	Guillemot	Egg	0.124	ug kg-1 lw	130				Sellström et al., 2003
2000	Baltic Sea	Guillemot	Egg	0.117	ug kg-1 lw	140				Sellström et al., 2003
2001	Baltic Sea	Guillemot	Egg	0.121	ug kg-1 lw	140				Sellström et al., 2003
2000-2002	Baltic Sea	Guillemot	egg		ug kg-1 lw	138				Lundstedt-Enkel et al., 2006b
2000-2002	Baltic Sea	Guillemot	muscle	0.029	ug kg-1 lw	64.7				Lundstedt-Enkel et al., 2006b
2000	Baltic Sea	Guillemot	muscle		ug kg-1 lw	66.7				Lundstedt-Enkel et al., 2005
2002	Svalbard	Guillemot		0.28	ug kg-1 lw	35.4				Murvoll et al., 2007
2002	Svalbard	Eider		0.21	ug kg-1 lw	6.23				Murvoll et al., 2007
1980	Sweden	Human	Milk		ug kg-1 lw	0.11				Fangstrom et al. 2008
1984	Sweden	Human	Milk		ug kg-1 lw	0.09				Fangstrom et al. 2008
1988	Sweden	Human	Milk		ug kg-1 lw	0.22				Fangstrom et al. 2008
1990	Sweden	Human	Milk		ug kg-1 lw	0.21				Fangstrom et al. 2008
1992	Sweden	Human	Milk		ug kg-1 lw	0.31				Fangstrom et al. 2008
1994	Sweden	Human	Milk		ug kg-1 lw	0.41				Fangstrom et al. 2008

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
1995	Sweden	Human	Milk		ug kg-1 lw	0.51				Fangstrom et al. 2008
1996	Sweden	Human	Milk		ug kg-1 lw	0.32				Fangstrom et al. 2008
1997	Sweden	Human	Milk		ug kg-1 lw	0.29				Fangstrom et al. 2008
1999	Sweden	Human	Milk		ug kg-1 lw	0.39				Fangstrom et al. 2008
2001	Sweden	Human	Milk		ug kg-1 lw	0.53				Fangstrom et al. 2008
2002	Sweden	Human	Milk		ug kg-1 lw	0.64				Fangstrom et al. 2008
2003	Sweden	Human	Milk		ug kg-1 lw	0.05				Fangstrom et al. 2008
2004	Sweden	Human	Milk		ug kg-1 lw	0.37				Fangstrom et al. 2008
1980	Sweden	Human	Milk		ug kg-1 lw	0.06				Fangstrom et al. 2008
1984	Sweden	Human	Milk		ug kg-1 lw	0.10				Fangstrom et al. 2008
1988	Sweden	Human	Milk		ug kg-1 lw	0.26				Fangstrom et al. 2008
1990	Sweden	Human	Milk		ug kg-1 lw	0.19				Fangstrom et al. 2008
1992	Sweden	Human	Milk		ug kg-1 lw	0.26				Fangstrom et al. 2008
1994	Sweden	Human	Milk		ug kg-1 lw	0.35				Fangstrom et al. 2008
1995	Sweden	Human	Milk		ug kg-1 lw	0.52				Fangstrom et al. 2008
1996	Sweden	Human	Milk		ug kg-1 lw	0.35				Fangstrom et al. 2008
1997	Sweden	Human	Milk		ug kg-1 lw	0.30				Fangstrom et al. 2008
1999	Sweden	Human	Milk		ug kg-1 lw	0.35				Fangstrom et al. 2008
2001	Sweden	Human	Milk		ug kg-1 lw	0.55				Fangstrom et al. 2008
2002	Sweden	Human	Milk		ug kg-1 lw	0.53				Fangstrom et al. 2008
2003	Sweden	Human	Milk		ug kg-1 lw	0.49				Fangstrom et al. 2008
2004	Sweden	Human	Milk		ug kg-1 lw	0.40				Fangstrom et al. 2008
~2007	Czech Republic	Human	fat tissue	0.83	ug kg-1 lw	1.2				Pulkrabová et al., 2009
~2007	Spain	Human	Milk		ug kg-1 lw	27				Eljarrat et al. 2009
2001	Sweden	Human	Milk		ug kg-1 lw	0.5				Cited in Eljarrat et al. 2009
2002-2003	Spain	Human	Milk		ug kg-1 lw	0.5				Cited in Eljarrat et al. 2009
2002-2003	Canada	Human	Milk		ug kg-1 lw	3.8				Cited in Eljarrat et al. 2009
2005	France	Human	Milk		ug kg-1 lw	3				Antignac et al. 2008
~2007	Norway	Human	Blood		ug kg-1 lw	4.1				Thomsen et al., 2008
~2007	Norway	Human	Blood		ug kg-1 lw	2.6				Thomsen et al., 2008
1999	Sweden	Bovine meat	Unknown	0.87	ug kg-1 lw	1.4				Remberger et al. 2004
1999	Sweden	Pork meat	Unknown	0.82	ug kg-1 lw	1.0				Remberger et al. 2004
1999	Sweden	Lamb	Unknown	0.81	ug kg-1 lw	4.1				Remberger et al. 2004
1999	Sweden	Chicken	Unknown	0.63	ug kg-1 lw	6.5				Remberger et al. 2004
1999	Sweden	Egg Yolk	Unknown	0.25	ug kg-1 lw	9.4				Remberger et al. 2004
1999	Sweden	Milk	Unknown	0.038	ug kg-1 lw	1.8				Remberger et al. 2004
1999	Sweden	Fish (mixed)	Unknown	0.06	ug kg-1 lw	48.0				Remberger et al. 2004

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
2004-2007	Norway	Farmed trout	Unknown	0.195	ug kg-1 lw	3.6				Knutsen et al., 2008
2004-2007	Norway	Herring	Unknown	0.114	ug kg-1 lw	3.9				Knutsen et al., 2008
2004-2007	Norway	Sardines	Unknown	0.291	ug kg-1 lw	2.7				Knutsen et al., 2008
2004-2007	Norway	Farmed salmon	Unknown	0.148	ug kg-1 lw	1.8				Knutsen et al., 2008
2004-2007	Norway	Mackerel	Unknown	0.257	ug kg-1 lw	3.1				Knutsen et al., 2008
2004-2007	Norway	Smoked salmon	Unknown	0.111	ug kg-1 lw	2.7				Knutsen et al., 2008
2004-2007	Norway	Shrimp	Unknown	0.012	ug kg-1 lw	1.1				Knutsen et al., 2008
2004-2007	Norway	Crab	Unknown	0.092	ug kg-1 lw	3.0				Knutsen et al., 2008
2004-2007	Norway	Bovine meat	Unknown	0.178	ug kg-1 lw	0.3				Knutsen et al., 2008
2004-2007	Norway	Pork meat	Unknown	0.248	ug kg-1 lw	0.1				Knutsen et al., 2008
2004-2007	Norway	Dairy products	Unknown	0.521	ug kg-1 lw	0.3				Knutsen et al., 2008
2004-2007	Norway	Hen egg	Unknown	0.098	ug kg-1 lw	1.6				Knutsen et al., 2008
2004-2007	Norway	Seagull egg	Unknown	0.094	ug kg-1 lw	70.4				Knutsen et al., 2008
2004-2007	Norway	Vegetable oil	Unknown	1	ug kg-1 lw	0.4				Knutsen et al., 2008
2004-2007	Norway	Banana	Unknown	0.001	ug kg-1 lw	0.04				Knutsen et al., 2008
2001-2003	Czech Republic	barbel (fish)	muscle	0.034	ug kg-1 lw	68				Pulkrabová et al., 2007
2001-2003	Czech Republic	barbel (fish)	muscle	0.048	ug kg-1 lw	174				Pulkrabová et al., 2007
2001-2003	Czech Republic	barbel (fish)	muscle	0.04	ug kg-1 lw	389				Pulkrabová et al., 2007
2001-2003	Czech Republic	barbel (fish)	muscle	0.036	ug kg-1 lw	101				Pulkrabová et al., 2007
2001-2003	Czech Republic	barbel (fish)	muscle	0.024	ug kg-1 lw	75				Pulkrabová et al., 2007
2002	Spain	barbel (fish)	muscle		ug kg-1 ww	309				Eljarrat et al., 2004
2002	Spain	barbel (fish)	muscle		ug kg-1 ww	750.4				Eljarrat et al., 2004
2002	Spain	barbel (fish)	liver		ug kg-1 ww	432.3				Eljarrat et al., 2004
2002	Spain	barbel (fish)	liver		ug kg-1 ww	554.4				Eljarrat et al., 2004
2002	Spain	barbel (fish)	liver		ug kg-1 ww	624.8				Eljarrat et al., 2004
2002	Spain	barbel (fish)	liver		ug kg-1 ww	483.9				Eljarrat et al., 2004
2002	Spain	barbel (fish)	muscle		ug kg-1 ww	89.5				Eljarrat et al., 2004
2002	Spain	barbel (fish)	muscle		ug kg-1 ww	529.7				Eljarrat et al., 2004
2002	Spain	barbel (fish)	muscle		ug kg-1 ww	0.02				Eljarrat et al., 2004
2002	Spain	barbel (fish)	liver		ug kg-1 ww	0.02				Eljarrat et al., 2004
2002	Spain	barbel (fish)	liver		ug kg-1 ww	0.02				Eljarrat et al., 2004
2002	Spain	barbel (fish)	muscle		ug kg-1 ww	0.02				Eljarrat et al., 2004
2002	Spain	bleak (fish)	whole		ug kg-1 ww	0.02				Eljarrat et al., 2005
2002	Spain	bleak (fish)	whole		ug kg-1 ww	0.02				Eljarrat et al., 2005
2002	Spain	bleak (fish)	whole		ug kg-1 ww	1501				Eljarrat et al., 2005
2002	Spain	bleak (fish)	whole		ug kg-1 ww	172				Eljarrat et al., 2005
2002	Spain	bleak (fish)	whole		ug kg-1 ww	760				Eljarrat et al., 2005

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
2002	Spain	bleak (fish)	whole		ug kg-1 ww	381				Eljarrat et al., 2005
2001-2003	Czech Republic	bream (fish)	muscle	0.041	ug kg-1 lw	2				Pulkrabová et al., 2007
2001-2003	Czech Republic	bream (fish)	muscle	0.023	ug kg-1 lw	34				Pulkrabová et al., 2007
2001-2003	Czech Republic	bream (fish)	muscle	0.035	ug kg-1 lw	39				Pulkrabová et al., 2007
2001-2003	Czech Republic	bream (fish)	muscle	0.046	ug kg-1 lw	161				Pulkrabová et al., 2007
2001-2003	Czech Republic	bream (fish)	muscle	0.021	ug kg-1 lw	108				Pulkrabová et al., 2007
2001-2003	Czech Republic	bream (fish)	muscle	0.021	ug kg-1 lw	328				Pulkrabová et al., 2007
2005	Czech Republic	bream (fish)	muscle	0.021	ug kg-1 lw	576.2				Hajslova et al., 2007
2001-2003	Czech Republic	chub (fish)	muscle	0.027	ug kg-1 lw	58				Pulkrabová et al., 2007
2001-2003	Czech Republic	chub (fish)	muscle	0.021	ug kg-1 lw	64				Pulkrabová et al., 2007
2001-2003	Czech Republic	chub (fish)	muscle	0.032	ug kg-1 lw	115				Pulkrabová et al., 2007
2001-2003	Czech Republic	chub (fish)	muscle	0.019	ug kg-1 lw	215				Pulkrabová et al., 2007
2001-2003	Czech Republic	chub (fish)	muscle	0.021	ug kg-1 lw	183				Pulkrabová et al., 2007
2001-2003	Czech Republic	chub (fish)	muscle	0.03	ug kg-1 lw	46				Pulkrabová et al., 2007
2005	Czech Republic	chub (fish)	muscle	0.018	ug kg-1 lw	116.7				Hajslova et al., 2007
2001-2003	Czech Republic	perch (fish)	muscle	0.007	ug kg-1 lw	14				Pulkrabová et al., 2007
2001-2003	Czech Republic	perch (fish)	muscle	0.01	ug kg-1 lw	42				Pulkrabová et al., 2007
2001-2003	Czech Republic	perch (fish)	muscle	0.006	ug kg-1 lw	82				Pulkrabová et al., 2007
2001-2003	Czech Republic	perch (fish)	muscle	0.008	ug kg-1 lw	114				Pulkrabová et al., 2007
2001-2003	Czech Republic	perch (fish)	muscle	0.007	ug kg-1 lw	123				Pulkrabová et al., 2007
2001-2003	Czech Republic	perch (fish)	muscle	0.009	ug kg-1 lw	177				Pulkrabová et al., 2007
2005	Czech Republic	perch (fish)	muscle	0.017	ug kg-1 lw	158.8				Hajslova et al., 2007
2001-2003	Czech Republic	trout (fish)	muscle		ug kg-1 lw	6				Pulkrabová et al., 2007
2001-2003	Czech Republic	trout (fish)	muscle		ug kg-1 lw	5				Pulkrabová et al., 2007
2001-2003	Czech Republic	trout (fish)	muscle		ug kg-1 lw	4				Pulkrabová et al., 2007
2004	Svalbard	glaucous gulls	plasma	0.0154	ug kg-1 ww	0.34				(Verrault et al. 2005)
2004	Svalbard	glaucous gulls	plasma	0.0148	ug kg-1 ww	0.32				Verrault et al. (2005)
2002	Svalbard	polar bears	plasma	0.0101	ug kg-1 ww					Verrault et al. (2005)
2002, 2004	Bear Island	glaucous gulls	plasma	0.0143	ug kg-1 ww	0.51				Verrault et al. (2005)
2002, 2004	Bear Island	glaucous gulls	plasma	0.0138	ug kg-1 ww	0.7				Verrault et al. (2005)
2002, 2004	Bear Island	gull eggs	egg fat	0.0961	ug kg-1 ww	13.3				Verrault et al. (2005)
1983	Hornoya	Herring gull	egg	0.11	ug kg-1 ww	1.8	1.8	< DL	< DL	Knudsen et al. (2005)
1993	Hornoya	Herring gull	egg	0.102	ug kg-1 ww	1.9	1.9	< DL	< DL	Knudsen et al. (2005)
2003	Hornoya	Herring gull	egg	0.097	ug kg-1 ww	9.3	9.3	< DL	< DL	Knudsen et al. (2005)
1983	Hornoya	A. puffin	egg	0.126	ug kg-1 ww	1.9	1.9	< DL	< DL	Knudsen et al. (2005)
1993	Hornoya	A. puffin	egg	0.117	ug kg-1 ww	4.9	4.9	< DL	< DL	Knudsen et al. (2005)
2003	Hornoya	A. puffin	egg	0.137	ug kg-1 ww	10.1	10.1	< DL	< DL	Knudsen et al. (2005)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
1983	Hornoya	Kittiwake	egg	0.098	ug kg-1 ww	1.7	1.7	< DL	< DL	Knudsen et al. (2005)
1993	Hornoya	Kittiwake	egg	0.096	ug kg-1 ww	3	3	< DL	< DL	Knudsen et al. (2005)
2003	Hornoya	Kittiwake	egg	0.095	ug kg-1 ww	10.8	10.8	< DL	< DL	Knudsen et al. (2005)
1983	Rost/Hekkingen	Herring gull	egg	0.095	ug kg-1 ww	1.4	1.4	< DL	< DL	Knudsen et al. (2005)
1993	Rost/Hekkingen	Herring gull	egg	0.083	ug kg-1 ww	3.7	3.7	< DL	< DL	Knudsen et al. (2005)
2003	Rost/Hekkingen	Herring gull	egg	0.104	ug kg-1 ww	11.6	11.6	< DL	< DL	Knudsen et al. (2005)
1983	Rost/Hekkingen	A. puffin	egg	0.122	ug kg-1 ww	1.1	1.1	< DL	< DL	Knudsen et al. (2005)
1993	Rost/Hekkingen	A. puffin	egg	0.122	ug kg-1 ww	2.2	2.2	< DL	< DL	Knudsen et al. (2005)
2003	Rost/Hekkingen	A. puffin	egg	0.137	ug kg-1 ww	6.1	6.1	< DL	< DL	Knudsen et al. (2005)
1983	Rost/Hekkingen	Kittiwake	egg	0.071	ug kg-1 ww	2.9	2.9	< DL	< DL	Knudsen et al. (2005)
1993	Rost/Hekkingen	Kittiwake	egg	0.094	ug kg-1 ww	7.1	7.1	< DL	< DL	Knudsen et al. (2005)
2003	Rost/Hekkingen	Kittiwake	egg	0.099	ug kg-1 ww	17.3	17.3	< DL	< DL	Knudsen et al. (2005)
1997	Bear Island	glaucous gull	egg	0.09	ug kg-1 ww	2.3	2.3	< DL	< DL	Knudsen et al. (2005)
2002	Bear Island	glaucous gull	egg	0.085	ug kg-1 ww	12	12	< DL	< DL	Knudsen et al. (2005)
1986	South Greenland	peregrine falcon	egg	0.07	ug kg-1 lw	< 0.8				Vorkamp et al. (2005)
1987	South Greenland	peregrine falcon	egg	0.094	ug kg-1 lw	34				Vorkamp et al. (2005)
1988	South Greenland	peregrine falcon	egg	0.189	ug kg-1 lw					Vorkamp et al. (2005)
1988	South Greenland	peregrine falcon	egg	0.224	ug kg-1 lw					Vorkamp et al. (2005)
1988	South Greenland	peregrine falcon	egg	0.203	ug kg-1 lw					Vorkamp et al. (2005)
1989	South Greenland	peregrine falcon	egg	0.107	ug kg-1 lw	7.8				Vorkamp et al. (2005)
1990	South Greenland	peregrine falcon	egg	0.063	ug kg-1 lw	4.1				Vorkamp et al. (2005)
1990	South Greenland	peregrine falcon	egg	0.073	ug kg-1 lw	230				Vorkamp et al. (2005)
1990	South Greenland	peregrine falcon	egg	0.063	ug kg-1 lw	9				Vorkamp et al. (2005)
1991	South Greenland	peregrine falcon	egg	0.075	ug kg-1 lw	< 1.1				Vorkamp et al. (2005)
1991	South Greenland	peregrine falcon	egg	0.068	ug kg-1 lw	10				Vorkamp et al. (2005)
1991	South Greenland	peregrine falcon	egg	0.052	ug kg-1 lw	22				Vorkamp et al. (2005)
1992	South Greenland	peregrine falcon	egg	0.093	ug kg-1 lw	1.2				Vorkamp et al. (2005)
1992	South Greenland	peregrine falcon	egg	0.048	ug kg-1 lw	< 1.1				Vorkamp et al. (2005)
1992	South Greenland	peregrine falcon	egg	0.068	ug kg-1 lw	26				Vorkamp et al. (2005)
1992	South Greenland	peregrine falcon	egg	0.056	ug kg-1 lw	32				Vorkamp et al. (2005)
1994	South Greenland	peregrine falcon	egg	0.075	ug kg-1 lw	2.4				Vorkamp et al. (2005)
1994	South Greenland	peregrine falcon	egg	0.036	ug kg-1 lw	2.1				Vorkamp et al. (2005)
1994	South Greenland	peregrine falcon	egg	0.067	ug kg-1 lw	77				Vorkamp et al. (2005)
1994	South Greenland	peregrine falcon	egg	0.079	ug kg-1 lw	67				Vorkamp et al. (2005)
1994	South Greenland	peregrine falcon	egg	0.057	ug kg-1 lw	< 0.1				Vorkamp et al. (2005)
1995	South Greenland	peregrine falcon	egg	0.065	ug kg-1 lw	< 0.8				Vorkamp et al. (2005)
1995	South Greenland	peregrine falcon	egg	0.063	ug kg-1 lw	3.2				Vorkamp et al. (2005)

Year	Location	Organism	Tissue	f_{LIPID}	Units	Total HBCD	α-HBCD	β-HBCD	γ-HBCD	Reference
1998	South Greenland	peregrine falcon	egg	0.058	ug kg-1 lw	2.6				Vorkamp et al. (2005)
1998	South Greenland	peregrine falcon	egg	0.076	ug kg-1 lw	< 0.8				Vorkamp et al. (2005)
1999	South Greenland	peregrine falcon	egg	0.039	ug kg-1 lw	< 0.9				Vorkamp et al. (2005)
1999	South Greenland	peregrine falcon	egg	0.06	ug kg-1 lw	< 1.2				Vorkamp et al. (2005)
1999	South Greenland	peregrine falcon	egg	0.04	ug kg-1 lw	< 0.1				Vorkamp et al. (2005)
2000	South Greenland	peregrine falcon	egg	0.036	ug kg-1 lw	14				Vorkamp et al. (2005)
2000	South Greenland	peregrine falcon	egg	0.028	ug kg-1 lw					Vorkamp et al. (2005)
2000	South Greenland	peregrine falcon	egg	0.039	ug kg-1 lw	2.7				Vorkamp et al. (2005)
2000	South Greenland	peregrine falcon	egg	0.046	ug kg-1 lw	< 0.1				Vorkamp et al. (2005)
2001	South Greenland	peregrine falcon	egg	0.065	ug kg-1 lw	< 0.8				Vorkamp et al. (2005)
2002	South Greenland	peregrine falcon	egg	0.069	ug kg-1 lw	1.6				Vorkamp et al. (2005)
2002	South Greenland	peregrine falcon	egg	0.053	ug kg-1 lw	< 1.0				Vorkamp et al. (2005)
2003	South Greenland	peregrine falcon	egg	0.07	ug kg-1 lw	27				Vorkamp et al. (2005)
2003	South Greenland	peregrine falcon	egg	0.06	ug kg-1 lw	< 0.1				Vorkamp et al. (2005)
2003	Svalbard	calanoid copepods	whole body	0.0996	ug kg-1 lw	NA				Sormo et al. (2006)
2003	Svalbard	Thysanoessa inermis	whole body	0.1242	ug kg-1 lw	NA				Sormo et al. (2006)
2003	Svalbard	Themisto libellula	whole body	0.0901	ug kg-1 lw	NA				Sormo et al. (2006)
2003	Svalbard	Gammarus wilkitzkii	whole body	0.0385	ug kg-1 lw	ND				Sormo et al. (2006)
2003	Svalbard	polar cod	whole body	0.1201	ug kg-1 lw	1.89				Sormo et al. (2006)
2003	Svalbard	ringed seal	blubber	0.8923	ug kg-1 lw	19.56				Sormo et al. (2006)
2003	Svalbard	polar bears	adipose tissue	0.8385	ug kg-1 lw	11.53				Sormo et al. (2006)
2002	Svalbard	polar bears	adipose tissue	0.91	ug kg-1 lw	44.4				Muir et al (2006)
1999 - 2001	East Greenland	polar bears	adipose tissue	0.9	ug kg-1 lw	44.5				Muir et al (2006)
1994 - 2002	Bering-Chukchi Sea	polar bears	adipose tissue	0.6	ug kg-1 lw	0.4				Muir et al (2006)
2002	eastern Arctic	zooplankton	whole body	0.0369	ug kg-1 lw	1.1	0.6		0.5	Tomy et al. (2008)
2000 - 2001	eastern Arctic	shrimp	whole body	0.075	ug kg-1 lw	1.9	1.4		0.5	Tomy et al. (2008)
2002	eastern Arctic	clam	whole body	0.0101	ug kg-1 lw	1.4	0.2		1.2	Tomy et al. (2008)
2000 - 2001	eastern Arctic	red fish	whole body	0.0181	ug kg-1 lw	2	1.4		0.6	Tomy et al. (2008)
2000 - 2001	eastern Arctic	polar cod	whole body	0.1419	ug kg-1 lw	0.42	0.4		0.02	Tomy et al. (2008)
1998	eastern Arctic	walrus	blubber	0.8879	ug kg-1 lw	0.6	0.2		0.4	Tomy et al. (2008)
1996	eastern Arctic	beluga	blubber	0.9232	ug kg-1 lw	1.4	1.2		0.2	Tomy et al. (2008)
2000	eastern Arctic	narwhal	blubber	0.8296	ug kg-1 lw	3.4	2.9		0.5	Tomy et al. (2008)

9.4 MASS BALANCE MODELS

9.4.1 General Background

All models, including the mass balance models used in the present study, are necessarily based on simplifying assumptions and cannot be expected to replicate the inherent complexity of actual systems. Model development, evaluation (sometimes termed validation), refinement, and application are therefore key elements in the evolutionary scientific process towards improved understanding. Models cannot provide "truths" or make decisions; however, they can be used to inform decisions, particularly if they are designed for a specific purpose. It must be recognized that measurements are also limited for regulatory decision-making because they are usually incomplete and often variable and uncertain. A strategy for improving environmental and human health assessments is to combine both models and measurements (McKone et al. 2007). Further detailed discussions on models, particularly for chemical regulatory purposes, are provided elsewhere (Mackay 2001; Webster et al. 2005). Mass balance modelling concepts for organic chemical evaluations are first briefly described followed by brief overviews for the mass balance models used in the present study.

Mass balance multi-compartment models use mathematical expressions to combine chemical transport processes (e.g., diffusion in water and advection by wind) and chemical transformation processes (e.g., microbial degradation, hydrolysis) with thermodynamic principles to quantify chemical fate, transport and behavior. The models are grounded in laws for the conservation of mass and energy. The fugacity concept has proven particularly useful in the development of environmental mass balance models (Mackay 2001). In particular, the fugacity approach facilitates development of the mass balance equations and the results are more readily interpreted. For example, since fugacity is an equilibrium criterion analogous to chemical potential, the equilibrium status of the system can be readily assessed.

In a simplistic sense the biosphere is comprised of a multitude of interconnected compartments and phases of various composition and size. Examples include the atmosphere, water bodies, bottom sediment, soil, biological organisms, tissues and cells. These compartments and phases can be viewed as a series of "boxes" in which chemicals can exist either in a fixed state, pass through or be in the process of transformation or reaction. In the environment most of these compartments are heterogeneous in nature (e.g., sediments and soils); however, some are relatively well-mixed phases (e.g., ponds). Properties such as volume, density and composition of each compartment in the mass balance equation must be defined. A single phase (e.g. air) can be modelled as a single box or as a number of boxes thus providing greater spatial resolution for the total air compartment. Representing heterogeneous concentrations in mass balance models can be difficult resulting in complex and parameter-intensive models; therefore, a typical simplifying assumption for model development is that concentrations in a particular box are homogeneously distributed. This is often referred to as the Continuous Stirred Tank Reactor (CSTR) assumption. In certain cases, particularly for substances that are

transformed rapidly, the application of this assumption requires further consideration (Warren et al. 2009).

Spatially, mass balance equations link the transport of chemical into and out of the various compartments (e.g. from air to water or water into a fish) and quantify processes within these compartments (e.g. reaction or formation). The model equations can be derived and solved as a function of time or at steady state. Steady state reflects a situation in which the chemical concentrations no longer change as a function of time, i.e., chemical input quantities equal chemical "loss" or output quantities. The steady state assumption is often used because it simplifies the mathematical and computational requirements to solve the equations. It is recognized that complex environmental systems are never truly at steady state; however, in certain cases "pseudo-steady state" conditions can be observed. Commercial chemicals released to the environment continuously or semi-continuously over years of production and use may approximate "pseudo-steady state" conditions, at least in regions where they are discharged.

Models can be developed, parameterized and applied to specific conditions with a view to describing an actual environment or situation. Alternatively, models can also be developed and applied in an evaluative sense in which the system is parameterized to be representative of general environmental conditions.

9.4.2 Supplementary Data for Globo-POP Modelling

Comparison of $eACP_{10}$ values calculate for t-, α-, β-, γ-HBCD where t-HBCD is based on the physical-chemical properties estimated for the technical mixture

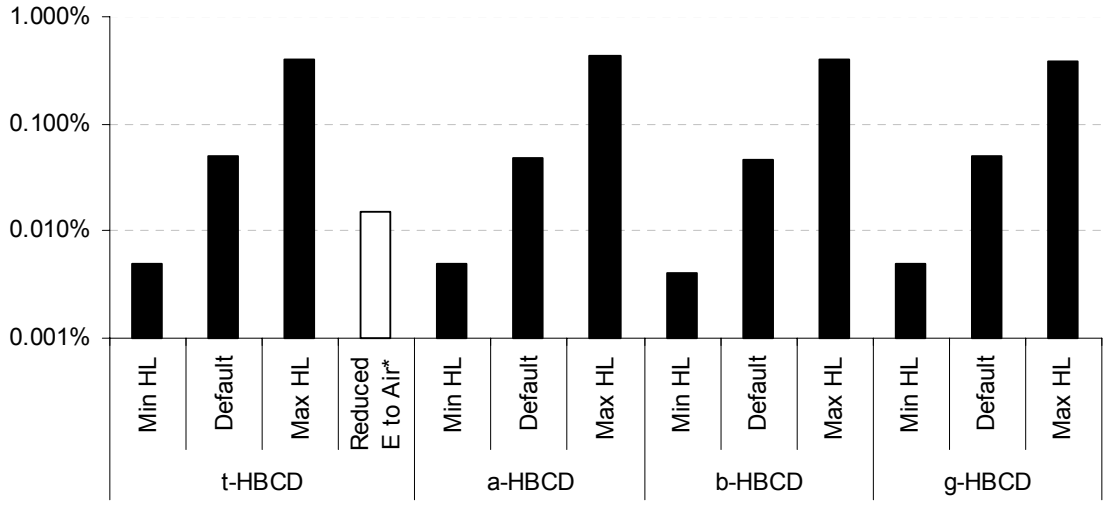

Figure 9.1. Comparison of $eACP_{10}$ values for t-, α-, β-, γ-HBCD

9.4.3 Supplementary Data for BETR-World Modelling

Table 9.9. Total Estimated Global Market Demand for HBCD in 2001 (AMAP, 2005).

Region	**HBCD**	**% total emission**
Americas	2800	16.77%
Europe	9500	56.89%
Asia	3900	23.35%
Rest of World	500	2.99%
Total	**16700**	

Table 9.10. Emissions to BETR-World Regions (kg/year).

	Region	**emission kg/year**	**% total emission**
1	NA - Continental Arctic	79	0.5%
2	NA - Canadian Provinces	513	3.27%
3	NA - Continental United States	2043	13%
4	NA - SA - Carribean	79	0.5%
5	SA - Centro	79	0.5%
6	SA - Arg. Chile	79	0.5%
7	EU - Europe	8942	56.89%
8	AS - Middle East	39	0.25%
9	AS - Russia	1313	8.35%
10	AS - Orient	2358	15%
11	OC - Oceania	38	0.24%
12	AF - Northern Africa	79	0.5%
13	AF - Southern Africa	79	0.5%
	Total Global	**15718**	

Table 9.11. Transport pathways of HBCD to the Arctic.

		UA-UA	LA-LA	FW-MW	MW-MW
1	NA - Continental Arctic	8.61%	0.88%	0.00%	0.03%
2	NA - Canadian Provinces	2.50%	0.78%	0.00%	1.99%
7	EU - Europe	17.89%	3.80%	0.06%	15.66%
9	AS - Russia	30.51%	6.83%	0.13%	0.90%
17	*North North Atlantic*	0.34%	0.16%	0.00%	8.93%
	Total	59.85%	12.45%	0.20%	27.50%

9.4.4 Supplementary Data for RAIDAR Modelling

For the application of RAIDAR in the present assessment there were some minor dietary preference changes to the default values. These changes were made so that the model predictions are more reflective of the regional environments from which the monitoring data were obtained. Feeding preferences for the avian piscivore (scavenger) were: 70% lower trophic level fish, 20% upper trophic level fish and 10% terrestrial invertebrates. Feeding preferences for the marine (aquatic) mammal were: 30% lower trophic level fish, 50% upper trophic level fish and 20% benthic invertebrates. Feeding preferences for the adult male human were changed from 4% upper trophic level fish to 2% upper trophic level fish and 2% lower trophic level fish. The remaining default human diet preferences were unchanged (Arnot and Mackay 2008).

9.5 Detailed Comments on HBCD Toxicity Data and Previous PBT Assessments

9.5.1 Water Solubility, Bioavailability and Cosolvents

There is some discussion in the European Commission (2008) draft report about water solubility issues in general as well as the differences in water solubility and perhaps other characteristics of the three main HBCD isomers (α-, β-, and γ-). As discussed in Section 2.3.2, FAVs for the water solubility of the α-, β-, and γ- isomers are 41, 15, and 2.5 $\mu g \cdot L^{-1}$, respectively, giving a water solubility estimate of 58 $\mu g \cdot L^{-1}$ for t-HBCD (the sum of all isomers). The γ- isomer makes up about 80% of t-HBCD. Thus, when the solubility limit for all components of the mixture is considered (i.e. 58 $\mu g \cdot L^{-1}$), there are undissolved precipitates of the β- and γ- isomers in the water column as they are substantially above their individual water solubility limits. Only by keeping the t-HBCD concentration near the water solubility limit for the γ- isomer, can it be assumed that all of the HBCD present is dissolved in the water. Thus, a water solubility of about 2 or 3 $\mu g \cdot L^{-1}$ represents a concentration at which all isomers of HBCD are truly dissolved and there are no precipates expected. Thus, there are unresolved and unquantified bioavailability issues for all testing data above the solubility of the γ-HBCD isomer. This issue has not been addressed in any of the previous reviews of the toxicity test data.

There are also a number of problems interpreting toxicity data for hydrophobic substances when cosolvents are used. Toxicity test design is based on a simple model

approach that allows the external exposure concentration to be used as a surrogate for the usually unknown amount of substance that enters the body of the organism, a portion of which ultimately reaches the site(s) of toxic action and initiates the adverse effect being examined. A key requirement for using such surrogate dose estimates in toxicity comparisons between various test results for various substances is that the toxicity modifying factors be the same in all cases (Mackay et al. 2001). Bioavailability of the test substance in the water column is a key toxicity modifying factor and any change in it, such as would be caused by the use of a cosolvent, changes the conditions and renders such test data not directly comparable with data obtained without cosolvent. If the nature and magnitude of any bioavailability change has been quantified, this would allow changes to be understood and an appropriate correction made. Unfortunately, such information does not appear to have been collected in any of the key HBCD studies.

Cosolvents affect the bioavailability of a substance in aqueous exposure media; however, to what degree is speculative without a greater quantitative understanding of the influence of the cosolvent in comparison to "pure" water phase exposures. The success of cosolvents in increasing effective bioavailablity is well established. In a variety of tests with hydrophobic chemicals no effects were reported unless the exposure to the substance is carried out with a cosolvent. This qualitative change is usually all that is reported, and contrary to guidance from the OECD (OECD 2000), key quantitative parameters are not reported. These oversights make it difficult to compare aqueous exposure measurements to other substances (relative toxicity) and against screening criteria that have not been developed to account for the influence of cosolvents on test data.

Bioavailability can also be changed by cosolvent in another way concurrent with the water solubility. Water solubility is determined in pure water, without dissolved or suspended organic material. Both toxicity and bioconcentration tests are carried out in water with living organisms which continuously release a variety of organic material to the water column. Food and feces also contribute to organic matter in the water column and certain exposure media used in algal and some invertebrate/bacterial testing contain other added substances that may be problematic. For very hydrophobic substances some portion of the total water column concentration will not be in solution because it partitions into these organic phases. Thus, the actual bioavailable fraction of chemical in the water is also a function of the organic matter in the water column. Substances in the dissolved/suspended organic phase are likely to be less bioavailable to normal respiratory uptake but may, in fact, be more bioavailable to some organisms in some cases via alternative uptake routes - e.g., diet. Depending on the amount and effectiveness of the cosolvent, this reduction in bioavailability may be counteracted or overwhelmed by increased "apparent water solubility" noted earlier. Both of these effects on bioavailability can occur at concentrations above and somewhat below the pure-water solubility limit. When water column concentrations of HBCD are above about 2 to 3 $\mu g \cdot L^{-1}$, the presence of undissolved precipitates of γ-HBCD (and β-HBCD isomer above about 15 $\mu g \cdot L^{-1}$), and possible interactions with solvents further complicate the problem.

In addition to the issue of the effect on the amount of substance taken up into the exposed organisms which affect amounts accumulated for either BCF or toxicity testing, it also

means that the total water column concentration is a confounded measure of the true exposure concentration experienced by the organisms. All in all, interpretation of aquatic testing results for very hydrophobic substances is very problematic and the simple process and general assumptions generally accepted for more water soluble substances should not be blindly adopted for candidate PBT substances. Since, essentially the same issues are applicable to evaluation of candidate POP substances, if the European PBT critieria or any screening criteria or evaluation schemes for Stockholm Convention and UN-ECE candidate POPs are to be employed in a scientifically-sound and fair decision-making process, a more thorough and detailed technical analysis and evaluation is needed. There is a substantial published literature on the effects of cosolvents produced by pharmaceutical researchers that should be brought to bear on this problem (e.g. (Jouyban 2008).

In summary, a variety of concerns regarding water solubility, use of co-solvent and bioavailablity render all available aquatic toxicity data for HBCD problematic since these issues confound accurate evaluation of the testing data. Concerns about the uncertainty that are introduced by these issues brings into question the reliability and validity for use of such compromised data in PBT, or for that matter POP, classification decisions.

9.5.2 Aquatic Toxicity Testing with Difficult Substances and Mixtures

Mixture toxicity remains a substantial challenge in toxicity, both in terms of a theoretical foundation and in practical application (McCarty and Borgert 2006b; McCarty and Borgert 2006a). In addition to the broad general challenge, there is another pertinent point about the use of cosolvents in toxicity testing with mixtures. The current OECD (2000) guidance document on testing of difficult substances and mixtures specifically advises against the use of cosolvents in cases such as encountered with HBCD (mixture of isomers).

"Water-miscible solvents provide a vehicle in which some poorly soluble substances can be dissolved to produce a stock solution which is more amenable to adding to, and mixing with, the test media. In particular, solvents could be helpful for hydrolytically unstable and highly viscous substances. However, because of the potential for interaction with the test substance resulting in an altered response in the test, their use should be restricted to situations where no other acceptable method of media preparation is available. If solvents are used, their effects on the test results, if any, need to be determined. It should be emphasized that solvents are not appropriate for mixtures where the use of the solvent can give preferential dissolution of one or more components and thereby affect the toxicity. This could also be true where the technical grade of a substance is tested and a toxic impurity dissolves preferentially." (OECD, 2000, p. 29)

Similarly, the use of generator systems such as generator columns is specifically contraindicated for toxicity testing of difficult substances and mixtures such as HBCD.

"Generator systems are not considered appropriate for substances containing components or impurities which differ in their water solubility. Differences in water solubility will

result in selective depletion of the more water soluble components from the column or disk matrix and their relative concentration in the water phase." (OECD, 2000, p. 23)

In summary, based on internationally recognized toxicity testing guidance it is clear that that most, if not all, of the aquatic toxicity data currently available for HBCD are unreliable due to lack of quantitative information about test-specific water solubility information, especially on the influence of cosolvents used with a difficult substance that is a mixture of isomers with different physical-chemical properties.

9.5.3 Critical Evaluation of PBT Assessments

Section 3.4.6 in the European Commission RAR (2008) entitled PBT-assessment outlines the screening process used in identifying substances with PBT characteristics and applies that policy to HBCD information. This PBT evaluation process is presented in detail in Section 4.4 Part II of the European Technical Guidance Manual (EC 2003). There are two general issues that must be raised with respect to the employed guidance: "inherent" properties and the use of NOEC data. First the issue of “inherent properties is discussed followed by the application of NOEC data without LOEC data.

Inherent Properties

There is an emphasis on screening information for PBT evaluation based on some relatively simplistic decision criteria. Although screening is a necessary first step, definitive categorization of a substance as a PBT requires a more thorough evaluation. However, the European Commission evaluative process indicates that screening may be sufficient to allow scientifically-sound PBT categorization appears to be based on a fundamental scientific error. There is repeated reference to "inherent" properties in the categories of persistence, bioaccumulation, and toxicity that appear to be the basis of enabling and justifying a relatively simple screening process as scientifically sound. The definition of the word "inherent" is instructive in this matter. There are various dictionary definitions with the following composite phrase providing a good perspective: an essential constituent or characteristic or a permanent and inseparable element in someone or something.

The property of toxicity is definitely not inherent to a substance. Toxicity requires an interaction - i.e., an exposure - with a living organism to occur, and then a given adverse effect still requires a sufficient degree of exposure (related to the amount accumulated and duration) for the effect to occur. Thus, there are many exposure scenarios for "inherently toxic" chemicals where no adverse effects will occur. Therefore, it is clear that the toxicity is not "inherent" to the substance. A more detailed review of this issue as it relates to PBT, derived from fundamental principles of chemistry, appears in Mackay *et al.* (Mackay et al. 2001). At the very least the term "inherent" is redundant as all substances are potentially toxic and the key determinant of exposure is clearly stated in a founding toxicological principle:

> "What is there that is not poison? All things are poison and nothing is without poison. Solely the dose determines that a thing is not a poison" - Paracelsus, 1564 (Deichmann et al. 1986)

The European Commission PBT assessment does not appear to fully consider the interaction between the "inherent" toxicity concept and the screening process. Where there are good toxicity data in terms of quality and scope, PBT screening may remove some substances from being categorized as PBTs as the toxicity test results indicate relatively low toxicity. However, that does not mean that substances that are screened in are necessarily PBTs.

Exposure-medium-based toxicity tests such as aquatic toxicity tests relate the adverse effects observed to a substance concentration in aqueous exposure media. That estimate is used as a surrogate dose metric for the usually unknown concentration in the body of the exposed organisms; specifically, the amounts at the usually unknown site(s) of toxic action. For organic chemicals the media-based dose surrogate is a product of two processes. One is the mode of toxic action of the substance, which determines the molar amounts of substance at the site(s) of toxic action in the organism that initiate the adverse effect in question. The other is the partitioning behavior of the substance, which determines bioaccumulation from the media in question.

To further illustrate this point, for a given toxicity endpoint an identical value can be obtained for a substance that combines low toxicity (i.e. baseline narcosis) and high hydrophobicity and a substance with higher toxicity (i.e. non-narcotic specific toxicity) and lower hydrophobicity. There is nothing "inherent" in an LC50 or chronic exposure-based toxicity estimate. The estimate is very explicitly a function of toxic potency and partitioning from the exposure medium. For this reason aquatic exposure toxicity metrics are not directly indicative of toxic potency, because of the inclusion of partitioning behavior, i.e., bioconcentration. Thus, some high K_{OW} chemicals can appear more toxic than lower K_{OW} chemicals because of higher bioaccumulation, rather than higher toxic potency. Knowledge of both the contribution of toxic mode of action/potency and the contribution of partitioning/bioaccumulation is imperative for a scientifically sound risk assessment. A number of publications have highlighted this issue and possible errors in interpreting toxicity data for hydrophobic chemicals in aquatic based exposure tests (e.g., (McCarty and Mackay 1993; Gobas et al. 2001; Maeder et al. 2004).

The use of screening trigger values for PBT assessments, or POPs assessments for that matter, do not consider partitioning because they use exposure criteria and thus many highly hydrophobic chemicals may be categorized as "T" when they are only baseline narcotic toxicants, the least toxic mode of action for organic chemicals. There appears to be little in the way of detailed consideration of the potency-partitioning issue in the European Commission (2008) PBT evaluation or risk assessment, even when it is clear that, due to the low water solubility of HBCD, this would provide valuable insights and a better understanding its toxic characteristics.

Using Lowest NOECs and NOECs without Associated LOECs

Although the European Commission (2008) report on HBCD provides a good overview of the available toxicity information, there appears to be little in the way of a thorough weight-of-evidence evaluation of the available data. In fact, the lowest toxicity values

from testing carried out with protocols judged acceptable are simply compared to the defined PBT toxicity trigger values. For aquatic data the PBT screening trigger values are:

"... a substance is considered to be potentially toxic when the L(E)C50 to aquatic organisms is less than 0.1 $mg \cdot L^{-1}$." while for the more definitive chronic endpoint the trigger is "... the long-term NOEC for marine or freshwater organisms is less than 0.01 $mg \cdot L^{-1}$."

The acute screening trigger of an LC50 or EC50 makes sense as these are a measure of some specific effect and the lowest effect level represents valuable information about real toxicity. The definitive chronic trigger defined as a NOEC level makes little sense as presented. NOECs are poor toxicity metrics with unreliable quantification since they do not refer to a known adverse dose-response relationship for an exposure level. In fact, they specifically refer to an exposure where no adverse effects have been found. NOECs may be found to be of some real use only when a corresponding known effect level is available. This problem is exacerbated by NOEC estimation methodology as, due to the generally low statistical power available in many toxicity tests, many NOECs can be considered to be largely statistical artifacts. The ultimate validity and utility of this metric has been widely questioned in the literature (OECD 1998; Crane and Newman 2000; Jager et al. 2006).

NOECs are commonly estimated as the exposure dose level employed in a test that is below the lowest exposure dose level where some adverse effects were reported. In such cases perspective on a NOEC estimate can be obtained by comparison to the known effect level. The difference between the NOEC and effect level can often be of the order of 10 or 100 times, but at least the NOEC can be put into perspective and an informed judgment made. A problem arises when the NOEC is the highest exposure level and there is no exposure level where adverse effects were reported. Here the NOEC is of little value for use in comparison with a trigger value as an actual effect level is not known. Where the range of available NOECs span the trigger value, risk assessors are placed in the difficult position of differentiating between the informational value of various NOECs, and making judgments based on that differentiation, when all of the NOECs indicate the same information: no toxic effects were observed.

The proposed use of NOECs in the European Commission RAR (2008) to identify candidate PBT substances is not considered to be scientifically defensible for a final PBT classification. Some additional direction to address NOEC issues, as discussed above, must be developed for final PBT and POP classification purposes. Additionally, the quality of the toxicity data must be more thoroughly evaluated than the simple observation that an acceptable testing protocol was employed. For example, results from aquatic toxicity tests for hydrophobic substances, such as HBCD, are difficult to interpret and to compare with substances that do not have low water solubility. Although an appropriate testing protocol may have been used, there is additional testing guidance (e.g., OECD, 2000) that must be followed before a specific test result should be judged as valid and acceptable.

9.5.4 Further Commentary on POP Assessments

The UN-ECE Protocal and UN Stockholm Convention do not contain clear quantitative endpoints for evaluating "T" as summarized in the following (UN-ECE 1998; UNEP 2001).

The screening criteria in Annex D of the Stockholm Convention for adverse effects are as follows:

(i) Evidence of adverse effects to human health or to the environment that justifies consideration of the chemical within the scope of this Convention; or

(ii) Toxicity or ecotoxicity data that indicate the potential for damage to human health or to the environment.

We interpret screening criterion (i) to imply some sort of risk assessment is required as evidence of adverse effects (i.e. comparisons of measured body burdens to relevant toxicity endpoints). Criterion (ii) is more vaguely expressed and hence it is unclear what sort of data or analysis is sufficient to "indicate the potential for damage". However, Annex D also contains the following statement:

"The proposing Party shall provide a statement of the reasons for concern
including, where possible, *a comparison of toxicity or ecotoxicity data with detected or predicted levels of a chemical resulting or anticipated from its long range environmental transport*, and a short statement indicating the need for global control."

With respect to the UN-ECE POPs protocol (UN-ECE 1998), the screening criterion for toxicity states the following:

1(b) Toxicity: potential to adversely affect human health and/or the environment

A more detailed assessment may occur following submission of the risk profile to the UN-ECE Executive Body, if, "further consideration of the substance is determined to be warranted", including the following evaluation:

2(b) Whether sufficient information exists to suggest that the substance is likely to have significant adverse human health and/or environmental effects as a result of its long range transboundary atmospheric transport

In summary, the present assessment considers assessing the potential for adverse effects in "remote regions" based on comparisons of exposure and effect levels (i.e. risk characterization / risk assessment) to be a more definitive assessment of a substance's POP potential in preference to comparisons of chemical properties against screening criteria.