Pollution from Hindustan Insecticides Ltd and other factories in Kerala, India: a follow-up study

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Technical note 05/03
December 2003

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1 EXECUTIVE SUMMARY

The Udyogmandal Industrial Estate is situated along the Periyar River in Kerala, on and near the island of Eloor. Among the factories on the island is Hindustan Insecticides Ltd, a Government of India enterprise that manufactures insecticides, including DDT. DDT was introduced in the 1940s and although its environmentally harmful properties became apparent during the 1950s and 1960s, it remains in use against insects carrying malaria and similar diseases in over 20 countries. However, in 2001, the Stockholm Convention, also known as the POPs Convention, required the global phase-out of DDT. The World Health Organisation is committed to working with countries such as India who still use DDT and who need to develop plans to protect public health adequately without it.

In 1999, Greenpeace conducted a survey of the pollution associated with the Hindustan Insecticides Ltd (HIL) factory and documented severe contamination of nearby creeks with organochlorine pollutants being discharged by HIL.

In 2002, a further set of samples was collected to see if there had been any improvement in the environmental situation and investigate how far through the creek system the pollution was travelling. Samples were analysed at the Greenpeace Research Laboratories at the University of Exeter, UK.

Analysis of samples by gas chromatography/mass spectrometry identified organochlorine and hydrocarbon compounds in all of the sediment samples. Only one, from a quiet offshoot called Panachi Thodu, did not contain any of the main components of DDT, though it did contain traces of several chemicals from the broader DDT group. Otherwise, sediments contained between 20 and 37 identifiable organochlorines as well as numerous hydrocarbons. A gradient of organochlorine pollution was seen from the plant to the south of the island, where the contamination enters the River Periyar.

Persistent organic pollutants such as DDT and related compounds are of particular environmental concern because not only are they toxic but they are also highly resistant to degradation and are liable to bioaccumulate. A number also biomagnify, meaning that they increase in concentration as they pass up the food chain, putting top predators and humans at particular risk.

In addition to the analysis to screen for organic pollution, concentrations of metallic elements were also determined in the samples collected. Panachi Thodu sediment was again the least polluted, because of the low flow rate. In the remainder of the sediments, metals were substantially elevated, particularly with mercury, copper and zinc. Lead, chromium and cadmium pollution was also present. All these metals are also entering the Periyar River via the creeks.

HIL, which handles predominantly organic chemicals, could not be the source of such severe metals pollution. However, at least three of the neighbouring plants will be discharging some metallic elements. Merchem, which makes and uses zinc-based compounds, is one such. There are also two government-operated plants, Indian Rare Earths (IRE) and Fertilisers and Chemicals Travancore (FACT). The pattern of metal contamination is most likely to be derived from phosphate rock, the major feedstock for phosphate fertilisers, pointing to FACT as the source of most of the metals pollution.

Moreover, a sediment sample collected in 1999 from the creek exiting the FACT premises contained metal pollution similar in pattern and extent to that of the most contaminated samples

from 2002. The gradient of pollution, however, did not correlate with distance away from FACT. Instead, the two sediment samples closest to the confluence of the creeks with the river were the most heavily contaminated. This phenomenon could be explained if the same type of metals pollution has been released into the creek that flows through FACT and into Unthi Thodu, the creek closest to the Periyar. Another possible hypothesis is that there are large intermittent inputs to the creek that passes through FACT and they move through the waterways in pulses.

This study elucidates the serious pollution of the creeks of the island of Eloor with toxic and persistent organic and inorganic compounds. Conditions do not appear to have improved at all in the three years since Greenpeace's first survey. Contaminants are for the first time recorded to be passing the whole breadth of the island to enter the Periyar River at its southern shore.

Many of these contaminants will enter the food chain and may contaminate locally produced food. Several Indian rivers have already been shown to be significant sources of organochlorine pollutants and some scientists estimate that India is the largest source to the oceans of numerous organochlorine pesticides including DDT and HCH, both of which are present in Eloor.

Hindustan Insecticides and the other nearby factories are therefore the source of pollution that is likely to have impacts both on the local and wider environment. To improve the situation, the Indian government needs to move to phase out DDT production and put in place measures to eliminate the sources of the other pollutants.

2 INTRODUCTION

In 2001, the international community made a groundbreaking commitment to eliminate some of the world's most environmentally harmful chemicals. The instrument they developed to achieve this goal was the Stockholm Convention on Persistent Organic Pollutants, usually known as the Stockholm Convention or the POPs Convention (UNEP 2001). It is the first multilateral environmental agreement to require the global elimination of polluting products.

To date, the Convention deals with twelve chlorinated chemicals or groups of chemicals. Most are pesticides and among them is the most notorious of all, DDT. DDT was first used as an insecticide in the 1940s and was credited with protecting US troops serving overseas during the Second World War from insects carrying diseases like malaria and typhus. After the war, it was increasingly used in agriculture. However, during the 1950s and 1960s, problems became evident: insect resistance to DDT was recorded and residues of the chemical started appearing in food (see eg Smith 1991). Birds were also severely impacted by DDT use. In some cases, so much DDT was applied that birds were killed outright (Carson 1962, Fry 1995, Cooper 1991), but DDT's breakdown product, DDE, also thinned eggshells, significantly reducing the number of chicks that hatched. This had a severe and long-term impact on bird species all over the world, with birds of prey often being most severely impacted since DDE biomagnifies and species at the top of the food chain receive the highest doses. Even in areas where DDT has been withdrawn, DDE persistence is such that bird populations continue to feel its effects for years or decades afterwards.

The popularity of DDT has been declining since the 1970s, when most western countries banned or severely restricted its use. The creation of the Stockholm Convention heralds its final elimination, once suitable anti-malaria strategies are in place for the countries that still use DDT against mosquitoes. The World Health Organisation is working with the United Nations Environment Program and other organisations to ensure that reliance on DDT is reduced without any adverse effect on public health (WHO 2001).

One of the world's few remaining DDT factories is situated close to the Periyar River, in the state of Kerala, South India. The river originates in the Periyar Lake near the border with the state of Tamil Nadu, receives several tributaries on its way and finally joins the Arabian Sea near the port city of Cochin. The river is a regionally important body of water and widely exploited for irrigation and fishing as well as supplying drinking water for many of the nearby peoples. In some parts of the river, especially the lower reaches, these vital uses are compromised by chemical industries discharging effluents either directly to the river or via lesser watercourses leading to the Periyar.

One of the largest industrial manufacturing centres, the Udyogmandal Industrial Estate, is located around the branch of the Periyar which passes to the north of Eloor, an island in the upper tidal reaches of the Periyar. Here industrial chemicals, leather and other goods are manufactured. Many of the factories are located on the mainland, but several others are clustered on the north of the island, including FACT (Fertilisers and Chemicals Travancore), IRE (India Rare Earths), Merchem and HIL (Hindustan Insecticides Limited).

Hindustan Insecticides Ltd. produces chlorinated pesticides such as DDT and endosulfan and it discharges industrial wastewaters into Kuzhikandam Thodu, which joins the Periyar River via Unthi Thodu. In 1999 Greenpeace analysed samples of sediments and water collected from Kuzhikandam Thodu and found that it was polluted by a mixture of the organochlorine chemicals including DDT and its metabolites, endosulfan and also by a range of other chlorinated compounds discharged from the factory (Labunska *et al.* 1999). Three years later, in 2002, Greenpeace revisited the HIL site to

establish whether any alterations in the pollution status of the environment around the factory had occurred.

3 METHODS AND SAMPLING SITES

A total of six samples were collected in the vicinity of the Hindustan Insecticides Ltd (HIL) site on 12th November 2002, including one sample of water/effluent from the creek upstream of HIL and five sediment samples from creeks downstream of HIL. A description of these samples is presented in Table 1 and the location of sampling points is presented in Figure 1.

Sample number	Sample type	Sample Location
IT02066	Sediment	Unthi Thodu at junction with the southern branch of Periyar river
IT02067	Effluent	Effluent channel leaving FACT, upstream of HIL
IT02068	Sediment	Kuzhikandam Thodu downstream of HIL and other factories
IT02069	Sediment	Unthi Thodi, just below Ammam Thuruth bridge
IT02070	Sediment	Panachi Thodu
IT02074	Sediment	Unthi Thodu, near Undhipalam

Table 1. Description of samples, Eloor, Kerala, India, 2002.

All samples were collected and stored in glass bottles that had been rinsed thoroughly with nitric acid and analytical grade pentane in order to remove all heavy metal and organic residues. Sediment samples were collected in 100ml bottles. The single aqueous sample was collected in a 1-litre bottle, ensuring that no air bubbles were present. All samples were immediately sealed and cooled upon collection and returned to the Greenpeace Research Laboratories for analysis. Detailed descriptions of sample preparation and analytical procedures are presented in Appendix 1.

4 RESULTS AND DISCUSSION

The results of organic screen analyses for samples collected in 2002 are presented in Table 2 and heavy metal analysis are presented in Table 4. A list of all the organic compounds that were reliably identified (greater than 90% spectral match quality) and groups of compounds tentatively identified is presented in Appendix 2.

4.1 Organic contaminants

Two effluent channels enter the HIL premises after passing through other factories. One passes through Indian Rare Earths, which processes mineral ores and through Merchem, which manufactures chemicals for the rubber and water treatment industries, and sulphur-based pesticide intermediates. It was not possible to access this channel. However, effluent sample IT02067 was collected from the other channel after is leaves the premises of FACT (Fertilisers and Chemicals Travancore Ltd) and before it enters HIL and Merchem territory. This sample contained only two organic compounds, which were present at such a low abundance that it was not possible to identify them to a high degree of reliability.

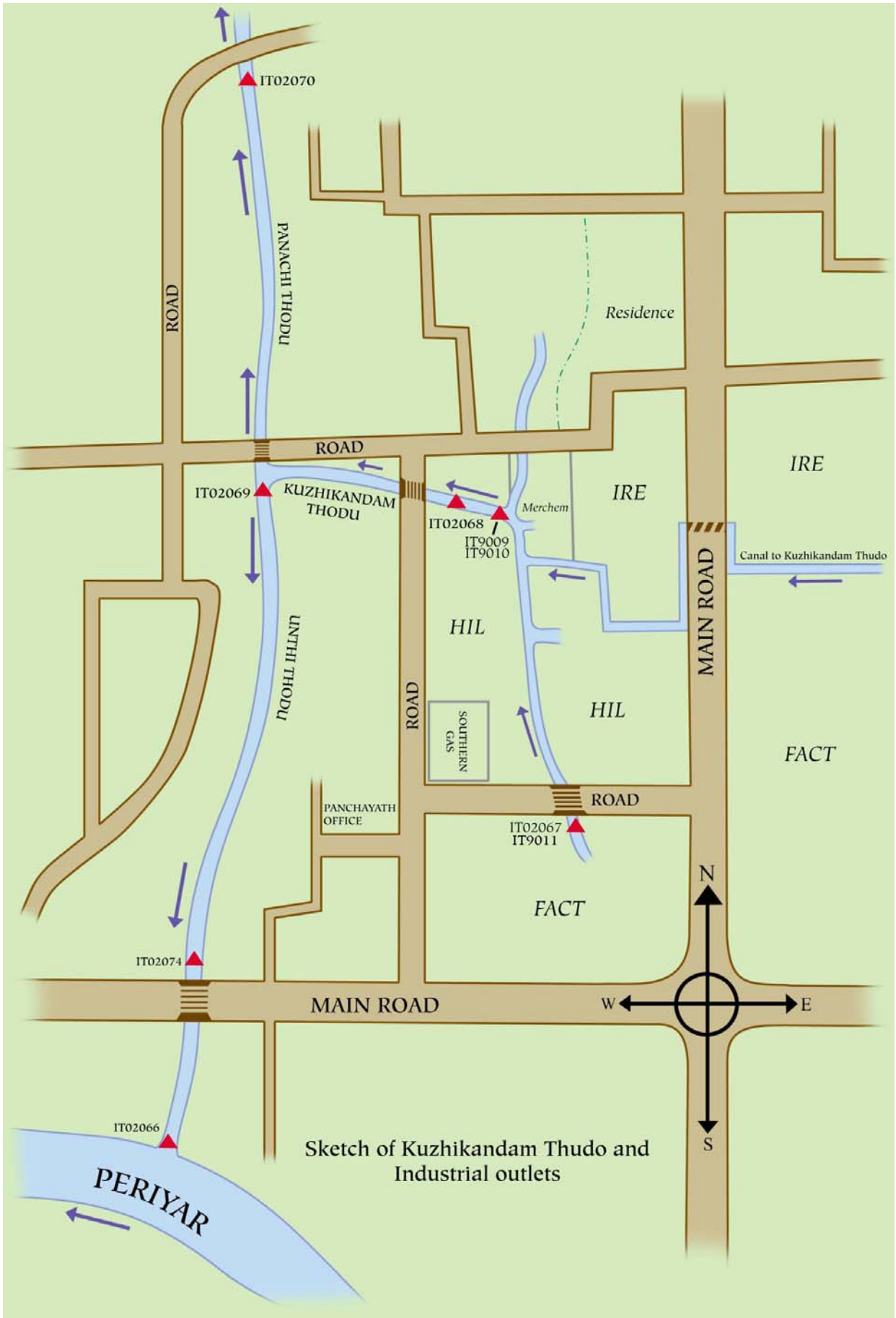

Figure 1. Location of sampling points, Eloor, Kerala, India, 1999 & 2002

Sample no.	IT02067	IT02068	IT02069	IT02074	IT02066	IT02070
Description	**Effluent**	**Sediment**	**Sediment**	**Sediment**	**Sediment**	**Sediment**
Number of organic compounds isolated	2	105	45	106	56	49
Number of organic compounds reliably identified (% of total isolated)	none	66(63%)	31(69%)	52(49%)	31(55%)	26(53%)
DDT and metabolites/degradation products						
o,p'-DDT		1	1	1		
p,p'-DDT		1	1		1	
o,p'-DDD		1	1	1	1	(1)
p,p'-DDD		1	1	1	1	(1)
m,p'-DDD			1	1		
o,p'-DDE		1	1	1	1	(1)
p,p'-DDE		1	1		1	(1)
DDMU		3	2	2	3	
DDMS			1			
DDOH		1		1		
DBM			1	1	1	1
DDM		1				
DCBP				1		
Benzene, 1,1'(1,2-dichloro-1,2-ethenediyl)bis-		1			1	
Benzene, 1,1'-(1-chloro-1,2-ethenediyl)bis-		1				
Ethane, 1,1-bis(p-chlorophenyl)-		1				
Benzene, 2,4-dichloro-1-(2-chloroethenyl)-			1			
Other chlorinated organic compounds						
Benzene, chloro-		(1)	(1)			
Benzenes, dichloro-		2, (1)	2, (1)	1, (2)	(2)	(2)
Benzenes, trichloro-		2	1, (1)	(2)	(2)	
Benzenes, tetrachloro-		(3)	(3)	(3)	(3)	
Benzene, pentachloro-		(1)	(1)	(1)	(1)	
Benzene, hexachloro-		(1)	(1)		(1)	
Benzene, 1,2-dichloro-3-ethyl-		1				
Benzene, 1,4-dichloro-3-ethenyl-		1				
Benzene, 1,1'-sulfonylbis[4-chloro-				1		
Benzene, 1-chloro-4-(chlorophenylmethyl)-				1		
Benzene, 1,1'-(2-chlororethylidene)bis-				1		
Benzene, 2,4-dichloro-1-(2-chloroethenyl)-		1				
Cyclohexanes, hexachloro-		4	1	(2)		
Butadiene, hexachloro-		1	(1)		(1)	
Biphenyls, chloro-		3				
Biphenyls, dichloro-		1				
Endosulfan ether				1		
Ethene, 1,1-diphenyl-2,2-dichloro-				1		
Styrene, octachloro-			(1)			
Methane, chlorodiphenyl-		2				
3-Butanone, 1,1-bis(4-chlorophenyl)-2,2-dimethyl-		1				
Non-chlorinated organic compounds						
Naphthalene and its derivatives		8				
Phthalate esters				1		
Biphenyl and its derivatives		1				
Alkylated benzenes				4		8
Benzothiazole						1
Methane, diphenyl-		1				
o-Terphenyl		1				
Sulfur, mol. (S8)		1				
Alkanols and its derivatives				5		
Linear aliphatic hydrocarbons		14	5	16	12	10

Table 2. Numbers of organic compounds identified in each sample using GC/MS screening in scan mode; numbers in parenthesis signify additional compounds identified only at trace levels using selective ion monitoring (SIM).

In the previous Greenpeace study in 1999, a sediment sample (IT9011) was collected from this same location. Of the sixteen compounds then isolated, only five were reliably identified. Those which were identifiable comprised three linear aliphatic hydrocarbons (3-hexadecene, 5-octadecene and dodecane) and two complex phenolic compounds: 2,4-bis(1,1-dimethylethyl)phenol and 2,6-bis(1,1-dimethylethyl)-4-methylphenol. No chlorinated compounds were found in samples collected in either 1999 or 2002 from the channel upstream of the Hindustan Insecticides factory.

Downstream of the factory, the channel is called Kuzhikandam Thodu, Thodu meaning "creek" or "canal" in Malayalam and Tamil. In contrast to the control samples, sediment sample IT02068, collected from Kuzhikandam Thodu immediately downstream of the HIL territory, contained a wide range of organic contaminants, many of them chlorinated. Of the 105 compounds isolated from this sample, 66 (63%) were identified to greater than 90% reliability. Thirty-nine of these, some 58% of those reliably identified, were chlorinated, including two isomers of DDT, twelve DDT metabolites, four isomers of hexachlorocyclohexane (HCH), including the gamma-isomer, better known as the insecticide lindane, hexachlorobutadiene (HCBD), mono- and dichlorobiphenyls and various chlorinated benzenes and toluenes.

This finding was similar to the earlier Greenpeace study, when effluent sample IT9009 and sediment sample IT9010 were collected from the same effluent channel. Table 3 presents a summary comparison of the organic compounds detected at this location in the two studies.

Groups of chlorinated compounds	**IT02068 sediment (2002 study)**	**IT9010 sediment (1999 study)**	**IT9009 effluent (1999 study)**
DDT and its metabolites	14	11	7
Chlorinated benzenes	4(7)	8	5
Chlorinated alkylbenzenes	3	3	n/d
Hexachlorocyclohexanes	4	4	2
Pentachlorocyclohexenes	n/d	1	n/d
Chlorinated cyclopentenes	n/d	n/d	1
Hexachlorobutadiene	1	1	1
PCBs	4	1	n/d
Endosulfan and its metabolites	n/d	3	n/d
Chlorinated benzophenones	n/d	5	n/d

Table 3. Groups of chlorinated organic compounds identified in the samples from Kuzhikandam Thodu in 1999 and 2002. Numbers in parentheses represent the number of compounds detected at trace levels using selected ion monitoring techniques; nd= not detected.

"Technical" DDT, as used in pesticide products, consists primarily of two isomers (p,p'-DDT and o,p'-DDT) with lesser concentrations of manufacturing by-products (DHHS 2002, ATSDR 2000, Smith 1991). These break down, albeit slowly, in the environment, to numerous different compounds, especially the highly persistent DDE. The presence of both DDT isomers and a wide range of the metabolites in both samples IT9010 and IT02068 is a clear indication of the ongoing release of technical DDT into the environment.

In addition, four isomers of hexachlorocyclohexane (HCH) were present on both occasions. India was for many years a major consumer of the pesticide known as BHC (Li 1999), which is in fact a technical mixture of HCH isomers, and concentrations in the Indian population are among the highest in the world (Allsopp *et al*. 1995 & 1998). Hindustan Insecticides was reported to be

manufacturing lindane (an insecticide containing the gamma HCH isomer extracted from the technical mixture) as recently as 10 years ago (Dinham 1993). However, India banned the use of technical HCH in 1997 (Kalra *et al*. 1999) and, at the time of writing, HCHs were absent from the list of chemicals manufactured that is provided on the Hindustan Insecticides Ltd web site (www.hil-india.com). It therefore seems most likely that the HCHs detected here represent long-term contamination resulting from earlier products and possibly continued runoff from contaminated areas or equipment within the plant.

Chlorinated benzenes may be produced as by-products of manufacturing organochlorines, including HCHs (Stringer & Johnston 2001, Stringer *et al.* 2002). Even more isomers (11 of a possible 12) were found in the sediments of Kuzhikandam Thodu in 2002 than in 1999, when eight were identified. Six isomers were detectable in the effluent in 1999. These chemicals exert a wide range of toxic effects on both the environment and humans (see detailed information in Appendix 3).

Hexachlorobutadiene, also found in all samples from this site (IT9009, IT9010 and IT02068) and low chlorinated PCB congeners (mono- and dichlorinated), detected in IT9010 and IT02068, are also believed to arise as by-products of the manufacture of other chlorinated chemicals (see eg Stringer & Johnston 2001).

Isomers of the chlorinated pesticide endosulfan and its metabolite endosulfan ether, which were detected in 1999 (sediment sample IT9010), were not detected at this location (IT02068) in 2002. However, endosulfan ether, a metabolite of endosulfan, was found further downstream in 2002 (IT02074). According to their website (www.hil-india.com) HIL manufactures endosulfan and exports it to Europe and South East Asia. Endosulfan has a shorter half-life environmental half-life than DDT compounds; 2 to 14 days under aqueous aerobic conditions and from 8 to 56 days under aqueous anaerobic conditions (Howard *et al.* 1991). However, endosulfan is noted for its toxicity to fish (Nowak & Sunderam 1991, Arnold *et al*. 1996), so a significant impact may still be occurring.

Further downstream, Kuzhikandam Thodu passes under Amman Thuruth bridge and splits into Panachi Thodu and Unthi Thodu. No samples were collected from these locations in 1999, but they were sampled in 2002 to establish how far from the factory the pollution could be traced.

Panachi Thodu flows only a short distance, ending in nearby fields. The sample from Panachi Thodu contained only five reliably identifiable DDT-group organochlorines and two other organochlorines. All except one were detected at trace levels and neither DDT isomer was present. This is the only sediment sample collected in the 2002 survey not to contain either isomer. Since this creek peters out in the fields, its flow-rate is presumed to be low, which would account for the lower level of contamination reaching it.

In contrast, Unthi Thodu, which flows south from Amman Thuruth bridge to join the Periyar, still shows significant organochlorine contamination. Although it contained fewer non-chlorinated hydrocarbons (5 compared with 26) than the sample from Kuzhikandam Thodu, the sediment below Amman Thuruth bridge (IT02069) contained nearly as many DDT compounds (12 compared with 14) and other organochlorines (14 compared with 26). The chlorobenzenes remained the most numerous group of non-DDT organochlorines.

Further down Unthi Thodu, near Undhipalam, sample IT02074 was collected. It contained ten DDT-group chemicals and sixteen other organochlorines. Interestingly, it also contained one of the

highest incidences of non-chlorinated hydrocarbons, though it was not possible to establish their source.

Finally, sample IT02066 was collected where Unthi Thodu joins the southern branch of the Periyar. The sediment at this location still contains ten reliably identifiable DDT group chemicals, including p,p'-DDT, the main constituent of technical DDT. A further ten organochlorines were identified at trace concentrations, including chlorinated benzenes and hexachlorobutadiene. This confirms for the first time that these contaminants are entering Periyar river via this route (see Figure 1).

4.2 Heavy metal contaminants

The metals analysed for in this study are naturally present in uncontaminated aquatic and terrestrial environments, though usaully at low concentrations. Apart from sample IT02070, all samples of sediment collected in 2002 contained a range of heavy metals at concentrations elevated above typical uncontaminated background levels, including cadmium, chromium, copper, lead, mercury and zinc (ATSDR 2000, Salomons & Forstner 1984). Sample IT02070 was the sediment sample least contaminated with organochlorines as well as metals, which is probably because Panachi Thodu where it was collected is a dead end with a low flow rate. The concentrations of all metals in this sample are within the typical range of concentrations found in uncontaminated environments (ATSDR 2000, Salomons & Forstner 1984), and can be taken to be indicative of local background sediment concentrations.

Sample no.	**IT02066**	**IT02067**	**IT02068**	**IT02069**	**IT02070**	**IT02074**
Concentrations	mg/kg dry weight	µg/l	mg/kg dry weight	mg/kg dry weight	mg/kg dry weight	mg/kg dry weight
Description	Sediment	Effluent	Sediment	Sediment	Sediment	Sediment
Cadmium	8	<10	<1	<1	<1	11
Chromium	169	<20	103	92	33	467
Cobalt	9	<20	5	6	4	13
Copper	119	26	52	53	10	292
Lead	160	<30	79	70	19	356
Manganese	131	316	97	74	43	175
Mercury	2.0	<1	0.8	0.7	<0.1	5.4
Nickel	32	<20	19	23	12	63
Vanadium	7	<10	27	28	24	96
Zinc	872	55	215	362	63	1419

Table 4. Concentrations of metallic elements in samples collected in Eloor, Kerala, India, 2002.

Apart from IT02070, the organochlorines were present in the greatest numbers in the sediments closest to the factory and thereafter showed a steady decline in numbers present from the HIL factory to the river. In contrast, the heavy metal pollutants did not show a clear gradient leading away from the industrial area under investigation. Concentrations of almost all metals were higher in the downstream samples, IT02074 and IT02066, than in the upstream samples IT02068 and IT02069. The highest overall level of metal contamination was in sample IT02074, the last sample but one before the creek reaches the Periyar River, with concentrations of mercury more than 54 times local background sediment concentrations (IT02070), while copper, zinc and lead were elevated by 29, 22 and 19 times respectively.

Despite the irregularity in the extent of metal contamination in the samples collected along the course of the creek system in 2002, the relative contribution of individual metals to the

contamination in the samples followed a consistent pattern. When compared to local background sediment concentrations, best represented by the concentrations in sample IT02070, the remaining samples contain elevated concentrations of most metals. For all contaminated samples, mercury concentrations are the most elevated, followed by either copper or zinc. The pattern continues for each sample, with lower but still significant elevations for lead followed by chromium. One exception was IT02066, which had a particularly high cadmium concentration.

The common pattern in the relative degree of contamination by each metal in all sediment samples strongly suggests the contaminants emanate from a common source. Merchem as well as the Indian Government operated IRE (Indian Rare Earths) and FACT (Fertilizers and Chemicals Travancore Ltd) have facilities within the Industrial Estate in the vicinity of the sampling area. Merchem manufactures various chemicals including zinc compounds and may be a source of zinc to the creek, though in 1999 locals reported that there were no regular discharges to Kuzhikundam Thodu from the Merchem plant (Labunska *et al.* 1999). IRE processes the mineral monazite, manufacturing compounds of rare earth elements and may discharge effluents containing metals such as lead and zinc.

FACT manufactures a range of fertilizer products including phosphate-based fertilizers derived from phosphoric acid (FACT 2003a, FACT 2003b). Fertilizers of this type are derived from phosphate ores (Kroschwitz & Howe-Grant 1995). The production of fertilizers using phosphate ore has been associated with the formation of waste streams contaminated with a range of heavy metals (EFMA 2000). The quantities of different trace metals in phosphate ores can vary significantly, but major contaminants generally include cadmium, chromium, copper, mercury, nickel, lead and zinc (EFMA 2000). Mercury is not the dominant contaminant in most phosphate ores, but this metal is typically present in very low concentrations in uncontaminated environments and therefore moderate inputs can lead to a high degree of elevation above background levels, as observed in the creek sediments.

Sample number and date	**IT9011 (1999 study)**	**IT9010 (1999 study)**	**IT9009 (1999 study)**
Concentrations	mg/kg dry weight	mg/kg dry weight	µg/l
Description	Sediment	Sediment	Effluent
Cadmium	1.7	n/d	<10
Chromium	570.7	57.6	120
Cobalt	9.5	5.7	<10
Copper	212.1	22.6	70
Lead	81.0	30.2	<30
Manganese	237.1	142.5	<10
Mercury	2.0	0.7	<2
Nickel	75.9	6.6	20
Vanadium	n/a	n/a	n/a
Zinc	622.4	67.9	510

Table 5. Concentrations of metallic elements in samples collected in Eloor, Kerala, India, 1999. n/a = not analysed, n/d = not detected.

The nature of heavy metal contamination found in the creeks suggests that FACT is the most likely source. Another factor pointing to FACT is that the sediment sample collected in 1999 from the creek leaving FACT (IT02011) contained high levels of contamination with a very similar profile to

samples collected in 2002. Data from samples collected in 1999 are presented in Table 5. The slight variation in the patterns between 1999 and 2002 (e.g. somewhat higher chromium and nickel in IT9011) could be explained by natural variation in the trace metal content of phosphate ores.

The inconsistency in the metal concentrations along the creeks could be explained in two ways. First, that the pollution has entered the creeks at more than one location, including one within or upstream of FACT and another in Unthi Thodu. Alternatively, there may be large but irregular inputs of heavy metals into the creek that flows through FACT, which have moved through the waterways in pulses.

Although it is not possible to conclusively attribute the reason for the increases and decreases in the concentrations of heavy metals along the Kuzhikandam-Unthi creek system, it is clear that a range of persistent and toxic heavy metals are being carried along them from the Udyogmandal Industrial Estate to the southern branch of the Periyar river.

5 CONCLUSIONS

Analysis of the water and sediment samples collected around the Hindustan Insecticides Ltd plant in Eloor, Kerala, in 2002, reconfirmed the contamination of Kuzhikandam Thodu with a wide range of hazardous chemicals including DDT, hexachlorocyclohexanes and heavy metals, which were previously found in the Greenpeace study conducted in 1999. This complex mixture of chemicals in the water and sediment of Kuzhikandam Thodu constitutes a long-term threat to aquatic life, fish, birds and humans, as many of these compounds are toxic, persistent and bioaccumulative.

There appears to have been no improvement in environmental conditions at this location in the intervening three years. Two major factors in this would be the extreme environmental persistence of metallic and organochlorine pollution, plus the continuing discharge of most, if not all of the detected pollutants, into the creeks.

Metals, being elements, cannot be broken down, although their chemical state may be altered and they may form compounds of varying toxicity depending upon the prevailing conditions. DDT and endosulfan are still listed by Hindustan Insecticides Ltd as products, so routine waste discharges are to be expected. However, it is also possible that HCH isomers, which are no longer listed as products of this facility, continue to leach from the site into the wider environment.

Metals pollution appears to be primarily due to the processing of phosphate ores for the manufacturing of chemical fertilisers at the FACT plant, through which one of the effluent channels passes before entering the HIL premises. Other facilities making or processing metal-containing materials include Indian Rare Earths (IRE) and Merchem, although neither of these is as likely to be able to produce the particular profile of metallic pollutants detected in the sediments.

In addition to updating the pollution status near the factory itself, this study documents conclusively for the first time that the organochlorine and heavy metal pollution from HIL is reaching the Periyar River directly via Kuzhikandam and Unthi creeks.

The ability of persistent organochlorine pollutants to accumulate in animal tissues has implications for the health of the local wildlife and human population. Animals living in the contaminated creeks or the Periyar itself are liable to accumulate pollutants, as are any that feed on contaminated land in or around the factory. From there, the pollutants can be passed on to animals higher up the

food chain, including fish-eating birds and birds of prey. People could be exposed through working at Hindustan Insecticides Ltd, using or handling the pesticides it produces or via eating contaminated foods such as fish, milk, eggs, meat or shellfish.

The impacts of DDT on birds were discussed earlier. Research shows that effects on human health are also possible. For example, the US government regard both hexachlorocyclohexanes and DDT as "reasonably anticipated to be a human carcinogen" (DHHS 2002). DDT metabolites, DDD and DDE, which are also present in the technical grade DDT as contaminants, can cause an increase in liver tumours in experimental animals (DHHS 2002).

An increased risk of spontaneous abortion was also associated with levels of DDE in maternal serum (Korrick *et al.* 2001). Most recently, DDT and DDE have been reported to have transgenerational effects on human fertility. The concentration of DDT in blood samples collected from women in the USA from 1960-1963 was correlated with an increase in the time it took their daughters to become pregnant. Conversely, DDE seemed it decrease time to pregnancy, although the effect was not as strong (Cohn *et al.* 2003).

The metals found to be contaminating the creeks exhibit a wide range of toxic effects on terrestrial and aquatic life, including humans. Many are able to accumulate in animals and fish, including cadmium, copper, mercury and zinc (ATSDR 2000, Goyer 1996). Furthermore, cadmium can also accumulate in plants and food crops (Jackson & Alloway 1992). Through these routes, these toxic and potentially toxic metals can enter the food chain and may result in human exposure.

Copper, chromium and zinc are essential nutrients at trace levels, though exposure to higher amounts can cause toxic effects in animals and humans (ATSDR 2000). Cadmium, lead and mercury, however, have no nutritional function and are highly toxic even at very low levels (ATSDR 2000, Goyer 1996). For humans and many animals, impacts on the liver and kidney have been observed for all these metals, while certain metals cause other major effects such as central nervous system damage from lead or mercury exposure, especially for developing children (ATSDR 2000, Goyer 1993). Furthermore, cadmium compounds and certain forms of chromium are known to be human carcinogens (DHHS 2002).

In addition to any local impacts, the discharges from Hindustan Insecticides Ltd and the other factories can be expected to add to the global burden of persistent organic and inorganic pollution. Of the metals in this study, mercury, lead and cadmium are particular of global concern due to their ability to be transported over very long distances, particularly through the atmosphere (UNECE 1998).

Sarkar *et al.* (1997) found that marine and estuarine sediments along the west coast of India are polluted by various organochlorine compounds, particularly chlorinated pesticides such as DDTs, HCHs, aldrin, dieldrin and endrin. The Periyar River, which was not included in that study, will add to the burden. In 1996, it was estimated that Indian rivers carried 180 tonnes (estimated range 66-548 tonnes) of DDT compounds and 229 tonnes (estimated range 159-456 tonnes) of HCHs to the sea. Based on these figures, fluxes of DDT and HCH from Indian rivers exceed those from all the other countries in the Asia-Pacific region (Bodo *et al.* 1996). Until the phase-out of DDT and other persistent products is implemented, this unhappy situation is set to continue.

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APPENDIX 1 ANALYTICAL METHODOLOGY

A1.1 ORGANIC ANALYSIS

A1.1.1 Preparation of samples for organic screen analysis

All solvents were of High Purity Grade (PRAG or low haloform). Glassware used in extraction and clean up procedures was cleaned in detergent, rinsed with tap water and deionised water, dried in the oven overnight at 105^0C, and rinsed three times with low haloform pentane.

Solid Samples

In preparation for analysis of extractable organic compounds, approximately 30g (wet weight) was weighed and transferred to a clean 100 ml glass bottle. Samples were spiked with deuterated naphthalene (an internal standard) at a concentration of 4.7 mg/kg. 15ml of pentane was added, followed by 5ml of acetone. All samples were then sonicated for 2 hours.

Extracts were decanted, filtered through a pre-cleaned hydrophobic phase separator filter and collected in reagent tubes. The samples were then acidified to pH 2 with 10% nitric acid. Following this, a second portion of 15ml of pentane was added, followed by 5ml of acetone and the extraction procedure repeated. Finally, both extracts obtained for each sample were combined and evaporated to a volume of approximately 3ml. 3ml of iso-propanol and 3ml of fresh prepared TBA-reagent (mixture of 3% tetrabutylammonium hydrogen sulphate and 20% anhydrous sodium sulphite in deionised water) were added to concentrated extract and the mixture shaken for 1 min. After shaking, 20ml of deionised water was added to reagent tube and the phases were allowed to separate. Finally, the organic layer was transferred with a Pasteur pipette into a pentane pre-washed Florosil column. The compounds were eluted with a 95:5 mixture of pentane: toluene and the eluent evaporated down to a volume of 2 ml under a stream of analytical grade nitrogen. 1-Bromonaphthalene was then added at a concentration of 10mg/l to provide an indication of GC/MS performance.

Aqueous Samples

Prior to the extraction, samples were spiked with deuterated naphthalene (an internal standard) at a concentration of 10mg/l. 20ml of pentane was added, and the sample agitated for 2 hours on a bottle roller to maximise contact between solvent and sample.

After separation of the phases, the solvent extract was filtered through a pre-cleaned hydrophobic phase separator filter and collected in a pre-cleaned reagent tube. The aqueous sample was acidified to pH 2 with 10% nitric acid, a second portion of 20ml pentane was added and the extraction procedure repeated. Both extracts were combined and cleaned up as described above for solid samples.

A1.1.2 Chromatographic Analysis

Organic compounds were identified qualitatively using Gas Chromatography Mass Spectrometry (GC-MS). Instrumentation was an Agilent 6890 Series gas chromatograph, interfaced with a Agilent Enhanced Chem-Station data system and linked to a Agilent 5973 Mass Selective Detector operated in SCAN mode. The identification of compounds was carried out by computer matching against Agilent Wiley7N and Pesticides Libraries of over 390,000 mass spectra combined with

expert interpretation. Also all extracts were analysed using selective ion monitoring (SIM) method against two standard solutions. The lists of compounds containing in Standard I and Standard II are presented below. All individual standards were obtained from Sigma Aldrich Co. Ltd., Supelco, UK.

Results are reported as either reliably or tentatively identified. Match qualities of 90% or greater against Agilent Wiley7N and Pesticides Libraries or identification confirmed against standard compounds (using retention times and mass-spectra obtained during calibration) are assumed to give reliable identifications. Tentative identification refers to qualities between 51% and 90% against Agilent Wiley7N and Pesticides Libraries only. Analytes yielding match qualities of 50% or less are assumed to be unidentified.

Compound	**Ions monitored**
Benzene, 1,3-dichloro-	146, 148, 111, 75
Benzene, 1,4-dichloro-	146, 148, 111, 75
Benzene, 1,2-dichloro-	146, 148, 111, 75
Benzene, 1,3,5-trichloro-	180, 182, 145, 74
Phenol, 2,4-dichloro-	162, 164, 63, 98
Benzene, 1,2,4-trichloro-	180, 182, 145, 109
Benzene, 1,2,3-trichloro-	180, 182, 145, 109
Dichlorvos	109, 185, 79, 47
Benzene, 1,2,3,5-tetrachloro-	216, 214, 218, 179
Benzene, 1,2,4,5-tetrachloro-	216, 214, 218, 179
Benzene, 1,2,3,4-tetrachloro-	216, 214, 218, 179
Benzene, pentachloro-	250, 252, 248, 215
alpha-HCH	181, 183, 219, 217
Benzene, hexachloro-	284, 286, 282, 249
Atrazine	200, 215, 202, 217
beta-HCH	181, 183, 219, 217
gamma-HCH	181, 183, 219, 217
delta-HCH	181, 183, 219, 217
o,p'-DDE	246, 248, 318, 176
p,p'-DDE	246, 318, 246, 316
o,p'-DDD	235, 237, 165, 199
p,p'-DDD	235, 237, 165, 199
o,p'-DDT	235, 237, 165, 199
p,p'-DDT	235, 237, 165, 199

Table A1.1.1 List of compounds in standard I used for SIM analysis

Compound	Ions monitored
Phenol	94, 66, 65, 95
Phenol, 2-chloro-	128, 64, 92, 39
Phenol, 2-methyl-	108, 79, 90, 51
Phenol, 3-methyl- and 4-methyl-	108, 107, 79, 77
Phenol, 2-nitro-	139, 65, 81, 109
Phenol, 2,5-dichloro-	162, 164, 63, 99
Phenol, 2,3-dichloro-	162, 126, 63, 99
Phenol, 4-chloro-	128, 65, 130, 100
Phenol, 2,6-dichloro-	162, 164, 63, 98
Butadiene, hexachloro-	225, 190, 260, 118
Phenol, 4-chloro-3-methyl-	107, 142, 77, 144
Phenol, 2,3,5-trichloro-	196, 198, 160, 97
Phenol, 2,4,6-trichloro-	196, 198, 97, 132
Phenol, 2,4,5-trichloro-	196, 198, 97, 132
Phenol, 2,3,4-trichloro-	196, 198, 97, 160
Phenol, 2,3,6-trichloro-	196, 198, 97, 132
Phenol, 3,5-dichloro-	162, 164, 99, 63
Phenol, 3,4-dichloro-	162, 164, 99, 63
Phenol, 2,3,5,6-tetrachloro-	232, 234, 230, 131
Phenol, 2,3,4,6-tetrachloro-	232, 234, 230, 131
Phenol, pentachloro-	266, 268, 264, 165
Dinoseb	211, 163, 147, 117
PCB-28	256, 258, 186, 150
Heptachlor	100, 272, 274, 137
PCB-52	292, 220, 290, 222
Aldrin	66, 263, 265, 261
Octachlorostyrene	308, 310, 380, 378
Chlordane I	373, 375, 272, 237
PCB-101	326, 324, 254, 328
Chlordane II	373, 375, 272, 237
PCB-81	292, 290, 294, 220
Dieldrin	79, 81, 263, 265
PCB-77	292, 290, 294, 220
Endrin	67, 317, 319, 345
PCB-123	326, 324, 254, 328
PCB-118	326, 324, 256, 328
PCB-114	326, 324, 256, 328
PCB-153	360, 362, 290, 358
PCB-105	326, 324, 254, 328
PCB-138	360, 362, 290, 358
PCB-126	326, 324, 254, 328
PCB-167	360, 362, 290, 358
PCB-156	360, 362, 290, 358
PCB-157	360, 362, 290, 358
PCB-180	396, 394, 324, 162
PCB-169	360, 362, 358, 145
PCB-170	396, 394, 324, 326
PCB-189	396, 394, 398, 324

Table A1.1.2. List of compounds in standard II used for SIM analysis

A1.2 HEAVY METAL ANALYSIS

A1.2.1 Preparation of samples for heavy metal analysis

All chemicals were of High Purity Aristar Grade. All glassware was cleaned in detergent, rinsed with tap water and deionised water, soaked in 10% nitric acid overnight, rinsed with deionised water and dried in an oven at 105°C.

Solid Samples

Samples were air dried until weighing readings became constant (approx. 5 days). They were then crushed using a pestle and mortar until homogenous and sieved through a 2-mm mesh. Approximately 0.5 g of sample was accurately weighed into a glass 100 ml boiling tube and to this 10 ml of deionised water was added, followed by 7.5 ml of concentrated hydrochloric acid and 2.5 ml of concentrated nitric acid. The samples were digested at room temperature overnight prior to being placed onto a Gerhardt Kjeldatherm digestion block (40 space) connected to a Gerhardt Turbosog scrubber unit (filled with 10% w/v sodium hydroxide). The samples were then refluxed at 130°C for four hours. To prepare samples for mercury analysis, this procedure was repeated in an identical manner other than using a digest temperature of 90°C.

After cooling to ambient temperature, the digests were filtered into volumetric flasks, diluted with deionised water, made up to a volume of 50 ml and mixed. A Standard Reference Material, BCR-143 (trace elements in a sewage sludge amended soil), certified by the Commission of the European Communities, Brussels, and a blank sample, were prepared with the batch of samples. All were prepared in 15% v/v hydrochloric acid and 5% v/v nitric acid.

One sample (IT02070) was analysed in duplicate to assess the reproducibility of the method. Furthermore, a certified reference material and a blank sample were separately prepared in an identical manner with the batch of samples. The standard reference material analysed was PACS-2 (marine sediment reference material for trace elements and other constituents), certified by the National Research Council of Canada. Recovery data for the standard reference materials are presented in Table A1.2.1

Reference material	Cd (%)	Cr (%)	Co (%)	Cu (%)	Hg (%)	Mn (%)	Ni (%)	Pb (%)	Zn (%)
PACS2	95	63	101	96	91	71	75	92	99

Table A1.2.1. Percentage recovery data for the standard reference material analysed

Aqueous samples

On arrival, 100ml of sample was transferred to a clean glass bottle and acidified with nitric acid (10% v/v). 50 ml of this solution was subsequently transferred to a 100ml boiling tube, placed onto the Gerhardt Kjeldatherm digestion block, and refluxed at 130°C for four hours. After cooling to ambient temperature, the digests were filtered into volumetric flasks, diluted with deionised water, made up to a volume of 50 ml and mixed. To prepare samples for mercury analysis, this procedure was repeated in an identical manner other than using 25 ml of each acidified sample solution and a digest temperature of 90°C.

A1.2.2 Inductively Coupled Plasma Atomic Emission Spectrometry (ICP-AES)

Following preparation, samples were analysed by Inductively Coupled Plasma Atomic Emission Spectrometry (ICP-AES), using a Varian Liberty-100 Sequential Spectrometer. The following metals were quantified directly: cadmium (Cd), cobalt (Co), copper (Cu), chromium (Cr), lead (Pb), manganese (Mn), nickel (Ni), vanadium (V) and zinc (Zn). A multi-element instrument calibration standard was prepared at a concentration of 10 mg/l, matrix matched to the samples (i.e. in 15% v/v hydrochloric acid and 5% v/v nitric acid). The calibration was validated using a quality control standard (8 mg/l), prepared internally from different reagent stocks. Any sample exceeding the calibration range was diluted accordingly, in duplicate, and re-analysed.

Mercury (Hg) was determined using Cold Vapour Generation ICP-AES. Ionic mercury, Hg (II), was reduced to elemental mercury, Hg (0), following reduction of the samples with sodium borohydride (0.6% w/v), sodium hydroxide (0.5% w/v) and hydrochloric acid (10 molar). The elemental mercury vapour was carried in a stream of argon into the spectrometer. Two calibration standards were prepared, at 10 µg/l and 100 µg/l, matrix matched to the samples (i.e. in 15% v/v hydrochloric acid and 5% v/v nitric acid for solid samples or 10% v/v nitric acid for aqueous samples). The calibration was validated using a quality control standard (80 µg/l), prepared internally from different reagent stock. Any sample exceeding the calibration range was diluted accordingly, in duplicate, and re-analysed.

APPENDIX 2 ORGANIC SCREENING RESULTS

Sample number: IT02069	**Sample number:** IT02070	**Sample number:** IT02074
Number of compounds isolated: 45	**Number of compounds isolated:** 49	**Number of compounds isolated:** 106
Sample type: Sediment	**Sample type:** Sediment	**Sample type:** Sediment
Compounds identified to better than 90%:	**Compounds identified to better than 90%:**	**Compounds identified to better than 90%:**
Benzene, 1,1'-(2-chloroethylidene)bis[4-chloro-	Benzene, 1,1'-(2-chloroethylidene)bis[4-chloro-	10-Methylnonadecane
Benzene, 1,1'-methylenebis[4-chloro-	Benzene, 1,1'-methylenebis[4-chloro-	1-Decanol, 2-hexyl-
Benzene, 1,2,3,4-tetrachloro- (SIM)	Benzene, 1,2,3,4-tetrachloro- (SIM)	1-Dodecanol, 2-methyl-, (S)-
Benzene, 1,2,3,5-tetrachloro- (SIM)	Benzene, 1,2,3,5-tetrachloro- (SIM)	1-Hexadecanol, 2-methyl-
Benzene, 1,2,4,5-tetrachloro- (SIM)	Benzene, 1,2,4,5-tetrachloro- (SIM)	1-Octadecene
Benzene, 1,2,4-trichloro-	Benzene, 1,2,4-trichloro-	1-Octanol, 2-butyl-
Benzene, 1,2-dichloro-	Benzene, 1,2-dichloro-	1-Tridecanol
Benzene, 1,3,5-trichloro- (SIM)	Benzene, 1,3,5-trichloro- (SIM)	Benzene, 1-(dodecyloxy)-2-nitro-
Benzene, 1,3-dichloro- (SIM)	Benzene, 1,3-dichloro- (SIM)	Benzene, 1,1'-(2-chloroethylidenene)bis-
Benzene, 1,4-dichloro-	Benzene, 1,4-dichloro-	Benzene, 1,1'-methylenebis[4-chloro-
Benzene, 2,4-dichloro-1-(2-chloroethenyl)-	Benzene, 2,4-dichloro-1-(2-chloroethenyl)-	Benzene, 1,1'-sulfonylbis[4-chloro-
Benzene, chloro- (SIM)	Benzene, chloro- (SIM)	Benzene, 1,2,3,4-tetrachloro- (SIM)
Benzene, hexachloro- (SIM)	Benzene, hexachloro- (SIM)	Benzene, 1,2,3,5-tetrachloro- (SIM)
Benzene, pentachloro- (SIM)	Benzene, pentachloro- (SIM)	Benzene, 1,2,4,5-tetrachloro- (SIM)
Butadiene, hexachloro- (SIM)	Butadiene, hexachloro- (SIM)	Benzene, 1,2,4-trichloro- (SIM)
Cyclohexane, .alpha.-hexachloro-	Cyclohexane, .alpha.-hexachloro-	Benzene, 1,2-bis(1-methylethyl)-
Eicosane	Eicosane	Benzene, 1,2-dichloro- (SIM)
Octadecane	Octadecane	Benzene, 1,3,5-trichloro- (SIM)
o,p'- DDE	o,p'- DDE	Benzene, 1,3-bis(1-methylethyl)-
p,p'- DDE	p,p'- DDE	Benzene, 1,3-dichloro- (SIM)
m,p'-DDD	m,p'-DDD	Benzene, 1,4-bis(1-methylethyl)-
o,p'- DDD	o,p'- DDD	Benzene, 1,4-dichloro-
p,p'- DDD	p,p'- DDD	Benzene, 1-chloro-4-(chlorophenylmethyl)-
o,p'- DDMU	o,p'- DDMU	Benzene, pentachloro- (SIM)
p,p'- DDMU	p,p'- DDMU	Benzeneethanol, 4-chloro-.beta.-(4-chlorophenyl)-
o,p'- DDT	o,p'- DDT	Benzophenone, 4,4'-dichloro-
p,p'- DDT	p,p'- DDT	Bis(2-ethylhexyl) phthalate
Pentadecane	Pentadecane	Cyclohexane, alpha.-hexachloro- (SIM)
Pentadecane, 7-methyl-	Pentadecane, 7-methyl-	Cyclohexane,.delta.-hexachloro- (SIM)
Styrene, octachloro- (SIM)	Styrene, octachloro- (SIM)	Decane
Tetracosane	Tetracosane	Docosane
		Docosane, 11-decyl-
Compounds tentatively identified:	**Compounds tentatively identified:**	Dodecane
		Dodecane, 6-methyl-
6-Dodecanone	6-Dodecanone	Eicosane
Benzene, 1,4-dichloro-2-(2-chloroethenyl)-	Benzene, 1,4-dichloro-2-(2-chloroethenyl)-	Endosulfane ether
Hexadecane, 2-methyl-	Hexadecane, 2-methyl-	Ethene, 1,1-diphenyl-2,2-dichloro-
Undecane, 5-methyl-	Undecane, 5-methyl-	Heptadecane
		Hexadecane
		Hexadecane, 2,6,11,15-tetramethyl-
		Octadecane
		o,p'-DDE
		p,p'-DDE
		o,p'-DDD
		p,p'-DDD
		o,p'-DDT
		o,p'-DDMU
		p,p'-DDMU
		Pentadecane
		Tetradecane
		Tridecane
		Undecane
		Compounds tentatively identified:
		Benzene, 1,1'-(chloroethenylidene)bis-
		Benzene, 1,1'-(dichloromethylene)bis-
		Ethanol, 2-(dodecyloxy)-
		Mirex
		Linear aliphatic hydrocarbons- 19 compounds

Table A2, part 1. List of compounds reliably identified and groups of compounds tentatively identified in the samples

Sample number: IT02066	**Sample number:** IT02067	**Sample number:** IT02068
Number of compounds isolated: 56	**Number of compounds isolated:** 2	**Number of compounds isolated:** 105
Sample type: Sediment	**Sample type:** Effluent	**Sample type:** Sediment
Compounds identified to better than 90%: Benzene, 1,1'-(1,2-dichloro-1,2-ethenediyl)bis-, (Z)- Benzene, 1,1'-(chloroethenylidene)bis[4-chloro- (2 isomers) Benzene, 1,1'-methylenebis[4-chloro- Benzene, 1-chloro-2-[2-chloro-1-(4-chlorophenyl]- Benzene, 1,2,3,4-tetrachloro- (SIM) Benzene, 1,2,3,5-tetrachloro- (SIM) Benzene, 1,2,4,5-tetrachloro- (SIM) Benzene, 1,2,4-trichloro- (SIM) Benzene, 1,2-dichloro- (SIM) Benzene, 1,3,5-trichloro- (SIM) Benzene, 1,4-dichloro- (SIM) Benzene, hexachloro- (SIM) Benzene, pentachloro- (SIM) Butadiene, hexachloro- (SIM) Dodecane Dodecane, 2,6,11-trimethyl- Eicosane Heneicosane Heptadecane Hexadecane Nonadecane o,p'-DDD o,p'-DDE p,p'-DDD p,p'-DDE p,p'-DDT Pentadecane Pentadecane, 2,6,10,14-tetramethyl- Tetracosane Tetradecane Tridecane **Compounds tentatively identified:** Decane, 2,6,7-trimethyl- Decane, 3,6-dimethyl- Hexadecane, 2-methyl- Hexane, 3,3-dimethyl- Tricosane Tridecane, 3-methyl- Tridecane, 6-propyl- Undecane, 5,7-dimethyl- Undecane, 5-methyl-	**Compounds identified to better than 90%:** None **Compounds tentatively identified:** None	**Compounds identified to better than 90%:** 1,1'-Biphenyl 1,1'-Biphenyl, 2,5-dichloro- 1,1'-Biphenyl, 2-chloro- 1,1'-Biphenyl, 3-chloro- 1,1'-Biphenyl, 4-chloro- 1,3-Butadiene, hexachloro- 1s,cis-Calamenene 3-Butanone,1,1-bis(4-chlorophenyl)-2,2-dimethyl- Benzene, 1,1'-(1,2-dichloro-1,2-ethenediyl)bis-,(Z)- Benzene, 1,1'-(1-chloro-1,2-ethenediyl)bis- Benzene, 1,1'-methylenebis[4-chloro- Benzene, 1,2,3,4-tetrachloro- (SIM) Benzene, 1,2,3,5-tetrachloro- (SIM) Benzene, 1,2,4,5-tetrachloro- (SIM) Benzene, 1,2,4-trichloro- Benzene, 1,2-dichloro- Benzene, 1,2-dichloro-3-ethyl- Benzene, 1,3,5-trichloro- Benzene, 1,3-dichloro- (SIM) Benzene, 1,4-dichloro- Benzene, 1,4-dichloro-2-ethenyl- Benzene, 1-chloro-2-[2-chloro-1-(4-chlorophehyl)ethenyl]- Benzene, 2,4-dichloro-1-(2-chloroethenyl)- Benzene, chloro- (SIM) Benzene, hexachloro- (SIM) Benzene, pentachloro- (SIM) Benzeneethanol, 4-chloro-.beta.-(4-chlorophenyl)- Cyclohexane, .alpha.-hexachloro- Cyclohexane, .beta.-hexachloro- Cyclohexane, .delta.-hexachloro- Cyclohexane, .gamma.-hexachloro- Dodecane Dodecane, 2,6,10-trimethyl- Ethane, 1,1-bis(p-chlorophenyl)- Heneicosane Heptadecane Hexadecane Hexadecane, 2-methyl- Methane, 2-chlorodiphenyl- Methane, 4-chlorodiphenyl- Methane, diphenyl- Naphthalene, 1,3-dimethyl- Naphthalene, 1,4,6-trimethyl- Naphthalene, 1,6,7-trimethyl- Naphthalene, 1,6-dimethyl- Naphthalene, 1,8-dimethyl- Naphthalene, 2,3,6-trimethyl- Naphthalene, 2,6-dimethyl- Naphthalene, 2-methyl- Nonadecane Octadecane o-Terphenyl o,p'-DDE p,p'-DDE o,p'-DDD p,p'-DDD o,p'-DDMU p,p'-DDMU o,p'-DDT p,p'-DDT Pentadecane Pentadecane, 2,6,10,14-tetramethyl- Sulfur, mol. (S8) Tetracosane Tetradecane Undecane **Compounds tentatively identified:** Benzene, 1,3-dichloro-2-ethenyl- Mirex Linear aliphatic hydrocarbons – 8 compounds

Table A2, part 2. List of compounds reliably identified and groups of compounds tentatively identified in the samples.

APPENDIX 3 TOXICOLOGICAL OUTLINES FOR KEY ORGANIC COMPOUNDS

A3.1 DDT AND ITS METABOLITES DDD AND DDE

Production and major uses

DDT, an organochlorine insecticide, was first widely used during the Second World War to control disease-carrying insects referred to as "vectors". Hence, DDT is often described as being used for "vector control". It was first synthesized in 1874, but only in 1939 did Mueller and his co-workers discover its insecticidal properties (ATSDR 2000). For a time DDT was also used in agriculture and silviculture (see eg Carson 1962, Fry 1995, Cooper 1991), but because of its environmental impact such uses have been almost universally banned. Currently, DDT is licensed almost exclusively for vector control. The best data currently available show that at least 24 countries use DDT for this purpose (WHO 2001) including the Russian Federation, China, South Africa, Zambia, Papua New Guinea, Ecuador, Mauritius, Costa Rica, the Comoro Islands, and the Korean Republic (UNEP 2000). Nonetheless, it is thought that some of the DDT manufactured ostensibly for vector control is in fact used for agricultural purposes (Zhou *et al.* 2001, WWF 1998).

Few DDT manufacturers are now left. At the beginning of the 1990s DDT was being manufactured by Hindustan Insecticides Ltd (India), by EniChem Synthesis S.p.A. (Italy) (Dinham 1993, FAO/UNEP 1991, RSC 1991). More recent reports also refer to producers in China, Mexico, Russia, South Korea and former Soviet Union States (WWF 1998, UNEP 2000).

Identity

The term "DDT" generally refers to technical DDT, which is a mixture of several compounds and may not always have the same composition. Technical DDT is manufactured by the condensation reaction of chloral hydrate with chlorobenzene in concentrated sulfuric acid. The main component is p,p'-DDT (65-80%), together with a variable mix of other compounds. These are reported by different sources to include 15-20% of o,p'-DDT (ATSDR 2000, DHHS 2002), 4% p,p'-DDE (Smith 1991, DHHS 2002) and traces of other compounds such as 1-(p-chlorophenyl)-2,2,2-trichloroethanol, o,o'-DDT and bis(p-chlorophenyl)sulfone (DHHS 2002). Technical DDT is a white amorphous powder that is odourless, but which may have a slight aromatic odour.

o,p'-DDT & p,p'-DDT
Molecular formula: **$C_{14}H_9Cl_5$**
CAS registry number: **o,p'-DDT [789-02-6]**
p,p'-DDT [50-29-3]

The chemical names of o,p'-DDT are:
Benzene, 1-chloro-2-[2,2,2-trichloro-1-(4-chlorophenyl)ethyl]- or
o,p'-dichlorodiphenyltrichloroethane

and of p,p-DDT are:
Benzene, 1,1'-(2,2,2-trichloroethylidene)bis[4-chloro- or
p,p'-dichlorodiphenyltrichloroethane

o,p'-DDD & p,p'-DDD
Molecular formula: $C_{14}H_{10}Cl_4$
CAS registry number: **o,p'-DDD [53-19-0]**
p,p'-DDD [72-54-8]

The chemical names of o,p'-DDD are:
Benzene, 1-chloro-2-[2,2-dichloro-1-(4-chlorophenyl)ethyl]- or
o,p'-dichlorodiphenyldichloroethane

and of p,p-DDD are:
Benzene, 1,1'-(2,2-dichloroethylidene)bis[4-chloro- or
p,p'-dichlorodiphenyldichloroethane

o,p'-DDE & p,p'-DDE
Molecular formula: $C_{14}H_8Cl_4$
CAS registry number: **o,p'-DDE [3424-82-6]**
p,p'-DDE [72-55-9]

The chemical names of o,p'-DDE are:
Benzene, 1-chloro-2-[2,2-dichloro-1-(4-chlorophenyl)ethenyl]- or
o,p'-dichlorodiphenyldichloroethene

and of p,p-DDE are:
Benzene, 1,1'-(dichloroethenylidene)bis[4-chloro- or
p,p'-dichlorodiphenyldichloroethene

Environmental fate

Most of the DDT (and DDT metabolites) found in the environment has resulted from historical use as a general insecticide. Large quantities of DDT were directly applied to agricultural soils (ATSDR 2000). However, contamination of soil, sediment, and of chicken eggs has been reported after DDT was used more specifically for vector control (Vieira *et al.* 2001, Torres *et al.* 2002). Additionally, agricultural applications of the pesticide dicofol, which is known to contain DDT isomers as impurities from production, may also contribute to environmental contamination by DDT and its related compounds (Pham *et al.* 1996). DDT is very highly persistent in the environment, with a reported half-life of between 2 and 15 years (Howard *et al*. 1991). It is largely immobile in most soils. Routes of loss and degradation include runoff, volatilisation, photolysis and biodegradation (aerobic and anaerobic). These processes generally occur only very slowly.

Breakdown products of DDT that have been found in the soil environment are predominantly DDD and DDE. These are also highly persistent and have similar chemical and physical properties to the parent compound (Howard *et al*. 1991). Due to an extremely low solubility in water, DDT will be retained to a greater degree by soils and soil fractions with higher contents of soil organic matter. DDT, together with its metabolites, has been detected in a wide variety of environmental media including soil, sediments, surface and groundwater, air, animal and plant tissues, food and also in working and domestic environments (ATSDR 2000, DHHS 2002, Zhou *et al.* 2001, Sarkar *et al.*1997, Pham *et al.* 1996).

Toxicity

Toxicity to humans

DDT is moderately to slightly toxic in studied mammalian species administered the insecticide by the oral route (RSC 1991, Meister 1992, ASTDR 2002). The primary target organ of DDT is the nervous system. High doses can cause trembling, increased susceptibility to cold and feelings of fearfulness. Convulsions result at the highest doses. Death can occur through respiratory arrest, though animals that survive a day or more after the last dose usually recover completely (Smith 1991). DDT has also been observed to cause chronic effects on the nervous system, liver, kidneys and immune systems in experimental animals (ASTDR 2000, WHO 1979).

DDT is poorly absorbed through the skin, with powder forms being far less easily taken up than oil-based formulations. DDT is readily absorbed through the gastrointestinal tract, with increased absorption in the presence of fats (ASTDR 2000). Inhalation exposure to powders may also take place though these may in fact be trapped in the upper reaches of the respiratory tract and be ingested rather than absorbed through the lungs (ATSDR 2000, Smith 1991).

DDT and its metabolites are lipophilic and, as a result, can bioaccumulate in fatty tissues. In people who do not actually work with DDT, food is generally the greatest source of exposure. Human occupational and dietary exposure to DDT may differ both in dose and in chemical character. The main ingredient of technical grade DDT, p,p'-DDT, is broken down in the environment or in the body to p,p'-DDE and smaller quantities of other chemicals. Occupational exposure to technical DDT is predominantly in the form of p,p'-DDT. Dietary exposure, especially in those countries where DDT is no longer used, is predominantly to p,p'-DDE, although there are several other minor breakdown products to which individuals will also be exposed (Longnecker *et al.* 1997, ATSDR 2000). p,p'-DDE is more persistent both in the body and the environment than p,p'-DDT (Smith 1991) and much of the environmental impact is attributable to this compound unless there has been recent exposure to technical DDT.

DDT and several related compounds are known to show endocrine disrupting activity. They exhibit different modes of action. Several are weakly oestrogenic. Of these, o,p'-DDT is the most active. p,p'-DDE, the compound likely to be present at the highest concentrations in most humans, is an anti-androgen (Longnecker *et al.* 1997), i.e. it works against the action of male sex hormones.

Symptoms observed in humans as a result of low to moderate exposure may include nausea, diarrhoea, increased liver enzyme activity, irritation (of the eyes, nose or throat), and excitability (ASTDR 2000).

The IARC has classified p,p'-DDT as possibly carcinogenic to humans (group 2B) and the US Department of Health and Human Services regards it as being "reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity in experimental animals" (DHHS 2002). There is inadequate direct evidence for the carcinogenicity of DDT in humans (DHHS 2002), but there are epidemiological studies available on the cancer risks associated with exposure to DDT. Some of the results have been equivocal. For example, an extensive review of human studies that has evaluated the breast cancer risk of

DDT exposure was carried out by Snedeker (2000). This established that most of the studies failed to confirm earlier observations of a significant relationship between serum and adipose tissue levels of DDE or DDT and breast cancer. Nevertheless, some studies showed that DDT and its metabolites were present at much higher levels in the blood of breast cancer patients, as compared to healthy females, irrespective of their age, diet or geographical distribution (Mathur *et al.* 2002).

It has been suggested that p,p'-DDT and p,p'-DDE could play a part in the pathogenesis of exocrine pancreatic cancer through modulation of K-*ras* activation. It was shown that patients with exocrine pancreatic cancer whose tumours had a K-*ras* mutation had significantly higher concentrations of p,p'-DDT, p,p'-DDE and other organochlorines than patients without such a mutation. It was suggested that organochlorines do not necessarily play a direct part in activation of K-*ras*. Rather, the compounds might enhance the effects of K-*ras* mutagens or might provide a growth advantage to the mutated cells (Porta *et al.* 1999).

Cohn and co-workers (2003) investigated the possible effects of DDT and DDE on the human reproductive system. They studied the daughters of subjects previously exposed to DDT and DDE. Their observations indicated that elevated levels of DDT in maternal blood were clearly associated with decreased chance of pregnancy in their daughters. For every 10 micrograms DDT per litre in maternal serum, the probability of pregnancy fell by 32%. However, quite unexpectedly, the chance of pregnancy increased by 16% with each increment of 10 micrograms per litre of maternal serum of DDE. The apparently antagonistic effects of DDT and DDE may explain why large changes in reproductive performance overall have not been noticed in human populations since the introduction and widespread use of DDT. A modest increased risk of spontaneous abortion was also found to be associated with elevated maternal serum DDE levels (Korrick *et al.* 2001). Another recent study (Hauser *et al.* 2003) which investigated the relationship between environmental chemicals exposure and semen parameters in adult men, concluded that there is only limited evidence of an inverse association between p,p'-DDE and sperm motility.

Environmental toxicity

Exposure of wildlife to DDT and its metabolites in the natural environment are primarily associated with the accumulation and persistence of these contaminants in both aquatic and terrestrial food chains. Ingestion of contaminated food results in the deposition of DDT/DDE/DDD in tissues with subsequent potential reproductive, developmental and neurological effects. The most important reproductive effect observed in wildlife concerns eggshell thinning in birds. It was found that p,p'-DDE causes the thinning of bird's eggshells through perturbation of calcium metabolism. Eggshell thinning caused by p,p'-DDE results in crushed eggs, or, if the egg is not crushed, the embryo can die of dehydration as too much water is lost through the thinned shell (Hickey & Anderson 1968, Newton 1995, Provini & Galassi 1999). Tests on 15 different toxic pollutants found that only p,p'-DDE has the ability to thin shells over an extended period (Haegele & Tucker 1974, Peakall & Lincer 1996). Although DDT primarily causes population decline through reproductive failure, it may also kill highly exposed birds directly (Carson 1962, Cooper 1995, Newton *et al.* 1982, Garcelon & Thomas 1997). Analysis of kestrels and sparrowhawks in the 1960s and 1970s suggest that some were being killed directly by p,p'-DDE exposure (Newton *et al.* 1982). Some bird populations which previously suffered from p,p'-DDE impacts of egg-shell thinning and egg breakage are no longer at such risk.

Studies in the UK on the grey heron, *Ardea cinerea L.*, (Newton *et al.* 1993) show that levels of DDE in herons or their eggs have significantly declined. A study on grey herons in France noted that levels of p,p'-DDE in eggs were lower than levels associated with reproductive effects reported in the wild or in laboratory studies (de Cruz *et al.* 1997). However, some effects of organochlorines in seabirds have been observed recently despite the general downward trend in many organochlorines. In the Arctic, present p,p'-DDE levels in Canadian tundra peregrines, *Fennoscandian merlin,* and white-tailed sea eagle are still causing significant egg shell thinning (de Wit *et al.* 1997).

There is evidence that DDT isomers and metabolites can cause reproductive and developmental effects in fish and other test animals (ASTDR 2000). DDT and related compounds can cause disturbance of the endocrine system resulting in thyroid dysfunction in birds and fish; decreased fertility in birds, fish and mammals; decreased hatching success in birds, fish, and turtles; gross birth deformities in birds, fish, and turtles; metabolic abnormalities in birds; behavioural abnormalities in birds; demasculinisation and feminization of male fish and birds; defeminization and masculinisation of female fish and birds; and compromised immune systems in birds and mammals (Colborn & Thayer 2000).

Legislation

DDT is controlled through a number of international legal instruments - notably the PIC Convention, the UNECE LRTAP POPs protocol, the Barcelona Convention, the Helsinki Convention, the IJC and the UNEP POPs Convention (The Stockholm Convention). DDT was included among other twelve POPs chemicals in the Stockholm Convention on Persistent Organic Pollutants (Annex B, part II) that was signed in May 2001 and is to take effect in the coming three to five years (UNEP 2001). According to this Convention production and use of DDT was restricted to disease vector control and to use as an intermediate in production of dicofol in accordance with Part II of Annex B.

After the Stockholm Convention was signed, The World Health Organisation (WHO 2001) set up an Action Plan with an aim to increase public health staff awareness and to reduce and/or eliminate use of the DDT in disease vector control. DDT is, of course, included under wider groupings of organochlorine pesticides or organohalogens under the various waste trade Conventions.

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A3.2 ENDOSULFAN

Production and major uses

Endosulfan is a chlorinated cyclodiene insecticide. It is used on food, non-food and forage crops as well as for wood preservation and in home gardening products. It is particularly effective against the Colorado beetle, a serious pest of potatoes (ATSDR 2000). It is compatible with many other active ingredients and may be found in formulations with other active ingredients such as dimethoate, malathion, methomyl, monocrotophos, pirimicarb, triazophos, fenoprop, parathion, petroleum oils, and oxine-copper. It is not compatible with alkaline materials due to hydrolytic breakdown (Herrmann 2003).

Endosulfan was first produced by Farbwerke Hoechst AG in the 1950s and was also manufactured in the USA by FMC Corporation. In 1984, worldwide production was estimated at 10,000 metric tonnes annually. Currently, within the UNECE region, only one company has been reported to produce endosulfan. Located at a site in Germany, the company produces approximately 5000 tpa of the pesticide (Herrmann 2003). There are, however further production sites reported in non-UNECE countries such as Israel, India, South Korea, and, most recently, in China.

Currently the use of endosulfan within the European Union is steadily decreasing. Almost 90 % of the 490 tpa consumed in 1999 were used in the Mediterranean. Spain was the main consumer of endosulfan within the European Union, accounting for almost half of the total volume, followed by Italy (about 20%) and Greece and France (about 15% each). The US uses an average annual amount of 626 tons (Herrmann 2003). Endosulfan is the only remaining pesticide of its class still used in the United States (DeLorenzo *et al.* 2001).

Several classes of pesticides have been reported as substitutes for endosulfan such as organophosphorous pesticides (methamidiphos, phosphomidon and dimethoate), organothiophosphates (oxydemethon-methyl) and pyrethroids (cyfluthrin and beta cyfluthrin) (Herrmann 2003).

Identity

Technical grade endosulfan is a brownish solid that consists of about four parts of α-endosulfan and one part of β-endosulfan. These two stereoisomers are also known as endosulfan 1 and endosulfan 2 (Metcalf 1995). The two endosulfan isomers have different physical properties (Smith 1991). Technical grade endosulfan may also contain up to 2% endosulfan alcohol and 1% endosulfan ether (ATSDR 2000). The main intermediates that are used in the manufacture of endosulfan are hexachlorocyclopentadiene, 1,4-dihydroxy-2-butene and thionyl chloride (Safe 1993, Weil & Sandeer 1997).

Molecular formula: $C_9H_6Cl_6O_3S$
CAS registry numbers: **Endosulfan [115-29-7]**
α-Endosulfan [959-98-8]
β-Endosulfan [33213-65-9]

The chemical names of α-endosulfan are:
(3alpha,5alpha,6alpha,9alpha,9alpha)-6,7,8,9,10,10-hexachloro-1,5,5a,6,9,9a-hexahydro-6,9--methano-2,4,3-benzodioxathiepin 3-oxide or
Hexachloro-5-norbornene-2,3-dimethanol, cyclic sulfite, endo-

The chemical names of β-endosulfan are:
(3alpha,5beta,6beta,9beta,9beta)-6,7,8,9,10,10-hexachloro-1,5,5a,6,9,9a-hexahydro-6,9--methano-2,4,3-benzodioxathiepin 3-oxide or
Hexachloro-5-norbornene-2,3-dimethanol, cyclic sulfite, exo-

Environmental fate

High levels of contamination by endosulfan are limited to the areas where it is manufactured, formulated, applied or disposed of. After endosulfan is applied to crops, it either can persist in the soil as a sorbed phase or be removed through a variety of physical, chemical and biological processes. Endosulfan is a lipophilic compound so it may also bioaccumulate in plants and animals. Studies suggest that secondary emissions of residual endosulfan are continuing to recycle in the global system while they slowly migrate and are redeposited via wet deposition in the Northern Hemisphere (US EPA 2002). Endosulfan has been detected in Arctic snow (Garbarino *et al.* 2002), seawater (Bidleman *et al.* 2003) and the atmosphere (Hung *et al.* 2001 & 2002). The latter studies concluded that the levels of α-endosulfan in the Arctic atmosphere did not decline over the 5 years of monitoring carried out.

The two major metabolites of endosulfan are endosulfan sulphate and endosulfan diol. Endosulfan sulphate is the major metabolite in soil, which is formed through oxidative microbial degradation (Smith 1991, Kullman & Matsumura 1996), and is more persistent than the parent isomers (DeLorenzo *et al.* 2001). Shetty and co-workers (2000) showed in laboratory experiments that the soil fungus, *M. thermo-hyalospora* MTCC 1384, was effective in degradation of endosulfan (more than 80% of applied endosulfan) with formation of the endosulfan diol as a major metabolite. They suggest that fungus *M. thermo-hyalospora* MTCC 1384 could be useful in the detoxification process since endosulfan diol can be further transformed to harmless compounds. Similar findings of a non-oxidative pathway of endosulfan degradation without the formation of endosulfan sulphate were reported in another study (Kwon *et al.* 2002). Pseudomonad microbes have been reported to biodegrade endosulfan to endosulfan alcohol and endosulfan ether (ATSDR 2000). Blue-green algae can transform endosulfan to endosulfan ether and endosulfan lactone (DeLorenzo *et al.* 2001). Additionally, some studies reported a previously unknown metabolite of microbial endosulfan degradation tentatively identified as either endosulfan dialdehyde (Kullman & Matsumura 1996) or endosulfan monoaldehyde (Sutherland *et al.* 2000).

Toxicity

Toxicity to humans

For the general population, the main source of exposure to endosulfan is via ingestion of food that contains endosulfan, as a result of direct pesticide application or through bio-concentration. Endosulfan residues found in food samples were reported by a number of US states in 1988 and 1989. In eight cases contamination was considered to be significant (Minyard & Roberts 1991). In Portugal, levels exceeding the EEC MRL of 1 mg/kg have been found in broccoli (Magalh *et al.* 1989). When administered in large doses endosulfan appears in milk, largely as the endosulfan sulphate (Smith 1991). This metabolite has been reported in cows' milk samples from various locations in Switzerland (Rappe *et al.* 1987). Endosulfan has also been detected in human milk samples from Bhopal in India (Sanghi *et al.* 2003). The levels of endosulfan were found to be the highest among the suite of pesticides analysed in this study with a mean value of 0.363±0.077 mg/l.

Stimulation of the CNS is the main characteristic of endosulfan poisoning. Symptoms of acute exposure include hyperactivity, tremors, decreased respiration, salivation, anaemia and a loss of co-ordination as well as the ability to stand (Smith 1991). Other signs of poisoning include gagging, vomiting, diarrhoea, agitation, convulsions and loss of consciousness. It was shown that endosulfan can induce apoptosis in a human T-cell leukemic cell line which may have direct relevance to loss of T cells and thymocytes *in vivo*. These data also strongly suggest that mitochondrial dysfunction and oxidative stress result from endosulfan toxicity (Kannan *et al.* 2000).

Endosulfan may be lethal to humans through inhalation and oral or dermal exposure. A tragic case of extensive food contamination occurred in Sudan in 1991. Maize, which had been intentionally treated with endosulfan for use as poisonous bait for birds, was instead used in baking flour, causing the deaths of thirty-one people (Dinham 1993). There are some other records of accidental and intentional poisoning. A 70 year-old woman died approximately three hours after taking 'drops' of an endosulfan formulation. An unsuccessful suicide resulted in an acute cardiac and convulsion stage, a sub-acute pulmonary and convulsive stage and finally a slow recovery. After one month's occupational exposure, a worker reported 'flu-like' symptoms and headaches followed the next day by collapse, convulsions and temporary unconsciousness and amnesia. Subsequent effects were consistent with epilepsy (Smith 1991).

Additionally, it has been discovered that endosulfan and endosulfan sulphate produce estrogenic effects on human breast estrogen-sensitive cells – effects that are comparable with those of DDT on the same cells (Soto *et al.* 1994).

Environmental toxicity

Exposure to endosulfan can result in both acute and chronic impacts on terrestrial and aquatic organisms (US EPA 2002). Endosulfan's persistence and its high toxicity to fish are of special concern and numerous studies have investigated the risks and adverse effects of run-off water on non-target organisms. Technical grade endosulfan 96h LD_{50} values of 2.0μg/l at 30^0C and 4.6μg/l at 35^0C have been determined for mosquito fish (Nowak & Sunderam 1991). The reported LC_{50} for rainbow trout is 1.4μg/l. The No Observed Effect Concentration (NOEC), based on growth and reproduction, is 49μg/l for *Daphnia cephalata*

(Barry *et al.* 1996). At concentrations greater than 50ng/l, pathological changes in mitochondria and peroxisomes have been observed as a result of endosulfan exposure consistent with its acting as a mixed-type inducer of MFO systems in fish hepatocytes (Arnold *et al.* 1996). *Channa punctatus* suffered ovarian steroidogenesis inhibition at sub-lethal concentrations (Inbaraj & Haider 1988). In catfish, the highest levels of accumulated endosulfan were found in the liver, the kidney and the brain. The distribution was probably influenced by the lipid content in the fish (Nowak 1991). Endosulfan sulphate is as toxic to aquatic organisms as its parent compound and apparently more potent than the β-isomer of endosulfan. It has been extensively researched because of links to fish deaths (Smith 1991).

Studies on experimental animals concluded that the main target of endosulfan is the central nervous system. However, the liver, the kidney, the gastrointestinal, haematopoietic and dermal systems and the developing foetus can also be affected (Nowak 1991). Moreover, it was found that α-endosulfan is more toxic than β-endosulfan (Smith 1991). Reported toxic effects of endosulfan in exposed animal livestock included ataxia, progressing to total inability to stand, blindness (with full recovery after a month), acute illness and death (Smith 1991). Neuromuscular symptoms associated with organochlorine toxicosis (such as abnormal posture and gait, apprehension, hypersensitivity, belligerence, loss of coordination and unceasing chewing movements) were observed in exposed cattle. These signs of intense nervous system stimulation may be caused by impairment of glutamine synthesis, resulting in elevated ammonia concentrations in the brain and enhanced neurotransmitter release at cholinergic synapses (Nowak 1991).

Oral LD_{50} values of 43 mg/kg for male rats and 18 mg/kg for female rats have been determined in laboratory tests. Observed sub-lethal effects included liver enlargement, seizures, reduced growth and survival, raised blood sugar, more aggressive behaviour, depressed immune responses and interstitial changes in the kidneys of male rats (Smith 1991). Oral doses of or exceeding 5mg/kg/day during gestation increased both the mortality rate of rat dams and the incidence of resorption and skeletal abnormality of their foetuses. Doses of 10mg/kg/day caused degeneration and weight loss of testes in the male rats (Smith 1991, ENDS 1996).

Endosulfan has also been recognised as possessing estrogenic activity in aquatic and terrestrial species. The observed effects caused by endosulfan exposure were as follows: impaired development in amphibians, reduced cortisol secretion in fish, impaired development of the genital tract in birds, and altered hormone levels, testicular atrophy and reduced sperm production in mammals (Herrmann 2003). It was also found that endosulfan inhibits the moulting of the cladoceran *Daphnia magna* (Zou & Fingerman 1997 & 1999).

Legislation

Endosulfan is subject to a number of regulations and action plans worldwide. In the EU it is regulated under Directive 91/414/EEC concerning the placing on the market of plant protection products (EEC 1991), which is currently under the peer review programme at the Community level (Herrmann 2003). If endosulfan is excluded from Annex I to the Directive (a list of active substances authorised for incorporation in plant protection products), use of any plant protection products containing endosulfan will cease in all EU member states. Until then, diverting national legislation continues to apply. This means that agricultural use of endosulfan is still authorised in 11 EU member states, while it is banned in all others. Worldwide, endosulfan has been banned in the following countries:

Denmark, Germany, Netherlands, Sweden, Belize, Singapore and the Brazilian state of Rondonia. Colombia and Indonesia are preparing for a ban on endosulfan. Its use is not allowed in rice fields in Bangladesh, Indonesia, Korea and Thailand. Use of endosulfan is restricted or severely restricted in: Canada, Finland, Great Britain, Kuwait, the Philippines, Russia, Sri Lanka, Thailand and Madagascar (PAN UK 2000).

Endosulfan and its metabolites have also been included in the OSPAR List of Chemicals for Priority Action (OSPAR 2002) as highly persistent, bioaccumulative, very toxic and potentially endocrine disruptive chemicals to all organisms. Endosulfan was proposed as a priority substance under the Water Framework Directive (EC 2000) and is subject to a review for identification as a possible "priority hazardous substance" before 31 December 2003.

Numerous international, national and state regulations and guidelines pertaining to endosulfan and its metabolites in air, water and other media are summarised in the toxicological profile for endosulfan prepared by the Agency for Toxic Substances and Disease Registry (ATSDR 2000).

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A3.3 HEXACHLOROCYCLOHEXANES (HCHS)

Production and major uses

Hexachlorocyclohexane (HCH) is an organochlorine insecticide that is used for the control of grasshoppers, insect pests of cotton and of rice, wireworms, and other soil pests. Technical grade HCH is a mixture of isomers: α-HCH, β-HCH, γ-HCH (also known as Lindane), δ-HCH, and ε-HCH. Lindane was widely used as an insecticide until the 1990s in almost all regions for treatment of poultry and livestock, and for control of household insects. It is still used as a scabicide and pediculocide, usually in the form of lotions, creams, and shampoos (ATSDR 2000).

Current production of lindane as well as marketed volumes is steadily decreasing Production is known to have definitely taken place in the Czech Republic, France, Germany, Spain, Poland and the U.S. Past production in Denmark, Monaco, the Netherlands, Switzerland and the United Kingdom is not clear. Only very rough estimates (if any) of years of production and on produced volumes exist for these countries. Since no current production could be identified in 2001 for any of the UNECE regions it was concluded that all continuing uses of lindane products are based either on imported active substance or domestic stockpiles. Germany, Spain, Switzerland, UK and the US declared that lindane is imported to their countries. Croatia prohibited the import of lindane from 2001 onwards. Export of formulated products took place from the US and from Germany. Lindane is not produced or manufactured in Canada. In Latvia lindane was imported and used from 1995 to 2000. Neither import nor use is reported after 2000 (Hauzenberger *et al.* 2002).

Identity

Manufacture of technical grade hexachlorocyclohexane involves photochlorination of benzene, which yields an isomeric mixture. The approximate isomer content is α-HCH (65-70%), β-HCH (7-10%), γ-HCH (14-15%), δ-HCH (approximately 7%), ε-HCH (1-2%) and other components (1-2%)(IPCS 1991). One of the other components present in technical grade HCH is heptachlorocyclohexane, which is thought to be largely responsible for the distinctive odour of the mixture (IPCS 1991). Lindane, the gamma isomer of HCH, is commercially produced by purification of the technical HCH mixture (Thomas & Colborn 1992, Safe 1993). Historically, this has led to large quantities of residues arising from lindane production.

Molecular formula:	**$C_6H_6Cl_6$**	
CAS registry numbers:	**α-HCH**	**[319-84-6]**
	β- HCH	**[319-85-7]**
	γ- HCH	**[58-89-9]**
	δ- HCH	**[319-86-8]**
	ε- HCH	**[6108-10-7]**

The chemical names of **α-HCH** are:
(1alpha,2alpha,3beta,4alpha,5beta,6beta)-1,2,3,4,5,6-hexachlorocyclohexane or
alpha-1,2,3,4,5,6-Hexachlorocyclohexane or
alpha-Hexachlorocyclohexane or
alpha-benzene hexachloride or
alpha-BHC

The chemical names of **β-HCH** are:
(1alpha,2beta,3alpha,4beta,5alpha,6beta)-1,2,3,4,5,6-hexachlorocyclohexane or
beta-1,2,3,4,5,6-Hexachlorocyclohexane or
beta-Hexachlorocyclohexane or
beta-benzene hexachloride or
beta-BHC

The chemical names of **γ-HCH** are:
(1alpha,2alpha,3beta,4alpha,5alpha,6beta)-1,2,3,4,5,6-hexachlorocyclohexane or
gamma-1,2,3,4,5,6-hexachlorocyclohexane or

gamma-hexachlorocyclohexane or
gamma-benzene hexachloride or
gamma-BHC or
Lindane

The chemical names of **δ-HCH** are:
(1alpha,2alpha,3alpha,4beta,5alpha,6beta)-1,2,3,4,5,6-hexachlorocyclohexane or
delta-1,2,3,4,5,6-Hexachlorocyclohexane or
delta-Hexachlorocyclohexane or
delta-benzene hexachloride or
delta-BHC

The chemical names of **ε-HCH** are:
(1alpha,2alpha,3alpha,4beta,5alpha,6beta)-1,2,3,4,5,6-hexachlorocyclohexane or
epsilon-1,2,3,4,5,6-Hexachlorocyclohexane or
epsilon-Hexachlorocyclohexane or
epsilon-benzene hexachloride or
epsilon-BHC or
Picloram

Environmental fate

Among the five isomers of HCH listed above, α-HCH, β-HCH, and γ-HCH are of the most concern in terms of the environmental toxicity. Lindane, the γ-HCH isomer, may be introduced into environment both as lindane itself or as a component of the technical mixture. It can undergo long-range transport and be deposited into aquatic systems, where it becomes localised in sediments. Lindane has been detected in air and in water (Bidelman *et al.* 1995), plants (Strachan *et al.* 1994), birds and mammals (Smith 1991, Junqueira *et al.* 1994), and humans (Pesendorfer *et al.* 1973, Acker 1974, Inoue *et al.* 1974, Pramanik & Hansen 1979).

In laboratory experiments it was shown that, in air or aqueous solution, lindane undergoes photodegradation with formation of 1,2,3-trichlorobenzene, γ-2,3,4,5,6-pentachloro-cyclohex-1-ene, and α-HCH and then can be completely mineralised to CO_2 and HCl under the UV light irradiation in the presence of TiO_2, Fe_2O_3 and other semiconductors (Zaleska *et al.* 2000).

Lindane can persist for a long time in the treated soils. Some sources report half-life for lindane in soils from 13 to 240 days (Howard *et al.* 1991) depending on the soil type; another study found that its half-life may be in order of 3 to 4 years (Martijn *et al.* 1993). In the soil lindane can undergo bioisomerization forming α-, β-, δ-HCH**,** but not ε-HCH (Waliszewski 1993). Isomers of HCH can be degraded in the soil by various bacteria (Sahu *et al.* 1990, Jacobsen *et al.* 1991, Imai *et al.* 1989). However, β-HCH appeared to be more resistant to degradation than α- and γ-isomers (Sahu *et al.* 1990).

HCH isomers may bioaccumulate in tissues of animals. The β-isomer is more persistent than other HCH isomers (see: Johnston 1989) as the α- and γ-isomers do not concentrate highly through the food chain.

Toxicity

Toxicity to humans

Human intake of HCH compounds is largely through food consumption (Toppari *et al.* 1995). HCHs are lipophilic compounds and they tend to bioaccumulate in the fatty tissues of organisms. In humans, therefore, lindane mostly concentrates in adipose tissue. Like many persistent organochlorines, HCH levels in the body have been found to increase with age (ASTDR 2000). It was also found that women chronically exposed to lindane or living in areas of intense usage, contained residues of lindane in breast milk (Larsen *et al.* 1994, Schlaud *et al.* 1995, Schoula *et al.* 1996). Other studies demonstrated that among three isomers detected in human breast milk (α-, β- and γ-HCHs) the β-isomer was the most ubiquitous. The generally less widespread nature of the α- and γ-isomers in comparison to β-HCH is due to the more rapid clearance of these isomers from the body (National Research Council 1993).

All isomers of HCH are toxic compounds. The α-, β- and δ-HCH isomers mainly act as a depressant of the nervous system (Smith 1991, Nagata *et al* 1996). β-HCH was reported as a possible representative of a new class of xenobiotics that produces estrogen-like effects through non-classical mechanisms and, therefore, may be of concern with regard to breast and uterine cancer risk (Steinmentz *et al.* 1996). Several authors have reported that the genotoxicity of lindane and other isomers of hexachlorocyclohexane is now clearly established (Mattioli *et al.* 1996, Dubois *et al.* 1997).

It is thought that humans chronically exposed to γ-HCH or similar insecticides and pesticides are much more susceptible to liver damage when using paracetamol as a pain reliever. Exposure may also cause a serious drop in the blood cell count (aplastic anaemia) or in the white blood cell count (agranulocytopenia) (Itinose *et al.* 1995).

Lindane and other HCH isomers are reasonably anticipated to be a human carcinogen (DHHS 2002).

Environmental toxicity

Hexachlorocyclohexanes introduced to the environment from industrial discharges, insecticide applications, or spills, can cause significant damage. Acute toxic effects may include the death of animals, birds, or fish, and death or low growth rate in plants (Bunton 1996, Smith 1991). It has been found that lindane and other organochlorine compounds can be transferred through the pathway soil-earthworm-bird/mammal (Romijn *et al.* 1994) causing secondary poisoning.

Acute poisoning of animals by lindane causes increased respiratory rate, restlessness accompanied by increased frequency of micturition, intermittent muscular spasms of the whole body, salivation, grinding of teeth and consequent bleeding from the mouth, backward movement with loss of balance and somersaulting, retraction of the head, convulsions, gasping and biting, and collapse and death usually within a day. Chronic toxic effects may include shortened lifespan, reproductive problems, lower fertility and changes in appearance or behaviour (Smith 1991). Lindane has also been shown to cause liver, lung, endocrine gland and other types of cancer in animals (Bunton 1996, Smith 1991).

Poisoning of animals by α-HCH is characterised by tremors of the extremities and an inability of the animals to make co-ordinated movements. α-HCH has been found to concentrate mainly in the white matter of the brain after intraperitoneal injection to experimental animals and stayed in the brain for more than 24 hours (Smith 1991).

The β-isomer produces lameness and a peculiar flaccidity in the entire musculature. Poisoning by δ-isomer is characterised by prostration, the animals remaining motionless for days (Smith 1991).

All of the isomers of HCH induce liver microsomal enzymes. It was found that α-HCH is more tumourigenic in mice and rats than β-, γ- and δ-isomers; and it was suggested that the α-isomer probably is responsible for the tumourigenic action of technical grade hexachlorocyclohexane. β-HCH was found to be carcinogenic only when administered together with a polychlorinated biphenyl (Smith 1991).

Lindane has high chronic toxicity to aquatic life. The insecticide load in surface waters does not ordinarily reach concentrations acutely toxic to aquatic fauna. The effects of the low insecticide concentrations often appear only after relatively long exposure times. Chronic exposure to insecticides, such as lindane, (Schulz *et al.* 1995) can be hazardous to freshwater macro-invertebrates even at unexpectedly low concentrations. The low-concentration effects may depend on both species and substance and therefore cannot be predicted from toxicity data at higher concentrations.

Legislation

Hexachlorocyclohexane is subject to European Community legislation as a toxic, persistent and bioaccumulative chemical. Stringer & Johnston (2001) presented a comprehensive overview of hexachlorocyclohexane legislation including instruments formulated under EU Council Directives together with the OSPAR, LRTAP and PIC Conventions. The limit values and quality objectives for discharges of hexachlorocyclohexane are set by the Council Directive 84/491/EEC (EEC 1984) as subsequently amended. The uses of hexachlorocyclohexane (including lindane) were severely restricted under the Persistent Organic Pollutants (POPs) Protocol, which was adopted in 1998 and has 36 contracting parties encompassing not only Europe but also Canada and the United States of America (UNECE 1998). The POPs Protocol is part of the 1979 Convention on Long-Range Transboudary Air Pollution (LRTAP), which falls under the auspices of the United Nations Economic Council for Europe.

Lindane is also included in the Annex III of the 1998 Rotterdam Convention on the Prior Informed Consent procedure (PIC procedure) among 27 other chemicals (FAO/UNEP 1998). Under the PIC procedure, countries should not export any chemical to any other country without first receiving explicit permission. In order to avoid unfair trade barriers arising through the implementation of the Convention, any country that has denied import of any chemical must also stop producing it domestically and may not import it from any country that is not a Party to the Convention.

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A3.4 CHLORINATED BENZENES

Production and major uses

Both technological changes and environmental concerns have severely affected the production of chlorobenzenes; today only monochlorobenzene and 1,2- and 1,4-dichlorobenzenes are manufactured in large quantities. These are often produced together, with the economically optimised reaction yielding approximately 85% monochlorobenzene, 10% 1,4-dichlorobenzene and 5% 1,2-dichlorobenzene. Monochlorobenzene yield can be increased to 90% by careful monitoring of the reaction mix density and recycling of unreacted benzene, but total elimination of dichlorobenzene formation is not economical. Should the primary interest be in the para-isomer, yield may be increased by use of a selective catalyst, or the mix can be further chlorinated to produce a mixture of 1,4-dichlorobenzene and 1,2,4-trichlorobenzene. These two products can easily be separated by distillation (Bryant 1993, CEC 1986).

One of the earliest uses of monochlorobenzene was as an intermediate for the explosive picric acid during the First World War (CEC 1986). It is used as a solvent and as an intermediate in chemical synthesis. In the US in the 1980s, the predominant use was for the production of ortho- and para-chlorobenzenes. Theses are used as intermediates for rubber chemicals, antioxidants, dyes and pigments, pharmaceuticals and agricultural chemicals. The fungicide benomyl, and carbofuran and the parathion group of insecticides are all derived from chlorobenzene. One previously important use was in the manufacture of DDT. Chlorobenzene production has fallen due to the development of other routes to aniline and phenol and the restriction of DDT use. By various routes, chlorobenzene is also used for the manufacture of specialty silicones, Grignard reagents and catalysts (Bryant 1993).

1,2-Dichlorobenzene is produced unavoidably in the production of monochlorobenzene, but it is also possible to maximise dichlorobenzene production to 98% of the reaction mixture using suitable catalysts or alternative production methods leading to specific isomers. It is used mainly in the production of dyes and pesticides after conversion to 1,2-dichloro-4-nitrobenzene or dichloroaniline. Other uses include the solvent phase in the production of toluene di-isocyanates, production of deodorants and disinfectants and on a small scale as a heat transfer fluid. According to Meek *et al.* (1994b), the largest use is in degreasing for the metal and automotive industries.

1,3-Dichlorobenzene is growing in importance as a starting product in the manufacture of dyes, pesticides and pharmaceuticals. However, this has not yet reached commercial importance. There are some other small, specialised uses, but larger markets have not been developed, mainly because 1,3-dichlorobenzene only occurs as a minor constituent (approx 1%) of the technical dichlorobenzene reaction mix, and to produce it by other routes is expensive (Bryant 1993). 1,4-Dichlorobenzene (p-dichlorobenzene) is used largely in the production of deodorant blocks and room deodorants. It is also used as a moth control agent, as an insecticide and an intermediate for production of insecticides and dyes. An emerging market is in the manufacture of poly(phenylene sulphide) resin (PPS), and minor uses are as a germicide, fungicideand extreme pressure lubricant (Bryant 1993, CEC 1986).

1,2,3- and 1,2,4-trichlorobenzene have been produced from the dehydrohalogenation of the unwanted isomers of the production of the pesticide 1,2,3,4,5,6-hexachlorocyclohexane. This is of limited application.

Tetrachlorobenzenes are no longer used or produced in Canada and releases come only from dielectric fluid spills and long-range transport. At one time, 1,2,4,5-tetrachlorobenzene was used in the production of 2,4,5-trichlorophenol on a large scale, but this use has now been discontinued (Giddings *et al.* 1994b).

Pentachlorobenzene is not produced anymore in the UN-ECE and it does not occur on the HPV-LPV Chemicals Data Base of the European Chemicals Bureau (ECB). No data are available on production, recent or past, outside the UN-ECE region (van de Plassche *et al.* 2002) with the exception of one source reporting that pentachlorobenzene is no longer manufactured or used in Canada (Giddings *et al.* 1994c).

Hexachlorobenzene (HCB) was used as a wood preservative, as a fungicide for treating seeds, and as an intermediate in organic syntheses (Budavari *et al.* 2000). HCB is not currently manufactured in the US as a commercial end-product (ATSDR 2000). However, hexachlorobenzene may be formed as an unwanted by-product in the synthesis of other organochlorine compounds and from high-temperature sources (Newhook & Meek 1994, Sala *et al.* 1999).

Identity

Chlorinated benzenes formed by direct chlorination of benzene in the liquid phase with a ferric chloride as a catalyst. Only limited control can be exerted over the final product mix. The distillation train used for separating the mixture has a limited resolving power and the distillates are always mixtures of close boiling isomers which can be further separated by crystallisation (see eg Bryant 1993). Distillation also gives rise to chlorinated tars.

Chlorobenzene, 1,2-dichlorobenzene and 1,3-dichlorobenzene are colourless liquids; 1,4-dichlorobenzene forms colourless crystals at room temperature (Ware 1988a & b). 1,2,3- and 1,3,5-trichlorobenzene are solids and 1,2,4-trichlorobenzene is a liquid (Budavari *et al.* 2000). Tetrachlorobenzenes, pentachlorobenzene and hexachlorobenzene are white solids (van de Plassche *et al.* 2002, ATSDR 2000).

12 chlorinated benzenes are possible, with substitution patterns as follows:
1 chlorine monochlorobenzene,
2 chlorines 1,2-di-, 1,3-di- and 1,4-dichlorobenzenes
3 chlorines 1,2,3-tri-, 1,2,4-tri- and 1,3,5-trichlorobenzenes
4 chlorines 1,2,3,4-tetra-, 1,2,3,5,-tetra- and 1,2,4,5-tetrachlorobenzenes
5 chlorines pentachlorobenzene
6 chlorines hexachlorobenzene

Molecular formula: **C_6H_5Cl**
CAS registry number: **Monochlorobenzene [108-90-7]**

Molecular formula: **$C_6H_4Cl_2$**
CAS registry numbers: **1,2-Dichlorobenzene [95-50-1]**
1,3-Dichlorobenzene [541-73-1]
1,4-Dichlorobenzene [106-46-7]

Molecular formula: **$C_6H_3Cl_3$**
CAS registry numbers: **1,2,3-Trichlorobenzene [87-61-6]**
1,2,4-Trichlorobenzene [120-82-1]
1,3,5-Trichlorobenzene [108-70-3]

Molecular formula: **$C_6H_2Cl_4$**
CAS registry numbers: **1,2,3,4-Tetrachlorobenzene [634-66-2]**
1,2,3,5-Tetrachlorobenzene [634-90-2]
1,2,4,5-Tetrachlorobenzene [95-94-3]

Molecular formula: **C_6HCl_5**
CAS registry number: **Pentachlorobenzene [608-93-5]**

Molecular formula: **C_6Cl_6**
CAS registry number: **Hexachlorobenzene [118-74-1]**

Mono- and dichlorobenzenes (DCBs)

Monochlorobenzene release to the environment is expected to derive from its use as a solvent, either through fugitive emissions or volatilisation from pesticides for which it used as a carrier. Thus, inhalation is thought to be a major route of exposure for humans since it is rarely if ever found in food. It bioaccumulates in algae, fish and aquatic invertebrates. Mammalian metabolites are reported to be p-chlorophenol, p-chlorocatechol and p-chlorophenyl mercapturic acid. Human exposure to monochlorobenzene causes CNS depression and respiratory tract irritation and animal studies have reported liver necrosis, renal toxicity and effects on the pancreas, blood and lymph and adrenal glands (Ware 1988a, Meek *et al.* 1994a). Canada has derived a TDI of 8.1ug/kg body weight/day;

estimated exposures (0.05-0.14ug/kg/day) are considerably lower than this (Meek *et al.* 1994a).

Ware (1988b) reports human symptoms after exposure to DCBs, but does not distinguish between isomers. Effects reported are anaemia, skin lesions, vomiting, headaches, eye and respiratory tract irritation, anorexia, weight loss, yellow atrophy of the liver, blood dyscrasias, porphyria, and chromosomal breaks in blood samples. Animal experiments recorded liver and kidney damage to be the most frequent effects, though high doses caused CNS perturbation and death through respiratory depression. The dichlorobenzenes are bioaccumulative in algae, aquatic invertebrates and fish (Ware 1988b). All three have also been reportedly found in blood (Ware 1988b).

Laboratory animals exposed to 1,2-dichlorobenzene exhibited hepatic, renal and haematological effects as well as lymphoid depletion of the thymus and spleen and multifocal mineralisation of both muscular and heart muscles (Ware 1988b, Meek *et al.* 1994b). Developmental toxicity was only observed at concentrations that were overtly toxic to the mother. Human toxicity data are sparse, but chromosomal aberrations, anaemia and leukemia have been reported (Meek *et al.* 1994b). Mammals metabolise 1,2-dichlorobenzene to phenols and catechols, most of which are excreted after conjugation with glucoronic or sulphuric acids. Mercapturic acids may also be produced. The primary metabolites in humans are conjugated phenols (Ware 1988b). 1,2-dichlorobenzene is found in air, food, breast milk and drinking water (Meek *et al.* 1994b). It is also toxic to higher plants, inducing abnormal mitosis (cell division) in onions (Ware 1988b).

Mammalian (and human) metabolism for 1,3-dichlorobenzene is as for 1,2-dichorobenzene above, but generally little is known about this 1,3-dichlorobenzene in comparison to the more commercially important dichlorobenzenes.

1,4-Dichlorobenzene may be absorbed both through the inhalation of vapours, through the skin and though consumption of contaminated food. Human symptoms include damage to the liver, kidneys and lungs. Accidental poisoning of children, presumably who have eaten moth repellent was widespread in the 1970s (CEC 1986). Once absorbed, 1,4-dichlorobenzene is stored in the adipose tissue and has been detected in human samples (CEC 1986, Ware 1988b). The metabolism of 1,4-dichlorobenzene by mammals varies from that of the other two isomers in that mercapturic acids are not formed. 1,4-dichlorobenzene causes abnormal mitosis in higher plants. 1,4-Dichlorobenzene has been reported in human adipose tissue, as well as in blood (Ware 1988b).

Trichlorobenzenes

Environmental regulations have curbed the use and discharge of trichlorobenzenes to the environment, as least in Europe and the USA (Harper *et al.* 1992, Bryant 1993). Not surprisingly, therefore, little research appears to have been carried out in comparison with some other chlorobenzenes. The general human population would probably receive their greatest exposure to trichlorobenzenes through inhalation. The toxicity of all three appear similar; they damage the liver, kidney and thyroid. There is some indication of slight fetotoxicity at high doses. There is little evidence of mutagenicity and too few data are available for the trichlorobenzenes to given a carcinogenicity classification (Giddings *et al.* 1994a). All three isomers are toxic to phytoplankton (Sicko-Goad *et al.* 1989a-d, Sicko-Goad & Andresen 1993a & b).

1,2,3-Trichlorobenzene has been detected in air, drinking water, food and breast milk (Giddings *et al.* 1994a) as well as industrially polluted surface waters (Harper *et al.* 1992), though it was not found in human adipose tissue from Canada (Hermanson *et al.* 1997). Little is known about its toxicity other than its ability to damage the liver, kidney and thyroid (Giddings *et al.* 1994a).

More information is available about 1,2,4-trichlorobenzene. According to Giddings *et al.* (1994a), only 1,2,4-trichlorobenzene has industrial application in Canada. It is imported for solvent and intermediate use. Environmental releases come from industrial discharges and from spillage of dielectric fluids. As mentioned above, it is toxic to the liver, thyroid and kidney. Liver and kidney weights and porphyrin excretion increase. In some studies, more severe liver damage has occurred, including necrotic and non-necrotic degeneration. 1,2,4-Trichlorobenzene may be found in all environmental media, though there is insufficient analytical data to tell how widespread contamination is and it was not found in human adipose tissue from Canada (Hermanson *et al.* 1997).

Giddings *et al.* (1994a) report 1,3,5-trichlorobenzene air, drinking water, food, breast milk, though it was not found in human adipose tissue from Canada (Hermanson *et al.* 1997). It can be found in association with industrial operations (Harper *et al.* 1992) including PVC industry (Johnston *et al.* 1993).

Tetrachlorobenzenes

Giddings *et al.* (1994b) reviewed toxicity and exposure data for the tetrachlorobenzenes. There are not expected to be large differences between the behaviour of the isomers. Uptake of 1,2,4,5-tetrachlorobenzene was studied in rainbow trout. It is not volatile enough to evaporate from water easily, and is accumulated by the fish, through its gills. Bioaccumulation depended upon the rate of activity and oxygen uptake of the fish, and only the low water solubility prevented significant toxicity occurring (Brauner *et al.* 1994).

The greatest exposure of the general population is probably through food. All isomers were found to affect the liver, kidney, thyroid and lungs, with 1,2,4,5-tatrachlorobenzene being the most toxic. Not enough information was available to classify tetrachlorobenzenes as to carcinogenicity.

In addition to the effects noted above, 1,2,4,5-tetrachlorobenzene has also caused changes in the spleen, thymus, lymph nodes and haematological parameters in animals (Giddings *et al.* 1994b). An increase in chromosomal aberrations was seen in workers exposed to 1,2,4,5-tetrachlorphenol at a pesticide manufacturing complex (Giddings *et al.* 1994b).

In rats, 1,2,3,4- and 1,2,3,5-tetrachlorobenzene caused reduction in the number of live offspring at concentrations too low to adversely affect the mother (Giddings *et al.* 1994b).

All isomers have been detected in ambient air, drinking water and food and 1,2,3,4- and 1,2,3,5-tetrachlorobenzene have been identified in breast milk (Giddings *et al.* 1994b), though none of the isomers were detected in Canadian human adipose tissue (Hermanson *et al.* 1997).

Pentachlorobenzene

Pentachlorobenzene has been detected in air, drinking water, food and breast milk (Giddings *et al.* 1994b), though according to Hermanson *et al.* (1997) it was found in less than 15% of human adipose samples collected in Ontario, Canada.

Animal studies demonstrate weight loss and effects on the liver, thymus, kidney, adrenal glands and digestive tract. Anaemia and malformation of sperm also occurred. There is some indication of fetotoxicity and developmental toxicity. The thyroid was impacted, with and thyroid hormone (free and total thyroxin) concentrations reduced. Pentachlorobenzene cannot be assigned a carcinogenicity classification because of lack of data. Pentachlorbenzene accumulates in, and is toxic to algae (Sicko-Goad *et al.* 1989d).

Hexachlorobenzene

Once introduced into environment, HCB strongly absorb to soil materials and almost no desorption take place (Bahnick & Doucette 1988). It is bioaccumulative and biomagnifies. It can be measured in ambient air, drinking water, soil, food and breast milk (Newhook and Meek 1994).

HCB is toxic to aquatic life, land plants, land animals and humans. It is listed by the IARC as a Group 2B carcinogen, i.e. possible carcinogen to humans and appears to be a tumour promoter. Hexachlorobenzene may damage the developing foetus, liver, immune system, thyroid and kidneys and CNS. The liver and nervous system are the most sensitive to its effects. Porphyria is a common symptom of HCB toxicity. High or repeated exposure may damage the nervous system, and can cause irritability, difficulty with walking and co-ordination, muscle weakness, tremor and/or a feeling of pins and needles on the skin. Repeated exposure, especially when skin effects occur, can lead to permanent skin changes, such as changes in pigmentation, tight, thickened skin, easy wrinkling, skin scarring, fragile skin, and increased hair growth, especially on the face and forearms (ATSDR 2000, Newhook & Meek 1994, van Birgelen 1998). Research suggests that HCB has dioxin-like toxicity and that, based on a preliminary toxic equivalence factor (TEF) of 0.0001, HCB could contribute significantly to the dioxin-type toxicity of human milk based on PCB/PCDD/PCDF toxicity equivalents. In many countries, this could mean an increase of 10%-60%, but in countries with high HCB exposure levels, the effects could be even greater. In Spain and the Czech Republic, inclusion of HCB in total breastmilk TEQ estimates could lead to totals 6 times higher than based only on PCBs and PCDFs. Slovakia and India also have very high HCB levels; other countries (eg Austria) high levels in previous decades. It has been suggested that more epidemiological studies should be undertaken, especially in the most highly contaminated countries (van Birgelen 1998).

With the exception of occupational settings, almost all human exposure occurs via food. The greatest body of information on HCB toxicity to humans derives from an incident in Turkey between 1955 and 1959, when HCB-treated grain was made into bread. More than 600 people experienced porphyria cutanea tarda. Children of exposed women had skin lesions and 95% of them died at less than one year old. In the long term (20-30 years), some people continued to have abnormal porphyrin biochemistry and neurological, orthopaedic and dermatological symptoms persisted. Hexachlorobenzene is also thought to have caused porphyria cutanea tarda in populations exposed industrially and through food (Newhook & Meek 1994). High concentrations of HCB were found in the air around a

chlor-alkali and organochlorine manufacturing plant at Flix in Spain and in blood of workers and local residents (Sala *et al.* 1999, Grimalt *et al.* 1994). One study found a significant elevation in incidence of cancer of the thyroid, soft tissues and at unspecified sites in the men of the community (Grimalt *et al.* 1994) and the authors of one study stated that HCB exposure was associated with specific health effects in the most highly exposed subjects (Sala *et al.* 1999).

Legislation

Hexachlorobenzene is the most regulated chemical among all chlorinated benzenes. The UNECE (1998) lists HCB alongside PCDD/Fs and PAHs as being the most important POPs emitted from stationary sources. HCB is one of twelve priority POPs intended for global action by the UN Environment Programme (UNEP) Governing Council. It is intended that HCB will be phased out worldwide under a convention currently being drawn up (UNEP 1995, 1997). Furthermore, HCB is included on Annex I of the Draft UNECE POPs Protocol under the Convention on Long-Range Transboundary Air Pollution (LRTAP)(UNECE 1998). Within the EC, discharges of HCB are controlled as stipulated by EC Council Directive 86/280/EEC, which amends Directive 76/464/EEC, regarding pollution caused by certain dangerous substances discharged into the aquatic environment (EEC 1986, 1976). HCB is also included in the list of priority hazardous substances agreed by the Third and Fourth North Sea Conferences (MINDEC 1990, 1995), where continuous reduction of all hazardous substances was agreed with the ultimate aim of reducing environmental concentrations of hazardous substances to near background levels (synthetic substances to zero) within the next 25 years. The 1998 Ministerial Meeting of the OSPAR Commission (OSPAR 1998a) further reinforced these objectives. HCB is included on the OSPAR 1998 List of Candidate Substances, Annex 3 of the OSPAR Strategy with regard to Hazardous Substances (OSPAR 1998b). In addition, HCB is regulated under the 1995 Barcelona Convention, the Rotterdam (PIC) Convention and the IJ has called for all uses to be eliminated.

Pentachlorobenzene is one of the priority substances of the recently adopted EU Water Framework Directive (EC 2000). The EU will propose community-wide water quality standards and emission controls for these priority substances. Within the list of these priority substances a group of priority hazardous substances is identified which are of particular concern for the freshwater, coastal and marine environment. These substances will be subject to cessation or phasing out of discharges, emissions and losses within an appropriate timetable that shall not exceed 20 years. Pentachlorobenzene is on the OSPAR DYNAMEC list in Group V: Substances with PTB properties, but which are heavily regulated or withdrawn from the market (OSPAR 2000). Recently pentachlorobenzene was proposed as a candidate for inclusion in the Protocol on POPs based on its potential for long-range atmospheric transport, persistence (in water, sediment and soil), bioaccumulation and (eco)toxicity (van de Plassche *et al.* 2002).

Trichlorobenzenes are regulated in the EU under the Council Directive 90/415/EEC, which sets limit values for discharges of all three isomers of trichlorobenzene. This Directive applies in particular to those trichlorobenzenes used as a solvent or colouring support in the textile industry, or as a component of the oils used in transformers (EEC 1990).

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A3.5 MERCURY (HG)

Production and major uses

Mercury is produced from mercuric sulfide cinnabar ore, and is also recovered from secondary sources such as mercury containing wastes (ATSDR 2000, UNEP 2002).

Uses of mercury are extensive, due to its unique properties of fluidity, its high surface tension and its ability to alloy with other metals. It is primarily used in the electrical industry in alkaline batteries, electric lamps and wiring and switching devices, such as thermostats and cathode tubes. It is also used in the chemical industry as a catalyst (Matthews 1996), and as the cathode in the chlor-alkali electrolytic separation of brine to produce chlorine and sodium hydroxide (caustic soda) (ATSDR 2000). Metallic mercury is also used in dental restorations, and in medical equipment such as thermometers and manometers (ATSDR 2000).

Mercuric oxide and sulphide are used as pigments in paints (ATSDR 2000). Mercury compounds historically were also used extensively as pharmaceuticals and agrochemicals, e.g. as components of antiseptics, diuretics, skin lightening creams, laxatives, anti-syphilitic drugs, fungicides, bactericides, wood and felt preservatives. However, due to the high toxicity of mercury, most of these applications are banned in most parts of the World

(ATSDR 2000, UNEP 2002). Mercury compounds are also present as trace components in bulk raw materials such as coal and phosphate ores (ATSDR 2000).

Identity

Mercury is a naturally occurring metal, it is a very rare metal found in the earth's crust at concentrations frequently below 0.03 mg/kg (Alloway 1990). Mercury can exist in several forms. Metallic mercury is a shiny, silver-white liquid that forms a colorless, odorless gas if heated. Mercury combines with other elements such as chlorine, sulfur, or oxygen to form inorganic mercury compounds or salts, which are usually white solids. Mercury also combines with carbon to make organic mercury compounds, the most common of which is methylmercury (ATSDR 2000, UNEP 2002).

Environmental Fate

Mercury is found at very low concentrations in many aquatic and terrestrial ecosystems (see Table A3.5). Elevations above natural, background concentrations are often found as anthropogenic emissions of mercury far exceed those from natural sources (Alloway 1990, Bryan & Langston 1992, UNEP 2002)

Environmental Matrix	Concentration	**Reference**
Seawater (open ocean)	0.001-0.004 µg/l 0.02 µg/l	Bryan & Langston 1992, WHO 1989 ATSDR 2000
Freshwater	<0.005 µg/l	ATSDR 2000
Marine sediment	0.02-0.1 mg/kg	WHO 1989
Freshwater sediment	0.2-0.35 mg/kg	Salomons & Forstner 1984
Soil	0.02-0.625 mg/kg	Alloway 1990, WHO 1989
Fish	<0.2 mg/kg	ATSDR 2000

Table A3.5. Background concentrations of mercury found in water, sediments, soil and fish.

Due to the fact that mercury is the only metal that can exist as both a liquid and a vapour at ambient temperatures, its environmental behaviour differs from that of most other toxic elements (ATSDR 2000, WHO 1989). Mercury can exist in three valence states, Hg (0) metallic, Hg (I) mercurous and Hg (II) mercuric. In the atmosphere, elemental mercury is by far the most common form, and as a vapour it is responsible for the long-range, global cycling of mercury (UNEP 2002). In addition, though to a far lesser degree, mercury may be associated with particulates, which can be moved by dry or wet deposition (ATSDR 2000, WHO 1991).

In the aquatic environment, mercury is most commonly found in the mercuric (II) state. Once released, its fate is dominated by rapid adsorption to soluble and particulate organic material. This is followed by flocculation, precipitation and final accumulation in the bottom sediment. Because of the strength with which mercury is bound to sediment,

exchange back to the water column is generally slight, although it can be accelerated in saline waters, and in the presence of high concentrations of sulphide (anoxic conditions) (ATSDR 2000, Bryan & Langston 1992). Mercury accumulation from sediments may therefore be a dominant pathway for uptake in aquatic organisms and accounts for relatively high concentrations in deposit feeders, in both freshwater and marine systems (Bryan & Langston 1992).

Inorganic mercury can be converted to organic forms by micro-organisms indigenous to soils, fresh water and marine sediments. The most common form of organic mercury is methylmercury (MeHg), which is soluble, mobile and quick to enter the aquatic food chain. Increased levels of mercury in the environment can increase the amount of methylmercury that these bacteria produce. Once in the food chain, MeHg is selectively retained at each level, resulting in mercury being one of the few metals that biomagnify in food chains (ATSDR 2000, WHO 1989, UNEP 2002). Mercury concentrations in top predators can be 10,000-100,000 times the concentrations found in ambient waters (ATSDR 2000). The significance of this bioaccumulation is that it is generally the most important source of non-occupational mercury exposure for humans (ATSDR 2000, WHO 1989).

Toxicity

All forms of mercury are extremely toxic, with the precise toxicity being dependant on the route of exposure and the form of mercury; metallic (elemental), inorganic or organic. Mercury is a non-essential trace metal, with no biochemical or nutritional function; it can be excreted from the body, but slowly and with residues tending to remain in certain organs, notably the brain and kidneys (WHO 1989).

Human toxicity

In the industrial context, the most significant route of exposure is generally though inhalation of elemental (metallic) mercury vapour, which is readily absorbed by the lungs (Bernard 1997, WHO 1991). For the general public, exposure to mercury is mainly through diet in the form of methyl mercury (UNEP 2002). The nervous system is highly sensitive to all forms of mercury (ATSDR 2000).

Acute inhalation of mercury vapour may cause nausea, vomiting, diarrhoea, increases in blood pressure or heart rate, skin rashes, eye irritation, corrosive bronchitis and pneumonitis. In extreme cases, high levels of exposure to metallic mercury vapours can result in death through respiratory failure (ATSDR 2000). If not fatal, acute exposure may also be associated with central nervous system (CNS) effects such as tremors, loss of memory and personality changes (ATSDR 2000, Goyer 1996). Long-term exposure to lower levels can also lead to similar effects on the CNS as well as renal (kidney) damage (Ratcliffe et al. 1996, Goyer 1996).

Effects of exposure to inorganic mercury salts are mainly on the central nervous system (CNS) and the kidney, though damage to the skin and stomach have also been reported (ATSDR 2000, Zalups & Lash 1994).

Exposure to methyl mercury (MeHg) can result in permanent damage to the CNS, kidneys, and the developing foetus (ATSDR 2000, UNEP 2002). Symptoms from high levels of exposure include brain damage, numbness of extremities, and paralysis, along with the loss

of hearing, speech and sight (D'Itri 1991). Children and foetuses are particularly sensitive to the harmful effects of metallic mercury on the nervous system from all exposure routes (ATSDR 2000).

Environmental toxicity

In the majority of cases, animal exposure to mercury is mainly through diet in the form of methyl mercury. The majority of the effects listed above for humans have also been reported in animal studies (ATSDR 2000).

Studies on the aquatic toxicity of mercury are numerous. Inorganic mercury is toxic to fish at low concentrations, with impacts seen at concentrations below 1μg/l in some cases (Bryan & Langston 1992, WHO 1989). Elevated levels can result in adverse reproductive effects in fish including the development of sex organs, reductions in embryo survival and increases in deformities (WHO 1989). High incidences of abnormalities in seabirds that correlate with mercury residues in their tissues have also been observed (Bryan & Langston 1992). Studies also show that MeHg is generally more toxic to aquatic organisms than inorganic forms of mercury (WHO 1989).

Legislation

Many national and international regulations and recommendations apply to levels of mercury in air, water and soil. Some examples of these are given below;

For occupational exposure to metallic mercury vapours the United States National Institute for Occupational Safety and Health (NIOSH) recommended airborne exposure limit of 0.05 mg/m^3 averaged over a 8-hour work shift (NIOSH 1992).

The United States Environment Protection Agency (USEPA) set a maximum permissible level for mercury in drinking water of 2 μg/l (ATSDR 2000), while the World Health Organisation recommends a guideline value of 1 μg/l (WHO 1993).

Of the many regulations and recommendations applicable to mercury in soil, standards set by the Dutch government are widely recognised. Multipurpose land is defined as seriously contaminated and needing intervention above a concentration of 10 mg/kg. In addition, a target value of 0.3 mg/kg is also set to provide long term negligible risks to the ecosystem through a fully sustainable soil quality (MHSPE 1994).

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A3.6 COPPER (CU)

Production and major uses

Copper is produced from ore, or recovered from secondary sources (i.e. scrap). The latter process is far less energy intensive, and therefore is playing an increasingly important role in terms of global copper production (UNEP 1993).

Metallic copper is principally used as an electrical conductor (copper cables and wires), however it is also widely employed in alloys such as bronze (copper and tin), brass (copper and zinc) and monel (copper and nickel), in corrosive-resistant and decorative plating, in munitions and in dental alloys. Its compounds, or 'salts' are used as chemical catalysts, wood preservatives, algicides, fungicides, anti-fouling paints, disinfectants, nutritional supplements in fertilizers and feeds, in petroleum refining and as printing inks and dyes (ATSDR 2000, UNEP 1993). Copper compounds are also present as trace components in bulk raw materials such as coal and phosphate ores (ATSDR 2000).

Identity

Copper (Cu) is an element that naturally occurs in the environment, principally as ionic compounds but also in elemental (metallic) form. Its abundance in the Earth's crust is in the range 24-55 mg/kg (Alloway 1990). In metallic form, Cu(0), copper is highly malleable and ductile, and is an excellent conductor of heat and electricity (ATSDR 2000). In ionic forms, copper can exist in a number of oxidation states; Cu(I), Cu(II) & Cu(III), as either simple salts or ionic complexes. Generally, copper salts are readily soluble in water especially under acidic conditions, with the cupric Cu(II) forms predominating (ATSDR 2000).

Environmental Fate

Copper is a relatively abundant "trace" metal, found at varying concentrations in nearly all uncontaminated aquatic and terrestrial ecosystems (see Table A3.3). However, as anthropogenic emissions of copper exceed those from natural sources, elevations above

these natural, background concentrations are often found (Alloway 1990, Bryan & Langston 1992).

Environmental Matrix	Concentration	Reference
Seawater (English Channel, Irish Sea, North Sea)	0.35-4.0 μg/l (coastal) 0.14-0.9 μg/l (open ocean)	Law *et al*. 1994
Seawater (background)	0.1 μg/l	Sadiq 1992, Bryan & Langston 1992
Freshwater, UK	<20 μg/l	Mance *et al*. 1984
Soil	20 –30mg/kg	Alloway 1990
Marine sediment	10-30 mg/kg	UNEP 1993, Bryan & Langston 1992
Freshwater sediment	45-50 mg/kg	Salomons & Forstner 1984

Table A3.3. Background concentrations of copper found in water, sediment and soil

Copper generally exists in natural waters either in the dissolved form as the cupric (+2) ion or as complexes with inorganic anions or organic ligands (e.g. carbonates, chlorides, humic and fulvic acids). Copper compounds may also be present in insoluble forms (e.g. hydroxide, phosphate, or sulphide) or adsorbed onto suspended particulate matter. Alternatively it can be adsorbed to bottom sediments or exist as settled particulates. The relative concentrations of each of these forms is dependant upon a number of chemical parameters, including pH, salinity, and the presence of organic ligands, inorganic anions and other metal ions. Studies have frequently shown that the concentration of free Cu(+2) ions is low compared to the level of copper associated with suspended and bottom sediments (ATSDR 2000, Mance *et al*. 1984).

In soils, copper is typically present in ionic forms which have a high affinity for sorption by organic and inorganic ligands (e.g. humic and fulvic acids, hydroxides of iron, aluminium and manganese), though it can also exist as soluble ions and complexes.

In a soluble form copper is far more bioavailable and far more likely to migrate through the environment than if it is bound to organic matter or present as an insoluble precipitate. Therefore, simple ionic compounds that predominate in municipal waste incinerator ash, coal fly, mine tailings and process wastes from phosphate fertilizer production, tend to be far more bioavailable and migratory than the more complex forms of copper in the materials processed by these industries (ATSDR 2000, Alloway 1990, Mance *et al*. 1984).

Toxicity and essentiality

Copper is an important element for plants and animals and is an essential dietary requirement in low doses. However, if humans, animals or plants are exposed to elevated levels of bioavailable copper, in either a single dose or through long term elevated exposure, bioaccumulation and toxic effects can occur (ATSDR 2000).

Human toxicity

Copper is an essential nutrient for humans. It is incorporated into numerous enzyme systems including those involved in haemoglobin formation, carbohydrate metabolism and melanin formation (ATSDR 2000). Human deficiency is characterised by anaemia, resulting from defective haemoglobin synthesis (Goyer 1996). Acute poisoning from excessive doses, however, can cause vomiting, hypotension, jaundice, coma and even death

(ATSDR 2000). Furthermore, inhalation of dust and vapours can irritate the nose, mouth and eyes, and cause headaches, dizziness, nausea and diarrhoea.

Copper homeostasis plays an important role in the prevention of copper toxicity in humans. Copper is readily absorbed from the stomach and small intestine; and after requirements are met several mechanisms prevent copper overload e.g. bile excretion (ATSDR 2000). Failure of this mechanism can occur following exposure to high levels of copper. This rare disease, known as Wilson's disease, is characterised by the excessive retention of copper in the liver and impaired copper excretion in the bile and can result in liver and kidney damage and haemolytic anaemia (ATSDR 2000).

Environmental toxicity

As for humans, copper is an essential nutrient for most terrestrial and aquatic organisms, with copper homeostasis playing an important role in the prevention of copper toxicity (ATSDR 2000). In addition to similar toxic effect to those report for humans, some animal studies have shown developmental and reproductive damage following exposure to high levels of copper. No such effects have been reported in humans (ATSDR 2000).

Aquatic toxicity to copper is well studied, and there is experimental evidence that a considerable number of species are sensitive to dissolved concentrations as low as 1-10 μg/l (Bryan & Langston 1992). For example, studies have shown that at levels of 2 μg/l the survival rate of young bay scallops was significantly affected; and concentrations of 5 μg/l were seen to induce abnormalities in the embryos of oysters and mussels. A similar concentration resulted in increased mortality in populations of the isopod crustacean *Idothea baltica* (UNEP 1993, Bryan & Langston 1992, Giudici *et al*. 1989). Other studies have reported reductions in the survival, growth and fertility of amphipods and copepods (Conradi & DePledge 1998, UNEP 1993), and embryonic sensitivity in fish exposed to levels of 25 μg/l (UNEP 1993, Mance *et al*. 1984). Furthermore, a study of species diversity in benthic communities from Norwegian fjords led to the conclusion that the most sensitive animals were missing from sites where sediment copper levels exceeded 200 mg/kg. Similar effects have been observed in estuaries of the UK (Bryan & Langston 1992).

Legislation

Many national and international regulations and recommendations apply to copper. Some examples of those concerning copper in drinking water and soil are given below;

Drinking water regulations and recommendations include the United States Environmental Protection Agency maximum permissible concentration of 1300 μg/l (ATSDR 2000) and the World Health Organisation recommended guideline value of 2000 μg/l (WHO 1993).

Many standards apply to levels of copper in soil. For example, widely recognised Dutch standards for multipurpose land which classify a copper concentration of 190 mg/kg as seriously contaminated and requiring intervention. These standards also set a target value of 36 mg/kg for a fully recovered sustainable soil quality, with the aim of providing long term negligible risks to the ecosystem (MHSPE 1994).

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A3.7 ZINC (ZN)

Production and major uses

Zinc is primarily produced from ores through smelting or electrolyses, but can also be recovered from secondary sources such as recycled scrap (ATSDR 2000, Kroschwitz & Howe-Grant 1995).

Zinc is one of the most extensively used "trace" metals (Nriagu 1990). It is most commonly employed as a protective coating for other metals e.g. galvanised steel, or as a component of bronze, brass and die-casting alloys. In addition, zinc compounds or 'salts' are widely employed as wood preservatives, herbicides, catalysts, analytical reagents, vulcanisation accelerators for rubber, and stabilisers in PVC. They can also be found in ceramics, textiles, fertilisers, paints, pigments, batteries and dental, medical, and household products (ATSDR 2000, Annema & Ros 1994, UNEP 1993, Budavari *et al.* 1989). Zinc compounds are also present as trace components in bulk raw materials such as coal and phosphate ores (ATSDR 2000).

Identity

Zinc (Zn) is a metallic element that is naturally present in the environment, principally as ionic compounds. It is found in the Earth's crust at an average concentration of 80 mg/kg, although some clay sediments and shales may contain higher concentrations (Alloway 1990, Salomons & Forstner 1984). In ionic forms, the divalent oxidation state Zn(II) predominates (ATSDR 2000).

Environmental Fate

Zinc is a relatively abundant metal, which is found at varying concentrations in nearly all uncontaminated aquatic and terrestrial ecosystems (see Table A3.7). However, as anthropogenic emissions of zinc far exceed those from natural sources, elevations above these natural, background concentrations are often found (Bryan & Langston 1992, UNEP 1993)

Environmental Matrix	Concentration	Reference
Seawater (open ocean) (coastal and estuarine)	<1 μg/l 0.3-70 μg/l	Bryan & Langston 1992, UNEP 1993
Freshwater	<50 μg/l	ATSDR 2000
Drinking water	0.02-1.2 mg/l	ATSDR 2000
Soil	50 mg/kg average (10-300 mg/kg)	Alloway 1990
Freshwater sediment	<100 mg/kg	ATSDR 2000, Salomons & Forstner 1984
Marine Sediment	<100 mg/kg	Bryan & Langston 1992, UNEP 1993

Table A3.7. Background concentrations of zinc found in water, sediments and soil.

In the environment zinc occurs primarily in the +2 oxidation state, either as the free (hydrated) zinc ion, or as dissolved and insoluble complexes and compounds (ATSDR 2000). In soils, zinc generally remains strongly sorbed, and in the aquatic environment it will predominantly bind to suspended material before finally accumulating in the sediment (ATSDR 2000, Bryan & Langston 1992, Alloway 1990). Re-solubilisation back into an aqueous, more bioavailable phase, is possible under certain physical-chemical conditions, e.g. the presence of soluble anions, the absence of organic matter, clay minerals and hydrous oxides of iron and manganese, low pH and increased salinity (ATSDR 2000). Zinc in a soluble form (e.g. sulphate or chloride, as present in incinerator ash, or ore processing wastestreams) is far more likely to migrate through the environment than if it is bound to organic matter or present as an insoluble precipitate (e.g. as in sewage sludge) (ATSDR 2000).

Toxicity and essentiality

Zinc is an essential nutrient for plants and animals and humans. However, if exposed to high concentrations of bioavailable zinc, significant bioaccumulation can results, with possible toxic effects (ATSDR 2000).

Human toxicity

Zinc is a nutritionally essential metal in the human diet, playing a role in enzymatic, structural and regulatory systems (Goyer 1996, Aggett & Comerford 1995). Due to its essentiality, dietary allowances of zinc are recommended; 15 mg/day for men, and 12 mg/day for women (ATSDR 2000). Deficiency can result in severe health consequences including growth retardation, anorexia, dermatitis, depression and neuropsychiatric symptoms (Aggett & Comerford 1995).

High doses of zinc in the diet, however, can induce a range of symptoms in humans, including pancreatic damage, anaemia, gastrointestinal distress and diarrhoea (ATSDR 2000, Goyer 1996). For example, humans taking supplements at higher than recommended doses (400-500 mg/day) suffered severe gastro-enteritis (Abernathy & Poirier 1997); and

humans who drank water from galvanised pipes over a prolonged period suffered irritability, muscular stiffness and pain, loss of appetite and nausea (UNEP 1993).

Exposure and toxic effects can also occur via inhalation of zinc fumes, primarily in an occupational situation. Inhalation of zinc oxide fumes presents the most significant effect, with symptoms including include chills and fever, profuse sweating and weakness. Attacks usually begin after 4-8 hours of exposure, and last between 24-48 hours (ATSDR 2000, Goyer 1996).

Environmental toxicity

Excessive oral exposure to zinc can cause a similar range of toxic effects in animals as described above for humans (ATSDR 2000, Goyer 1996). For example, animal studies involving doses 1,000 times higher than the RDA, taken over a period of a month, resulted in anaemia and injury to the pancreas and kidney. Furthermore, rats that ate very large amounts of zinc became infertile (ATSDR 2000).

In the aquatic environment, appreciable quantities of zinc can have a direct disrupting effect on the external cell membranes or cell walls of organisms, resulting in rapid mortality (UNEP 1993). Prolonged exposure to lower sub-lethal concentrations can also affect aquatic organisms. For example, studies have shown that at concentrations as low as 15 μg/l, carbon fixation rates in natural phytoplankton populations were depressed. Others observed that the growth of cultured diatoms was inhibited at 20 μg/l (Bryan & Langston 1992).

Plant studies have shown that although an essential element for higher plants, in elevated concentrations zinc is considered phytotoxic, directly affecting crop yield and soil fertility. Soil concentrations ranging from 70-400 mg/kg are classified as critical, above which toxicity is considered likely (Alloway 1990).

Legislation

Many national and international regulations and recommendations apply to zinc. For example, a number of recommended maximum concentrations exist for zinc in drinking water, including those of the United States Environmental Protection Agency (5 mg/l) (ATSDR 2000) and the World Health Organisation guideline value (3 mg/l) (WHO 1993).

Regarding soil concentrations, Dutch legislation sets widely recognised standards. For multipurpose land, a concentration of 720 mg/kg is classified as seriously contaminated and requiring intervention. To provide long term negligible risks to the ecosystem and achieve a fully sustainable soil quality, a target value of 140 mg/kg is also set (MHSPE 1994).

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A3.8 LEAD (PB)

Production and major uses

Lead is primarily obtained from lead sulphide ores, but is also obtained from the smelting of secondary sources including recycled lead from electric storage batteries (Dudka & Adriano 1997).

Uses of lead and its compounds are extensive. As a metal, it has historically been used in pipes for water distribution, or as containers for storing for corrosive. Alloys of lead are used in welding, printing and as anti-friction metals; great quantities, both of the metal and its dioxide, are also used in electric storage batteries. Other uses include cable coverings, ammunition, and in the manufacture of tetraethyl lead, used as an anti-knock compound in petrol. Compounds of lead are used as paint pigments, PVC stabilisers, pesticides, varnishes, lubricants, as glazes for pottery and porcelain, and in leaded glass crystal (Budavari *et al.* 1989, ATSDR 2000). Lead compounds are also present as trace components in bulk raw materials such as coal and phosphate ores (ATSDR 2000).

Identity

Lead (Pb) is a naturally occurring metallic element that is present in the environment in low levels, principally in ionic forms; the average concentration of lead in the Earth's crust is approximately 16 mg/kg (Alloway 1990). Lead can exist in many forms; as a malleable metal Pb(0), in ionic forms with oxidation states +2 and +4 as either simple salts or ionic complexes, or as organic lead compounds such as tetraethyl lead (ATSDR 2000).

Environmental Fate

Lead is present in nearly all uncontaminated aquatic and terrestrial ecosystems, though typically at very low levels (see Table A3.4). Environmental levels can, however, far

exceed natural background concentrations as a result of anthropogenic (ATSDR 2000, Bryan & Langston 1992).

Environmental Matrix	Concentration	Reference
Seawater (estuarine waters around England and Wales)	0.024-0.880 μg/l	Law *et al*. 1994
Seawater (open ocean)	0.005-0.071 μg/l	ATSDR 2000, Bryan & Langston 1992, Law *et al*. 1994
Freshwater (mean value from 39,490 measurements)	3.9 μg/l	ATSDR 2000
Drinking water	<5-30 μg/l	ATSDR 2000
Soil	10-30 mg/kg	Alloway 1990
Freshwater / marine sediment	20-30 mg/kg	ATSDR 2000

Table A3.4. Background concentrations of lead found in water, sediments and soil

When lead is released into the environment it has a long residence time compared with most pollutants, and tends to accumulate in soils and sediments. In such cases, due to low solubility, it can remain accessible to the food chain and to human metabolism far into the future (Sauve *et al.* 1997, ATSDR 2000, Alloway 1990). The bioavailability of lead is highly dependent on speciation. Of the two stable oxidation states of lead (+2 and +4), the environmental chemistry is dominated by the Pb^{+2} ion, its compounds and complexes. In general, the free +2 ion is more bioavailable than inorganic complexes, and therefore any factor which increases complexation and decreases the concentration of the free ion affects the bioavailability adversely. Organic forms of lead are also present in the environment, from direct inputs (manufacture, transport and storage of leaded petrol and consequent car exhaust emissions) and the possible chemical / biological methylation of inorganic lead in anaerobic sediments (Sadiq 1992, Forsyth *et al.* 1991).

Lead tends to form ionic compounds with anions having low solubility, such as hydroxides, carbonates, and phosphates. In addition, the solubility of lead compounds is dependent on many factors including pH and salinity. The amount of lead remaining in solution in surface waters is often low, with the major fraction bound to suspended or bottom sediment particles (ATSDR 2000). In soils and sediments, lead is often in the form of relatively stable organic-metal complexes, with most of the lead strongly retained. Re-entry to surface waters can occur as a result of erosion of lead-containing soil particulates, or through formation of relatively soluble lead sulphate at the soil / sediment surface (ATSDR 2000, Sadiq 1992, Alloway 1990).

Plants and animals can accumulate lead from water, soil and sediment, with organic forms being more easily absorbed than inorganic (ATSDR 2000).

Toxicity

Lead is a highly ubiquitous toxic metal with no known biochemical or nutritional function. It is highly toxic to plants, animals and humans (Alloway 1990, ATSDR 2000, Goyer 1996).

Human toxicity

The toxic effects of lead are the same, irrespective of whether it is ingested or inhaled. Symptoms include nervous system disorders, anaemia and decreased haemoglobin

synthesis, cardiovascular disease, and disorders in bone metabolism, renal function and reproduction. Of particular concern is the effect of relatively low level exposure on cognitive and behavioural development in children (Pirkle *et al.* 1998, ATSDR 2000, Bernard *et al.* 1995, Goyer 1993, Nriagu 1988). Exposure to lead is measured through levels in blood; it is currently thought that there may be no level of blood-lead that does not produce a toxic effect, particularly in the developing central nervous system (ATSDR 2000, Goyer 1993).

Environmental toxicity

Animals studies have reproduced many of the toxic effects listed for humans (Blus *et al.* 1991, ATSDR 2000, WHO 1989). Lead is also toxic to all aquatic biota, and even though it is not one of the most environmentally mobile metals there is appreciable evidence showing the bioavailability of sediment-bound lead to deposit feeding species. This is in addition to directly accumulated lead from sea and fresh waters (Bryan & Langston 1992, Sadiq 1992). Studies involving invertebrates often report effects at μg/l (parts per billion) concentrations, including reduction in growth, fertility and reproduction as well as mortality(WHO 1989).

Legislation

As a result of its toxicity and ubiquity in the environment, lead is covered by many national and international regulations and recommendations. For example, to address lead in drinking water, the United States Environmental Protection Agency (USEPA) sets a maximum permissible concentration of 15 μg/l (ATSDR 2000), while the World Health Organisation currently recommends a guideline level of 10 μg/l (WHO 1993).

Of the many regulations regarding soil contamination, standards set by the Dutch government are widely recognised. For multipurpose land, a lead concentration of 530 mg/kg is considered as seriously contaminated and requiring intervention. To provide long term negligible risks to the ecosystem through a fully recovered sustainable soil quality, a target value of 85 mg/kg is also set (MHSPE 1994).

To address the environmental and health concerns associated with lead, the goal of some more recent legislation is the phase out of lead through its substitution with less toxic alternatives. For example, Denmark has recently imposed a wide-ranging ban on the import, marketing and manufacture of lead and products containing lead (MEE 2000).

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A3.9 CHROMIUM (CR)

Production and major uses

Chromium is produced from chromite ore in two different processes, as ferrochrome from the reduction of chromite with coke in an electric arc or through oxidation, leaching, precipitation and reduction to produce chromium metal. Ferrochrome is an iron-chromium alloy that is used directly as an additive to produce chromium-steels, commonly known as stainless steels (ATSDR 2000, Greenwood & Earnshaw 1984).

The majority of chromium is used in alloys, including stainless steel that contains varying amounts of iron, chromium and nickel, depending on the properties required in the final product. About 15% is also used in the general chemical industry, where chromium compounds or 'salts' are commonly used as tanning agents, textile pigments and preservatives, anti-fouling paints, catalysts, corrosion inhibitors, drilling muds, high temperature batteries, fungicides, wood preservatives, and in metal finishing and electroplating (ATSDR 2000, Alloway 1990, Greenwood & Earnshaw 1984). Chromium compounds are also present as trace components in bulk raw materials such as coal and phosphate ores (ATSDR 2000).

Identity

Chromium (Cr) is a metallic element that naturally occurs in the environment, principally as ionic compounds, with an average abundance in the Earth's crust of 100 mg/kg (Alloway 1990). Chromium does not naturally exist in elemental form, Cr(0). Predominant valence states for ionic chromium include the divalent Cr(II), trivalent Cr(III) and hexavalent Cr(VI) forms (ATSDR 2000). When in the divalent chromous Cr(II) state, oxidation readily occurs to give the trivalent chromic Cr(III) state. Generally trivalent chromic Cr(III) compounds

are insoluble in water, while many chromate Cr(VI) compounds are readily soluble in water (ATSDR 2000).

Environmental Fate

Chromium is found in varying concentrations in nearly all uncontaminated aquatic and terrestrial ecosystems (see Table A3.2). However, in areas associated with anthropogenic emissions, ecosystem levels can far exceed natural, background concentrations (Alloway 1990, Bryan & Langston 1992).

Environmental Matrix	Concentration	Reference
Seawater (open ocean)	0.057-0.234 μg/l	Bryan & Langston 1992
Freshwater	1.30 μg/l	ATSDR 2000
Drinking water	0.4-8.0 μg/l	ATSDR 2000
Marine sediment	30-200 mg/kg	Bryan & Langston 1992,
Freshwater sediment / suspended particulates	1-500 mg/kg	ATSDR 2000
Soil	<1-100 mg/kg 4-80 mg/kg	Alloway 1990 Dudka & Adriano 1997

Table A3.2. Background concentrations of chromium found in water, sediment and soil

Although many different oxidation states of chromium exist in the environment, only the trivalent (III) and hexavalent (VI) forms are of biological importance. In aquatic environments chromium (VI) will be present predominantly in a soluble form. These soluble forms may be stable enough to undergo transport between different media, however chromium (VI) will eventually be converted to chromium (III) by reducing species (ATSDR 2000, Kimbrough *et al.* 1999). The trivalent chromic form is generally insoluble in water and tends to rapidly precipitate or adsorb onto suspended particles and bottom sediments. In this form, therefore, chromium does not generally migrate significantly in natural systems. However, changes in the chemical and physical properties of an aquatic environment can result in changes to the redox equilibrium, enabling oxidation of chromium (III) to chromium (VI) (Richard & Bourg 1991).

In soils, chromium (III) is relatively immobile due to its strong adsorption capacity onto soils. In contrast, chromium (VI) is highly unstable and mobile, since it is poorly adsorbed onto soils under natural conditions (Mukherjee 1998). Changes in speciation between chromium (III) and chromium (VI) are important processes affecting the speciation and hence the bioavailability and toxicity of chromium in soils (Mukherjee 1998).

Whilst chromium (III) is an essential trace element in animals, chromium (VI) is non-essential and toxic at low concentrations. Thus, because oxidation processes can result in the formation of chromium (VI), anthropogenic activities that release either chromium (III) or chromium (VI) are equally non-desirable. Even if chromium (III) is discharged into the environment, there is no guarantee that it will remain in this chemical state. For example, the landfilling of chromium (III) tannery waste with other acidic industrial wastes, or domestic sewage, which on decomposition can yield acidic conditions, can result in the oxidation of chromium (III) to chromium (VI) (Mukherjee 1998, Outridge & Sheuhammer 1993, UNEP 1991, Richard & Bourg 1991).

Toxicity and essentiality

The trivalent chromic Cr(III) form is an essential trace nutrient for animals and humans, though large doses may be harmful. In contrast, hexavalent chromate Cr(VI) is non-essential, highly toxic, and in some cases carcinogenic (ATSDR 2000).

Human toxicity

Chromium (III) is an essential nutrient for humans being required for glucose, protein and fat metabolism, with deficiency able to cause weight loss and impairment of the body to remove glucose from the blood (ATSDR 2000, Goyer 1996). The minimum human daily requirement of chromium (III) for optimal health is not known, but a daily ingestion of 50-200 μg/day has been estimated to be safe and adequate. The ingestion of very large doses may, however, be harmful to humans (ATSDR 2000).

Chromium (VI) is non-essential and toxic. Compounds are corrosive, and allergic skin reactions readily occur following exposure, independent of dose. Short-term exposure to high levels can result in ulceration through skin contact, perforations of respiratory surfaces through inhalation and irritation of the gastrointestinal tract through ingestion. Damage to the kidney and liver have also been reported (ATSDR 2000).

In addition, long-term occupational exposure to airborne levels of chromium higher than those in the natural environment has been associated with lung cancer, and the United States Department of Health and Human Services has classified chromium (VI) compounds as 'known to be human carcinogens' (DHHS 2002). In addition, the International Agency for Research on Cancer (IARC) classifies chromium (VI) compounds as carcinogenic to humans (1990). Individuals at most risk include those in chromate and chromium pigment industries as well as chromium-alloy workers, stainless steel welders, and chrome-platers (Kimbrough 1999, DHHS 2002).

Environmental toxicity

As for humans, chromium (III) is an essential nutrient for animals and plants while chromium (VI) is generally toxic. Studies have also shown an increased risk of cancer for some animals exposed to chromium (VI) through inhalation (ATSDR 2000).

The aquatic toxicology of chromium is also dependent upon speciation, with chromium (III) far less biologically available and toxic than chromium (VI). Negative effects have been observed in aquatic organisms exposed to dissolved chromium (VI) at concentrations below 40 μg/l. Furthermore, both chromium (III) and chromium (VI) have been shown to accumulate in many aquatic species, especially in bottom-feeding fish, bivalves, mussels and clams (Kimbrough *et al.* 1999).

Legislation

Many national and international regulations and recommendations apply to chromium. Some examples of those concerning chromium in drinking water and soil are given below;

For drinking water, the United States Environmental Protection Agency (USEPA) sets a maximum permissible concentration for chromium of 100 μg/l (ATSDR 2000), while the World Health Organisation currently recommends a guideline level of 50 μg/l (WHO 1993).

Of the many national regulations concerning chromium in soil, Dutch standards are widely recognised. For multipurpose land, these classify a concentration of 380 mg/kg as seriously contaminated and requiring intervention. For a fully recovered sustainable soil quality, a target value of 100 mg/kg is also set with the aim of providing long term negligible risks to the ecosystem (MHSPE 1994).

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A3.10 CADMIUM (CD)

Production and major uses

Cadmium is produced as a by-product during the processing of zinc, copper and lead ores (ATSDR 2000). This metal is primarily used for the production of nickel-cadmium batteries, and for metal plating. It is also used in pigments for glasses and plastics, as a stabiliser in polyvinyl chloride (PVC), and as a component of various alloys (ATSDR 2000, WHO 1992). In addition to intentional use, cadmium compounds are also present as trace components in bulk raw materials including coal and phosphate ores. Such unintentional uses can result in significant cadmium releases to various media through incineration, coal

combustion and phosphate ore processing (ATSDR 2000, WHO 1992). Total anthropogenic emissions of cadmium are estimated to exceed natural emissions by approximately 10 times (Elinder 1992).

Identity

Cadmium (Cd) is a relatively rare element that naturally occurs in the environment in low concentrations, principally as ionic compounds. It is present in the Earth's crust with an average abundance of 0.1 mg/kg, though some sedimentary rocks and marine phosphates can accumulate higher levels (Alloway 1990, WHO 1992). In metallic form, cadmium Cd(0) is a silver-white metal. In inorganic forms, cadmium almost exclusively exists as divalent Cd(II) compounds, as either simple salts or ionic complexes. The majority of cadmium salts are practically insoluble in water, with major exceptions being nitrates, sulfates and halogenates (ATSDR 2000).

Environmental Fate

Cadmium is a rare metal, found naturally as very low concentrations (see Table A3.1). However, as anthropogenic emissions far exceed those from natural sources, elevations above these natural, background levels, are often found (Alloway 1996, Bryan & Langston 1992).

Environmental matrix	Concentration	Reference
Freshwater, groundwater, drinking water	<1 μg/l	ATSDR 2000, WHO 1992
Seawater (open ocean)	0.02-0.12 μg/l	Sadiq 1992, Bryan & Langston 1992
Seawater (coastal)	0.01-0.17 μg/l	Bryan & Langston 1992
Marine sediment	<1 mg/kg	Sadiq 1992, Salomons & Forstner 1984
Estuarine sediment	0.2 mg/kg	Bryan & Langston 1992
River sediment	1 mg/kg	Salomons & Forstner 1984
Soil	0.01-2.0 mg/kg	Alloway 1990, ATSDR 2000

Table A3.1. Background concentrations of cadmium found in water, sediment and soil

In aquatic environments cadmium is more mobile than most other metals. It is also bioaccumulative and persistent in the environment with a residence half-life of 10-30 years (ATSDR 2000). In surface and groundwater cadmium primarily exists as either the +2 hydrated Cd(II) ion, or as ionic complexes with other inorganic or organic substances. While soluble forms may migrate in water, cadmium in insoluble complexes or adsorbed to sediments is relatively immobile. Similarly, cadmium in soil may exist in soluble form in soil water, or in insoluble complexes with inorganic and organic soil constituents (ATSDR 2000, WHO 1992). In soils, the agricultural use of phosphate fertilisers or cadmium-containing sewage sludge, can dramatically increase soil cadmium concentrations (Cabrera *et al.* 1994, Jackson & Alloway 1992).

When cadmium is present in bioavailable forms, bioaccumulation has been observed for both aquatic and terrestrial organisms. Studies have shown accumulation in aquatic animals, such fish and invertebrates, at concentrations hundreds to thousands of times higher than in the water (ATSDR 2000). Cadmium accumulation has also been reported in earthworms, poultry, cattle, horses, and wildlife (Jackson & Alloway 1992, ATSDR 2000,

WHO 1992). Furthermore, cadmium is readily available in plants including food crops such as grain, rice and vegetables, and there is a clear association between the cadmium concentration in soil and the plants grown on that soil (Elinder & Jarup 1996, Jackson & Alloway 1992). This accumulation of cadmium in the food chain has important implications for human exposure (ATSDR 2000).

Toxicity

Cadmium is highly toxic to plants, animals and humans, having no known biochemical or nutritional function (Alloway 1990, ATSDR 2000, WHO 1992).

Human toxicity

The kidney is the main target organ of cadmium toxicity in humans, following extended exposure (ATSDR 2000, Elinder & Jarup 1996, Goyer 1996, Iwata *et al.* 1993, WHO 1992). Recent studies have demonstrated impacts at lower levels of exposure than previously anticipated (Hellstrom *et al.* 2001, Jarup *et al.* 2000).

Other toxic effects of cadmium include disruption to calcium mechanisms in the body which can result in calcium deficiency in long-term occupational exposed workers, leading to osteoporosis, or osteomalacia (softening of the bones) (ATSDR 2000, Goyer *et al.* 1994, WHO 1992). Similar cadmium induced skeletal disorders have occurred though non-occupational exposure in cadmium-contaminated areas (Alloway 1996). Oral exposure in high doses can also result in severe irritation to the gastrointestinal epithelium, nausea, vomiting, salivation, abdominal pain, cramps and diarrhoea (ATSDR 2000). In addition, cadmium appears to play a role in the development of hypertension (high blood pressure) and heart disease (ATSDR 2000, Goyer 1996, Elinder & Jarup 1996).

The inhalation of high levels of cadmium oxide fumes or dust is intensely irritating to respiratory tissue, and acute high-level exposures can be fatal. Typical non-fatal symptoms can include severe tracheobronchitis, pneumonitis, and pulmonary oedema, which can develop within hours of exposure (ATSDR 2000, Goyer 1996, WHO 1992).

There is also sufficient evidence to show a relationship between exposure to cadmium and lung cancer, particularly for occupationally exposed workers through inhalation. An association may also exist for prostate, renal and bladder cancers (DHSS 2002, Goyer 1996). The US Department of Health and Human Services lists cadmium and cadmium compounds as known to be human carcinogens (DHSS 2002). Similarly, cadmium and cadmium compounds are listed by the International Agency for Research on Cancer (IARC) as carcinogenic (IARC 1994).

Environmental toxicity

As for humans, the kidney has been found the main target organ of cadmium toxicity in animal studies (ATSDR 2000, Elinder & Jarup 1996, Goyer 1996, Iwata *et al.* 1993, WHO 1992). Cadmium also has an adverse effect on the growth of plants, including food crops. Elevated exposure can lead to stunted growth and toxic signs on crops including lettuce, cabbage, carrot and radish (Alloway 1990, WHO 1992).

A range of toxic effects following cadmium exposure has been reported for many aquatic organisms. For example, some species of phytoplankton are very sensitive to cadmium, with inhibition of growth observed at water concentrations as low as 1 μg/l (Bryan & Langston 1992). Deleterious effects have also been reported in limpets, copepods and isopods, fish and invertebrates at concentrations as low as 5 μg/l. Effects include reductions in reproduction rates, changes in immune function and depressed growth (Bryan & Langston 1992, Thuvander 1989). Furthermore, toxicity from cadmium contaminated sediments has been observed in some aquatic mollusks at relatively low cadmium concentrations (Bryan & Langston 1992).

Animal studies have also demonstrated respiratory injury following inhalation exposure to cadmium (ATSDR 2000, WHO 1992). The International Agency for Research on Cancer (IARC) lists cadmium compounds as carcinogenic in experimental animals (IARC 1994).

Legislation

Many national and international regulations and recommendations apply to cadmium. Those concerning cadmium in drinking water include the United States Environmental Protection Agencys (USEPA) maximum permissible concentration of 5 μg/l (ATSDR 2000) and the World Health Organisation recommended guideline concentration of 3 μg/l (WHO 1993).

The concentration of cadmium in soil is addressed in many national regulations. For example, widely recognised Dutch standards for multipurpose land classify a concentration of 12 mg/kg as seriously contaminated requiring intervention. A target value of 0.8 mg/kg is also defined for a fully recovered sustainable soil quality that will provide long term negligible risks to the ecosystem (MHSPE 1994).

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