

既存化学物質の生態影響に関する情報

平成20年12月19日 化審法3省合同会議

官報公示 整理番号	CAS No.	物質名称	頁
4-44	843-55-0	1, 1-ビス(4-ヒドロキシフェニル)-シクロヘキサン	1
3-2254	3194-55-6 25637-99-4	1, 2, 5, 6, 9, 10-ヘキサブロモシクロドデカン	13

要 約

試験委託者 : 環境省

表 題 : 1,1-ビス(4-ヒドロキシフェニル)-シクロヘキサンの
藻類 (*Pseudokirchneriella subcapitata*) に対する生長阻害試験

試験番号 : A060512

試験方法 : 本試験は「新規化学物質等に係る試験の方法について<藻類生長阻害試験, ミジンコ急性遊泳阻害試験及び魚類急性毒性試験>」(平成15年11月21日 薬食発第1121002号, 平成15・11・13製局第2号, 環保企発第031121002号, 最終改正: 平成18年11月20日) に準拠して実施した。

- 1) 供試生物 : 単細胞緑藻類 (*Pseudokirchneriella subcapitata*)
 2) 試験用水 : 試験ガイドライン推奨培地
 3) 暴露期間 : 72時間
 4) 培養方式 : 止水式 (開放系), 振とう培養 (100 rpm)
 5) 初期生物量 : 前培養した藻類 5×10^3 cells/mL
 (指数増殖期の藻類乾燥重量: 1.7×10^{-8} mg/cell, n=7)
 6) 試験温度 : 22 °C (暴露期間中の変動範囲は ± 2 °C以内)
 7) 照明 : 65~75 $\mu\text{E}/\text{m}^2/\text{s}$, 白色蛍光灯で連続照明 (液面付近)
 8) 試験濃度 (設定値) :

試験区	濃度 (mg/L)
対照区	—
助剤対照区	—
濃度区 1	0.24
濃度区 2	0.51
濃度区 3	1.1
濃度区 4	2.3
濃度区 5	4.8*

助剤: *N,N*-ジメチルホルムアミド 95 $\mu\text{L}/\text{L}$
(濃度一定, ただし対照区は使用せず)

公比: 2.1

* : 試験液調製可能最高濃度

9) 分析法 : 高速液体クロマトグラフ (HPLC) 法

結 果

1) 試験液および試験培養液中の被験物質濃度

測定値の設定値に対する割合は、暴露開始時の試験液において 88~96%、暴露開始後 72 時間の試験培養液において 43~79%であり、暴露期間中に濃度減少が認められた。この減少理由の詳細は不明であるが、藻類が関与した被験物質の変化、または藻体への吸着の可能性が考えられた。

2) 生長速度の比較による阻害濃度

阻害濃度の算出には測定値の時間加重平均値を用いた。

半数生長阻害濃度 ErC50(0-72h) : >3.64 mg/L* (95%信頼区間 : 算出不可)

最大無影響濃度 NOECr(0-72h) : 0.92 mg/L

*試験最高濃度区は、試験液調製可能最高濃度 (4.8 mg/L, 測定値の平均値 : 3.64 mg/L) であり、阻害率が<50%であったため、「>試験最高濃度」という結果となった。

3) 藻類の形態観察

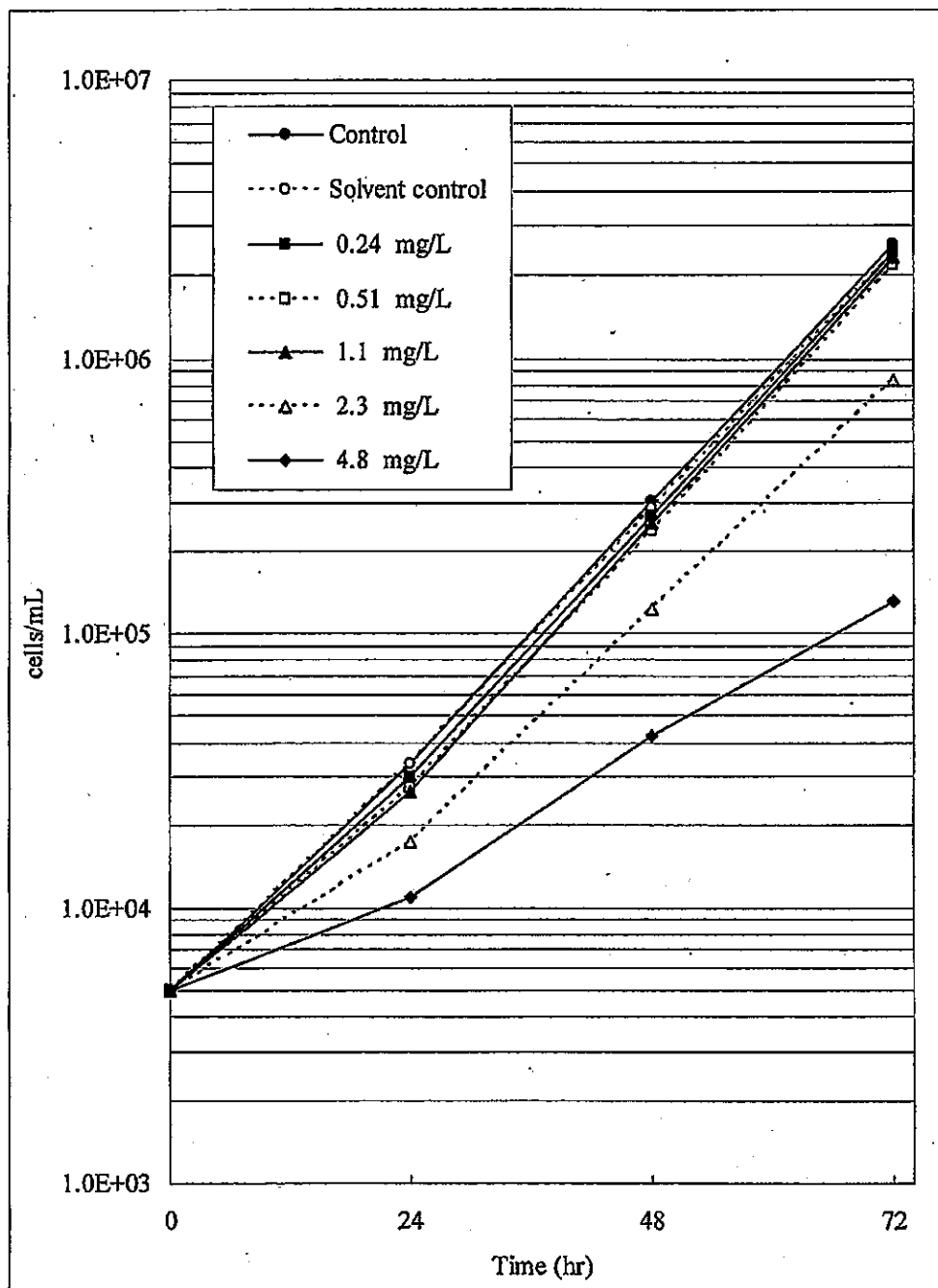
暴露開始後 72時間の顕微鏡下での細胞形態観察の結果、全ての濃度区において、細胞形態の変化(収縮, 膨張, 破裂等)や細胞凝集は認められず、また、対照区および助剤対照区との相違もなかった。

Table 4 Measured Concentration of the Test Substance in Test Cultures

Test Group	Nominal Concentration (mg/L)	Measured Concentration (mg/L) (Percent of Nominal)				Mean ^a Measured Concentration (mg/L) (Percent of Nominal)
		0 Hour	24 Hours	48 Hour	72 Hours	
Control	--	<0.02	<0.02	<0.02	<0.02	---
Solvent control	--	<0.02	<0.02	<0.02	<0.02	---
Conc.1	0.24	0.23 (96)	0.22 (92)	0.21 (88)	0.18 (75)	0.21 (88)
Conc.2	0.51	0.48 (94)	0.48 (94)	0.45 (88)	0.39 (76)	0.45 (88)
Conc.3	1.1	1.02 (93)	0.94 (85)	0.92 (84)	0.79 (72)	0.92 (84)
Conc.4	2.3	2.08 (90)	2.00 (87)	2.00 (87)	1.82 (79)	1.98 (86)
Conc.5	4.8	4.20 (88)	3.88 (81)	4.02 (84)	2.04 (43)	3.64 (76)

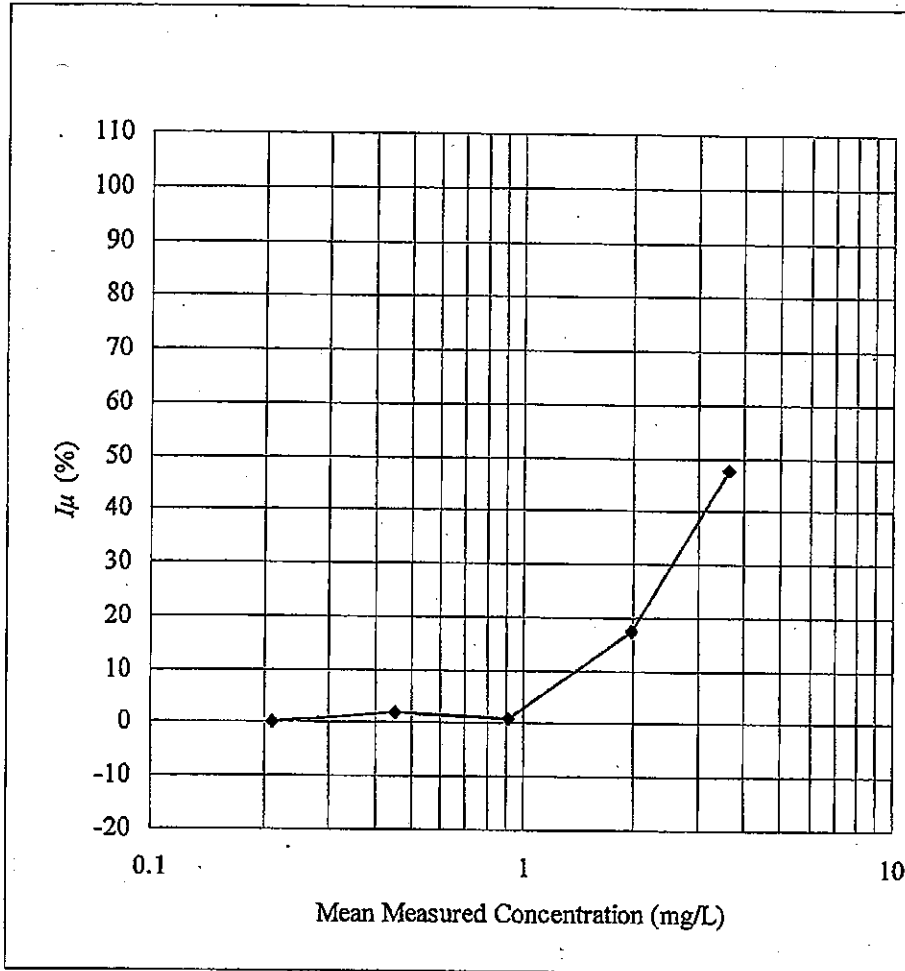
a : Time weighted mean

Figure 1 Growth Curve of *Pseudokirchneriella subcapitata*
(Mean biomass vs time during the 72-hour exposure)



Values in legend are given in the nominal concentration.

Figure 2 Concentration-Inhibition Curve Based on I_{μ} values Calculated from the Growth Rates



要約

試験委託者： 環境省

表 題： 1,1-ビス(4-ヒドロキシフェニル)-シクロヘキサンのオオミジンコ
(*Daphnia magna*) に対する急性遊泳阻害試験

試験番号： A060513

試験方法： 本試験は、「新規化学物質等に係る試験の方法について<藻類生長阻害試験、ミジンコ急性遊泳阻害試験及び魚類急性毒性試験>」（平成15年11月21日薬食発第1121002号、平成15-11-13製局第2号、環保企発第031121002号、最終改正：平成18年11月20日）に準拠して実施した。

- 1) 供試生物： オオミジンコ (*Daphnia magna*)
- 2) 試験用水： Elendt M4 medium
- 3) 暴露期間： 48時間
- 4) 暴露方式： 半止水式 (24時間後に試験液の全量を交換)
- 5) 供試生物数： 20頭/試験区 (5頭/容器)
- 6) 試験温度： 20±1℃
- 7) 照明： 室内光, 16時間明 (800 lux 以下) / 8時間暗
- 8) 試験濃度 (設定値) :

試験区	濃度 (mg/L)
対照区	—
助剤対照区	—
濃度区1	0.47
濃度区2	0.84
濃度区3	1.5
濃度区4	2.6
濃度区5*	4.7

公比 1.8

助剤：N,N-ジメチルホルムアミド, 95 μL/L
(濃度一定, ただし対照区は使用せず)

*: 試験用水に対する溶解度

- 9) 分析方法： 高速液体クロマトグラフ (HPLC) 法

結果：以下の結果は、測定値をもとに算出した。

48時間 半数遊泳阻害濃度 (EC50) : 1.81 mg/L (95%信頼限界 1.59~2.08 mg/L)

Table 5 Measured Concentrations of the Test Substance in Test Solutions

(Semi-Static Condition)

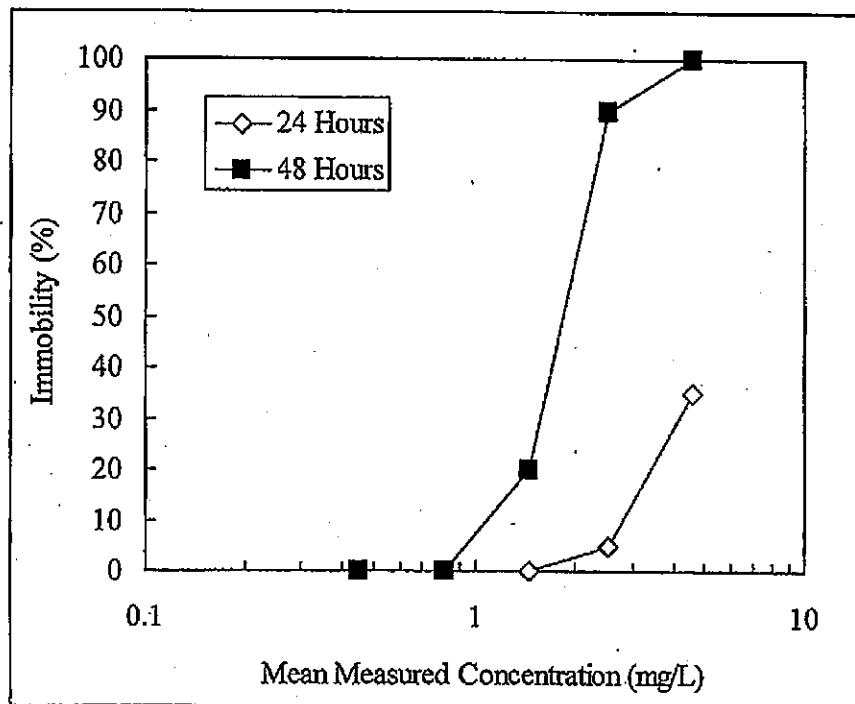
Test Group	Nominal Concentration (mg/L)	Measured Concentration (mg/L)				Mean ^a
		0 Hour New	24 Hours Old	24 Hours New	48 Hours Old	
		(Percent of Nominal, %)				
Control	--	<0.04	<0.04	<0.04	<0.04	--
Solvent Control	--	<0.04	<0.04	<0.04	<0.04	--
Conc.1	0.47	0.45 (96)	0.46 (98)	0.46 (98)	0.45 (96)	0.45 (96)
Conc.2	0.84	0.81 (96)	0.81 (96)	0.82 (98)	0.83 (99)	0.82 (98)
Conc.3	1.5	1.45 (97)	1.44 (96)	1.48 (99)	1.46 (97)	1.46 (97)
Conc.4	2.6	2.51 (97)	2.49 (96)	2.51 (97)	2.57 (99)	2.52 (97)
Conc.5	4.7	4.53 (96)	4.52 (96)	4.57 (97)	4.65 (99)	4.57 (97)

a: Time-weighted mean

New: New test water freshly prepared

Old: Old test water immediately prior to renewal or at the end of the exposure

Figure 1 Concentration-Immobility Curve



要 約

試験委託者： 環境省

表題： 1,1-ビス (4-ヒドロキシフェニル) -シクロヘキサンのヒメダカ (*Oryzias latipes*) に対する急性毒性試験

試験番号： A060514

試験方法： 本試験は「新規化学物質等に係る試験の方法について〈藻類生長阻害試験、ミジンコ急性遊泳阻害試験及び魚類急性毒性試験〉」（平成15年11月21日薬食発第1121002号，平成15・11・13製局第2号，環保企発第031121002号，最終改正：平成18年11月20日）に準拠して実施した。

- 1) 供試生物： ヒメダカ (*Oryzias latipes*)
- 2) 試験用水： 脱塩素水道水
- 3) 暴露期間： 96時間
- 4) 暴露方式： 半止水式 (24時間毎に試験液の全量を交換)
- 5) 供試生物数： 10尾/試験区
- 6) 水温： 24±1℃
- 7) 照明： 室内光，16時間明 (1000 lux 以下) / 8時間暗
- 8) 試験濃度 (設定値)：

試験区	濃度 (mg/L)
対照区	—
助剤対照区	—
濃度区1	0.48
濃度区2	0.85
濃度区3	1.5
濃度区4	2.7
濃度区5	4.8

公比：1.8

助剤：N,N-ジメチルホルムアミド，95 μL/L (濃度一定，ただし対照区は使用せず)

- 9) 分析方法： 高速液体クロマトグラフ (HPLC) 法

結果：

以下の結果は，被験物質濃度の測定値をもとに算出した。

96時間半数致死濃度 (LC50)： 1.82 mg/L (95%信頼限界 1.46 ~ 2.25 mg/L)

Table 5 Measured Concentrations of the Test Substance in Test Water

Test group	Nominal conc. (mg/L)	Measured concentration (mg/L)					Mean
			0 - 24 hr	24 - 48 hr	48 - 72 hr	72 - 96 hr	
Control		New	<0.02	<0.02	<0.02	<0.02	
		Old	<0.02	<0.02	<0.02	<0.02	
Solvent control		New	<0.02	<0.02	<0.02	<0.02	
		Old	<0.02	<0.02	<0.02	<0.02	
Conc.1	0.48	New	0.41	0.45	0.46	0.44	0.42 [88%]
		Old	0.41 (100%)	0.41 (91%)	0.39 (85%)	0.42 (95%)	
Conc.2	0.85	New	0.82	0.80	0.77	0.76	0.75 [88%]
		Old	0.73 (89%)	0.71 (89%)	0.72 (94%)	0.71 (93%)	
Conc.3	1.5	New	1.39	1.36	1.37	1.38	1.34 [89%]
		Old	1.30 (94%)	1.30 (96%)	1.28 (93%)	1.31 (95%)	
Conc.4	2.7	New	2.48	2.47	2.47	2.49	2.46 [91%]
		Old	2.33 (94%)	2.44 (99%)	2.42 (98%)	2.58 (104%)	
Conc.5	4.8	New	4.47	--	--	--	4.31 [90%]
		Old	4.16 (93%)	-- (--)	-- (--)	-- (--)	

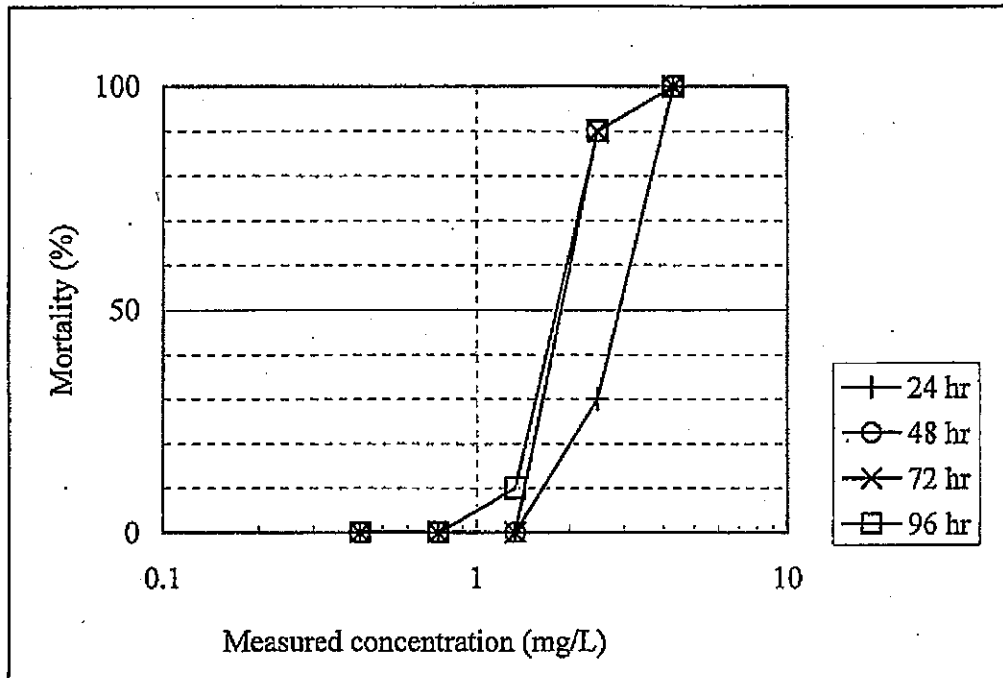
New: New test water freshly prepared .

Old: Old test water immediately prior to renewal or at the end of the exposure
(Percent of New)

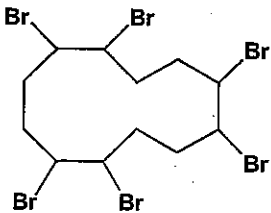
Mean: Time-weighted mean
[Percent of Nominal]

--: Not measured because all fish were dead.

Figure 1 Concentration-Mortality Curve



SIDS INITIAL ASSESSMENT PROFILE

CAS No.	25637-99-4, 3194-55-6
Chemical Name	Hexabromocyclododecane (HBCDD)
Structural formula	

SUMMARY CONCLUSIONS OF THE SIAR

Human Health

Available studies demonstrate that HBCDD can be absorbed from the gastro-intestinal tract. The highest concentrations are subsequently reached in adipose tissue and muscles followed by liver, and with much lower concentrations present in lung, kidney, blood, brain, and gonads. At long-term exposure, higher concentrations are achieved in females than in males, but the substance is bioaccumulating in both sexes. Of the three diastereoisomers constituting HBCDD, the α -form is much more accumulating than the others (the relative bioaccumulation factor is 99:11:1 for α , β and γ , respectively). The time to reach steady-state seems to be in the order of months. HBCDD can be metabolised, and three polar metabolites are identified. Elimination of HBCDD and its metabolites mainly occur via faeces with a minor part excreted in urine. The absorption, both after oral and inhalation exposure, is set to 100 %, whereas a value of 2 % (granules) or 4 % (powder) is set for the dermal absorption, depending on the size of the particles occurring at the exposure situation.

HBCDD has demonstrated a low acute toxicity. The minimal lethal dose is greater than 20 g/kg for both dermal and oral routes of administration, and greater than 200 mg/L at inhalation for 4 hours.

HBCDD is mildly irritating for the eyes, but not irritating to skin.

Two Magnusson-Kligman studies performed with HBCDD of unknown specification have given positive results. However, studies on HBCDD of known specification by the major producers of HBCDD, has shown negative results both in a Magnusson-Kligman test and in a Local Lymph Node assay. Overall, HBCDD is therefore not regarded as a skin sensitiser.

No repeated dose toxicity studies with inhalation or dermal exposure as route of administration are available.

A 28-day repeated dose toxicity study has been performed using a benchmark model design with oral administration of dissolved HBCDD. There were five dose groups, the highest dose group achieved 200 mg/kg bw/day. The result showed organ weight increase of the liver, the thyroid, and the pituitary. Enzyme induction in the liver was likely the cause of the effects, as hepatic enzyme induction leads to increased excretion of T4, compensatory activation of the pituitary, increased serum TSH concentration and thereby activation of the thyroid.

Other repeated dose toxicity studies, both 28- and 90 days with oral exposure of HBCDD particles, support the liver and thyroid being the main target organs. The NOAEL of 1000mg/kg bw/day from the 90 days study would normally be preferred, but the uncertainties introduced in the evaluation of this study by the dosing of HBCDD-particles to the animals, leads to the choice of a NOAEL from a 28-days study.

Overall, a NOAEL/BMD-L of 22.9 mg/kg/day for liver weight increase is decided for repeated dose toxicity.

HBCDD did not induce mutations in the Ames test, and was negative in both an *in vitro* chromosome aberration test and an *in vivo* micronucleus test. Therefore, it can be concluded that HBCDD lacks significant genotoxic potential *in vitro* as well as *in vivo*. Based on one available lifetime assay, it is not possible to assess the carcinogenic potential of HBCDD. However, the available database gives no

reason for further exploration of this endpoint.

Two developmental toxicity studies have failed to demonstrate any fetotoxicity, teratogenic potential, or adverse effects from HBCDD on development postpartum. A NOAEL of >1000mg/kg/day (highest dose tested) is decided for developmental toxicity.

A study on developmental neurotoxicity in adult mice exposed as pups at day 10 postpartum has been conducted. It indicates that HBCDD may cause statistically significant changes in spontaneous behaviour, learning, and memory defects at the dose 0.9 mg/kg/day. The study is published, but has not been performed according to OECD Test Guideline or GLP and thus would benefit from being confirmed by other laboratories. Overall, the study thus indicates that the substance is a possible developmental neurotoxicant.

Functional observation batteries and motor activity tests in the 28-day and 90-day studies showed no evidence of neurotoxicity.

The reproductive toxicity is not fully tested because there are no fertility studies available. However, the 90-day study included investigation on the reproductive organs including histopathological examination, semen analysis and estrous cycle monitoring. At the highest dose (1000 mg/kg/day) a statistically significant increase in absolute and relative prostate weight was observed, but no accompanying microscopical changes were detected. Females showed signs of inhibited oogenesis in most of the follicles and sparse ripening follicles in the ovaries at exposure to 4700 mg/kg/day in a 28-day study not conducted in accordance with present standards. The effect on the prostate and the oogenesis has not been confirmed in any other studies and the inhibited oogenesis occurred at a very high dose.

Environment

Technical grade HBCDD is generally produced from *cis trans, trans-1,5,9-cyclododecatriene* (CDT), one of four CDT isomers, (CAS No. 27070-59-3). The reaction, *trans*-addition of bromine to the double bounds of CDT, results in the three diastereomers α -, β - and γ -HBCDD. The final distribution of the diastereomers in technical HBCDD varies with a range of about 70-95 % γ -HBCDD and 5-30 % α - and β -HBCDD. The major impurities are tetrabromocyclododecene and isobutanol. HBCDD is a white odourless solid. The log n-octanol/water partition coefficient (log Kow) of HBCDD was determined to 5.6 at 25±0.05°C. The composite sample produced by mixing equal amounts of three commercial HBCDD technical products contained 8.5 % β -, 6.0 % α - and 79.1 % γ -HBCDD (total HBCDD 93.6%). There was no information on the identity and properties of the remaining 6.4%. A vapour pressure of 6.3·10⁻⁵ Pa at 21°C is used in the assessment. The melting point range varies from approximately 172-184 °C for a crude product to 201-205 °C for the highest melting version following crystallisation. Melting points for the individual diastereomers have been determined to 207-210°C for γ -HBCDD, 171-181 °C for α -HBCDD and 169-172 °C for β -HBCDD. The water solubility of technical HBCDD has been determined to 3.4µg/l at 20°C. However, this value mainly reflects the water solubility of γ -HBCDD. The different diastereomers have different water solubilities, 49 µg/l for α -HBCDD, 15µg/l for β -HBCDD and 2µg/l for γ -HBCDD. The value of 66µg/l, which is the sum of the water solubilities of the three diastereomers, is used for the technical HBCDD mix of diastereomers in the EUSES calculations as a worst case estimate.

The log Koc of HBCDD is calculated to be 4.66 thus, HBCDD is predicted to absorb strongly to organic carbon (i.e. soil and sediment). The substance has a low potential to evaporate from the aquatic surface and evaporation is therefore considered as a less important route of dispersion. Despite this two different studies have shown that HBCDD has a low long-range transport potential (LRTP). The distance was estimated to be in the range of 760-2550 km. The LRTP potential is confirmed by findings of HBCDD in biota in the arctic. Calculations (EUSES model) indicate that the overall removal of HBCDD in a sewage treatment plant is approximately 80%. The major part is expected to be adsorbed to the sludge.

The log Kow of HBCDD of 5.6 indicates a potential for accumulation in living organisms. Results from two studies on fish support each other. One of the studies gave a BCF value of 18100 for fathead minnow. The other study, where two different test concentrations were employed, gave BCF values of 8974 and 13085 in the high and low dose group, respectively. When estimated with kinetic modelling the BCF-values from the study were calculated to be 16450 and 21940, in the two dose groups respectively. An overall BCF of 18,100 is chosen as representative from these studies. One existing study on earthworms (*Eisenia fetida*) shows that HBCDD is taken up in the worm tissue. Although not valid for making conclusions about the magnitude of bioconcentration of HBCDD in earthworms it shows that the uptake of α -HBCDD (BAF: 0.3-0.8) was more than one order of magnitude higher than for γ -HBCDD (BAF: 0.005—0.02). This is in line with what has been observed also for other biota e.g. mammals and fish where α -HBCDD is the dominating diastereomer despite constituting only 6% of the technical product and in most cases having the lowest concentrations in the abiotic environment i.e. sediment.

There are lots of monitoring studies showing uptake of HBCDD in biota. Data exists from freshwater invertebrates, freshwater fish, plants and birds, where the concentrations range between 0.025-28, 0.03-9432, 1.5-11114 and 0.002-160 $\mu\text{g}/\text{kg}$ ww, respectively. Moreover, there are data from brackish and marine biota including invertebrates (0-329 $\mu\text{g}/\text{kg}$ ww), fish (0.001-89 $\mu\text{g}/\text{kg}$ ww), and marine birds mainly eggs (0-100 $\mu\text{g}/\text{kg}$ ww). The highest concentrations of HBCDD in marine biota are measured in marine mammals with concentrations ranging from 0.5-6404mg/kg ww. This is a strong indication that HBCDD biomagnifies in the marine food chain. Monitoring data also indicate that the concentrations of HBCDD are increasing. The mean concentrations measured in Atlantic puffin, Herring gull and Kittiwake in the North of Norway have increased with a factor of about 5-8 over 20 years. The mean concentrations measured in eggs from Guillemot from St. Karlsö in the Baltic Sea has approximately doubled from 8 μg HBCDD/kg ww in the early 1970-ties to about 16 μg HBCDD/kg ww in the late 1990's. The increase has levelled out since the mid 1990's. Also for marine mammals the data indicate increasing tissue concentrations. The median concentrations in the blubber of harbour porpoises stranded or dying due to physical trauma in the UK increased from below 100 $\mu\text{g}/\text{kg}$ lw in the mid 1990's to 9400 $\mu\text{g}/\text{kg}$ lw in 2003. HBCDD has also been detected in adipose tissue of polar bears from Svalbard in concentrations 5-45 μg HBCDD/kg ww.

The hydrolysis of HBCDD has not been studied. Hydrolysis should however, not be considered as a significant route of environmental degradation for this substance due to the low water solubility and high partitioning to organic carbon. Furthermore, the dissipation of HBCDD in abiotic aerobic water sediment studies was very slow. No studies on abiotic degradation of HBCDD in air, i.e. photodegradation exist. The route is however considered to have low environmental significance because of low vapour pressure of HBCDD.

Standard ready and inherent biodegradation tests show no biodegradation over a 28d test period under aerobic conditions. Several studies on biodegradation of HBCDD in sediment and soil are available giving different results. Two of these are considered reliable. In the first study the HBCDD concentrations ranged between 34-89 $\mu\text{g}/\text{kg}$ sediment dw (which is comparable to the mean concentrations of HBCDD in sediment if sediments affected by point sources are excluded) and 25 $\mu\text{g}/\text{kg}$ dw in soil. Due to the low concentrations only the degradation of the γ -diastereomer could be followed. The half life for γ -HBCDD in aerobic sediment was 11 and 32 days in two different sediments and approximately 1 day in anaerobic sediment at 20 °C. The half life for γ -HBCDD in aerobic and anaerobic soil at 20 °C was 63 days and 7 days, respectively. No degradation products were detected neither in sediment nor soil. In the second study the HBCDD concentration was 4.3 – 4.7 mg/kg dw in sediment (which is comparable to levels in sediment measured close to some point sources) and 3 mg/kg dw in soil. In soil no degradation was observed whereas in sediment the half-life of total HBCDD at 20°C was 101 days under aerobic conditions and 66 days under anaerobic conditions. α - and γ -HBCDD had similar half lives under aerobic conditions but γ -HBCDD disappeared with a half life of 66 days compared to 113 days for α -HBCDD under anaerobic conditions, indicating that α -HBCDD may be more stable than γ -HBCDD. Results from degradation studies in sewage sludge support this indication where α -HBCDD degraded more slowly than γ -HBCDD in two studies out of three. The transformation pathway of HBCDD was identified as being a step-wise dehalogenation, via tetrabromocyclododecene and dibromocyclododecadiene to 1,5,9-cyclododecatriene. The degradation of the transformation product 1,5,9-cyclododecatriene has been studied in a modified ready biodegradation test. The test substance was coated on silica gel and incubated for up to 77 days. During this period significant amounts of CO₂ (70%) of were formed indicating that the substance is biodegradable.

Five studies on the toxicity of HBCDD for algae are available. The 72h-EC₅₀ value of 52 $\mu\text{g}/\text{l}$ is considered as the most realistic and reliable result. From corresponding experiments on invertebrates (2 studies) and fishes (3 studies), a 48h-EC₅₀ of >3.2 $\mu\text{g}/\text{l}$ for a crustacean (*Daphnia magna*) and a 96h-EC₅₀ value of \geq 2.5 $\mu\text{g}/\text{l}$ for rainbow trout (*Oncorhynchus mykiss*) was determined respectively. The toxicity of HBCDD to aquatic organisms has also been obtained with QSAR for *Daphnia magna*. The result show a 48h-EC₅₀=140 $\mu\text{g}/\text{l}$, which is far above the water solubility of HBCDD. Two long term tests are available for HBCDD. The first study, a reproduction test (21d) on *Daphnia magna* reports a NOEC of 3.1 $\mu\text{g}/\text{l}$. In the other study, a fish early life stage test on *Oncorhynchus mykiss*, no effects were seen at the highest tested concentration, which was 3.7 $\mu\text{g}/\text{l}$. Two studies on sediment dwelling organisms are available, both considered reliable. The lowest NOEC value is 3.1mg/kg dw sediment for the reduction of total numbers of worms (*Lumbriculus variegatus*).

There are three studies available on terrestrial organisms from three trophic levels, soil micro-organisms, plants, and soil dwelling organisms. No effects on soil micro-organisms (nitrification) were seen in any treatment giving a NOEC >1000mg HBCDD/kg dw soil. No effects could be determined for the plants in the highest concentration tested 6200 μg HBCDD/kg soil. The test species were corn (*Zea mays*), cucumber (*Cucumis sativa*), onion (*Allium cepa*), ryegrass, (*Lolium perenne*), soybean (*Glycine max*), and tomato (*Lycopersicon esculentum*). A NOEC value of 128mg HBCDD/kg dry soil for reproduction

of earthworms (*Esenia fetida*) (56d) is concluded for the terrestrial environment.

Exposure

HBCDD was in 2006 only produced at one site in EU15¹, located in the Netherlands. The total annual (2005) EU15 production of HBCDD is around 6 000 tonnes. No information on export of HBCDD from the EU has been provided. Countries outside the EU15 known to produce HBCDD are the USA and Japan. HBCDD is imported to the EU15 from the USA (around 5 000 tonnes per year). Data from Japan indicate that the consumption of HBCDD in Japan is about 2 000 tonnes per year.

The main uses of HBCDD are in the polymer and textile industries. HBCDD can be used on its own or in combination with other flame retardants e.g. antimony trioxide and decabromodiphenyl ether. HBCDD is used in four principal product types, which are Expandable Polystyrene (EPS), Extruded Polystyrene (XPS), High Impact Polystyrene (HIPS) and Polymer dispersion for textiles.

According to industry information, the main use (90%) of HBCDD is in polystyrene (PS). The predominant use of PS is in rigid insulation panels/boards for building and construction (EPS and XPS). About 2 % of the total use of HBCDD is in "high impact polystyrene" (HIPS). Most of the flame retarded HIPS-products are used in electrical and electronic appliances e.g. audio visual equipment cabinets (video and stereo equipment), distribution boxes for electrical lines in the construction sector, refrigerator lining.

Textiles with back-coating containing HBCDD can be used for e.g. flat and pile upholstered furniture (residential and commercial furniture), upholstery seatings in transportation, draperies, and wall coverings, bed mattress ticking, interior textiles e.g. roller blinds, automobile interior textiles and car cushions.

Humans may be exposed to HBCDD at the workplace, from use of consumer products, and indirectly from the environment via food, soil, water and air. The highest exposures undoubtedly occur in the workplace environment. Still, there are no occupational exposure limits for HBCDD. The available information about releases of HBCDD from waste and waste management is limited. Possible routes of release are as dust particles from demolition of buildings, leakage from landfilled material and from waste remained in the environment. The emission today are assumed to be limited, due to that HBCDD has only been used in the last decades, but may increase in the future depending on how the waste will be handled.

RECOMMENDATIONS AND RATIONALE FOR THE RECOMMENDATION AND NATURE OF FURTHER WORK RECOMMENDED

Human Health: The chemical is a candidate for further work. The chemical possesses properties indicating a hazard for human health with regard to repeated dose toxicity and possible developmental neurotoxicity. Therefore, member countries are invited to perform an exposure assessment and if then indicated a risk assessment. Note: A risk assessment performed in the EU in the context of the EU Existing Chemicals Regulation is in progress.

Environment: The chemical is a candidate for further work. The chemical possesses properties indicating a hazard for the environment (acute aquatic toxicity to algae, chronic toxicity to *Daphnia*, high bioaccumulation potential). Therefore, member countries are invited to perform an exposure assessment and if indicated a risk assessment. Note: A risk assessment performed in the EU in the context of the EU Existing Chemicals Regulation is in progress.

¹ Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Luxemburg, Netherlands, Portugal, Sweden, Spain, United Kingdom