Euro Chlor Risk Assessment for the Marine Environment

OSPARCOM Region - North Sea

Hexachlorobutadiene

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Euro Chlor
representing the chlor-alkali industry

EURO CHLOR RISK ASSESSMENT FOR THE MARINE ENVIRONMENT

HEXACHLOROBUTADIENE (HCBD)

OSPARCOM Region – North Sea

EXECUTIVE SUMMARY

Euro Chlor has voluntarily agreed to carry out risk assessments of 25 chemicals related to the chlorine industry. The risk assessments were targeted on the marine environment, specifically for the North Sea. The assessments are carried out according to the methodology laid down in the EU Risk Assessment Regulation (1488/94) and the Guidance Documents of the EU Existing Substances Regulation (793/93). The exercise consists of the collection and evaluation of data on effects on aquatic organisms and environmental fate. Basically, the adverse effect data are derived from laboratory toxicity tests and the exposure data from monitoring programs. Finally, the risk is indicated by comparing the "predicted environmental concentrations" (PEC) as indices of exposure with the "predicted no effect concentrations" (PNEC) as indices of effect. This PEC/PNEC ratio is considered as the risk quotient (RQ) for the marine environment. If RQ < 1 it is presumed that the likelihood of an adverse effect is very low. An RQ > 1 is a cause for concern, necessitating a further refinement of the risk assessment and eventually for reducing the risks.

In the case of hexachlorobutadiene (HCBD) which has a low water solubility (i.e. about 3.2 mg/l) and a relatively high log octanol/water partition coefficient (i.e. about 4.9) it was necessary, in addition to the aquatic phase, to consider the risks associated with the potential of HCBD to bioconcentrate in marine organisms, to partition to sediments and to produce toxicity in sediment dwelling organisms, and to produce toxicity in predators up the food chain, e.g. water to fish to fish-eating mammals (i.e. risk of secondary poisoning).

Therefore, to assess the risk posed by HCBD in the marine environment four approaches have been used.

1. Assessment of risk for the aquatic compartment

To assess the risk posed by HCBD to organisms living in the marine environment the Predicted No Effect Concentration, i.e. PNEC, derived from toxicology studies with representative aquatic organisms was compared with the Predicted Environmental Concentration of HCBD in marine surface waters, i.e. PEC_{water}.

A PNEC value of 130 ng/l was derived from the results of toxicological studies in organisms representing three different trophic levels, i.e. aquatic plants, invertebrates and fish.

The values of 5 and 12 ng/l for typical and worst case PEC_{water} are based on the monitoring data available for North Sea coastal and estuarine waters and for rivers which discharge to the North Sea, respectively.

The derived PEC_{water} values for the marine surface water are below the ambient water quality criteria of 450 ng/l recommended by the US EPA for the protection of human health from potential carcinogenic effects (US EPA, 1980). They are also below the acceptable concentration of 100 ng/l established by Environment Canada for the protection of aquatic organisms and wildlife (Environment Canada, 1983). HCBD has been deleted from the Canadian Environmental Contaminants Act List of Priority Chemicals because levels of

HCBD in the aquatic ecosystem were not high enough to merit further investigation.

Based on the available toxicological and monitoring data the PEC/PNEC ratios are lower than 1 both in typical (0.038) and worst case (0.092) approaches. These ratios indicate that the levels of HCBD in surface waters are unlikely to pose a risk to marine organisms living in the North Sea

2. Assessment of risk to fish species as evaluated by bioconcentration and monitoring data

To address the potential for HCBD to bioconcentrate in fish the bioconcentration factor (BCF) and the no effect concentration (NOEC) was used to calculate a critical body burden (CBB) which predicts the level of HCBD that may be present within tissues of the organism without causing a toxic effect.

$$CBB = NOEC \times BCF$$

For this calculation a BCF of 17,000 l/kg was used and a NOEC of 6.5 µg/l

$$CBB = 6.5\ (\mu g/l) \times 17{,}000\ l/kg = 111\ mg/kg\ wet\ weight.$$

To assess the risk of toxicity due to bioconcentration, the calculated CBB was compared with the concentrations of HCBD measured in marine fish collected at various locations around the UK which ranged from non-detectable to 0.4 µg/kg flesh. The comparison showed that the actual concentrations of HCBD in marine fish are well below the critical body burden associated with toxic effects indicating that risks to fish through bioconcentration are unlikely. This supports the above conclusion on low risks of HCBD to marine surface water organisms.

3. Assessment of risk to organisms living in sediment

A $PNEC_{sediment}$ was derived from the $PNEC_{aquatic}$ by applying the equilibrium partitioning method according to the TGD resulting in a $PNEC_{sediment}$ of 24.4 µg/kg dry weight.
Based on marine monitoring data the predicted environmental concentration of HCBD in sediment, i.e. $PEC_{sediment}$, was estimated from available monitoring data.

The majority of available sediment monitoring data on HCBD indicate levels less than 1 µg/kg, with a typical mean of 1.1 and a 90-percentile of 4 µg/kg dry weight, respectively. This means that PEC/PNEC ratios for typical and worst-case exposure are 0.045 and 0.16, respectively, indicating that unacceptable risks of HCBD to sediment organisms are unlikely.

4. Assessment of risk to fish-eating predators (biomagnification)

To assess the risk posed to predators eating fish contaminated with HCBD the Estimated Daily Intake of HCBD through eating fish, i.e. EDI_{fish}, was compared with the Predicted No Effect Level of HCBD for predatory species i.e. $PNEC_{oral/food}$.

Three values have been used to determine the NOAEL for HCBD:
A $PNEC_{oral/food}$ from a chronic toxicity in the rat: 0.2 mg/kg body wt/day
A $PNEC_{oral/food}$ from sub-chronic toxicity in Japanese quail: 3 mg/kg body wt/day
A $PNEC_{oral/food}$ for reproductive toxicity in the rat: 20 mg/kg body wt/day

The EDI_{fish} was calculated by multiplying the Predicted Environmental Concentration of HCBD in fish, i.e. $PEC_{fish,}$ with the feeding rate (FR) of the predators.

Based on biomonitoring data the PEC_{fish} was estimated to be approximately 0.4 µg HCBD/kg body weight. Combining this with the feeding rates of predatory species, i.e. 0.15 for the mink and 0.11 for the eagle, gives EDI_{fish} of 0.06 µg HCBD/kg body weight/day for the mink and 0.04 µg HCBD/kg body weight/day for the eagle. As the estimated daily intakes of HCBD are however several orders of magnitude below the no adverse effect levels there is little risk of toxicological consequences associated with predators eating fish contaminated with HCBD.
While the mink and ferret are considered to be more sensitive to reproductive toxicants than laboratory rodents the data show that even allowing for species sensitivity there is little risk of reproductive toxicity occurring in fish eating mammals.
As HCBD is metabolized and excreted rapidly the risk of bioaccumulation and secondary poisoning is low.

General Conclusion

The calculated PEC/PNEC ratios for HCBD for the various scenarios are summarized in the table below.

Summary table for PEC/PNEC ratios – Hexachlorobutadiene

Compartment	**PEC**	**PNEC**	**PEC/PNEC**
Aquatic			
Typical	5 ng/l	130 ng/l	0.038
worst case	12 ng/l	130 ng/l	0.092
Fish (CBB approach)	0-0.4 µg/kg	111 mg/kg	$0\text{-}3.6*10^{-6}$
Sediment			
Typical	1.1 µg/kg d.w.	24.4 µg/kg d.w.	0.045
worst case	4 µg/kg d.w.	24.4 µg/kg d.w.	0.16
	EDI	**NOAEL**	**EDI/PNEC**
Predators			
- Rodent (chronic toxicity)	0.06 µg/kg bw	200 µg/kg bw	0.0003
- Quail (sub chronic toxicity)	0.04 µg/kg bw	3,000 µg/kg bw	0.00001
- Rat (reproductive toxicity)	0.06 µg/kg bw	20,000 µg/kg bw	$0.3*10^{-6}$

In conclusion the calculated PEC/PNEC ratio for surface waters is less than 1 indicating that the levels of HCBD measured in marine surface waters are unlikely to represent a risk to the marine environment in the North Sea region. The assessment also indicated that toxicity to fish due to bioconcentration of HCBD (uptake from water) is unlikely. Similarly there is little risk of toxic effects occurring in fish eating mammals or birds. The lack of sediment toxicity data made it necessary to use the equilibrium partitioning method to estimate $PNEC_{sediment}$. The PEC/PNEC ratio for sediment for typical and worst-case exposures were below 1, indicating that risks to sediment organisms are unlikely.

Overall the data are supportive of the conclusion that the levels of HCBD in the marine environment do not pose an unacceptable risk and as environmental concentrations of HCBD continue to decline then so does any residual risk. This conclusion is supported by the decision of Environment Canada to delete HCBD from the Canadian Environmental Contaminants Act List of Priority Chemicals on the basis that levels of HCBD in the aquatic ecosystem were not high enough to merit further investigation.

1. INTRODUCTION: PRINCIPLES AND PURPOSES OF EURO CHLOR RISK ASSESSMENT

Within the EU a programme is being carried out to assess the environmental and human health risks for "existing chemicals", which also include chlorinated chemicals. In due course the most important chlorinated chemicals that are presently in the market will be dealt with in this formal programme. In this activity Euro Chlor members are cooperating with member state rapporteurs. These risk assessment activities include human health risks as well as a broad range of environmental scenarios.

Additionally Euro Chlor has voluntarily agreed to carry out limited risk assessments for 25 prioritized chemicals related to the chlorine industry. These compounds are on lists of concern of European Nations participating in the North Sea Conference. The purpose of this activity is to explore if chlorinated chemicals presently pose a risk to the marine environment especially for the North Sea situation. This will indicate the necessity for further refinement of the risk assessments and eventually for additional risk reduction programmes.

These risk assessments are carried out specifically for the marine environment according to principles laid down in the EU Risk Assessment Regulation (1488/94) and the Guidance Documents of the EU Existing Substances Regulation (793/93), (TGD, 1996). In addition the potential for HCBD to produce toxicity as a result of bioconcentration has been assessed using the methodology described by Nendza (1997) with the determination of the Critical Body Burden. Moreover, as HCBD has the potential to bioaccumulate the assessment includes an evaluation of the risk of secondary poisoning as a result of predators eating fish contaminated with HCBD.

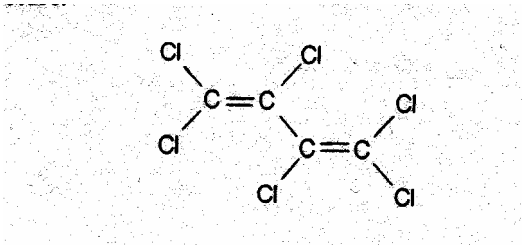
The exercise consists of the collection and evaluation of data on effects and environmental concentrations. Basically, the effect data are derived from laboratory toxicity tests and exposure data from analytical monitoring programmes. Where necessary, the exposure data are backed up with calculated concentrations based on emission models. Finally, the risk is indicated by comparing the "predicted environmental concentrations" (PEC) with the "predicted no effect concentrations" (PNEC) expressed as risk quotients (RQ) for the relevant compartments of the marine environment. This PEC/PNEC ratio is considered as the risk quotient (RQ) for the marine environment. If $RQ < 1$ it is presumed that the likelihood of an adverse effect is very low. An $RQ > 1$ is a cause for concern, necessitating a further refinement of the risk assessment and eventually for reducing the risks.

2. DATA SOURCES

The data used in this risk assessment are primarily derived from the published literature, from country specific chemical monitoring programs (for exposure data), the IUCLID Data Sheet And the IPCS document on HCBD (WHO IPCS, 1994).

3. COMPOUND IDENTIFICATION

Description	CAS number:	87-68-3
	EINECS No.:	201-765-5
	IUPAC Name:	hexachlorobutadiene (HCBD)
	Appearance:	clear colourless liquid
	Molecular Formula:	C_4Cl_6
	Formula weight:	261
	Structural Formula:	



EU Labelling

According to Annex 1 of Directive 93/72/EEC hexachlorobutadiene is classified: as harmful in contact with skin or if swallowed (R21/22), irritating to eyes and respiratory system (R36/37), possible risk of irreversible effects (R40), may cause sensitisation by skin contact (R43), very toxic to aquatic organisms. May cause long-term adverse effects in the aquatic environment (R 50/53).

4. PHYSICO-CHEMICAL PROPERTIES

Table 1 gives the major chemical and physical properties of hexachlorobutadiene which were adopted for the purpose of this risk assessment.

Table 1: Physical and chemical properties of HCBD

Property	Value
Molecular weight	260.8 (g/mol)
Vapour pressure	20 Pa at 20°C
Log-octanol-water partition coefficient (log Kow)	4.78 to 4.9
Log Koc	3.95-4.05
Water solubility	3.2 mg/l at 20°C
Henry Constant	1630 $Pa.m^3$/mol at 25°C

(Data from WHO, IPCS Environmental Health Criteria Document on HCBD, 1994)

5. COMPARTMENT OF CONCERN BY MACKAY LEVEL I MODEL

The risk assessment presented here focuses on the marine environment, with special attention for the North Sea conditions where appropriate. Although this risk assessment focuses on the aquatic environment, it should be borne in mind that all environmental compartments are inter-related.

An indication of the partitioning tendency of a compound can be defined using a

Mackay level I calculation obtained through the ENVCLASS software distributed by the "Nordic Council of Ministers". This model describes the ultimate distribution of the compound in the environment (Mackay & Patterson, 1990, Pedersen *et al.*, 1994).

Hexachlorobutadiene has quite a high vapour pressure which consequently results in a Mackay level I calculation indicating that it will partition mainly to air (98%), despite its $\log_{Kow}$ which is also relatively high..

The data used for calculation are shown in *Appendix 1* and the results of the calculation for HCBD are given in Table 2.

Table 2 : Partition of hexachlorobutadiene into different environmental compartments according to Mackay level I calculation
(Mackay & Patterson, 1990)

Compartment	**%**
Air	97.8
Water	0.2
Soil	1.0
Sediment	1.0

6. PRODUCTION, USES AND EMISSIONS

6.1. Production and uses

Historically HCBD was used as a solvent for rubber and other polymers, heat transfer fluids, transformer liquid, hydraulic fluid and washing liquor for removing hydrocarbons (WHO IPCS, 1994). It has also been used in agriculture as a seed dressing and fungicide for a variety of crops and has found applications in a number of manufacturing processes such as production of aluminium and graphite rods. Due to concerns about persistence, potential to bioaccumulate and toxicological properties the use of HCBD in such applications has now virtually ceased, although it is possible that HCBD may still be in use in some parts of the world.

6.2. Emissions

While the commercial production of HCBD use has been virtually eliminated it is still generated inadvertently as a by-product of tetrachlorethene and tetrachloromethane production. With improved manufacturing processes HCBD is no longer detectable in either of these products.

The main routes by which HCBD enters the environment during processing are the atmosphere and hydrosphere. Emissions in Europe in 1997 represented 2 kg/y in air and 100 kg/y in water, based on a survey of about 76 sites from the European chlorine industry. This represents a reduction of 98% and 97% respectively, since 1985 (Euro Chlor, 2001).

6.3. Applicable Regulations

In the EU, hexachlorobutadiene emissions to water are governed by EC Directive 76/464 on pollution caused by certain substances and by Council Directive 88/347/EEC setting limits to environmental releases of certain hazardous chemicals, including HCBD. HCBD emissions from perchloroethylene plus carbon tetrachloride production are limited to 1.5 g/t total production capacity (as from January 1990) and for trichloroethylene and perchloroethylene production the Water Quality Objective of 0.1 µg/l must be respected. The European Council of Vinyl Manufacturers (ECVM) set a voluntary limit value of 10 µg/l in waste water discharge from EDC/VCM/PVC production plants to be committed before end 2003 (ECVM, 1998).
A number of regulatory standards for acceptable levels of HCBD in water have been established by different countries and authorities. For example, the Surface Water Quality Objective defined by the EU Directive 88/347 for HCBD is 0.1 µg/l. A similar value is proposed by CSTEE (1994). The WHO drinking water guideline is 0.6 µg/l based on an evaluation of animal carcinogenicity data.

7. EFFECTS ASSESSMENT

In order to assess the risk posed by HCBD in the marine environment it is necessary to consider 4 scenarios:

1. Assessment of risk for aquatic organisms
2. Assessment of risk to fish species by bioconcentration
3. Assessment of risk to organisms living in sediments
4. Assessment of risk to fish-eating predators (biomagnification).

To assess the risk HCBD poses to the marine environment, it is necessary to examine the available toxicological information and to determine a Predicted No Effect Concentration for organisms living in the marine aquatic environment (i.e. $PNEC_{marine}$), a Predicted No Effect Concentration for organisms living in sediment (i.e. $PNEC_{sediment}$) and a Predicted No Effect Concentration for species eating fish contaminated with HCBD (i.e. $PNEC_{oral/food}$).

7.1. Aquatic Toxicity

As a first approach, this chapter only considers the following three trophic levels: aquatic plants, invertebrates and fish.

The evaluation of the data was conducted according to the quality criteria recommended by the European authorities (Commission Regulation 1488/94/EEC). The evaluation criteria are given in *Appendix 1*.

A summary of all data is given in *Appendix 3*.
In total 25 data for fish, 7 data for invertebrates and 2 data for algae were found. Of these data 5, 0 and 0 respectively were considered valid for risk assessment purposes. For the respective taxonomic groups 7, 3 and 0 should be considered with care, and 13, 4 and 2 data respectively, were judged as not valid for risk assessment or could not be assigned due to lack of information.

It is necessary to distinguish the acute studies (LC_{50}/EC_{50}) from chronic studies (NOEC/LOEC). In the tables presented in *Appendix 3* the data are ranked based on class (fishes, invertebrates, algae), criterion (LC_{50}/EC_{50}, NOEC/LOEC), environment (freshwater, saltwater) and validity (1-4).

The different trophic levels are reviewed hereafter.

7.1.1. Marine fish

Four acute toxicity studies are reported for 3 marine fish species.

Two were conducted under static conditions without analysis (US EPA, 1980) and are considered non-valid as no precautions to avoid volatile losses were reported. The source was secondary, citing unpublished US EPA data.

One study with *Cyprinodon variegatus* under static conditions showed a 96h LC_{50} of 3.6 mg/l. In this study methanol was used as solvent resulting in concentrations at or above the maximum water solubility of 3.2 mg/l.
Only the result of 1 study with *Limanda limanda* was expressed as measured concentrations in a flow-through system designed to test compounds of high volatility. Although this was not completed in accordance with current guidelines, the result is considered valid and gives a 96h LC_{50} of 0.45 mg/l which is the lowest toxicity value for marine fish (Pearson & McConnell, 1975).

No long-term studies are available.

7.1.2. Freshwater fish

Fifteen acute toxicity studies are reported for 11 freshwater fish species. Three flow-through studies based on measured concentrations were considered valid without restriction; two of these, on *Pimephales promelas* (Walbridge *et al*., 1983 and Geiger *et al*., 1985), gave similar LC_{50} values of 0.1 and 0.09 mg/l, respectively. A study with *Brachydanio rerio* gave an LC_{50} of 0.24 mg/l (Roederer *et al*., 1989)

Of the studies classified as validity category 2 (to be used with care), a semi-static study with *Carassius auratus* (Leeuwangh *et al*., 1975) employed only partially closed vessels with limited analysis. However, this gave an LC_{50} of 0.09 mg/l, based on measured concentrations, equal to the lowest value with *P. promelas*. The remaining category 2 studies, which gave more limited information on analysis of the solutions or were based on nominal concentrations, produced higher LC_{50} values.

The seven studies which were considered not valid for this risk assessment, due to lack of analysis or insufficient detail on measures to prevent volatile losses, gave higher LC_{50} values.

Of the studies considered valid, the 96 hour LC_{50} to *P. promelas* (and *C. auratus*) of 0.09 mg/l is the lowest acute toxicity value for freshwater fish (Geiger *et al*., 1985 and Leeuwangh *et al*., 1975).

Five long-term studies (and one short-term sublethal study) are reported. The lowest NOEC reported (0.003 mg/l, Leeuwangh *et al*., 1975) was considered not valid for risk

assessment because the non-standard, biochemical endpoint (liver enzyme activity of *C. auratus*) represents a response to the substance but not necessarily an adverse effect. The NOEC for growth from the same study was similar to, but higher than, the NOEC from an early lifestage test with *P. promelas* (Benoit *et al*., 1982) which was considered valid without restriction and used a flow-through system with analysis of the test solutions.

The 28d NOEC for hatching and survival of *P. promelas* was 0.0065 mg/l, based on measured concentrations (Benoit *et al.*, 1982). This is the lowest NOEC value for freshwater fish.

7.1.3. Marine invertebrates

Three acute toxicity studies are reported for 3 marine invertebrates species. One of them was conducted under static conditions in closed vessels with analysis of the test compound and is considered valid but should be used with care. The remaining studies (US EPA, 1980) do not give detailed information on the test duration or conditions (and cite unpublished US EPA data), but were based upon nominal concentrations in static tests. They are therefore considered not valid.

The lowest valid acute toxicity value for a marine invertebrate is for *Elminius modestus* with a 48h LC_{50} of 0.87 mg l-1 (Pearson & McConnell, 1975).

No long-term toxicity study is reported for marine invertebrates.

7.1.4. Freshwater invertebrates

Four acute toxicity values are reported for freshwater invertebrates. Two were considered not valid, being based on nominal concentrations in static tests (Knie *et al.*, 1983 and Slooff *et al*., 1983); in addition, the study with *Dreissena polymorpha* (Slooff *et al*., 1983) used a non-standard endpoint (shell closure response threshold). Two studies reported together (Leeuwangh *et al*., 1975) were considered valid with restrictions, to be used with care. Although the vessels appear to have been only partially closed, with glass lids, the tests were semi-static and the test solutions were analysed.

The lowest acute toxicity for freshwater invertebrates is a 96h LC_{50} to *Asellus aquaticus* of 0.13 mg/l (Leeuwangh *et al*., 1975).

No long-term toxicity study is reported for freshwater invertebrates.

7.1.5. Marine algae

No toxicity studies are reported for marine algae.

7.1.6. Freshwater algae

Two studies are reported with freshwater algae. Both are considered non-valid. A study with *Haematococcus pluralis* was of short duration (4 hours) and was a static test using nominal concentrations (Knie *et al*., 1983). A study with *Scenedesmus quadricauda* (Bringmann & Kuehn, 1977) employed closed vessels but with a significant headspace, and the endpoint (toxicity threshold) was non-standard. However, this endpoint is approximately equivalent to a NOEC and the result (>25 mg/l) is probably sufficient to indicate that algae are not the most sensitive

trophic group for this compound.

7.1.7. PNEC for marine environment

There is insufficient data to reliably compare the sensitivity of marine and freshwater organisms to hexachlorobutadiene. However, from an evaluation of the available toxicity data for other chlorinated aliphatic compounds (e.g. Calow, 1998), it is reasonable to conclude that the sensitivity of marine and freshwater organisms is quite similar.

A summary of the valid data selected for the derivation of PNEC values at different levels is given in Table 3. This table summarises the PNEC values derived from acute, chronic and ecosystem studies. When these studies are available, it is generally acknowledged that the latter are closer to real world than the former. As far as the North Sea is concerned, acute exposure is not relevant because of the absence of local sources.

The final PNEC for the risk assessment of hexachlorobutadiene is 0.13 µg/l.

This value is close to the proposed water quality objective of 0.1 µg/l proposed by CSTEE (1994) and WHO-IPCS (1994).

Table 3: Summary of ecotoxicity data selected for the PNEC derivation of HCBD, with the appropriate assessment factors

Available valid data	Assigned assessment factor	Lowest toxicity values
Short-term LC50 from two trophic levels (fish, invertebrates)	1000	- *L. limanda*, LC50, 96h = 0.45 mg/l, (Pearson & McConnell, 1975) - *P. promelas*, LC50, 96h = 0.09 mg/l, (Geiger *et al*., 1985) - *E. modestus*, LC50, 96h = 0.87 mg/l, (Pearson & McConnell, 1975) - *A. aquaticus*, LC50, 96h = 0.13 mg/l, (Leeuwangh *et al*., 1975).
	PNEC = 0.09 µg/l	
Long-term NOEC from 1 species representing one trophic level (fish) Algae less sensitive	50	- *P. promelas*, NOEC, 28d = 0.0065 mg/l, (Benoit *et al.*, 1982) - Algae > 2 mg/l (Knie *et al*., 1983)
	PNEC = 0.13 µg/l	

7.2. Toxicity in sediment

Toxicity studies on HCBD for sediment organisms are not available. Therefore the $PNEC_{sediment}$ was derived from the $PNEC_{aquatic}$ by applying the equilibrium partitioning method according to the TGD (1996). The details of this calculation are given in

Appendix 6.

The $PNEC_{sediment}$ derived in this way is 24.4 mg/kg dry weight.

7.3 Secondary Poisoning – Effect Assessment

As food can be a significant source of exposure for a substance such as HCBD with a low water solubility and high lipid solubility, this risk assessment also addresses whether or not HCBD present in the marine environment contributes to adverse effects in predatory animals higher up the food chain which feed on marine fish.
To estimate the risk posed by HCBD via uptake through the food chain it is necessary to have information on the $PNEC_{oral/food}$; which represents the level of HCBD present in food (in this case fish) which can be consumed by predatory species higher in the food chain without producing adverse effects.

7.3.1. Estimation of PNEC for birds

The WHO IPCS (1994) reports that the only reliable test with birds is a 90-day study with Japanese quail receiving HCBD in the diet. The study indicated that chick survival was decreased at 10 mg/kg diet although egg production, percentage of fertile eggs and percentage of hatchable eggs was unaffected by this treatment. No effects were seen at 3 mg/kg body weight which, for the purpose of this risk assessment, is considered the no observed adverse effect level NOAEL for birds.

7.3.2. Estimation of PNEC for mammals

The main target organs for toxicity of HCBD are the liver and the kidneys. On the basis of short and chronic (2 year) toxicity studies in rats and mice the WHO (WHO IPCS, 1994) concluded that the no observed adverse effect level (NOAEL) is 0.2 mg/kg body weight per day.
Studies to investigate reproductive effects in experimental rats have reported reduced birth weight and neonatal weight gain but only at doses producing maternal toxicity, i.e. 20 mg/kg body weight. No data are available on other mammalian species such as the mink or ferret, which are reported to be more sensitive to reproductive toxicants than the rat or mouse.
For the purpose of this risk assessment 0.2 mg/kg is considered the NOAEL for chronic effects and 20 mg/kg is considered the NOAEL for reproductive toxicity.

7.4. Persistence

In air, HCBD persists until it is either degraded photochemically or adsorbed to particulate matter and deposited to water or soil. Estimates of its half-life in air based on photochemical degradation through reactions with hydroxyl radicals and ozone range from 60 days (ATSDR, 1994) to three years (Howard *et al*., 1991).

Class & Ballschmiter (1987) calculated that HCBD would have a tropospheric half-life of 840 days in the northern hemisphere and 290 days in the southern hemisphere, based on a hydroxyl radical rate constant of 2×10^{-14} cm^3/molecule per second and a hydroxyl radical concentration of 7×10^5 molecules/cm^3 in the north and 17 x 105 molecules/cm^3 in the south.

Tabak et al. (1981) found that HCBD was completely removed within seven days of exposure under aerobic conditions. Approximately 70% adsorption to sludge and 10% degradation was found to occur within 8 days in a pilot low-loaded biological sewage treatment plant (Schröder, 1987). Degradation of HCBD is very slow under anaerobic conditions (Johnson & Young, 1983; Govind *et al.*, 1991; Howard *et al.*, 1991). The half-life of HCBD in water is proportional to the amount of organic matter in the aqueous media; in natural water, the half-life is estimated to be 4-52 weeks (Howard *et al.*, 1991).

7.5. Bioaccumulation

A number of investigators have examined the ability of HCBD to bioconcentrate in fish. For example Oliver and Niimi (1983) reported bioconcentration factors of 5,800 and 17,000 in rainbow trout (*Salmo gairdneri*) maintained for over a 100 days in aqueous solutions containing HCBD at concentrations of 0.1 ng/l or 3.4 ng/l, respectively, steady state having been achieved after 69 days at the low exposure and 7 days at the high exposure. Laseter *et al.* (1976) however, reported that the accumulation of HCBD in mollies (*Poecilia latipinna*) and the largemouth bass (*Micropterus salmoides*), held for 10 days in water containing concentrations of HCBD (10 to 59 µg/l), although variable was fairly low with concentrations normally below 50. A study by Pearson and McConnell (1975) to measure accumulation of HCBD in plaice (*Pleuronectes platessa*) and dabs (*L. limanda*) maintained for up to 3 months in water containing HCBD (1.7 µg/l) reported concentration factors of about 500 to 700 for flesh and 7,000 to 10,000 for the liver. Overall the data do indicate a potential for bioaccumulation, although the results appear to be dependent on dose, length of exposure and species tested.

Two of the publications, i.e. Laseter *et al*. (1976) and Pearson and McConnell (1975), looked at biomagnification by feeding fish on food contaminated with HCBD. Laseter *et al.* (1976) fed largemouth bass (*M. salmoides*) mollies (*P. latipinna*) containing approximately 0.02 µg/g of HCBD ad libitum for seven days. In the study conducted by. Laseter *et al.* (1976) no clear evidence of bioconcentration was seen, however the results were variable and inconclusive. In the investigation carried out by Pearson and McConnel (1975) plaice (*P. platessa*) were fed minced mussels contaminated with HCBD (about 0.002 µg/g) for 88 days. No evidence of bioaccumulation was seen.

The accumulation of HCBD by crayfish (*Procambarus clarki*) was investigated by Laseter *et al.* (1976) using both laboratory and field studies. In the laboratory investigations, 10 days exposure at 2 to 4 µg/l the concentration factors were relatively low with males having a mean concentration factor of about 12 and the females about 59. In the field studies crayfish were maintained for 17 days in pond water containing 4.6 µg/l HCBD. Concentration factors varied between 7 to 300, with females again accumulating more than the males. In a recovery study crayfish maintained in non contaminated water for 12 days were found to have lost about 95% of the HCBD from their tissues.

A study by Oliver (1987) measured HCBD uptake by oligochaete worms maintained in sediments spiked with HCBD; pore water concentration 0.032 µg/l. The results indicated that steady state was reached within 4 to 11 days of exposure with an average bioconcentration factor of 29,000 based on dry weight of which about 8% is lipid.

A 7-day laboratory experiment with the green alga (*Oedogonium cardiacum*) reported that the algae accumulated HCBD to a concentration approximately 160-fold greater than that in the surrounding water (Laseter *et al.*, 1976).
Studies in mammalian species have shown that when rats received oral doses of HCBD as part of a mixture of seven different chlorinated hydrocarbons, there was no evidence of accumulation in the selected organs examined (Jacobs *et al.*, 1974). No other data have been found concerning the accumulation of HCBD in mammalian tissues.

In addition to bioconcentration of HCBD from the surrounding environment a number of authors have examined data to determine if HCBD might biomagnify through the food chain. For example Goldbach *et al.* (1976) examined levels of HCBD in fish of prey and found that concentrations in fish such as pike and perch were in fact lower than the prey fish. The authors also found no correlation between age and HCBD residues. Based on these findings they concluded there is no significant biomagnification to higher trophic levels. This conclusion is supported by Pearson and McConnell (1975) who concluded that there was little evidence for biomagnification of HCBD up the food chain. Similarly Laseter *et al.*, (1976) concluded that HCBD was not concentrated to a great extent and accumulated irregularly.

8. EXPOSURE ASSESSMENT

The exposure assessment is essentially based on exposure data from analytical monitoring programmes which have measured HCBD in a number of water systems including the marine environment.

8.1. Concentration of HCBD measured in water

A study of HCBD in 108 samples of sea water collected in 1983 and 1984 from the Dutch coast of the North Sea reported an average HCBD concentration of 0.28 ng/l (Van de Meent, 1986). A survey of Liverpool Bay carried out by Pearson and McConnell (1975) reported average concentrations of 4 ng/l with maximum levels of 30 ng/l. Studies of HCBD concentrations in German rivers in 1984 and 1985 reported that surface waters of the River Rhine and Elbe contained 10 to 20 and 10 to 150 ng/l (IUCLID, 1994) while Goldbach *et al.* (1976) reported that levels of HCBD near the mouth of the river IJssel in The Netherlands were about 130 ng/l.

The statistical analysis of the monitoring data of the EU-COMMPS database, which contains more than 10,000 measured HCBD concentrations from rivers of six European countries (B,D,E,GR,UK,NL) over the period 1994-1997, shows a mean value and a 90-percentile of the concentration distribution at 6 and 12 ng/l respectively. The distribution of the measured concentrations is illustrated in Fig. 1 hereafter. It is to be pointed out that only 13% of the measured values are above detection limit.

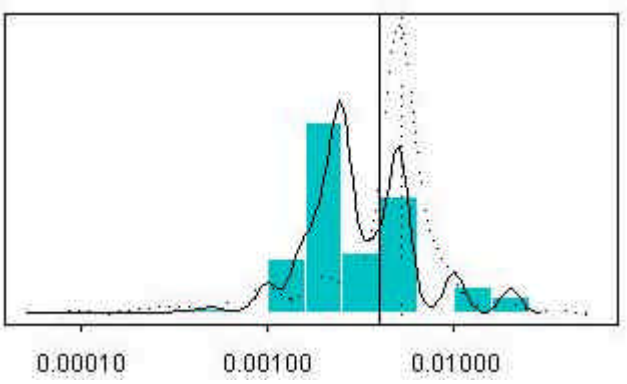


Fig.1: Distribution of HCBD concentrations in European surface waters in µg/l.

As illustrated in *Appendix 4*, between 1993 and 1996 concentrations in estuaries as reported in recent studies (WRc, 1998 and EU COMMPS database, 1998) vary from 0.4 to 90 ng/l, but typical values are in the range of 1 to 5 ng/l.

For the purpose of this marine risk assessment, a typical and a worst case PEC_{marine} of 5 ng/l and 12 ng/l are used in the calculations.

8.2. Concentrations of HCBD measured in sediments

There have been a number of studies measuring HCBD in sediments. For example samples collected around Hamburg contained <0.1 to 1.8 µg HCBD/kg dry weight of sediment while a study conducted 1980 to 1981 reported levels of 2 to 5 µg HCBD/kg dry weight in sediment collected from the Rhine (IUCLID, 1994). Pearson and McConell (1975) examined the concentrations of HCBD in marine sediments and while a few samples indicated concentrations above 1 µg/kg the majority were below 0.5 µg/kg.

Levels of HCBD in main estuaries and river sediments in Europe have been reported in the EU-COMMPS database over the period 1994-1997. A statistical analysis of about 500 measured concentrations concludes that the mean and the 90-percentile values of HCBD concentrations in sediments in Europe are 1.1 and 4 µg/kg respectively. The distribution of concentrations is illustrated in Fig.2 hereafter. It is to be pointed out that more than 50% of the measured values are under the detection limit.

Recent measurements from EU COMMPS database, reported in Appendix 4, indicate HCBD concentrations in estuarine or coastal sediment varying between < 0.2 and 3 µg/kg, typical values being close to 1µg/kg. (EU COMMPS, 1998).

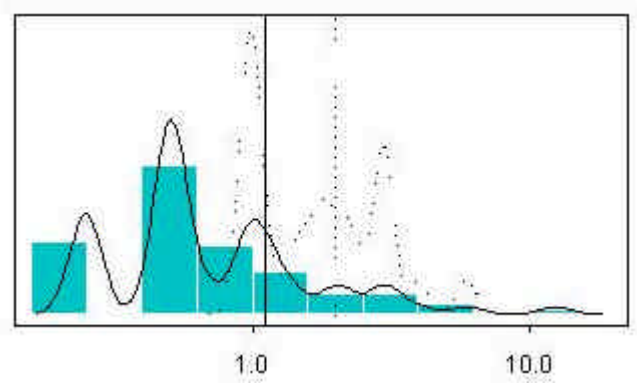


Fig.2: Distribution of HCBD concentrations in sediments of European rivers, in µg/kg

8.2.1. Calculation of predicted environmental concentration of HCBD in sediment (i.e. $PEC_{sediment}$)

Based on these data, typical and worst case $PEC_{sediment}$ values of 1 and 4 µg/kg dry weight are used in the calculations to assess the risk.

8.3. Concentrations of HCBD measured in marine fish

The only information on marine fish is reported by Pearson and McConnell (1975) who analysed fish, collected in the Liverpool Bay and Thames Estuary areas, for tissue concentrations of HCBD. Of the 15 samples of fish which were analysed, HCBD was not detected (limit of detection 0.001 ng/kg) in 10 samples and of the remaining 5 samples the highest level detected in the flesh was 0.4 µg/kg.

8.3.1. Calculation of predicted environmental concentration of HCBD in fish (i.e. PEC_{fish})

For the purpose of this marine risk assessment an upper average PEC_{fish} of 0.4 µg/kg has been used.

9. RISK ASSESSMENT

The marine risk assessment on HCBD described in this report is largely based on the methodology laid down in the EU Risk Assessment Regulation (1488/94) and the Guidance Documents of the EU Existing Substances Regulation (793/93). Basically the assessment consists of comparing toxicological data, derived mainly from laboratory toxicity tests, with exposure data from analytical monitoring programs. If the "predicted environmental concentrations" (PEC) calculated from the exposure data is less than the "predicted no effect concentrations" (PNEC), derived from the toxicological data, then the prediction is the risks are low. If the PEC exceeds the PNEC, then further refinement of the risk assessment may be necessary or eventually risk reduction may be necessary.

As HCBD has a fairly low water solubility (about 3.2 mg/l) and a relatively high log octanol/water partition coefficient (about 4.9) consideration was also given to potential

risks of HCBD to:

- bioconcentrate in marine organisms
- partition into sediments and produce toxicity in sediment dwelling organisms
- produce toxicity in predators up the food chain, e.g. water to fish to fish-eating mammal, (i.e. risk of secondary poisoning).

To assess the risk posed by HCBD in the marine environment 4 approaches have been used:

9.1 Assessment of risk to the aquatic compartment

To assess the risk posed by HCBD to organisms living in the marine environment the Predicted No Effect Concentration, i.e. PNEC, derived from toxicology studies with representative aquatic organisms was compared with the Predicted Environmental Concentration of HCBD in marine surface waters, i.e. PEC_{water}.
A PNEC value of 130 ng/l was derived from the results of toxicological studies in organisms representing three different trophic levels, i.e. aquatic plants, invertebrates and fish.

The values of 5 and 12 ng/l for typical and worst case PEC_{water} are based on the monitoring data available for North Sea coastal and estuarine waters and for rivers which discharge to the North Sea, respectively.
The derived PEC_{water} values for the marine surface water are below the ambient water quality criteria of 450 ng/l recommended by the US EPA for the protection of human health from potential carcinogenic effects (US EPA, 1980). They are also below the acceptable concentration of 100 ng/l established by Environment Canada for the protection of aquatic organisms and wildlife (Environment Canada, 1983). HCBD has been deleted from the Canadian Environmental Contaminants Act List of Priority Chemicals because levels of HCBD in the aquatic ecosystem were not high enough to merit further investigation.

Based on the available toxicological and monitoring data the PEC/PNEC ratios are lower than 1 both in typical (0.038) and worst case (0.092) approaches. These ratios indicate that the levels of HCBD in surface waters are unlikely to pose a risk to organisms living in the North Sea.

9.2 Assessment of risk to fish species as evaluated by bioconcentration and monitoring data

To address the potential for HCBD to bioconcentrate in fish the bioconcentration factor (BCF) and the no effect concentration (NOEC) was used to calculate a critical body burden (CBB) which predicts the level of HCBD that may be present within tissues of the organism without causing a toxic effect.

$$CBB = NOEC \times BCF$$

For this calculation a BCF of 17,000 l/kg was used and a NOEC of 6.5 µg/l

CBB = 6.5 (µg/l) x 17,000 l/kg = 111 mg/kg wet weight.

To assess the risk of toxicity due to bioconcentration, the calculated CBB was compared with the concentrations of HCBD measured in marine fish collected at various locations around the UK which ranged from non-detectable to 0.4 µg/kg flesh. The comparison showed that the actual concentrations of HCBD in marine fish are well below the critical body burden associated with toxic effects indicating that risks to fish through bioconcentration are unlikely.

9.3 Assessment of risk posed by potential to partition to sediments

An estimation of a $PNEC_{sediment}$ led to the level of 24.4 µg/kg dry weight. The majority of available sediment monitoring data on HCBD indicate levels less than 1 µg/kg, with a typical mean of 1.1 and a 90-percentile of 4 µg/kg dry weight, respectively. This means that PEC/PNEC ratios for typical and worst-case exposure are 0.045 and 0.16, respectively, indicating that unacceptable risks of HCBD to sediment organisms are unlikely.

9.4 Assessment of risk to fish-eating predators (biomagnification)

Although there is little evidence for biomagnification of HCBD in the food chain, it is important to assess the risk posed to predators eating fish contaminated with HCBD. For this purpose, the Estimated Daily Intake of HCBD associated with fish-eating, i.e. EDI_{fish}, was compared with the Predicted No Effect Level of HCBD ($PNEC_{oral/food}$) for predatory species.

Three $PNEC_{oral/food}$ values have been used in the risk assessment:

1) A $PNEC_{oral/food}$ from a chronic toxicity in the rat: 0.2 mg/kg body wt/day
2) A $PNEC_{oral/food}$ from sub-chronic toxicity in Japanese quail: 3 mg/kg body wt/day
3) A $PNEC_{oral/food}$ for reproductive toxicity in the rat: 20 mg/kg body wt/day.

The EDI_{fish} was calculated by multiplying the Predicted Environmental Concentration of HCBD in fish, i.e. PEC_{fish} with the feeding rate (FR) of the predators:
$EDI = PEC_{fish} \times FR$.

Based on biomonitoring data the $PEC_{(fish)}$ was estimated to be approximately 0.4 µg HCBD/kg body weight while the feeding rates for predatory species such as the mink and eagle have been estimated at 0.15 and 0.11 kg/kg body weight for the mink and eagle respectively (US EPA, 1992). Using these values a mink eating fish contaminated with HCBD will ingest 0.06 µg HCB/kg body weight/day while the eagle will be exposed to 0.04 µg HCBD/kg body weight/day. The assessment shows that the estimated daily intakes of HCBD *via* eating contaminated fish are several orders of magnitude below the $PNEC_{oral/food}$ for chronic toxicity in the rat, the $PNEC_{oral/food}$ for subchronic toxicity in the quail and the NOAEL for reproductive toxicity in the rat. These results thus indicate there is little risk of toxicological effects for predatory species eating fish contaminated with HCBD.

For further refinement of the risk assessment it is also possible to compare the sensitivities of laboratory species with the predatory species of concern. Regarding predatory birds the quail is apparently more sensitive to chemical toxicants than environmentally relevant species such as gulls or eagles (Cowan *et al.*, 1995) thus the NOAEL from the quail toxicity study can be extrapolated directly to predatory bird species. In the case of reproductive toxicology environmentally relevant predatory species such as the mink and otter are reported to be more sensitive to reproductive toxicants than commonly used laboratory species such as the rat (Aulerich and Ringer, 1977). As there are no reproductive toxicological data in the mink or otter it is necessary to use the rat information which shows an approximately 50,000-fold difference between the estimated daily intake of HCBD associated with eating contaminated fish and the NOAEL for reproductive toxicology. With such a large margin of safety even allowing for differences in species sensitivity the data indicate that risk of reproductive toxic effects in predatory species such as the mink and otter are unlikely.

The risk assessment for HCBD would not be complete without an examination of the potential for secondary poisoning in species such as fish eating birds or mammals at the top of the food chain. Investigations examining the potential bioaccumulation of HCBD have concluded that there is no evidence of bioaccumulation via the food chain (Goldbach *et al*., 1976, Pearson and McConnell, 1975). This lack of bioaccumulation is associated with the short half life of HCBD in fish (Goldbach *et al.*, 1976) and the rapid metabolism and excretion in mammals and birds eating fish (WHO IPCS, 1994). Studies have also shown that HCBD is rapidly cleared in invertebrates species with data in an oligochaete worm indicating a half life of less than 5 days (Oliver, 1987). Even though biokinetic and metabolic data which have shown that HCBD metabolic processes can be saturated (WHO IPCS Environmental Health Criteria on Hexachlorobutadiene, 1994) this will not occur at low environmental exposure concentrations. Overall these data support the conclusion that the risk of bioaccumulation and secondary poisoning of HCBD is low.

10. CONCLUSION

The calculated PEC/PNEC ratios for HCBD for the various scenarios are summarised in the table below. The calculated PEC/PNEC ratio for surface waters is less than 1 indicating that the levels of HCBD measured in marine surface waters are considered not to represent a risk to the marine environment in the North Sea region. The assessment also indicates that fish toxicity resulting from bioconcentration of HCBD (uptake from water) is unlikely while there is little risk of general toxicity occurring in fish eating mammals or birds. The lack of sediment toxicity data made it necessary to use the equilibrium partitioning method to estimate $PNEC_{sediment}$. The PEC/PNEC ratio for sediment for typical and worst-case exposures were below 1, indicating that risks to sediment organisms are unlikely.

Overall the data are supportive of the conclusion that the levels of HCBD in the marine environment do not pose an unacceptable risk and as environmental concentrations of HCBD continue to decline then so does any residual risk. This conclusion is supported by the decision of Environment Canada to delete HCBD from the Canadian Environmental Contaminants Act List of Priority Chemicals on the basis

that levels of HCBD in the aquatic ecosystem were not high enough to merit further investigation.

Summary table for PEC/PNEC ratios – Hexachlorobutadiene

Compartment	**PEC**	**PNEC**	**PEC/PNEC**
Aquatic			
Typical	5 ng/l	130 ng/l	0.038
worst case	12 ng/l	130 ng/l	0.092
Fish (CBB approach)	0-0.4 µg/kg	111 mg/kg	$0\text{-}3.6*10^{-6}$
Sediment			
Typical	1.1 µg/kg d.w.	24.4 µg/kg d.w.	0.045
Worst case	4 µg/kg d.w.	24.4 µg/kg d.w.	0.16
	EDI	**NOAEL**	**EDI/PNEC**
Predators			
- Rodent (chronic toxicity)	0.06 µg/kg bw	200 µg/kg bw	0.0003
- Quail (sub chronic toxicity)	0.04 µg/kg bw	3,000 µg/kg bw	0.00001
- Rat (reproductive toxicity)	0.06 µg/kg bw	20,000 µg/kg bw	$0.3*10^{-6}$

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APPENDIX 1

Environmental quality criteria for assessment of ecotoxicity data

The principal quality criteria for acceptance of data are that the test procedure should be well described (with Reference to an official guideline) and that the toxicant concentrations must be measured with an adequate analytical method.

Four cases can be distinguished and are summarised in the following table according to criteria defined in IUCLID system).

Table: Quality criteria for acceptance of ecotoxicity data

Case	Detailed Description of the test	Accordance With scientific guidelines	Measured concentration	Conclusion: reliability level
I	+	+	+	[1] : valid without restriction
II	±	±	±	[2] : valid with restrictions; to be considered with care
III	Insufficient or -	-	-	[3] : invalid
IV	the information to give an adequate opinion is not available			[4] : not assignable

The selected validated data LC50, EC50 or NOEC are divided by an assessment factor to determine a PNEC (Predicted No Effect Concentration) for the aquatic environment.

This assessment factor takes into account the confidence with which a PNEC can be derived from the available data: interspecies- and interlaboratory variabilities, extrapolation from acute to chronic effects.

Assessment factors will decrease as the available data are more relevant and Refer to various trophic levels.

APPENDIX 2

Ultimate distribution in the environment according to Mackay level 1 model (details of calculation)

Fugacity Level I calculation

Chemical: Hexachlorobutadiene

Fugacity Level I calculation

Chemical: Hexachlorobutadiene

Temperature (C)	20
Molecular weight (g/mol)	260.80
Vapour pressure (Pa)	20
Solubility (g/m3)	3.20
Solubility (mol/m3)	0.01
Henry's law constant (PA.m3/mol)	1630
Log octanol water part. coefficient	4.90
Octanol water part. coefficient	79432.82
Organic C-water part. coefficient	32567.46
Air-water partition coefficient	0.67
Soil-water partition coefficient	977.02
Sediment-water partition coefficient	1954.05
Amount of chemical (moles)	1
Fugacity (Pa)	.39710364E-6
Total VZ products	2518234.23

Phase properties and compositions:

Phase properties and compositions:

Phase	Air	Water	Soil	Sediment
Volume (m3)	.600OE+10	.7000E+7	.4500E+5	.2100E+5
Density(kgm3)	.12056317E+2	.1000E+4	.1500E+4	.1500E+4
Frn org carb.	.0000E+0	.0000E+0	.20000000E-1	.40000000E-1
Z mol/m3.Pa	.41029864E-3	.61349693E-3	.59940106E+0	.11988021E+1
W mol/Pa	.24617918E+7	.42944785E+4	.26973047E+5	.25174844E+5
Fugacity	.39710364E-6	.39710364E-6	.39710364E-6	.39710364E-6
Conc mol/m3	.16293108E-9	.24362186E-9	.23802434E-6	.47604869E-6
Conc g/m3	.42492427E-7	.63536583E-7	.62076749E-4	.12415349E-3
Conc ug/g	.35244946E-5	.63536583E-7	.41384499E-4	.82768999E-4
Amount mol	.97758652E+0	.1705353OE-2	.10711095E-1	.99970226E-2
Amount %:	97.76	0.17	1.07	1.00

APPENDIX 3

SUMMARY TABLE OF ECOTOXICITY DATA ON HEXACHLOROBUTADIENE

1. FISH

Species	Duration h(hours)/d (days)	Type of Study	Criterion (LC50/EC50 NOEC/LOEC)	Concentration (mg/l)	Validity	Comments	Reference
EC50/LC50 STUDIES							
1. Freshwater							
Pimephales promelas	96h	AFT	LC50	0.1	1	Secondary sources give as 0.102 mg/l.	Walbridge *et al.* (1983), Benoit *et al.* (1982), EPA (1980)
Pimephales promelas	96h	AFT	LC50	0.09	1		Geiger *et al.* (1985)
Brachydanio rerio	96h	AFT	LC50	0.24	1	To GLP & OECD 203, *Data abstracted from IUCLID*	Roederer *et al.* (1989)
Carassius auratus	96h	ASS	LC50	0.09	2	Partial closure of vessels (glass lid); no aeration. Mean of analysis at 0 and 24h.	Leeuwangh *et al.* (1975), EPA (1980)
Lepomis macrochirus	96h	AFT	LC50	0.324	2	Also 192h LC50 of 0.318 mg/l	Call *et al.* (1983), EPA (1980)
Oncorhynchus mykiss	96h	AFT	LC50	0.32	2	Also 192h LC50 of 0.121 mg/l	Call *et al.* (1983), EPA (1980)
Brachydanio rerio	48h	NFTC	LC50	1	2		Slooff (1979), Walbridge *et al.* (1983)
Poecilia latipinna	30.5h	AS	LC50	4.5	2	Limited method information.	Laseter *et al.* (1976)
Poecilia reticulata	14d	NSSC	LC50	0.41	3	Closed system with air space.	Koenemann (1981)
Ictalurus punctatus	96h	NS	LC50	0.76	3	Limited method information	Mayer & Ellersieck (1986)
Lepomis macrochirus	96h	NS	LC50	0.76	3	Limited method information	Mayer & Ellersieck (1986)
Salmo gairdneri	96h	NS	LC50	0.25	3	Limited method information	Mayer & Ellersieck (1986)
Leuciscus idus	48h ?	N?S?	LC50	>3	3	Limited information. DIN 38412 (L15)	Knie *et al.* (1983)
Leuciscus idus	48 h ?	N?S?	LC50	470	3	Limited information. DIN 38412 (L15)	Juhnke & Luedemann (1978)
Poecilia latipinna	96h	AFT	LC50	1.6	3	Limited method information.	Laska *et al.* (1978), Walbridge *et al.* (1983)

SUMMARY TABLE OF ECOTOXICITY DATA ON HEXACHLOROBUTADIENE

1. FISH

Species	Duration h(hours)/ d(days)	Type of Study	Criterion (LC50/EC50 NOEC/LOEC)	Concentration (mg/l)	Validity	Comments	Reference
EC50/LC50 STUDIES							
2. Saltwater							
Limanda limanda	96h	AFT	LC50	0.45	1	Method designed for compounds with high volatility	Pearson & McConnell (1975)
Cyprinodon variegatus	96h	A	LC50	3.6	2	Limited method information	Dow Chemical Company (1978)
Lagodon rhomboides	no data	NS	LC50	0.399	3	No details. Cites unpublished EPA data.	US EPA (1980)
Cyprinodon variegatus	no data	NS	LC50	0.557	3	No details. Cites unpublished EPA data.	US EPA (1980)

SUMMARY TABLE OF ECOTOXICITY DATA ON HEXACHLOROBUTADIENE

1. FISHES

Species	Duration h(hours)/ d(days)	Type of Study	Criterion (LC50/EC50 NOEC/LOEC)	Concentration (mg/l)	Validity	Comments	Reference
NOEC/LOEC STUDIES							
1. Freshwater							
Pimephales promelas	28d	AFT	NOEC	0.0065	1	Method: US EPA. NOEC for larval survival and weight.	Benoit *et al.* (1982), Walbridge *et al.* (1983)
Carassius auratus	67d	ASS	NOEC	0.0096	2	NOEC for growth. Partial closure of vessels (glass lid); no aeration. Mean of analysis at 0 and 24h.	Leeuwangh *et al.* (1975)
Carassius auratus	49d	ASS	NOEC	0.003	3	NOEC for liver enzyme activity - significance uncertain. Partial closure of vessels (glass lid); no aeration. Mean of analysis at 0 and 24h.	Leeuwangh *et al.* (1975)
Brachydanio rerio	24h	NFTC	Detection limit	0.05	3	Respiration (gill beat) monitor	Slooff W. (1979)
Brachydanio rerio	14d	A	NOEC	0.005	4	OECD 204 - Data abstracted from IUCLID	Roederer *et al.* (1989)
Brachydanio rerio	14d	A	LOEC	0.014	4	OECD 204 - Data abstracted from IUCLID	Roederer *et al.* (1989)
2. Saltwater							
No data available							

SUMMARY TABLE OF ECOTOXICITY DATA ON HEXACHLOROBUTADIENE

2. INVERTEBRATES

Species	Duration h(hours)/ d(days)	Type of Study	Criterion (LC50/EC50 NOEC/LOEC)	Concentration (mg/l)	Validity	Comments	Reference
EC50/LC50 STUDIES							
1. Freshwater							
Asellus aquaticus	96h	ASS	LC50	0.13	2	Partial closure of vessels (glass lid); no aeration. Mean of analysis at 0 and 24h.	Leeuwangh *et al.* (1975)
Lymnaea stagnalis	96h	ASS	LC50	0.21	2	Partial closure of vessels (glass lid); no aeration. Mean of analysis at 0 and 24h.	Leeuwangh *et al.* (1975)
Daphnia magna	24h	NS	EC50	0.5	3	Limited information. Method DIN 38412 (L11).	Knie *et al.* (1983)
Dreissena polymorpha	<8h	NS	Detection limit	0.15 - 0.41	3	Shell valve closure. Recirculating system.	Slooff *et al.* (1983)
2. Saltwater							
Eliminius modestus	48h	ASC	LC50	0.87	2	Method designed for compounds with high volatility. Nauplii tested.	Pearson & McConnell (1975)
Mysidopsis bahia	?	NS	LC50	0.059	4	Not on IUCLID.	US EPA (1980)
Palaemonetes pugio	?	NS	LC50	0.032	4	Not on IUCLID.	US EPA (1980)
NOEC/LOEC STUDIES							
1. Freshwater							
No data available							
2. Saltwater							
No data available							

SUMMARY TABLE OF ECOTOXICITY DATA ON HEXACHLOROBUTADIENE

3. AQUATIC PLANTS

Species	Duration H(ours)/ d(days)	Type of Study	Criteria (LC50/EC50 NOEC/LOEC)	Concentration mg/l	Validity	Comments	Reference
EC50/LC50 STUDIES							
1. Freshwater							
Scenedesmus quadricauda	8 d	NSC	toxicity threshold	>25	3		Bringmann & Kuehn (1977)
Haematococcus plura lis	4h	NS	EC10	>2	3		Knie *et al.* (1983)
2. Saltwater							
No data available							
NOEC/LOEC STUDIES							
1. Freshwater							
No data available							
2. Saltwater							
No data available							

LIST OF ABBREVIATIONS USED IN TABLES

A	=	Analysis
C	=	Closed system or controlled evaporation
O	=	Open vessels
h	=	hour(s)
d	=	day(s)
MATC	=	Maximun acceptable toxicant concentration
N	=	nominal concentration
S	=	static
SS	=	semistatic
F-T	=	flowthrough

Validity column:

1	=	valid without restriction
2	=	valid with restrictions: to be considered with care
3	=	invalid
4	=	not assignable

Appendix 4

MONITORING DATA HCBD

A. CONCENTRATIONS IN COASTAL WATER AND ESTUARIES, in ng/l

COUNTRY	WATER TYPE		HCBD CONCENTRATION
United Kingdom	Coastal		<5
	Estuaries	Mersey	<5-92
		Lune	<5
		Kent	<10
		Ribble	<5
		Waver	<5
		Wyre	<5
Belgium	Coastal		2-15
	Estuary	Schelde	<5-7
Germany	Estuaries	Elbe	<0.1-2
		Weser	<2
		Rhine (NL Border)	<1-4
The Netherlands	Estuary	Maas/Rhine	<1-6
		Schelde	<1-3
		Ijsselmeer	<1

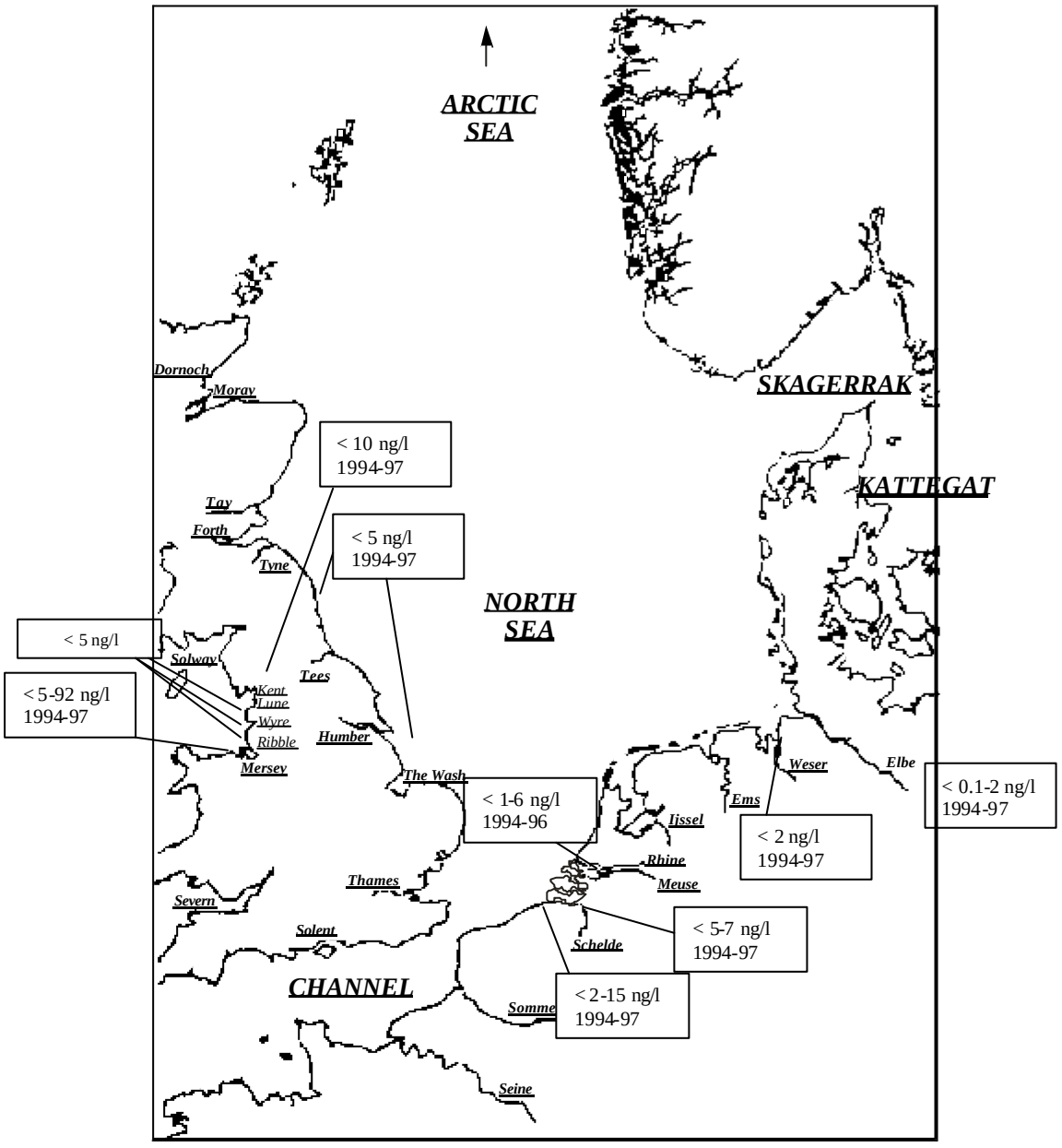
B. CONCENTRATIONS IN ESTUARINE SEDIMENTS, in µg/kg dw

Country	Sediment type	Location	HCBD Concentration
Germany	Estuary	Weser	<2
		Rhine(NL border)	<1-3
United Kingdom	Coastal		<0.3
	Estuary	Tay	<0.2
The Netherlands	Estuaries	Ijsselmeer	<1-1
		Maas/Rhine	<1-2
		Schelde	1
		Ijmuiden	<1

All data from COMMPS database linked to the Fraunhofer study (1998) and covering the period 1994-1997

APPENDIX 5

NORTH SEA MONITORING DATA ON PENTACHLOROPHENOL



APPENDIX 6

CALCULATION FOR A PNEC SEDIMENT

The PNEC for sediment can be calculated from the aquatic toxicity data ($PNEC_{water}$), according to the method described in the EU TGD, based on equilibrium partitioning theory (Di Toro *et al*, 1991). This calculation requires knowledge of the organic carbon/ water partition coefficient (K_{oc}) and the characteristics of the sediment must also be defined, in particular the weight fraction of organic carbon in the sediment (F_{oc}). The TGD default for freshwater sediment is F_{oc} = 0.05 (5% organic carbon). Although this level, or higher, is typical of estuaries, particularly in the upper, silt-rich area near to the riverine input, the F_{oc} tends to decrease towards the mouth of the estuary and the coastal sea, declining to 1% or less in coarse, sandy offshore sediments. Therefore, for these purposes, a value of 2% (F_{oc} = 0.02) is selected, as a "reasonable worst-case" average for estuarine and coastal areas, since it is likely that the majority of the monitoring data (and the highest levels of contaminants) are found in these regions. It should be noted that the affinity of hydrophobic chemicals for organic carbon will result in a general positive correlation between organic matter content and contaminant concentration. Thus, although the calculated $PNEC_{sediment}$ would be lower if the F_{oc} was lower than 2%, the exposure level (PEC) in such sediment is also likely to be lower.

For substances with a K_{oc} value of 2000 or above, the $PNEC_{sediment}$ is directly proportional to F_{oc}. Therefore, if the available monitoring data specifies the organic carbon level of the sediment, the PNEC can be simply corrected to the same carbon level. (For K_{oc} <2000, the proportionality is not exact, due to the TGD method of calculation, but is a sufficiently good approximation for these purposes).

Using the quantitative structure activity relationship (QSAR) for calculating K_{oc} from K_{ow} for non polar hydrophobic organics ($\log K_{oc} = 0.81 \times \log K_{ow} + 0.1$), $\log K_{oc}$ for HCBD is estimated to be 3.97 (K_{oc} = 9371) for $\log K_{ow}$ 4.78.

The $PNEC_{sediment}$ is calculated from $PNEC_{water}$ as follows:

$PNEC_{sed} = K_{sed\text{-}water}/RHO_{sed} \times PNEC_{water} \times 1000$ mg/kg wet weight
where: $PNEC_{water}$ is the predicted no-effect concentration in water (mg/l) (0.00013 mg/l)
RHO_{sed} is the bulk density of wet sediment (1300 kg/m^3)
$K_{sed\text{-}water} = F_{solidsed} \times Kp_{sed}/1000 \times RHO_{solid}$
where: $F_{solidsed}$ = fraction of solid in sediment set at 0.2 m^3/m^3

and $K_{p(sed)}$ = solid-water partition coefficient in sediment = $F_{oc(sed)} \times K_{oc}$
= 0.02 x 9371 = 187.4

RHO_{solid} is the density of the solid phase (2500 kg/m^3)

So $K_{sed-water}$ = (0.2 m^3/m^3 x 187.4/1000 x 2500 kg/m^3) = 93.7

Therefore, $PNEC_{sed}$ = 93.7/1300 x 0.00013 mg/l x 1000
= 0.00937 mg/kg wet weight = 9.37 µg/kg wet weight

The fixed sediment characteristics define a sediment wet/dry ratio of 2.6. Therefore:

$PNEC_{sed}$ = 9.37 x 2.6
= 24.4 µg/kg dry weight