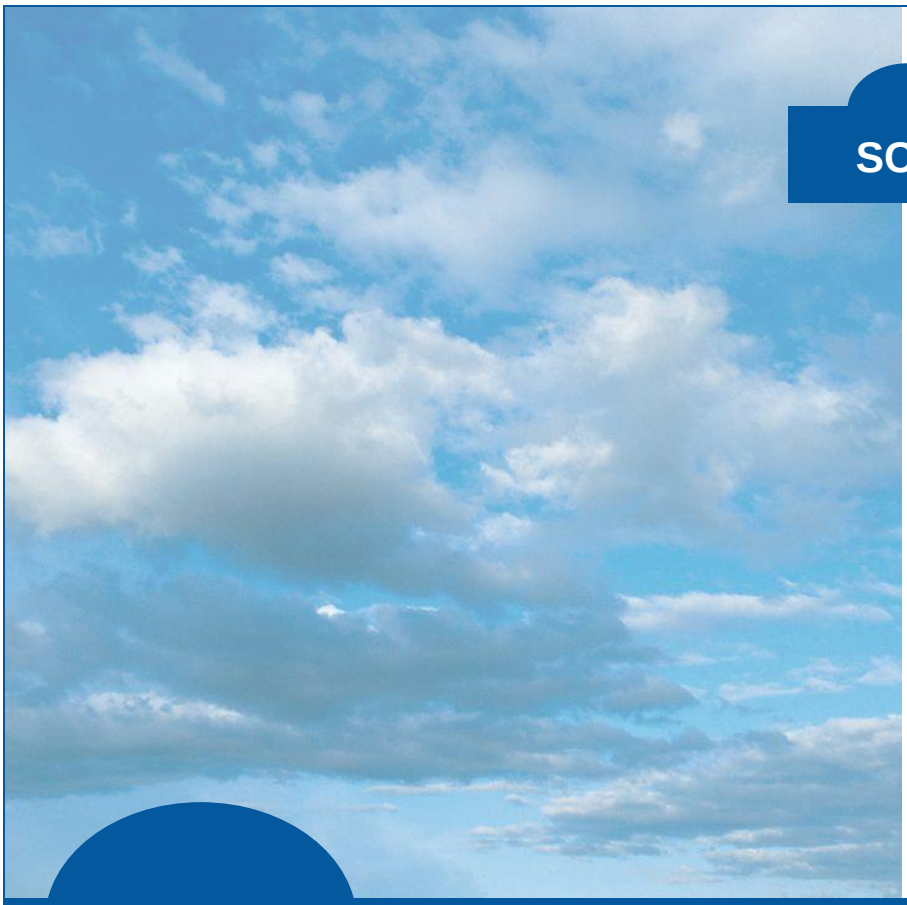

SCIENCE DOSSIER

Hexachlorobutadiene - Sources, environmental fate and risk characterisation

Professor André Lecloux

October 2004

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Foreword

The Monitoring & Environmental Chemistry Working group (MECW) is a science group of Euro Chlor, which represents the European chlor-alkali industry. The main objectives of the group are to identify both natural and anthropogenic sources of chlorinated substances, study their fate, gather information on the mechanisms of formation and degradation in the environment and achieve a better knowledge of the persistence of such substances. The MECW often uses external specialists to assist in developing reports that review the state of existing knowledge of the different aspects mentioned. The principal investigator collects information from the scientific literature and available data regarding natural or anthropogenic emissions in the environment, with the objective to cover all the aspects described above.

Dr André Lecloux is Professor in advanced inorganic materials at the University of Liège and is currently a scientific advisor to Euro Chlor. His past roles have been as general manager of R&D for Solvay in Germany (1991-1995) prior to joining Euro Chlor as science director for six and a half years. Between 1996 and 1998 he was seconded to Cefic to start up the endocrine modulator research programme, which has since become a key part of the chemical industry's Long-range Research Initiative. In 1999, Prof. Lecloux was presented with the Golden Award of the Association des Ingénieurs de l'Université de Liège in recognition of his scientific contribution to academia and industry.

Much has been written about hexachlorobutadiene (HCBD) and politicians have subjected it to extensive regulation. Its status is currently being reviewed as to whether or not it should be listed as a Persistent Organic Pollutant. *Hexachlorobutadiene – Sources, environmental fate and risk characterisation* examines how HCBD is created, its characteristics and how it impacts on the environment and human health. One of the main findings is that the substance is indeed toxic and prone to long range transport via air. Despite its bioaccumulating properties, there is no evidence to suggest that it is prone to biomagnification.

Summary

This science dossier examines the sources of HCBD, a substance currently subject to much regulation with its candidature as a possible Persistent Organic Pollutant further putting it in the spotlight. The fate of HCBD concerns the chlor-alkali industry in particular because its primary source is as an unintended by-product in the manufacture of chlorinated hydrocarbons (e.g. perchloroethylene, trichloroethylene and carbon tetrachloride). Currently HCBD is not commercially produced in the United Nations Economic Commission for Europe (UNECE) region. In fact, commercial production has ceased within Europe (Euro Chlor 2000/BUA 1991).

It is estimated that 1% to 3% of HCBD formed in the 1970s was released into the environment during that time. Some was emitted to water in industrial effluent and some to air from stacks.

HCBD is toxic and is likely to bio-accumulate but does not bio-magnify in the food chain. It may persist in air, at least until it comes into contact with OH radicals or is photo-chemically degraded or deposited in water or soil when adsorbed on particulate matter. In water, sediment or soil containing organic matter, HCBD may biodegrade.

There are no known natural sources of the HCBD; WHO, EPA and Environment Canada have concluded that the low levels of HCBD in the environment do not present a danger to human life or health.

1. Introduction

Hexachlorobutadiene has historically been subject to extensive regulation. It has recently come under renewed scrutiny in various regulatory forums as a potential candidate Persistent Organic Pollutant (POP). The aim of this study is to describe the sources, properties and environmental fate of hexachlorobutadiene and any potential risks to the environment or human health.

2. Physical-chemical properties of hexachlorobutadiene

Hexachlorobutadiene (HCBD) has the empirical molecular formula C_4Cl_6 and the structural formula shown in Figure 1. HCBD is a non-flammable, incombustible, clear, colourless, oily liquid at room temperature with a mild turpentine-like odour (HSDB, 2000). The compound is poorly soluble in water, but miscible with ether and ethanol.

Property	Value	Reference
CAS registry number	87-68-3	
Molecular weight	260.76 g/mol.	
Melting point	-21°C	Montgomery and Welkom, 1990
Boiling point	215°C	Montgomery and Welkom, 1990
Density at 20°C	1.55 g/cm³	HSDB 1993
Water solubility at 25°C	3.20 mg/l	Gradiski *et al.*, 1975; Mackay *et al.*, 1998
Water solubility at 20°C	2.55mg/l	Montgomery and Welkom, 1990
Vapour pressure at 20°C	20 Pa	Pearson and McConnell, 1975; Mackay *et al.,*1998
Henry's law constant	1,044 Pa·m³/mol	Shen, 1982; Mackay *et al*., 1998
log K_{oc}	3.67	ATSDR, 1994; Mackay *et al*., 1998
log K_{ow}	4.78	ATSDR, 1994; Mackay *et al.*, 1998
Auto-ignition temperature	610°C	IPCS, 1994

Table 1: Properties of hexachlorobutadiene

The poor solubility in water of HCBD, its high vapour pressure, its high log K_{oc} and log K_{ow} values determine its behaviour and fate in environmental media.

To compare environmental concentrations from various sources and in different media, conversion factors should be used and the following values are recommended (IPCS, 1993): 1 ppbv of hexachlorobutadiene = 10.67 µg/m^3 air, and 1 µg of hexachlorobutadiene per m^3 air = 0.094 ppbv at 25 °C and 101.3 kPa (760 mm Hg).

Synonyms for HCBD include 1,1,2,3,4,4-hexachloro-1,3-butadiene, hexachloro-1,3-butadiene, perchlorobutadiene and perchloro-1,3-butadiene. Previous common trade names were Dolen-Pur; C-46, UN2279 and GP-40-66:120 (U.S. EPA, 1980 and 1991a).

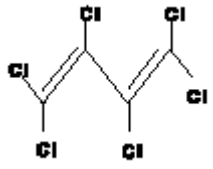

Figure 1: Structure of HCBD

The substance can be detected and determined quantitatively by gas chromatographic methods. Detection limits are 0.03 µg/m^3 in air, 0.001 µg/dm^3 in water, 0.7 µg/kg wet weight in soil or sediment and 0.02 µg/dm^3 in blood, (IPCS 1993).

3. Fluxes in the environment

3.1 Possible sources

The primary source of hexachlorobutadiene is inadvertent production as a by-product of the manufacture of chlorinated hydrocarbons such as perchloroethylene, trichloroethylene and carbon tetrachloride, where it occurs in the heavy fractions (US EPA, 1980; Kusz *et al.*, 1984; Yang, 1988; Choudhary, 1995; US EPA, 2002). Several reports (US EPA, 1980; Environment Canada, 2000; VROM, 2002) indicate the possibility of HCBD forming as by-product during the production of vinyl chloride, allyl chloride and epichlorohydrin. This is in fact extremely unlikely due to the relatively reductive conditions existing in these processes. The reports may have resulted from the fact that wastes from all processes carried out at one site are normally combined before being destroyed by incineration or otherwise disposed of.

During the 1970s and 1980s, large amounts of HCBD were been produced as by-product of chlorination processes involving organic compounds. Annual worldwide production of the compound in heavy fractions was estimated to be 10,000 tonnes in 1982 (CCOHS, 2001). Larger quantities of the chemical were reportedly generated in the US as waste by-product from the chlorination of hydrocarbons: about 4,000 tons in 1975 and 14,000 tons in 1982 (US EPA, 1982b; HSDB, 1993). In North America (US and Canada), HCBD has never been manufactured as a commercial product. In the US, small quantities of HCBD were imported - mostly from Germany - as commercial product: about 250 tons/year in the late 1970s and 75 tons/year in 1981 (Howard, 1989; ATSDR, 1994). In the past, HCBD was also imported into Canada for use as a solvent (Environment Canada, 1979), but is no longer imported or used (Environment Canada, 1997c).

According to a recent study (VROM, 2002), there is probably no current commercial production of HCBD in the United Nations Economic Commission for Europe (UNECE) region of 55 member countries. No data are available on production outside the UNECE region. According to Euro Chlor (2002) and BUA (1991) commercial production of HCBD has been virtually eliminated in Europe.

Among production processes leading to formation of HCBD as an unavoidable by-product, it seems that the only remaining significant source in the UNECE region is the low-pressure chlorolysis for combined production of perchloroethylene and tetrachloromethane. The residue obtained from the process is generally destroyed by incineration at 1,200 ^{o}C or recycled through high temperature chlorolysis to carbon tetrachloride and perchloroethylene (Markovec and Magee, 1984).

HCBD can also be released from magnesium production plants and from other non-chemical industries (Deutscher and Cathro, 2001; Lenoir *et al.,* 2001), but no information on quantities is available.

There are no known natural sources of HCBD (Environment Canada 2000; US EPA, 2002)

3.2 Uses

The large amounts of HCBD produced as by-product provided an incentive to develop industrial applications. Prior to 1975, the largest use of HCBD was for the recovery of “snift” (chlorine-containing gas in chlorine plants, Gulko, 1972; HSDB, 1993) and as a wash liquor for removing certain volatile organic compounds from gas streams (Verschueren, 1983). HCBD is no longer used for these purposes (IARC, 1979; US EPA, 1982d; ATSDR, 1994).

HCBD was also used as a chemical intermediate in the manufacture of rubber compounds, and in production of chlorofluorocarbons and lubricants (IARC 1979; US EPA, 1980; Verschueren, 1983; Manahan, 1992). Lesser quantities were used as solvent in rubber manufacturing (US EPA, 1982d), as well as for transformer and hydraulic fluids, fluid for

gyroscopes, heat transfer liquid, laboratory reagents, and as a wash liquor for removing C4 and higher hydrocarbons (Hawley, 1981).

It also had widespread application as a fumigant for protecting grapevines against the parasitic insect pest, *Phylloxera*, in the former Soviet Union and to a lesser extent in France, Italy, Greece, Spain and Argentina. Widespread use no longer occurs (IARC, 1979; Howard, 1989; NTP, 1991; IPCS, 1994; ATSDR, 1995).

3.3 Emissions to the environment

3.3.1 Historical data (before 1985)

In the 1970s, formation of HCBD as a by-product was estimated to be equivalent to 1.5% of total perchloroethylene production (Brown *et al.*, 1975), and the fraction of HCBD released to the environment during its industrial life cycle has been estimated to range between 1 and 3% (SRI, 1984). Some of this waste was emitted to the aquatic environment in industrial effluents and to air from stacks.

Using a simple model describing the troposphere, the global annual emission rate was calculated to be 3200 tonnes/year of HCBD based on air sampling data in 1985 (Class & Ballschmiter, 1987). This level of release is most probably linked to the use of HCBD as fumigant in agricultural applications.

Historically, removed lining (ebonite) and graphite electrodes from chlorine electrolysis cells also generated solid waste containing traces of HCBD, which were handled carefully because they also contained traces of mercury.

3.3.2 More recent data (from 1988 to 2002)

Releases of HCBD to the environment can potentially occur *via* unintentional emissions from the production of chlorinated solvents, from current or historical disposal of waste containing HCBD, from magnesium production, or from any remaining commercial uses.

Recent data are available only for Western Europe and North America. Levels of environmental release of HCBD in other countries from the UNECE region or elsewhere are unknown. Since chlorinated solvents are produced in many parts of the world, potential unintentional emissions of HCBD to air and water might still occur in these countries.

In Canada, since the closing of the country's two perchloroethylene plants in 1985 and 1992, there have been no major point sources of HCBD (Environment Canada, 2000). Current Canadian sources are minor but potentially numerous. They include possible releases in landfill leachates and releases during refuse combustion. Based on the monitoring of discharge and effluent streams from organic chemical manufacturing plants in Ontario between 1989 and 1991, a total loading to water was estimated to be 20 g/day (OME, 1992).

The Great Lakes Commission reports HCBD air emissions of 15 pounds in 1997, mainly from manufacture of rubber-based products (GLC, 1997).

Until recently, the most significant point source of HCBD in Canada appeared to be the Cole Drain, which discharges into the St Clair River and includes outfalls from an industrial landfill and a few industrial companies. Loadings from the Cole Drain appear to have decreased from 140 g/day in 1985 (OME, 1991) to 30 g/day in 1995, corresponding to a maximum concentration of 0.9 µg HCBD/l in the final mixing chamber discharge in 1995. Since 1998, discharge from the Cole Drain has been practically eliminated as a result of remediation activities. The industrial landfill that was the primary source of HCBD in the Cole Drain was completely remediated and decommissioned, and the bed of the Cole Drain itself was remediated and restored in 1998 (Environment Canada, 2000; Sarnia Lambton, 2000).

In the United States, facilities are required to report the pounds per year of HCBD released into the environment both on- and off-site for inclusion in the Toxics Release Inventory (TRI). The on-site quantity is subdivided into air emissions, surface water discharges, underground injections and releases to land. The TRI data are useful in giving a general

idea of release trends; however, they are not exhaustive due to exclusion of releases from smaller industries. The current threshold reporting quantity of 25,000 lbs/yr was adopted in 1990, down from 75,000 lbs/yr in 1988 (TRI, US EPA, 2000a).

Over the period 1988–1998, air emissions constituted most of the on-site releases; surface water discharges initially increased, peaked in 1992–1993, and then decreased significantly through the late 1990s (see Table 2 below, US EPA, 2000b).

More recent data indicates that in 1998 the majority of releases came from the chemical sector (1,300 kg) and from the electrical, gas and sanitary services sectors (460 kg); in 1999 and 2000, total releases to air of 2,635 kg and 1,936 kg, respectively, were reported. The load to the atmosphere, however, does not include all possible releases from every type of industrial facility (ATSDR, 1994).

Some 15,000 tons of HCBD appear to have been produced in 2000 as by-product, but recycled in production processes or treated and destroyed as waste, mainly by incineration on-site or in publicly-owned treatment works. Only 1,835 kg of HCBD was released in waste. In the US, most of the disposal waste from chlorinated hydrocarbon manufacturing processes is incinerated.

Year	On-Site releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1998	2,421	5	0	0	510	2,936
1997	1,415	9	299	0	200	1,923
1996	2,381	256	952	0	310	3,899
1995	3,310	661	434	0	252	4,657
1994	1,410	351	201	0	430	2,392
1993	1,747	1,200	520	0	12	3,479
1992	4,134	1,911	738	0	5	6,788
1991	3,410	681	200	2	4,263	8,556
1990	4,906	715	330	0	45	5,996
1989	4,628	622	330	1	26,343	31,924
1988	2,508	153	220	0	19,640	22,521

Table 2: Environmental Releases (in pounds) of Hexachlorobutadiene in the US, 1988-1998 (Source: US EPA (2000b)

These TRI data for HCBD were reported from eight US states. However, HCBD contamination has been found in remote areas far from obvious physical discharge sources in at least 14 states (Howard, 1989; US EPA, 2000b).

In Europe, from recent surveys by Euro Chlor (Lecloux, 2003; Euro Chlor, 2004) at 76 European chlor-alkali production sites, HCBD emissions to water were reported to have decreased from 100 kg/year in 1997 to 2.4 kg/year in 2002. Emissions to air decreased from 2 kg/year to close to zero over the same period. This represents a reduction of more than 99% compared to 1985. According to the first edition of the EU-EPER database in 2001, there were no emissions of HCBD to air but emissions to water of 25.63 kg from all manufacture of organic chemicals and solvents (EU-EPER, 2004).

In 1990, HCBD emissions in Germany from the use of perchloroethylene as a solvent were estimated to be <0.56 and 620 kg/year to air and water, respectively, and discharges into Rhine and Elbe rivers were estimated at 70 and 150 kg/year, respectively (BUA, 1991).

Historical landfill storage of heavy fractions from the production of chlorinated organic substances can also lead to secondary HCBD emissions or leachates. In the UK, for example, environmental contamination with HCBD was recently detected around a disused dump. HCBD was detected in the underlying strata and groundwater and in indoor air in properties close to the site (COT, 2000).

4. Occurrence of hexachlorobutadiene in the environment

4.1 Air

In **Canada** HCBD was detected (detection limit 0.1 µg/m^3) in 153 of 9,231 samples (i.e., less than 2%) of outdoor air from 46 sites across Canada surveyed from 1989 to early 1997. It has not been detected at any of these sites since 1994. The maximum concentration measured was about 4 µg/m^3 in Windsor in 1992. Mean concentrations at each site, calculated by assuming a concentration of one-half the detection limit of 0.1 µg/m^3 in those samples not showing detectable levels of HCBD, ranged from 0.05 to 0.07 µg/m^3; concentrations in remote areas were less than 1 pg/m^3 (Dann, 1997; Environment Canada, 2000). HCBD has rarely been detected in recent monitoring programmes in areas far away from former sources.

For the United States, concentration data on HCBD in air have been summarised by the US EPA (1998a). Maximum air levels in off-plant property, at a plant boundary, and within a plant were reported to be 22 ppt, 938 ppt, and 43,000 ppt, respectively (Li *et al.*, 1976). The reported average concentration of HCBD, based on 72 samples from urban and source dominated areas, was 36 ppt (0.38 µg/m³; Shah and Heyerdahl, 1988; Shah and Singh, 1988). A number of cities had HCBD levels ranging from 2 to 11 ppt (0.02 to 0.12 µg/m³; Pellizzari, 1978; Singh *et al*., 1980, 1982). Niagara Falls had higher HCBD levels, with concentrations up to 37 ppt (0.39 µg/m^3) found in ambient air levels and up to 38 ppt (0.41 µg/m^3) found in the basement air of homes near industrial and chemical waste disposal sites (Pellizzari, 1982). HCBD concentrations in ambient air were also measured in monitoring data for the Urban Area Source Program (US EPA, 1994). Concentrations of HCBD were reported at a minimum detection level of 540 µg/m³ in Columbus, Ohio, and up to 1,000 µg/m³ in Cincinnati, Ohio, from 1989 to 1991. However, a recent monitoring study at six sampling locations in Columbus, Ohio, failed to detect HCBD in air (Spicer *et al*., 1996).

Class and Ballschmiter (1987) reported that the troposphere of the Northern Hemisphere contained an average concentration of 0.17 ppt (2 ng/m³) HCBD at 18 locations sampled from 1982 to 1986. The detection limits in this survey were between 0.01 and 0.1 ppt.

In Europe, a recent study by the Swedish EPA (2001) indicates that the HCBD concentration in air varies from 2 to 5 pg/m³ in three locations. These can be regarded as "background levels".

A study of air contaminants in Porto Alegre, Brazil (Grosjean and Rassmussen, 1999) did not find detectable levels of HCBD (detection limit 100 ppt) at any of 46 sampling locations.

4.2 Water

The highest reported concentration of HCBD in Canadian surface waters was 1.3 µg/l, which was measured in the St. Clair River in 1984 (OME/MDNR, 1991). Levels have decreased substantially (i.e. 500-fold) since 1984, based on a measurement of 0.0027 µg/l downstream from the Cole Drain in 1994, the highest concentration reported that year. Since 1990, concentrations of HCBD in surface water from southern Ontario have generally been less than 0.001 µg/l (L'Italien, 1996; Environment Canada, 2000). A maximum concentration of 24 mg/kg dry weight was measured in suspended sediments from the St. Clair River in 1985 (Oliver and Kaiser, 1986). In 1989, the highest level detected was 0.01 mg/kg dry weight (Chan, 1993). In the Great Lakes area of Canada, much lower levels of HCBD, around 1 ng/litre, were measured.

In the United States, HCBD has been detected in surface waters at a very low percentage of the ATSDR monitoring sites. However, releases have been reported through the TRI, and HCBD has also been detected in public water system samples, but occurrence estimates are low with only 0.13% and 0.05% of all samples containing HCBD in two different studies. Significantly, the values for the median and 99th percentile concentrations

of all samples are less than the lowest requested reporting level. For samples containing HCBD, the median concentration at various locations varies in the range 0.25-0.30 µg/l, while the 99th percentile concentration is in the range of 1.5-10 µg/l (US EPA, 2002).

In Europe, a study of HCBD in 108 samples of seawater 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 *et al.*, 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 Rivers Rhine and Elbe contained 10 to 20 and 10 to 150 ng/l, respectively (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.

A statistical analysis (Govaerts *et al.*, 2000 and 2004) of the monitoring data of the EU COMMPS database (1998), 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 90th percentile of the concentration distribution of 6 and 12 ng/l, respectively. The distribution of the measured concentrations is illustrated in Figure 2 below. It is of particular interest that only 13% of the measured values are above detection limit. The values under the detection limit (DL) have been replaced by DL/2.

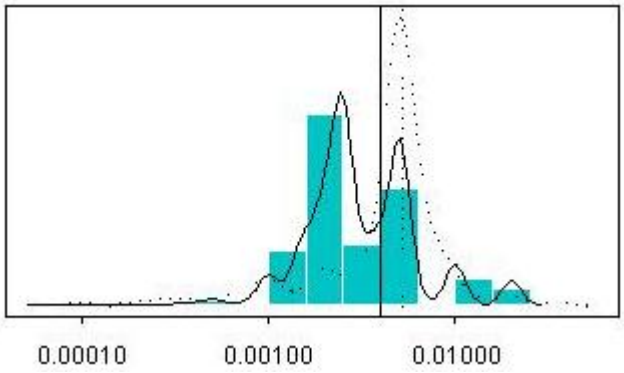

Figure 2: Distribution of HCBD concentrations in European surface waters in µg/l. Data below the detection limit (DL) have been replaced by DL (dotted line) or DL/2 (solid line), with the mean values represented by vertical lines.

Between 1993 and 1996, concentrations reported in estuaries (WRc, 1998 and EU COMMPS database, 1998) varied from 0.4 to 90 ng/l, with typical values in the range of 1 to 5 ng/l.

In a study (Meharg *et al.*, 1998) on the Humber catchments in the UK in 1995-1996, only one sample of water out of 280 samples analysed for HCBD was above the limit of detection of 0.04 ng/l. The author concluded that HCBD "does not constitute an environmental problem to the southern Humber rivers".

HCBD has not been detected in drinking water (detection limits ranging from 0.7 pg/l to 5 µg/l) in most provincial monitoring programs in Canada. It was detected (detection limit 1 ng/l) in only five out of 2,994 samples of treated drinking water from 143 sites across Ontario surveyed in 1991–1995; the maximum concentration measured was 6 ng/l (OMEE, 1996).

4.3 Sediment

In Canada, the greatest contamination by HCBD in sediment reportedly occurred in a few "hot spots". In the St Clair River, Ontario, the HCBD concentration prior to 1986 was 430 mg/kg dry weight. In 1985, HCBD was also detected (detection limit not specified) in 59 of 65 sampling sites in the same area, with the lowest reported concentration being 0.1 µg/kg dry weight (Oliver and Pugsley, 1986).

The highest concentration measured in recent years (1994) in the same area was 310 mg/kg dry weight, downstream from the Cole Drain. In the top 5 cm of sediment in the St

Clair River in 1994, concentrations of HCBD ranged from <0.001 to 243 mg/kg dry weight, with a geometric mean of 0.640 mg/kg dry weight (Bedard and Petro, 1997). In these samples, the 99th, 95th and 90th percentile values were 194, 60.9 and 18.7 mg/kg dry weight, respectively, while the median was 0.9 mg/kg dry weight. Bottom sediment levels here were reported to be up to 0.120 mg/kg dry weight. Older sediment layers from around 1960 contained higher concentrations (up to 0.550 mg/kg wet weight). The sediment concentration was demonstrated to increase with particle size in the sediment. In 1994, HCBD was detected (detection limit 1 µg/kg dry weight) in 148 of 153 samples (Farara and Burt, 1997).

In the United States, as reported by the US EPA (1999b), HCBD was not detectable in any of 196 sediment samples, based on a detection limit of 0.5 mg/kg for the analyses (Staples *et al.*, 1985). Sediments from the Niagara River contained 2.9 to 11 µg/kg HCBD (Oliver and Bourbonniere, 1985). Sediments from the Great Lakes were reported to contain levels of HCBD typically ranging from 0.08 to 120 µg/kg (McConnell *et al*., 1975).

Several studies have investigated HCBD levels in sediments from sites in Louisiana. HCBD levels ranged from less than 0.05 µg/kg to 0.40 µg/kg (Abdelghani *et al.*, 1995). These concentrations were well below the action levels of 4,000 µg/kg for sediment (US EPA, 1991a). However, several hot spots were identified in Baton Rouge and Lake Charles areas, with measured concentrations ranging from 2 to 82 mg/kg (US EPA, 1992b; Prytula and Pavlostathis, 1996a; Gess and Pavlostathis, 1997; Chen *et al*., 1999).

In Europe, 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 from 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 estuarine and river sediments in Europe have been reported in the EU COMMPS database over the period 1994-1997. Statistical analysis (Govaerts *et al*., 2000 and 2004) of about 500 measured concentrations indicated that the mean and the 90th percentile values of HCBD concentrations in sediments in Europe are 1.1 and 4 µg/kg, respectively. The distribution of concentrations is illustrated in Figure 3. Interestingly, more than 50% of the measured values are below the detection limit (DL), and in the statistical analysis these were replaced by DL/2. Recent measurements from EU COMMPS database indicate that HCBD concentrations in estuarine or coastal sediment vary between < 0.2 and 3 µg/kg, with typical values close to 1µg/kg (EU COMMPS, 1998).

Recent data for suspended solids collected from the Rhine-Meuse river basin indicate HCBD levels ranging from <3.4 to 19 µg/kg (Hendriks *et al*., 1998).

Lee *et al*. (2000) analysed surface sediment samples collected from 40 stations along the Kaohsiung coast in southern Taiwan. HCBD was found at a level of a few µg/kg in the most polluted locations.

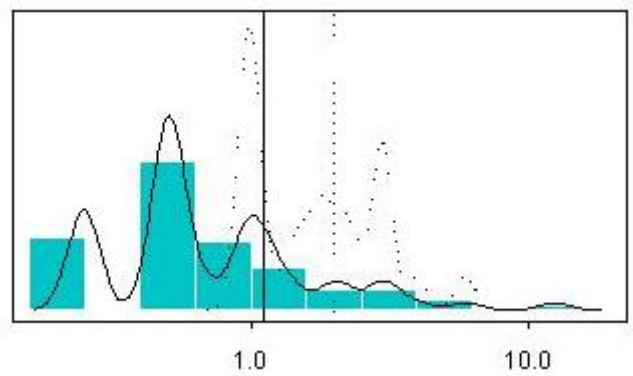

Figure 3: Distribution of HCBD concentrations in sediments of European rivers, in □g/kg dry weight. Data below the detection limit (DL) have been replaced by DL (dotted line) or DL/2 (solid line), with the mean values represented by vertical lines.

4.4 Soils

In the only identified relevant survey of soils in Canada, HCBD was neither detected (detection limit 0.05 µg/g dry weight) in 24 samples of agricultural soils from across the country, nor in six samples from areas that had repeatedly received heavy applications of pesticides (Webber and Wang, 1995).

No data was found on the concentration of HCBD in either soil or dust.

4.5 Biota

In Canada, levels in rainbow trout collected from Lake Ontario in 1981 ranged from 0.06 to 0.3 µg/kg wet weight (mean 0.2 µg/kg; Oliver and Niimi, 1983). Levels of up to 10 µg/kg wet weight were detected in composite samples of coho salmon (*Oncorhynchus kisutch*) collected from the Great Lakes in 1980 (Clark *et al.*, 1984). The maximum concentration of HCBD in caged mussels, *Elliptio complanata*, following three weeks of exposure on the sediment surface near three industrial areas of the St Clair River was 36 µg/kg wet weight (Kauss & Hamdy, 1985; OME/MDNR, 1991).

Concentrations of HCBD in aquatic organisms, birds and mammals indicate bioaccumulation but not biomagnification. In polluted waters, levels of over 1000 µg/kg wet weight have been measured in several species and 120 mg/kg (lipid base) in one species. Present levels generally remain below 100 µg/kg wet weight away from industrial outflows.

More recently, HCBD has been detected at low levels in lake trout, forage fish and invertebrates from Lake Superior in Canada (Environment Canada, 2000). The levels of HCBD in food web samples of Lake Superior measured in 1998 are given in Table 3 (Muir, 2003a).

HCBD in µg/kg wet weight	Mean value	Standard deviation
Lake trout	0.08	0.11
Smelt	0.10	0.11
Herring	0.08	0.06
Sculpin	1.49	0.80
Diporeia	0.01	0.01
Zooplankton	0.01	0.01
Mysis	0.03	0.03

Table 3: HCBD levels in food web samples of Lake Superior in 1998 (Muir, 2003a)

HCBD has been measured in beluga blubber (Muir, 2003a). Levels vary from 278 µg/kg of lipid in the St Lawrence River estuary to less than 0.1 µg/kg lipid in Northern Quebec (East Hudson Bay).

HCBD has also been detected in biota in northern Canada, which may indicate that HCBD is subject to long-range transport. However, levels are low, varying from 0.03 to 0.33 µg/kg with an average of 0.07 µg/kg in blubber of ringed seals from the Hudson Strait area of northern Quebec, Ungara Bay and Labrador (Environment Canada, 2000; Muir, 2003a).

In the United States, in Louisiana, tissue concentrations of HCBD were 226.33 ± 778.40 µg/kg in fish collected from a contaminated study site and 6.84 ± 10.41 µg/kg in fish collected from the corresponding control site. In other studies, fish samples from the Mississippi River were reported to contain HCBD levels ranging from 100 to 4,700 µg/kg (Laska *et al*., 1976; Yip, 1976; Yurawecz *et al.*, 1976). HCBD was generally not detected in fish from the Great Lakes (DeVault, 1985; Camanzo *et al*., 1987), with the exception of trout from Lake Ontario, which were reported to contain 60 to 300 µg/kg (Oliver & Niimi, 1983). HCBD was not detected in 51 biota samples catalogued in the STORET database (Staples *et al*., 1985).

The National Study of Chemical Residues in Fish (NSCRF), conducted by EPA's Office of Water, was undertaken to determine the occurrence of selected pollutants in fish from various locations across the United States. Pollutants were measured in bottom-feeding

and game fish at nearly 400 sites between 1986 and 1989 (US EPA 1992a; Kuehl *et al*., 1994). Targeted sites were chosen near areas with significant industrial, urban, or agricultural activity, including more than 100 sites near pulp and paper mills. Fish species chosen for sampling were those routinely consumed by humans and/or those expected to bioaccumulate organic contaminants. HCBD was detected in fish at 3% of the 362 sites sampled. The mean and standard deviation of HCBD concentrations in fish from all samples at all sites were 0.6 µg/kg and 8.7 µg/kg, respectively (Kuehl *et al*., 1994). Only four sites' concentrations were above 2.5 µg/kg, all of which were near organic chemical manufacturing plants (US EPA, 1992a)

In Europe, the only information on marine fish was 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 analysed, HCBD was not detected (limit of detection 0.001 ng/kg) in 10 samples and of the remaining 5 samples, the highest tissue level detected was 0.4 µg/kg.

Hendricks *et al*. (1998) evaluated HCBD levels in zebra mussel (Dreissena polymorpha) and eel (Anguilla anguilla) from approximately 30 locations in the Rhine-Meuse river basin. In zebra mussel, HCBD levels were 0.24 µg/kg at a background location and ranged from 0.95 to 14 µg/kg wet weight within the study area. In eel, HCBD levels were found to range from 5 to 55 µg/kg wet weight within the study area. A recent report on the status of the Rhine river (CIPR, 2002) indicates typical concentrations of HCBD in eel between 1 and 3 µg/kg wet weight and in roach between 1 and 2 µg/kg wet weight.

4.6 Food

Food may be contaminated with HCBD *via* environmental sources or by contact with contaminated water during food processing activity (DiNovi, 1997).

Data on levels of HCBD in foodstuffs are limited primarily to older studies. Concentrations of HCBD in beverages, bread, butter, cheese, eggs, fruits, meats, milk, oils and potatoes ranging from non-detectable to 3.7 µg/kg (grapes) were reported in the United Kingdom (McConnell *et al.*, 1975). In Germany, concentrations of HCBD in chicken, eggs, fish, margarine, meat and milk ranged from non-detectable to 42 µg/kg (egg yolk; Kotzias *et al.*, 1975).

IARC (1979) reported concentrations of HCBD in food sampled in the United Kingdom of 0.08 µg/kg in fresh milk, 2 µg/kg in butter, 0.2 µg/kg in cooking oil, 0.2 µg/kg in light ale, 0.8 µg/kg in tomatoes, and 3.7 µg/kg in black grapes.

HCBD was not detected in samples of eggs or vegetables and was detected in only 1 of 20 samples of milk produced in the vicinity of organic chemical manufacturing plants in the United States (detection limits 5 or 40 µg/kg; Yip, 1976). In a survey of human breast milk from five regions in Canada, HCBD was not detected in any of 210 samples analysed (detection limit 1.2 µg/l; Mes *et al.*, 1986).

In a recent pilot multimedia exposure study, samples of domestic air, tap water, beverages and food from 44 households in the Toronto area were analysed for HCBD. None of the samples contained detectable amounts (Zhu, 1997).

4.7 Waste

Waste streams containing HCBD generated as a by-product of certain chlorinated hydrocarbon production typically contain 33-80% HCBD (US EPA, 2002). These wastes are disposed off by various methods. Over the last decade, disposal practices have shifted from landfill to incineration. Incineration reportedly achieves greater than 99.9% destruction efficiency of HCBD (US EPA, 1982d). Historical dumping sites may contain significant amounts of HCBD, which could potentially act as secondary sources of emissions if not well confined. Improved industrial and waste treatment processes, including better improvements in containment facilities and spill prevention, have resulted in greatly reduced loadings of HCBD in the environment since the early 1980s (Environment Canada, 2000).

5. Behaviour of hexachlorobutadiene in the environment

5.1 Environmental partitioning and transport

5.1.1 Modelling

By applying a simple Mackay level I calculation (Mackay & Patterson, 1990; Pedersen *et al.*, 1994) to HCBD, its ultimate distribution at equilibrium in the environment can be estimated. It appears that 97.8% of HCBD partitions to air, 0.2% to water, 1.0% to soil and 1.0% to sediment.

If a steady-state, fugacity EQC Level III model (DMER and AEL, 1996) is used to estimate the distribution of HCBD in the environment, it shows that HCBD tends to remain in the environmental compartment into which it is released, indicating that equilibrium, as described by the simple model, is very difficult to reach.

If HCBD is emitted into air, more than 98% would stay in the air, about 1% would migrate to soil and less than 1% to water and sediment. If released to soil, about 99% would remain in the soil and about 1% would end up in air. If released to water, about 70% would remain in the water, with about 15% migrating to both air and sediment, and less than 1% to soil. The predicted distributions suggest that little inter-compartmental transport occurs when HCBD is discharged to air or soil. By comparison, disposal to water has the potential for significant transport of HCBD to the air *via* volatilisation and to sediment *via* adsorption on particulate matter.

5.1.2 Monitoring and field data

Transfer across ***soil-air boundaries*** has, however, been observed in a field study in the former USSR: concentrations of HCBD in air above a vineyard were found to be 0.08 and 0.003 mg/m^3 at 1 day and 3 months, respectively, following a spring application of 250 kg/ha. Volatilisation of the compound from light soils was more rapid than from heavy soils (Litvinov & Gorenshtein, 1982).

The value of Henry's constant indicates possible transfer of the compound across ***water-air boundaries*** leading to a wide distribution, with aerial transport playing a major role (McConnell *et al.*, 1975). In a model experiment, a loss of 25% of HCBD in 28 minutes was recorded when a 20-mg/litre aqueous-methanolic solution was gently stirred at 22°C (Hellmann, 1987a).

Adsorption to sediments and suspended particulates is an important factor in the fate of HCBD in water (US EPA, 1991a) and has been studied in various conditions, including field experiments where HCBD concentrations were measured simultaneously in water and in sediment (Leeuwangh *et al.*, 1975; Laseter *et al.*, 1976; Oliver & Charlton, 1984). These studies show that soil-water partition coefficients can range over 2 to 4 orders of magnitude, assuming equilibrium. The degree of adsorption to soil is highly dependent on the organic matter content and is less pronounced in sandy soils. It can be concluded that the compound does not migrate rapidly in soils and will accumulate in sediment. It should be noted that the micro-particles onto which HCBD is absorbed may themselves migrate to the sub-surface, resulting in facilitated transport.

Several studies indicate the possibility of ***atmospheric long-range transport*** and transboundary movement of HCBD. Evidence for such transport was provided by Mudroch *et al.* (1992), who found that HCBD was present at concentrations ranging from 0.01 to 0.23 ng/g at various sediment depths in samples taken in 1987 from Great Slave Lake in Northern Canada. Similarly, in the USA, TRI releases were reported in only eight states, but HCBD has been detected in site samples in fourteen states distributed nationwide (ATSDR, 2000). Dispersion of HCBD in the atmosphere has been confirmed by detection of HCBD at locations distant from emission sources (IPCS, 1994).

5.2 Behaviour in air

HCBD is released to air *via* chemical manufacturing and processing and by waste incineration (HSDB, 2000). The high organic carbon partition coefficient (Koc) of HCBD indicates that it could adsorb to airborne particulate matter with a high organic content. However, according to Bidleman (1988), significant adsorption onto particles can occur only if the vapour pressure is below 10^{-2} Pa. As the vapour pressure of HCBD is 20 Pa, it will be found in air mainly as a vapour and much less in association with atmospheric particulates.

HCBD absorbs light within the solar spectrum. The extent of mineralisation of the compound adsorbed on silica gel and exposed to oxygen was examined following irradiation with ultraviolet light. After 6 days, 50-90% mineralization to hydrogen chloride and/or chlorine, and carbon dioxide was observed (IPCS, 1994). These experiments indicate that HCBD can undergo quite rapid photolysis when adsorbed on particulate matter.

Experimentally, a half-life of one week was determined when HCBD was exposed to air in flasks outdoors. This relatively short disappearance time is possibly due to heterogeneous reactions on the vessel walls, as suggested by the authors of the report. Hydrogen chloride was found to be the main degradation product after exposure of samples to xenon arc radiations (wavelength > 290 nm; Pearson & McConnell, 1975). However, the authors were using such unrealistically high substrate concentrations (much higher than those present in the real atmosphere) that the degradation became “autocatalytic” due to the formation of Cl atoms.

Like the structurally similar chemical, perchloroethylene, HCBD is expected to react with hydroxyl radicals and to a much lesser extent with ozone *via* addition to double bonds (Atkinson and Carter, 1984; Atkinson, 1987). Estimates of its half-life in air based on photochemical degradation through reactions with hydroxyl radicals range from 60 days (ATSDR, 1994) to three years (Howard *et al.*, 1991). The Atmospheric Oxidation Program of the EPIWIN software suite estimates a half-life of about one year with respect to reaction with OH radicals and about 450 years for reaction with ozone.

By using a steady-state mathematical model for the troposphere and on the basis of gas chromatographic analysis of air samples from sites far away from anthropogenic sources, Class and Ballschmiter (1987) calculated that HCBD would have a tropospheric half-life of 840 days (2.3 years) in the Northern hemisphere and 290 days (0.8 years) in the southern hemisphere, based on a hydroxyl radical rate constant of 2 □□10^{-14} cm³/molecule per second and a hydroxyl radical concentration of 7 □□10^{5} molecules/cm³ in the north and 17□10^{5} molecules/cm³ in the south. Another mass-balance calculation based on monitoring data suggests that the half-life of atmospheric HCBD is about 1.6 years in the Northern hemisphere (HSDB, 2000).

Fricke *et al.* (1995) estimated a theoretical reaction rate constant between HCBD and OH radicals of 2 x 10^{-14} s^{-1}. Via a QSAR approach, the estimated half-life in air is 356 days based on 12-hour day light and an OH concentration of 1.5□10^{6} OH/cm^{3}.

Modelling and monitoring data suggest that the release rate of HCBD to the atmosphere in the Northern hemisphere was approximately 3,200 tons/year in 1985 (Class & Ballschmiter, 1987). This was mainly due to the use of HCBD in agricultural applications during that period. Based on the “background” concentrations recently observed in Sweden (Swedish EPA, 2001), the same model indicates that the atmospheric burden has drastically dropped to about 10 ton/yr in 2000. Dispersion of HCBD in the atmosphere has been confirmed by detection of HCBD at locations distant from emission sources (IPCS, 1994).

Whilst these data indicate that HCBD meets the criteria for persistence in air (half-life □2 days) used by most regulatory bodies (Environment Canada, 1999; UNEP, 2001), reaction with hydroxyl radicals is likely to be an important removal process and sink for HCBD in the troposphere (HSDB, 2000).

Worst-case calculations were made by UNEP (2000) to determine if HCBD has the potential to contribute to ***depletion of stratospheric ozone, ground-level ozone formation or climate change***. The Ozone Depletion Potential (ODP) was calculated to be

0.07 (with CFC11 = 1), the Photochemical Ozone Creation Potential (POCP) was estimated to be 0.01 (with ethylene = 100) and the Global warning Potential (GWP) was calculated to be 0.037 (with CFC11 = 1). These figures imply that HCBD is not likely to contribute significantly to ground-level ozone formation, but it does have the potential to contribute to depletion of stratospheric ozone and to climate change. Some substances currently subject to the Montreal Protocol have ODP values similar to the one calculated for HCBD, however, there is general agreement that at these ODP values, substances should not be automatically subject to control. Other criteria such as quantities emitted have to be part of the decision making.

In conclusion, HCBD persists in air until it reacts with OH radicals, is photochemically degraded or is deposited to water or soil when adsorbed on particulate matter. The main degradation process is the reaction with OH radicals leading to a half-life in air of about one to two years.

5.3 Behaviour in water

HCBD is released to surface- and ground-water via industrial effluents, by leaching from landfills or soil, or by urban runoff (ATSDR, 1994).

HCBD is highly resistant to hydrolysis in the absence of appropriate solvents, although it is readily degraded by ethanolic alkali (Roedig & Bernemann, 1956). The measured hydrolysis rate of HCBD in a 1:1 acetone-water mixture gives a half-life of over 1800 hours (Hermens *et al.*, 1985).

In a review on biodegradation of organic compounds, Van Agteren *et al*. (1998) conclude that HCBD is a recalcitrant substance under aerobic conditions while under anaerobic conditions, reductive dechlorination has been observed. No half-lives are presented. This is in line with the general observation that a higher degree of chlorination will favour anaerobic degradation (Beurkens, 1995).

These results are, however, contradicted by a study of Tabak *et al.* (1981), who found that solutions of 5 to 10 mg HCBD/l completely disappear within seven days of exposure in a flask test with wastewater microbiota under aerobic conditions. Whilst volatilisation losses were found to be minimal, these tests did not give definitive results because their designs could not easily differentiate removal or degradation *via* abiotic processes (e.g., adsorption, hydrolysis) from that *via* biodegradation. These results suggest, however, that HCBD would biodegrade at a slow-to-moderate rate in aqueous environments. A study by Schröder (1987) gives the same indication, because approximately 70% adsorption to sludge and 8% degradation was found to occur within eight days in a pilot low-loaded biological sewage treatment plant.

The half-life of HCBD in water appears to depend on the amount of organic matter in the aqueous media; in natural waters, the half-life is estimated to be 4–52 weeks (Howard *et al.*, 1991). The US EPA (2002) reports a half-life in water of 30 days. On the basis of data for Dutch surface waters, the disappearance time of HCBD is estimated to be 3-30 days in rivers and 30-300 days in lakes and groundwater. In this field experiment, it is not possible to differentiate removal from degradation. However, it seems that turbulence, and therefore increased aerobic biodegradation, volatilisation and adsorption, account for the shorter half-lives in river water (Zoeteman *et al.*, 1980).

Anaerobic degradation of HCBD at 100 mg/litre was not observed in 48 hour batch assays at 37°C using an inoculum from a laboratory digester (Johnson and& Young, 1983), indicating that the degradation is very slow even under anaerobic conditions (Govind *et al.*, 1991).

Calculations with the Syracuse BIOWIN model indicate that HCBD does not biodegrade rapidly (linear and non-linear model): the primary biodegradation takes “weeks” and HCBD is recalcitrant to ultimate biodegradation. Based on the structure of HCBD it can be expected that a dechlorination step is necessary before aerobic biodegradation can occur.

In conclusion, the information available on persistence of HCBD in water is inconsistent. Several data suggest that HCBD may biodegrade in natural waters containing organic

matter, in particular if there is high turbulence. It is not clear if the degradation is aerobic, anaerobic or both. The reported half-lives in water vary from one month to one year. It seems that a reductive dechlorination step is needed before aerobic degradation can occur, but that this step is also relatively slow.

5.4 Behaviour in sediment

Sediments can be considered as a sink for HCBD released to water because the compound is strongly adsorbed onto sediments with high organic content and, according to Environment Canada (2000), is not likely to persist. However, measured values for half-life in sediment are not available.

According to Stupp and Paus (1999), the degradation of polychlorinated organic substances could occur *via* two main mechanisms, aerobic and anaerobic. This is also true for perchlorinated substances such as HCBD. The contaminant can be used directly as energy source by micro-organisms, or be transformed as a co-metabolite in an enzymatic process (Scholz-Muramatsu & Flemming, 1991), leading to more rapid degradation in the presence of organic matter. However, in the study on column experiments packed with river Rhine sediments, Bosma *et al*. (1994) found no biodegradation under aerobic conditions within a period of 3 years. This is explained by stronger adsorption in aerobic conditions and thus lower bioavailability.

However, in the same type of column experiments, removal of HCBD was observed under anaerobic conditions after an acclimation time of four months (Bosma *et al.*, 1994). The main reaction product was 1,2,3,4-tetrachloro-1,3-butadiene (>90%). This substance may be further degraded aerobically. Similarly, sequential reductive dechlorination of HCBD was achieved by a culture enriched from contaminated estuarine sediment under anaerobic conditions (Booker and Pavlostathis, 2000). The predominant HCBD dechlorination products were isomers of tri- and dichloro-1,3-butadiene. Traces of a monochloro-1,3-butadiene isomer were also detected. Extensive dechlorination of HCBD was achieved, the dechlorination rates being enhanced at higher initial HCBD levels (1.5 *versus* 0.4 mg HCBD/l).

A desorption study of HCBD from contaminated sediments (Prytula and Pavlostathis, 1996b) indicated that the extent and rate of desorption was strongly correlated to the organic carbon-based partition coefficient of the contaminants. A significant fraction of the solid-phase contaminant concentration was found to be non-labile and not bioavailable. This leads to long-term persistence of these compounds, including HCBD, in natural contaminated sediments. It appears that desorption could be the rate-determining step in sediment remediation technologies

Naturally occurring micro-organisms mediate the reductive dechlorination of sediment-bound chlorinated organic compounds in laboratory experiments (Prytula, and Pavlostathis, 1996a) However, because of the strong contaminant adsorption, a low level of biotransformation was obtained. On the other hand, microbial reductive dechlorination of the sediment-bound contaminants leads to the long-term release of less chlorinated and more mobile compounds. Addition of bioavailable organic matter during the test resulted in rapid dehalogenation of available contaminant. This is in line with the enhanced degradation rate in water when organic matter is present and with the general observation that the higher the level of chlorination, the higher the probability of anaerobic degradation (Beurkens, 1995).

In conclusion, information available on persistence of HCBD in sediment shows that removal through biodegradation occurs at a slow rate. It is suggested that the first step of HCBD degradation in sediment containing organic matter is reductive biodegradation. A combined anaerobic-aerobic degradation process seems then to occur. The observed persistence is mainly due to strong adsorption to the sediment and reduced bioavailability, desorption being the rate-determining step. This explains the difficulty of defining a half-life.

5.5 Behaviour in soil

HCBD can be released to soil by disposal of industrial waste in landfill operations (ATSDR, 1994). Volatilisation from soil surfaces is thought to be a primary process for loss of HCBD

from soil (Tabak *et al*., 1981). However, as HCBD readily adsorbs to soil organic particles, volatilisation from highly organic soils is predicted to be low (HSDB, 2000).

The half-life of HCBD in soil depends upon the chemical, physical and biological heterogeneity of the soil and climatic conditions. Environment Canada (2000) reports half-lives in soils of 4-26 weeks referring to a study by Howard *et al.* (1991) on aerobic biodegradation rates. The US EPA (2002) reports that HCBD readily breaks down in soil.

Howard *et al*. (1991) suggested that HCBD may not biodegrade in anaerobic zones of soil and that evaporation would be a significant transport mechanism from soil surfaces. In a dune infiltration study in the Netherlands (Piet, 1980), HCBD was found to be mobile in sandy soils, with an average residence time of 100 days and little biodegradation.

Fragiadakis *et al.* (1979) examined residues of radio-labelled HCBD in soil–plant systems and observed that 4% of the original radioactivity was bound in non-extractable residues in the top 50 cm of soil after two years, suggesting potential for long-term accumulation. The remaining 96% of the original radioactivity was unaccounted for and was believed to have volatilised.

Soil organic matter content is likely to be an important factor in biodegradation time, since adsorption of HCBD to organic matter will significantly decrease its bioavailability to micro-organisms. In the absence of significant biodegradation or other loss processes, persistence of HCBD in soil may allow migration of the compound into groundwater, particularly in sandy soils (US EPA, 1984).

In conclusion, it is difficult to predict a half-life in soil due to the strong adsorption of HCBD. As observed for sediment, the presence of organic matter favours a reductive biodegradation but also reduces bioavailability.

5.6 Behaviour in biota

Considering the low water solubility of 3.2 mg/litre and the high log K_{ow} of 4.78, a strong bio-concentration potential would be expected. Both laboratory and field data support this prediction. In flow-through laboratory tests with algae, crustaceans, molluscs and fish in fresh or marine waters, bio-concentration factors (on a wet weight basis) were between 71 and 17,000. This wide range can be explained in part by species differences in metabolism or differences in exposure concentrations (ATSDR, 1994).

The results also appear to be highly dependent on the exposure period, with a steady state was clearly demonstrated in only one of these tests (Leeuwangh *et al.*, 1975; Pearson & McConnell, 1975; Laseter *et al.*, 1976; Oliver & Niimi, 1983). Oliver & Niimi (1983) exposed rainbow trout *(Salmo gairdnerii)* to aqueous solutions of HCBD at 0.10 and 3.4 ng/litre and found average bio-concentration factors of 5,800 and 17,000, steady states having been reached after 69 and seven days, respectively. Laseter *et al.* (1976) reported, however, that the accumulation of HCBD in mollies (*Poecilia latipinna*) and the largemouth bass (*Micropterus salmoides*), kept for 10 days in water containing concentrations of HCBD (10 to 59 µg/l), although variable, was fairly low with concentrations normally below 50 µg/l. A study by Pearson and McConnell (1975) to measure accumulation of HCBD in plaice (*Pleuronectes platessa*) and dab (*Limanda limanda*) kept for up to three months in water containing HCBD (1.7 µg/l) reported concentration factors of about 500 to 700 for muscle and 7,000 to 10,000 for liver.

The bioaccumulation factors found in plankton, crustaceans, molluscs and fish in surface waters are comparable to those observed in the laboratory: available bioaccumulation factors based on wet weight vary between 33 and 11,700 (Goldbach *et al.*,1976; Laseter *et al.*, 1976).

When oligochaete worms were exposed *via* spiked Lake Ontario sediments to a pore water concentration of 32 ng/litre in a flow-through system, steady state was reached within four to 11 days and the average bioconcentration factor was 29,000, based on dry weight, of which about 8% is lipid (Oliver, 1987).

A seven-day laboratory experiment with green alga (*Oedogonium cardiacum*) showed that the algae accumulated HCBD to a concentration approximately 160-fold greater than that in the surrounding water (Laseter *et al.*, 1976).

HCBD also bio-concentrates in aquatic invertebrates, but to a somewhat lesser degree than in fish, with a maximum reported BCF of 2000 for the mussel, *Mytilus edulis* (Pearson & McConnell, 1975). Contamination of water by HCBD led to uptake of the substance by caged mussels in the St Clair River (Kauss and Hamdy, 1985; OME/MDNR, 1991).

The bioconcentration of HCBD by crayfish (*Procambarus clarki*) was investigated by Laseter *et al.* (1976) using both laboratory and field studies. In the laboratory investigations, with 10 days' exposure at 2 to 4 µg/l concentrations, mean concentration factors between 12 and 59 were observed. In field studies, crayfish were kept for 17 days in pond water containing 4.6 µg/l HCBD and the concentration factors varied between 7 and 300.

In a recovery study (Laseter *et al.*, 1976), crayfish kept in non-contaminated water for 12 days were found to have lost about 95% of the HCBD from their tissues. HCBD was eliminated from the tissues of goldfish (*Carassius auratus*) with a half-life of 6.3 days (Leeuwangh *et al.*, 1975). Bioconcentration factors (BCFs) were much greater at higher exposure levels than at lower concentrations, so the rates of detoxification and elimination by fish should be considered as concentration-dependent.

Limited accumulation of HCBD was observed in the fat of rats when they received oral doses of HCBD as part of a mixture of seven chlorinated hydrocarbons for 4 to 12 weeks (each compound was administered at 2 or 4 mg/kg body weight per day). Fat concentrations of up to 8 mg/kg were observed at the higher dose rates (Jacobs *et al.*, 1974). 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.

HCBD does not appear to bio-accumulate in plants. In a field study with radio-labelled HCBD, no significant degree of accumulation occurred in roots, leaves or stems of potato or carrot plants (Fragiadakis *et al.*, 1979).

Biomagnification, the tendency of a substance to concentrate within a food chain, was not observed for HCBD in laboratory tests or in field experiments (IPCS, 1994). No biomagnification was seen when levels in fish were compared with those in detritus and several invertebrates (Goldbach *et al.*, 1976). Similarly, levels of HCBD in predatory fish such as pike and perch were found to be lower than concentrations in the prey fish (Goldbach *et al.,* 1976). The authors also found no correlation between age and HCBD residues. Based on these findings, they concluded that there is no significant biomagnification at higher trophic levels. This conclusion was confirmed by a trophodynamic analysis in the Lake Ontario ecosystem (Oliver & Niimi, 1988), supported by Pearson and McConnell (1975) and by the measurements made by Muir (2003a) in an aquatic food chain and described in section 4.5.

According to Environment Canada (1983), although HCBD accumulates in the tissues of freshwater aquatic invertebrates and fish, it does not biomagnify within food chains because of its fast depuration rate in fish (Goldbach *et al.*, 1976) and its rapid metabolism and excretion in mammals and fish-eating birds (IPCS, 1994).

Concentrations of HCBD in aquatic organisms, birds and mammals indicate bioaccumulation but no bio-magnification (IPCS 1994; Environment Canada, 2000).

6. Temporal trends

Clear downward trends in atmospheric HCBD concentration can be inferred by comparing the values of 1,200-4,600 pg/m³ observed in 1985 by Class and Ballschmiter (1987) in the Northern hemisphere to the background concentration of 2-5 pg/m³ measured in Sweden in 1999-2000 (Swedish EPA, 2001). There is thus an apparent reduction in atmospheric HCBD concentration of more than 99%, indicating a dramatic decrease in global emissions.

Whilst there are few historical data on HCBD, by combining data published by Durham and Oliver (1983) and Williams *et al*. (2000 and 2001), Bailey successfully described temporal trends in Niagara sediment and suspended solids (see *Figure 4*) during the 20th century. The highest levels are observed between 1960 and 1970.

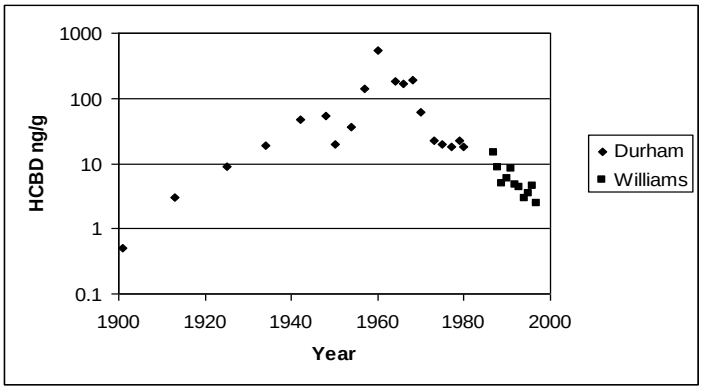

Figure 4: Concentration of HCBD in Niagara sediments and suspended solids during the 20th century (Bailey, 2003)

According to Muir (2003a), similar trends (see *Fig.5),* but at lower concentrations, have been observed in sediment cores from the western basin of Lake Ontario. This shows maximum HCBD levels in the late 1970s. The historical profile is similar to the PCB one but with concentration levels several hundred times lower (Muir, 2003a).

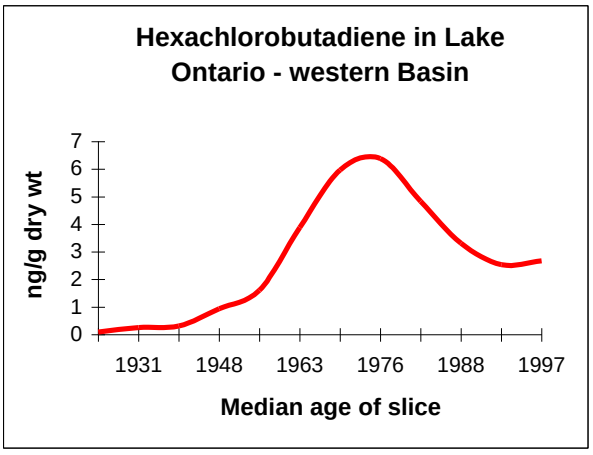

Figure 5: HCBD concentration in core sediment of Lake Ontario Western Basin (Muir, 2003a)

It seems that there is no data on temporal trends in HCBD concentrations in biota. However, archives of fish and wildlife samples exist in Canada, which could be further analysed if necessary (Muir, 2003b).

7. Environmental toxicity

The purpose of this study is not to discuss in detail the results of all available toxicity tests, but to present an overview of the key ecotoxicological studies as an indication of possible effects of HCBD on the environment, and corresponding threshold levels below which these effects are not likely to appear. This will be based on several review papers (IPCS, 1994; Environment Canada, 2000; US EPA, 2002; Euro Chlor, 2002).

7.1 Pelagic organisms

7.1.1 Acute studies

For ***marine fish***, the valid[1] study with *Limanda limanda* gives a 96h LC_{50} of 0.45 mg/l which is the lowest toxicity value for marine fish (Pearson & McConnell, 1975).

For ***freshwater fish*** species, three flow-through studies based on measured concentrations are considered to be valid; 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). The other studies produced similar or higher LC_{50} values (Leeuwangh *et al*., 1975; Euro Chlor, 2002). The lowest acute toxicity value for freshwater fish is 0.09 mg/l (Environment Canada, 2000).

For ***marine invertebrates***, the lowest valid acute toxicity value was obtained for *Elminius modestus* with a 48h LC_{50} of 0.87 mg/l (Pearson & McConnell, 1975).

For ***freshwater invertebrates***, the lowest acute toxicity is a 96h LC_{50} to *Asellus aquaticus* of 0.13 mg/l (Leeuwangh *et al*., 1975; Environment Canada, 2000).

Studies reported with ***freshwater algae*** (Bringmann & Kuehn, 1977; Knie *et al*., 1983) are considered as non-valid, but their results are sufficient to indicate that algae are not the most sensitive trophic group for HCBD.

7.1.2 Long term studies

The lowest no observed effect concentration (NOEC) reported (0.003 mg/l, Leeuwangh *et al*., 1975) was considered not valid for assessing the risk because the biochemical endpoint (liver enzyme activity of C. auratus) represents a response to the substance but not necessarily an adverse effect. The NOEC from an early life stage test with P. promelas (Benoit *et al*., 1982) is considered valid without restriction and used a flow-through system with analysis of the test solutions.

The 28 day 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.

HCBD preferentially accumulates in liver of fish (Pearson & McConnell, 1975). Once in the liver, it can be biotransformed into polar metabolites that will reach the kidneys *via* the bile and could become nephrotoxic in fish (Anders and Jakobson, 1985; Yang, 1988; IPCS, 1994).

In most cases, freshwater fish and marine crustaceans are more sensitive than their marine and freshwater counterparts, respectively (Environment Canada, 2000). No chronic toxicity data were identified for aquatic invertebrates.

It is interesting to note that both the Estimated No Effect Value (ENEV) as proposed by Environment Canada (2000) and the Predicted No Effect Concentration (PNEC) as derived

[1] The validity check is based on criteria described in EU-TGD 1996

by Euro Chlor (2002) following the EU Technical Guidance Document approach (EU-TGD, 1996) are exactly the same i.e. 0.13 µg/l for pelagic organisms. This value is close to the proposed water quality objective of 0.1 µg/l proposed by CSTEE (1994) and IPCS (1994), using the same set of data.

7.2 Benthic organisms

No acute or chronic toxicity studies on benthic organisms have been identified for HCBD. In the absence of such data, the water-sediment equilibrium partitioning approach is sometimes used, as surrogate, to estimate a level which is likely to be safe to benthic organisms. The principle behind this approach is the observation that sediment organic carbon is the main factor influencing partitioning of non-polar organic compounds into sediments (Di Toro *et al.*, 1991). Considering a mean carbon content of 2% and a Koc value of 10 000 (Euro Chlor, 2002) or 80,000 l/kg (Environment Canada, 2000), a PNEC value of 24 µg/kg dry weight and an Estimated No Effect Value (ENEV) of 210 µg/kg dry weight for benthic organisms, respectively, have been derived. The difference between the two figures depends on the assumed Koc value. However, without experimental data it is not possible to draw any definitive conclusions on the toxicity level of HCBD to benthic organisms.

7.3 Predators

As food can be a significant source of exposure for predators to a substance such as HCBD, with a low water solubility and high lipid solubility, it is important to address the possible adverse effects of HCBD in predatory animals higher up the food chain, feeding on fish, and the corresponding thresholds at which no effect is seen.

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

For mammals, the main target organs for toxicity of HCBD are the liver and the kidneys.

Environment Canada (2000) concludes that HCBD is moderately *acutely* toxic, with LD50s of 65-116 mg/kg body weight in mice, 200-580 mg/kg body weight in rats and 90 mg/kg body weight in guinea pigs (Murzakaev, 1963; Gulko *et al.*, 1964; Gradiski *et al.*, 1975; Kociba *et al.*, 1977a, 1977b). In the only *short-term* study identified in which animals were exposed to HCBD by inhalation, renal proximal tubular degeneration and adrenal cortical degeneration were noted in rats exposed to 25 ppm (267 mg/m^3) HCBD and above for up to 15 days. Renal toxicity was not observed at lower concentrations (5 ppm [53 mg/m^3] or 10 ppm [107 mg/m^3]; Gage, 1970).

On the basis of subchronic and chronic (two-year) toxicity studies in rats and mice, IPCS (1994) concluded that the NOAEL is 0.2 mg/kg body weight per day. Similarly, Environment Canada (2000) indicates that the No-Observed-Effect Level (NOEL) and the Lowest-Observed-Effect Level (LOEL) for chronic effects on the kidney are considered to be 0.2 and 2.0 mg/kg body weight per day, respectively.

Studies to investigate reproductive effects in rats have reported reduced birth weight and neonatal weight gain but only at doses producing maternal toxicity, i.e. 20 mg/kg body weight. Chronic oral administration of up to 20 mg HCBD/kg-bw per day did not induce histopathological changes in the testes or ovaries or effects on oestrous cycle or sperm parameters in mice or rats (Kociba *et al*., 1977a; NTP, 1991). 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.

Although the results of available studies are not completely consistent, there is some limited evidence that HCBD could be *genotoxic* under certain conditions (Environment Canada, 2000). The results of early standard Ames tests were negative (De Meester *et al.*, 1980; Stott *et al.*, 1981; Haworth *et al.*, 1983; Reichert *et al.*, 1983), but HCBD was observed to induce gene mutations in *Salmonella typhimurium* (Reichert *et al.*, 1984).

Despite the observation of these mutations in *Salmonella,* it has not been firmly established whether the initial step in kidney tumour formation is a result of genetic damage or epigenetic events, possibly in the mitochondria (Stott *et al.*, 1981; Schrenk and Dekant, 1989; Dekant *et al.*, 1990; Henschler and Dekant, 1990).

The limited available results in rodents do not indicate that *neurological* effects or effects on the *immune system* are critical endpoints associated with exposure to HCBD.

In conclusion, on the basis of available data, an oral intake of 0.2 mg/kg body weight per day is considered by Euro Chlor (2002) as the NOAEL for chronic effects in birds and mammals, and an oral intake of 20 mg/kg body weight per day is considered as the NOAEL for reproductive toxicity.

8. Risk of HCBD to the environment and human health

In this section, the outcome of three main studies for assessing the risk of HCBD to the environment and human health are summarised (US EPA 1998a; Environment Canada, 2000; and Euro Chlor, 2002). Interestingly, the results of these three studies are very similar and consistent.

Basically, a risk assessment consists of comparing the no-effect levels for various environmental compartments derived from toxicological data (see section 7) with estimated exposure levels, for example, derived from analytical monitoring programs (see section 4). If the exposure levels are lower than the no-effect levels, the risks are considered to be low or negligible. If the exposure levels exceed the no-effect levels, risk reduction measures may be necessary.

As HCBD has a fairly low water solubility and a relatively high octanol/water partition coefficient, consideration should also be given to potential risks of HCBD *via* bioconcentration in living organisms, which could produce toxic effects in predators, and *via* partition into sediments, which could produce toxicity in sediment-dwelling organisms.

For ***pelagic organisms***, the estimated no-effect value (ENEV) proposed by Environment Canada (2000) and the predicted no-effect concentration (PNEC) reported by Euro Chlor are both 130 ng/l. On the basis of more recent monitoring data, typical environmental concentrations in the Northern hemisphere are in the range of 1 to 12 ng/l. This indicates that the levels of HCBD in surface waters are unlikely to pose a risk to organisms living in fresh and marine waters of the Northern hemisphere for which data are available. The risks are even lower for freshwater invertebrates, since they appear to be somewhat less sensitive to HCBD than fish (Environment Canada 2000).

The critical body burden (CBB), which predicts the level of HCBD that can be present within an organism's tissues without causing a toxic effect, is the product of the no observed effect concentration (NOEC) and the bioconcentration factor (BCF) of any specific organism. It has been used by Euro Chlor (2002) to assess the risk of toxicity due to bioconcentration in fish. As reported, BCF values range from 70 to 17,000 l/kg (see section 5.6), the lowest NOEC value for fish is 6.5 µg/l (see section 7.1), and the calculated CBB varies from 450 to 111,000 µg/kg wet weight. As concentrations of HCBD measured in fish (see section 4) generally remain well below 100 µg/kg wet weight, with typical values ranging from 0.01- 5 µg/kg wet weight, it appears that the actual concentrations of HCBD in fish are well below the critical body burden associated with toxic effects. It is therefore concluded that risks to fish through bioconcentration are unlikely, at least in Northern hemisphere where monitoring data are available (Euro Chlor 2002).

For ***benthic organisms***, in the absence of experimental toxicological data, the no-effect level is estimated to be 24 µg/kg dry weight or 210 µg/kg dry weight, depending on the Koc value used in the partitioning approach (see section 7.2). The majority of recent available sediment monitoring data on HCBD (see section 4) indicates levels of 0.1 to 10 µg/kg dry weight. This leads to a safety factor varying between 2.4 and 21, indicating, according to Euro Chlor (2002), that unacceptable risks of HCBD to sediment organisms are unlikely.

It should, however, be pointed out that several highly contaminated sites have been identified in Canada (St Clair River) and in the US (Baton Rouge area) where HCBD concentrations have been reported at between 10 and 300 mg/kg. In these conditions, sensitive benthic organisms could experience adverse effects. Remediation programmes are considered for these "hot spots" (Environment Canada, 2000).

For ***fish-eating predators***, whilst 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 (Euro Chlor, 2002). One approach is to estimate the daily intake (DI) associated with fish-eating and to compare it with the lowest no-effect level for predatory species (as given in section 7), i.e. 0.2 mg/kg body weight/day for chronic toxicity. The DI was

calculated by multiplying the typical observed concentration in fish (0.01- 5 µg/kg wet weight) with the feeding rate of predators, estimated at 0.15 and 0.11 kg/kg body weight for the mink and eagle respectively (US EPA, 1992). This leads to a maximum daily intake between 0.55 and 0.75 µg HCBD/kg body weight per day, several orders of magnitude below the no-effect level for chronic toxicity.

Based on these data, it is concluded that there is little risk of toxicological effects for predatory species eating fish contaminated with HCBD. Overall these data support the conclusion that the risk of bioaccumulation and secondary poisoning for HCBD is low at present concentrations in Northern hemisphere.

For ***humans***, a NOAEL of 0.05 mg/kg body weight per day has been derived by IPCS (1994), based on the assumption that a margin of safety of 150 between the estimated NOAEL in humans and the maximum total daily intake is sufficient to protect the general population against the adverse effects of HCBD. This report also concluded, based on a IARC study (1979), that there is limited evidence HCBD carcinogenicity in animals (one study in one rodent strain) and insufficient evidence in humans.

According to Environment Canada (2000), food and possibly air appear to be the major routes of population exposure to HCBD. Based on exposure for the various age groups derived from limited available monitoring data, total daily intakes of HCBD from air, food and drinking water have been estimated at 0.01 to 0.2 µg/kg body weight per day. This estimated average daily intake from environmental sources is less than a Tolerable Intake[2] (TI) of 0.34 µg/kg body weight per day calculated from the NOAEL for humans. Probabilistic estimates of exposure via inhalation of ambient air give an intake for children of 0.09µg/kg - bw per day. It is concluded that HCBD is not present in the environment in quantities or under conditions that may constitute a danger to human life or health.

The magnitude of intake from fish consumption when HCBD is present in fish tissue is estimated by US EPA (1998a) to be 0.15 ng/kg/day for adults and 0.24 ng/kg/day for children. The corresponding intakes from drinking water are 8.6 and 30 ng/kg/day, respectively. Intakes by inhalation from ambient air are much higher: they are estimated to be 120 and 630 ng/kg/day for adults and children, respectively. As noted by US EPA (1998a), these estimates may be indicative of the magnitude of HCBD intake from air in urban and source-dominated areas where the chemical is present. It is likely that there would typically be no chronic exposure to HCBD *via* non-fish foods (US EPA, 1998a). The data indicates that most intake of HCBD by the general population occurs *via* inhalation. It should be noted, however, that the concentration data used to estimate respiratory exposure are old and that the releases have been greatly reduced in the meantime.

The Reference Dose (RfD)[3] for HCBD and the dose that corresponds to a lifetime excess cancer risk of $1x10^{-6}$ is 0.2 µg/kg/day (US EPA, 1998a). Under these conditions, it is concluded that it is unlikely that HCBD will occur with a frequency or at concentrations that are of concern for public health.

[2] A Tolerable Intake is the level of intake to which it is believed a person may be exposed daily over a lifetime without deleterious effect.

[3] The RfD is “an estimate (with uncertainty spanning approximately an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects over a lifetime” (U.S. EPA, 1993).

9. Conclusions

The main conclusions that can be drawn from this review are:

- HCBD should be considered as toxic, liable to bio-accumulate and prone to long range transport *via* air. However, concentrations of HCBD in aquatic organisms, birds and mammals indicate bioaccumulation but no biomagnification within the food chain (IPCS 1994; Environment Canada, 2000).

- There are still some doubts about persistence of HCBD in the environment. HCBD persists in ***air*** until it either reacts with OH radicals, or is photochemically degraded or deposited in water or soil when adsorbed on particulate matter. The main degradation process is the reaction with OH radicals, leading to a half-life in air of about one to two years.
    - On persistence in ***water***, there are conflicting results but several data suggest that HCBD may biodegrade in natural waters containing organic matter, in particular if there is high turbulence. It is not clear whether the degradation is aerobic, anaerobic or both. The proposed half-lives in water vary from one month to one year. It seems that an initial reductive dechlorination step is needed before aerobic degradation can occur.
    - Similarly, it is suggested that reductive biodegradation of HCBD may occur in ***sediment*** containing organic matter. A combined anaerobic-aerobic degradation pathway is likely. The observed persistence is mainly due to strong adsorption to the sediment and reduced bioavailability. This explains the difficulty of defining a half-life.
    - It is difficult to predict a half-life in ***soil*** due to the lack of data and strong adsorption of HCBD. As observed for sediment, the presence of organic matter favours a reductive biodegradation but also reduced the bioavailability.
- In the UNECE region, where HCBD is no longer produced commercially and remaining uses are insignificant, there are very limited releases into the environment. HCBD is still an unintentional by-product of the joint manufacture of perchloroethylene and carbon tetrachloride but releases are now well controlled by applying recycling processes and high temperature incineration technology to reduce landfill deposition. In Europe, this has resulted in releases of HCBD to air and water of about 25 kg in 2001, representing a decline of more than 99% compared to 1985. Both modelling and monitoring data indicate a drastic decrease (more than 99%) in Northern hemisphere emission levels over the past 15 years to an estimated 10 tonnes/year. Other sources of HCBD are minor: they include possible releases from old landfill leachates and releases during refuse combustion.
- Although the last AMAP report (2000) did not mention HCBD, it has been detected in Europe and North America in air, water, sediment, soils and biota. As described in section 4, the most recent observed levels are low and indicative of a strong decrease in environmental concentrations. Several heavily contaminated areas have been identified. These should still be considered as “hot spots”, but are well managed through local remediation programmes and do not require additional, global regulations. Contaminated solid waste arising from old chlorine electrolysis cells can also be handled by applying existing local or regional regulations.
- Multimedia models show that HCBD tends to remain in the environmental compartment into which it is released. Little inter-compartmental transport occurs when HCBD is discharged to air or soil, but, in contrast, disposal to water has potential for significant transport of HCBD to the air *via* volatilisation and to sediment *via* adsorption on particulate matter. In view of its strong adsorption potential to organic matter, the compound accumulates in sediment and does not migrate rapidly in soils. This is confirmed by monitoring approaches.

The predicted no-effect concentration (PNEC) based on the EU Technical Guidance Document (1996) or the estimated no-effect values (ENEV) calculated according to the Canadian scheme (Environment Canada, 2000), has been determined to be 0.13 µg/l in water for pelagic organisms. As there is a lack of sediment toxicity data, the equilibrium

partitioning method has been used to estimate the no-effect level for benthic organisms and values of 24 or 210 µg/kg dry weight in sediment have been calculated depending on the K_{oc} value chosen (10,000 or 80,000 l/kg). In most cases, freshwater fish and marine crustaceans are more sensitive than their marine and freshwater counterparts, respectively. Toxicity data are still missing for sediment-dwelling organisms.
On the basis of available data, an oral intake of 0.2 mg/kg body weight per day is considered as the NOAEL for chronic effects in birds and mammals. This leads to a tolerable intake (TI) of 0.2 µg/kg body weight for top predators, including humans.

- Outside of "hot spot" areas, current levels of HCBD in the Northern hemisphere are unlikely to pose a risk to organisms living in fresh and marine waters or sediments or to fish-eating birds and mammals. This conclusion is based on a comparison of observed environmental concentrations in water, sediment and biota with no-effect concentrations (IPCS, 1994; Environment Canada, 2000; Taylor *et al.*, 2003). As environmental concentrations of HCBD continue to decline, so will any residual risk.
- WHO (1994), US EPA (2002) and Environment Canada (2000) also concluded that HCBD is not present in the environment in quantities or under conditions that may constitute a danger to human life or health, including potential carcinogenic effect.

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