RELATÓRIO

Metal and organic pollution associated with the Bayer facility in Nova Iguaçu, Rio de Janeiro, Brazil 2000

Labunska, I., Stringer, R. & Brigden, K.

Greenpeace Research Laboratories, Department of Biological Sciences, University of Exeter, Exeter, UK.

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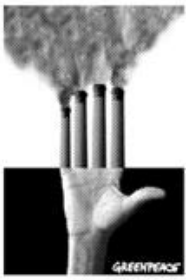
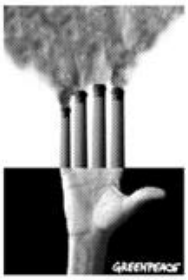




EXECUTIVE SUMMARY

The Bayer SA plant at Nova Iguaçu, Rio de Janeiro State, Brazil, manufactures chemicals and is also the location of a hazardous waste incinerator and a waste landfill. During August 2000, nine samples were collected from in and around the plant. The samples included five sediments (two of which were control samples collected upstream of Bayer site), two industrial wastewaters, and two samples of solid waste.

Alkanes were the dominant compounds in the control sediment samples from upstream of the Bayer site. This may indicate pollution of the sediments by petroleum, as alkanes are principal components of refined petroleum products. Several other organic compounds detected in the control samples, such as 1,4-dichlorobenzene, dihydrocholesterol and galaxolide, probably entered the Sarapui river with sewage discharges. The presence of beta-HCH in one of the control samples may indicate past usage of technical hexachlorocyclohexane as a pesticide because this isomer was also detected in all sediment samples from the Sarapui river further downstream. However, it was also present in one sample of waste from the Bayer landfill. Heavy metals found in the control samples were in the range of typical background levels.

Two samples were collected at the point of discharge from the Bayer treatment plant; one effluent and one sediment. Organic compounds found in the effluent sample were: derivatives (both halogenated and non-halogenated) of benzenamine and benzene, including 4-trifluoromethoxyaniline; Fyrol PCF- a flame retardant belonging to the class of chlorinated alkyl phosphate esters; several aromatic organic compounds which are used in organic synthesis (quinoline derivatives, 2-(methylthio)benzothiazole and diphenyl ether); and also polycyclic aromatic hydrocarbons (PAHs). 4-trifluoromethoxyaniline is amongst a wide range of chemicals produced by Bayer for the pharmaceutical industry. Copper, manganese, mercury and zinc were also found in the effluent sample at elevated levels.

A wide range of organochlorine compounds was found in the sediment sample including PCBs, DDT derivatives, HCH isomers and trace amounts of chlorinated benzenes. Moreover, this sample contained PAHs, benzaldehyde, benzenemethanol, a range of cyclic and linear hydrocarbons, and an organotin compound. The sediment sample had highly elevated levels of copper, cadmium, lead, mercury and zinc in comparison with typical background levels for freshwater sediments. Nickel and chromium concentrations were slightly elevated in this sample. These results demonstrate that the Bayer treatment plant does not provide efficient treatment of wastes and is a source of Sarapui river pollution. It is also possible that some of the contaminants, such as the PCBs, originated in the incinerator.

Two further two samples, one effluent and one sediment, were collected at another point of discharge from the Bayer site located near to the landfill, downstream of the discharge from the treatment plant. The metals and organic analyses indicated far less pollution from this source. Concentrations of the toxic heavy metal lead were elevated in both the effluent and the

sediment. However, only two organic compounds were detected in the effluent sample: 1,4-dichlorobenzene and 2-(methylthio)benzothiazole. The sediment sample contained beta-HCH and trace amounts of DDT derivatives. Beta-HCH appears to be a background contaminant in this area, but the DDT compounds were only detected in the sediment samples closest to the Bayer discharge points. The source of the phenolic compounds and DEHP, which were also found in the sediment sample, remains unclear.

The single sediment sample that was collected from the Sarapui river downstream of the Bayer site contained a similar pattern of both organic and heavy metal contaminants to that in the sediment sample by the discharge from the Bayer treatment plant, although at lower concentrations. Organic contaminants detected included PCBs, chlorinated benzenes, HCH isomers and PAHs. In addition, this sample contained alkylated benzenes and many aliphatic hydrocarbons that could indicate local contamination by mineral oils.

Two samples of waste were collected from the Bayer landfill. One contained almost all of the organic compounds that were detected in the sediment and effluent samples collected from the discharge near the Bayer treatment plant. These included chlorinated benzenes and benzenamines, brominated benzenamines, PCBs, an organotin compound, aliphatic hydrocarbons and beta-HCH. Other organic compounds found in this sample were numerous; PAHs, dibenzothiphene, biphenyl, diphenyl ether, alkylated benzenes and dibenzofuran. The concentrations of several of the heavy metals in this sample were higher than in the rest of the samples collected during this survey. Copper, lead, and zinc concentrations were particularly high.

The second sample of waste from the Bayer landfill contained a completely different pattern of organic and heavy metals contaminants, indicating that the landfill receives wastes from a variety of sources. The greatest concern attaches to the extremely high levels of mercury found in this sample. The rest of the metals considered in this study were detected at typical background levels for soil. Only a few organic compounds, mostly aliphatic hydrocarbons, were reliably identified in this sample.

1 INTRODUCTION

Bayer S.A. is a German-based company with operations in all six continents of the world, though most of their facilities are in Europe, the Americas and the Far East. The group is involved in the manufacturing of products for the healthcare market, as well as chemicals and polymers (Bayer 2000a). The Brazilian Chemical Industry Directory for 1999/2000 (ABIQUIM 1999) reports that there are four Bayer group plants in Brazil. Three are owned by Bayer S.A. and manufacture a range of chemicals from inorganics (iron oxide) to chlorinated agrochemicals (trichlorfon). The fourth facility is owned by Bayer Polímeros and manufactures ABS, MBS and SAN resins. In addition to this, the Bayer S.A. site at Nova Iguaçu, Rio de Janeiro State, also has a hazardous waste incinerator plant and a waste landfill.

2 SAMPLING PROGRAM

During August 2000, nine samples were collected in association with the Bayer SA facility, Nova Iguaçu, Rio de Janeiro State, Brazil. The samples included five sediments, two industrial wastewaters and two samples of solid waste.

2.1 General Sampling Procedures

All samples were collected and stored in pre-cleaned glass bottles that had been rinsed thoroughly with nitric acid and analytical grade pentane in order to remove all heavy metals and organic residues. Sediment and solid waste samples were collected in 100ml bottles, and the water samples were collected in 1-litre bottles. All sediment, solid waste and water samples were immediately sealed and cooled upon collection. The samples were returned to the Greenpeace Research Laboratories for analysis. Detailed description of sample preparation and analytical procedures are presented in Appendix 1.

2.2 Sample Descriptions

Description of the samples collected is given in Table 1, and a map of the locations from which they were collected is present as Figure 1.

Sample Number	Sample Description	Sample Location
AM0102	sediment	Tributary to Sarapui river, upstream from Bayer
AM0103	sediment	Sarapui River, upstream from Bayer
AM0100	effluent	Discharge pipe near effluent treatment plant
AM0101	sediment	Sarapui river, near AM0100
AM0098	effluent	Discharge pipe near landfill
AM0099	sediment	Adjacent to AM0098
AM0097	sediment	Sarapui River, downstream from site
AM0104	solid waste	landfill
AM0105	solid waste	landfill

Table 1. Description of samples collected in and around the Bayer site in Rio de Janeiro. Samples are presented in two groups; those associated with the rivers and effluent discharges, arranged with those furthest upstream listed before those further downstream and; two samples of waste from the landfill inside the site.

Figure 1. Map of sampling locations.

3 RESULTS AND DISCUSSION

The results of the organic screen analysis and heavy metals analysis are presented in Table 2, including a breakdown of the groups of organic compounds reliably identified in the samples.

For more information on the common sources, environmental behaviour and toxicological outlines for key pollutants detected during this study, see Appendices 2 and 3.

3.1.1 The Rio Sarapui and associated samples

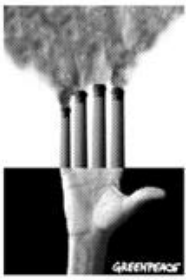
3.1.1.1 Control samples AM0102 and AM0103

Two control sediment samples were collected upstream of the Bayer site: sample AM0102 from the tributary to the Sarapui river and AM0103 from the Sarapui river itself (see Figure 1). 34 organic compounds were isolated from sample AM0102 and 20 of them were reliably identified. Similarly, 40 organic compounds were isolated from the sample AM0103, of which, 18 were reliably identified. Linear aliphatic hydrocarbons (alkanes) were predominant in both samples (14 in AM0102 and 11 in AM0103). This may indicate pollution of the sediments by petroleum, as alkanes are principal components of refined petroleum products (Overton 1994).

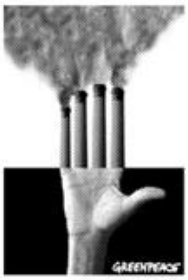
1,4-Dichlorobenzene was also detected in both samples AM0102 (at trace levels only) and AM0103. This compound is used in the production of deodorant blocks and room deodorants, so it is frequently found in sewage (Chapman *et al.* 1996). The presence of dihydrocholesterol, a compound that occurs in human faeces (Budavari 1989), in the sample AM0102 also confirms that the tributary of Sarapui river receives sewage.

Galaxolide, a polycyclic synthetic musk compound, which also known as Musk 50 or HHCB, was reliably identified in the sediment sample collected from Sarapui river (AM0103). Polycyclic synthetic musks are increasingly replacing synthetic nitro musks such as musk xylene (MX) and musk ketone (MK). World-wide production of polycyclic musks is estimated at 6 000 tonnes per year. Like MX and MK, polycyclic musks are detectable in many aquatic systems, particularly those receiving sewage (Rimkus 1999). Galaxolide is a highly persistent compound has a structure very similar to that of a natural human pheromone. It is absorbed directly through the skin and therefore may pose a health risk, though the potential health impacts of this compound have yet to be fully investigated (Kallenborn *et al.* 1999).

Two polycyclic aromatic hydrocarbons (PAHs) were reliably identified in these sediments: phenanthrene (in samples AM0102 and AM0103) and pyrene (in sample AM0103 only). PAHs are a group of compounds found in coal and oil. They are also formed during the combustion of coal, oil and gas (ATSDR 1997).



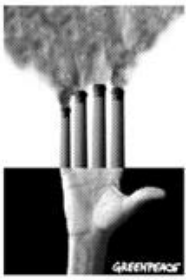
Sample Number	AM0102	AM0103	AM0100	AM0101	AM0098	AM0099	AM0097	AM0104	AM0105
Sample Type	sediment	sediment	effluent	sediment	effluent	sediment	sediment	waste	waste
Location	tributary to Sarapui, upstream of Bayer	Rio Sarapui, upstream of Bayer	discharge pipe near treatment plant	by discharge pipe near treatment plant	discharge pipe near landfill	by discharge pipe near landfill	Rio Sarapui down-stream of Bayer	from landfill	from landfill
METAL	mg/kg	mg/kg	ug/l	mg/kg	ug/l	mg/kg	mg/kg	mg/kg	mg/kg
Cadmium	<1	1	<20	30	<20	<1	1	70	9
Chromium	12	23	<20	2286	<20	426	64	455	52
Cobalt	2	6	<20	34	<20	10	10	19	<2
Copper	10	27	739	1115	<20	30	114	53888	<2
Lead	<3	<3	<30	426	57	53	96	5480	7
Manganese	93	287	236	1048	253	525	401	771	18
Mercury	0.15	0.20	4.2	22	<0.5	0.52	1.17	3.33	244
Nickel	<2	9	59	178	<20	37	23	683	26
Zinc	79	139	292	1884	13	192	527	25553	21
No. of organic compounds isolated	34	40	35	79	2	41	128	118	66
No. of organic compounds reliably identified	20(59%)	18(45%)	17(49%)	64(81%)	2(100%)	13(32%)	71(55%)	74(63%)	11(17%)
Groups of organic compounds reliably identified									
ORGANOHALOGEN COMPOUNDS									
Benzene, 1,2-dichloro-				*				1	
Benzene, 1,4-dichloro-	*	1		*	1	1	*	1	*
Benzene, 1,2,4-trichloro-				*				*	
Benzene, 1,2,3,5-tetrachloro-							*	*	
Benzene, 1,2,4,5-tetrachloro-								*	
Benzene, 1,2,3,4-tetrachloro-							*	*	
Benzene, pentachloro-							*	*	
Benzene, hexachloro-								1	
Benzenamines, trichloro-								1	
Benzenamine, 4-(trifluoromethoxy)-			1						
Benzenamine, 2,4,6-tribromo-								1	
Benzenamine, 2,6-dibromo-4-chloro-								1	
Benzamide, 4-chloro-			1						
Benzene, isocyanato-4-(trifluoromethoxy)-			1						
Trichlorobiphenyl isomers				1			*	*(2)	
Tetrachlorobiphenyl isomers				3			2, *(4)	*(2)	
Pentachlorobiphenyl isomers				8			8, *(3)	*(8)	
Hexachlorobiphenyl isomers (other than 138&153)				11			1, *(7)	*(6)	
Heptachlorobiphenyl isomers (other than 180)				5			*	*(3)	
Octachlorobiphenyl isomers				2					
PCB-138				1			1	*	
PCB-153				1			1	*	
PCB-180				1			*	*	
Fyrol PCF			1						
o,p'-DDE				1					
p,p'-DDE				1		*			
o,p'-DDD				*					
p,p'-DDD				1		*			
p,p'-DDT				1		*			
alpha-HCH				*		*	*		
beta-HCH	1			1		1	1	1	
gamma-HCH							*		
delta-HCH							*		
Phenol, 2,5-dichloro-								*	



Sample Number	AM0102	AM0103	AM0100	AM0101	AM0098	AM0099	AM0097	AM0104	AM0105
Sample Type	sediment	sediment	effluent	sediment	effluent	sediment	sediment	waste	waste
Location	tributary to Sarapui, upstream of Bayer	Rio Sarapui, upstream of Bayer	discharge pipe near treatment plant	by discharge pipe near treatment plant	discharge pipe near landfill	by discharge pipe near landfill	Rio Sarapui down-stream of Bayer	from landfill	from landfill
PAHs									
Naphthalene and/or its derivatives			3	4				6	
Anthracene and/or its derivatives								2	
Acenaphthene			1					1	
Phenanthrene and/or its derivatives	1	1						2	
Pyrene and/or its derivatives		1	1	1			1	1	
9H-Fluorene and/or its derivatives								1	
Fluoranthene			1	1				1	
ORGANOSULPHUR COMPOUNDS									
Dibenzothiophene and/or its derivatives								1	
Benzothiazole, 2-(methylthio)-			1		1				
PHENOLIC COMPOUNDS									
Phenol						1			
Phenol, 3-methyl-						1			
PHTHALATE ESTERS									
DEHP						1			
DiBP									1
OTHER AROMATIC COMPOUNDS									
Alkylated benzenes			1				5	4	
Biphenyl and/or its derivatives								1	
Diphenyl ether			1					1	
Benzaldehyde	1	1		1		1			
Benzenemethanol	1	1		1					
1H-Indole and/or its derivatives						1			
Galaxolide (Musk 50)		1							
Dibenzofuran								1	
Benzamine, N-methyl-			1						
ALIPHATIC HYDROCARBONS									
Linear	14	11		6		1	19	14	9
Cyclic			1	6			1		
MISCELLANEOUS									
Terpenoids						1			
Quinoline derivatives			2						
Stannane, tetracyclohexyl-				1				1	
Dihydrocholesterol	1								

*Table 2. Organic chemicals and heavy metals identified in samples collected in and around the Bayer site in Rio de Janeiro. For the groups of organic compounds reliably; # signifies the number of compounds reliably identified using the general GC/MS screening method; *(#) signifies compounds identified only at trace levels using a selective ion monitoring (SIM) method, with the number of compound in parentheses denoting how many compounds within that group were identified. Metal concentrations are given in mg/kg dry weight for solid samples and ug/l for liquid samples. Samples are presented in two groups; those associated with the rivers and effluent discharges, arranged with those furthest upstream listed before those further downstream and: two samples of waste from the landfill inside the site.*

Two aromatic compounds, benzenemethanol and benzaldehyde, were found in both sediment samples AM0102 and AM0103. Benzenemethanol, which also called benzyl alcohol, is a liquid with faint aromatic odor and sharp burning taste. It is produced by the reaction of sodium or potassium carbonate with benzyl chloride. Its main uses include manufacture of other benzyl compounds, pharmaceutical aid (antimicrobial), as a solvent, in perfumery and in



flavouring (Budavari 1989). Benzaldehyde is an artificial essential oil of almond. It is used in the preparation of certain aniline dyes and of other products, including perfumes and flavourings. It is also used as a solvent. Benzaldehyde is narcotic in high concentrations and it may cause contact dermatitis (Budavari 1989).

In addition, the beta-isomer of hexachlorocyclohexane (beta-HCH) was detected in sample AM0102. The presence of beta-HCH at this location might due to the use of technical HCH as an insecticide in the past, since HCH can persist for many years in the environment (Martijn *et al.* 1993) and the beta-isomer is the most persistent of the HCH isomers (ATSDR 1997).

Concentrations of all heavy metals considered in this study were in the range of the environmental background levels for both control sediment samples AM0102 and AM0103.

3.1.1.2 Effluent sample AM0100 and associated sediment AM0101

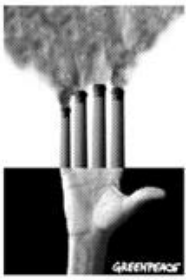
These two samples were collected from a discharge point to the Sarapui river close to the Bayer treatment plant. 35 organic compounds were isolated from effluent sample AM0100 and 17 of these were reliably identified. Groups of organic compounds found in this sample included derivatives (both halogenated and non-halogenated) of benzene and benzenamine (aniline), polycyclic aromatic hydrocarbons (PAHs), chlorinated alkyl phosphate esters, quinoline derivatives, as well as 2-(methylthio)benzothiazole, diphenyl ether and cyclic alkanes (see Table 2).

Benzenamine, 4-(trifluoromethoxy)-, also called 4-trifluoromethoxyaniline, is produced by the Bayer company (Bayer 2000a). A technical datasheet of the Bayer company for 4-trifluoromethoxyaniline (Bayer 2000b) states the applications for this compound as "pharmaceutical agents, pharmaceutical industry". Also in the same technical datasheet "Toxic" hazardous symbol is applied to 4-trifluoromethoxyaniline.

Three more nitrogen-containing aromatic compounds were detected in sample AM0100: N-methylbenzenamine, 4-chlorobenzamide and isocyanato-4-(trifluoromethoxy)benzene. These chemicals might also be associated with the production of pharmaceuticals or their intermediates, since Bayer produces a large number of similar compounds (Bayer 2000a).

Several polycyclic aromatic hydrocarbons (PAHs) were also detected in the effluent sample AM0100, including naphthalene and its methylated derivatives, acenaphthene, pyrene and fluoranthene. PAHs are components of crude oil and may also be formed during incomplete combustion of coal, oil and gas (Overton 1994).

Three isomers of tris(monochloropropyl) phosphate (TMCPPs) have been identified in the effluent sample AM0100 to a high degree of reliability. In fact, these compounds were the most abundant in the chromatogram of this sample. One of the applications of TMCPPs is as a flame retardant in rigid and flexible polyurethane foams (NRC 2000). TMCPPs are usually



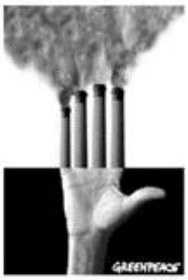
sold as mixtures under various trade names including Antiblaze 80, Amgard TMCP, Fyrol PCF and Hostaflam PO 820. The mixture of TMCPP isomers identified in sample AM0100 was closely matched with the industrial Fyrol PCF produced by Akzo Nobel company. It is likely that Fyrol PCF is used by the Bayer company in the production of polymers, and treatment procedures employed do not eliminate this flame retardant from the effluents.

A few more organic compounds, such as diphenyl ether, 1-methoxy-2-methylbenzene, quinoline derivatives, cyclic alkenes and 2-(methylthio)benzothiazole, were reliably identified in effluent sample AM0100. These compounds and/or their derivatives may be used as reagents in organic synthesis (Budavari 1989).

79 organic compounds were isolated from the sediment sample AM0101 collected by the pipe discharging near to the Bayer treatment plant. 64 compounds were reliably identified and 33 of them were polychlorinated biphenyls (PCBs) - from tri- to octachlorinated congeners. This sample was also compared to a standard of the industrial PCB mixture Aroclor 1254 (chlorine content 52-54%). The range of PCB congeners detected in the sample showed that sample contains additional PCB congeners to those present in the Aroclor 1254 - octachlorinated biphenyls. This might indicate the presence of two different industrial PCBs mixtures (also known as askarels) of a higher chlorine content, or a mixture of two such technical mixes (eg Aroclor 1254 (54% chlorine) and Aroclor 1260 (60% chlorine)).

Trace amounts of chlorinated benzenes were also found in sediment sample AM0101 which might be due to their use as solvents in PCB-containing transformer oils (Swami *et al.* 1992, de Voogt & Brinkman 1989).

The chlorinated pesticide DDT and its derivatives were reliably identified in this sediment sample, though some of them were present only at trace levels (see Table 2). Technical DDT comprises several derivatives and p,p-DDT is usually the main component (ATSDR 1997). The pattern of DDT derivatives found in this sample probably shows historical contamination because the most abundant derivative found in the sample was p,p'-DDE, a breakdown product of p,p'-DDT. The same explanation could be given for the isomers of hexachlorocyclohexane (HCH) detected in this sample. Beta-HCH was more abundant than alpha-HCH, which was detected only at trace levels. It is known (Safe 1993) that technical HCH contains alpha-HCH as a main component, and the beta-HCH is the most resistant to biodegradation (ATSDR 1997). DDT derivatives were also detected further downstream in the sediment sample (AM0097), but only at trace levels. HCH isomers were detected in both sediment samples from the Sarapui river downstream of Bayer site (AM0101 and AM0099), in one sample from the landfill (AM0104), and even in the sediment sample from the tributary to Sarapui upstream of Bayer site (AM0102). The source of these compounds to the Sarapui river is unclear. It is possible that either the Bayer treatment plant has discharged DDT and HCH in the past or that contamination of the river basin was due to DDT and HCH application as pesticides in this area.



PAHs found in the sediment sample (AM0101) in general matched those that been identified in the effluent sample (AM0100). The possible sources of PAHs are described above.

Benzaldehyde and benzenemethanol also were detected in sample AM0101. However, the same compounds were found in the control samples (AM0102 and AM0103). Therefore, it may indicate either transport of these compounds from the upstream or discharge from the Bayer treatment plant. Aliphatic hydrocarbons (alkanes), both linear and cyclic, that been identified in the sample AM0101 might indicate contamination by petroleum products.

The organotin compound tetracyclohexylstannane (tetracyclohexyltin) was reliably identified in the sediment sample AM0101. Little information is available on this particular compound. Organotin compounds are extensively used as antifouling paint biocides, fungicides, antifeedants, acaricides, disinfectants, heat and light stabilisers for rigid PVC, catalysts for polyurethane foams and transesterification reactions (Blunden & Chapman 1986). The tetraorganotins do not have any large-scale commercial outlet, but are important intermediates in the manufacture of other organotins (Blunden & Chapman 1986).

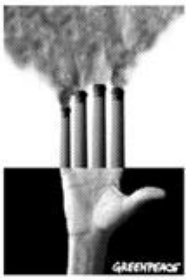
Several heavy metals were detected in the effluent sample AM0100 at high levels. Copper concentrations were more than 30 times higher than typical background levels for freshwaters (Mance *et al.* 1984), manganese was about 20 times higher (Bowen 1966), mercury was more than 800 times higher (ATSDR 1997) and zinc about 6 times higher (ATSDR 1997). Sediment sample AM0101 also has highly elevated levels of copper, cadmium, lead, mercury and zinc in comparison with typical background levels for freshwater sediments (Salomons & Forstner 1984, ATSDR 1997): 22 times for copper, 30 times for cadmium, approximately 20 times for lead, approximately 100 times for mercury and approximately 20 times for zinc. Nickel and chromium concentrations were also slightly elevated in this sample.

The high levels of heavy metals in the effluent (AM0100) indicate that waste treatment procedures employed on the Bayer treatment plant are not efficient for heavy metal removal. This leads to the contamination of the river water and sediments by heavy metals because they tend to bind to suspended material and accumulate in the bottom sediments (ATSDR 1997, Bryan & Langston 1992).

Heavy metals exert a broad range of toxic effects on humans, terrestrial and aquatic life and plants. A number of these metals also have the potential to bioaccumulate, including cadmium, chromium, lead, mercury and zinc (ATSDR 1997, Kimbrough *et al.* 1999, MINDEC 1995). In addition, certain forms of cadmium and chromium have carcinogenic properties (DHHS 2000).

3.1.1.3 Effluent sample AM0098 and associated sediment AM0099

Only two organic compounds were isolated and reliably identified in the effluent sample AM0098 - 1,4-dichlorobenzene and 2-(methylthio)benzothiazole. 1,4-Dichlorobenzene was



detected in every sample in this study except AM0100. 2-(Methylthio)benzothiazole was also detected in effluent sample AM0100. Possible sources of these compounds were described above (see sections 3.1.1.1 and 3.1.1.).

Sediment sample AM0099, which was collected near the pipe where effluent sample AM0098 was collected, also contained fewer organic compounds (a total 41 were isolated) than the sediment sample AM0101 collected by the pipe from the treatment plant. Moreover, fewer (13) were reliably identified.

Sediment sample AM0099 contained DDT derivatives, though fewer than in sample AM0101 collected further upstream, and only at trace concentrations. Alpha- and beta-HCH isomers, benzaldehyde and 1,4-dichlorobenzene were also detected in sample AM0099 as well as in sample AM0101. These contaminants might therefore have been transported from upstream, or might indicate local input.

Several organic compounds were reliably identified in sample AM0099 that were not present in sample AM0101: neophytadiene, phenol, 3-methylphenol (or meta-cresol), 1H-indole, and bis(2-ethylhexyl) phthalate (DEHP). Neophytadiene is a terpenoid compound that may occur naturally. IH-Indole is a compound that can be obtained from the 240-260^{0}C fraction from coal tar; it is also present in faeces (Budavari 1989).

Phenol is mainly a man-made chemical, although it is found in nature in animal wastes and organic material. The largest single use of phenol is to make plastics, but it also is used to make caprolactam and bisphenol A (used to make epoxy and other resins). Phenol has been found in materials released from landfills and hazardous waste sites, and it has been found in the groundwater near these sites (ATSDR 1997). Cresols are natural products that are present in many foods and in animal and human urine. They are also present in wood and tobacco smoke, crude oil, and coal tar. In addition, cresols are man-made and used as disinfectants and deodorisers, as a solvent and as starting chemicals for making other chemicals (ATSDR 1997). It is a possibility that phenolic compounds found in this sample might be associated with the production at the Bayer company. However, other sources should not be discounted since these compounds were not detected in effluent sample AM0098.

DEHP was one of the phthalates produced in the greatest quantities in the past (Menzert & Nelson 1986). It was mainly used as a plasticizer in the production of soft PVC (Cadogan *et al.* 1993). It is also used in inks and dyes (Jobling *et al.* 1995). However, since phthalates are the most abundant man-made chemicals in the environment (Jobling *et al.* 1995) and can persist for a long period of time, it is not possible to ascribe a source for this chemical in sample AM0099.

The range and concentrations of heavy metals detected in both effluent sample AM0098 and sediment sample AM0099 were much lower than those found in the samples AM0100 and AM0101 (see Table 2). The levels of lead were 10 times higher in the effluent sample and

about 2 times higher in the sediment sample in comparison with the background levels for freshwaters and freshwater sediments correspondingly (ATSDR 1997). Manganese levels were about 20 times higher in the effluent sample than the levels considered normal for unpolluted freshwater (Bowen 1966). All other metals considered in this study were in the range of background levels.

It is not known what part of the Bayer plant discharges effluent through this pipe. Therefore, it is unclear what the source of these metals in the effluent sample could be. A primary use of lead, at least in the past, was as pipe-work for water distribution. Compounds of lead are also used as paint pigments, PVC stabilisers, pesticides, varnishes, lubricants, as glazes for pottery and porcelain and in leaded glass crystal, in cable coverings and in the manufacture of tetraethyl lead (Budavari *et al.* 1989, ATSDR 1997).

3.1.1.4 Sediment sample AM0097 downstream of Bayer site

The pattern of organic contaminants in sediment sample AM0097 was very similar to that in the sediment sample (AM0101) collected by the pipe from the treatment plant further upstream. 128 organic compounds were isolated from sample AM0097. 71 compounds were reliably identified and 37 of those were polychlorinated biphenyls (trichlorinated to heptachlorinated congeners). This sample was also compared to an Aroclor 1254 standard mixture (chlorine content 52-54%) and showed a very good match. Octachlorinated congeners of PCBs were not detected in this sample. Four isomers of HCH (alpha-, beta, gamma- and delta-HCH) were reliably identified in this sample with the beta-HCH being the most abundant of the isomers. Only one representative of PAHs, pyrene, was detected in this sediment sample.

Four chlorinated benzenes (1,4-dichlorobenzene, two tetrachlorinated isomers and pentachlorobenzene) were detected in sample AM0097 at trace levels. As discussed above (see sections 3.1.1.1 and 3.1.1.2), tri- and tetrachlorinated benzenes are used as a solvent in transformer oils containing PCBs and this may explain the presence these compounds in the sediment sample. Pentachlorobenzene is also present in dielectric fluids and can enter the environment through spillage of atmospheric transport (Giddings *et al.* 1994c).

In contrast with sample AM0101, more aliphatic hydrocarbons and alkylated benzenes were detected in sample AM0097, which might show local contamination by petroleum products.

The distribution of heavy metals found in the sediment sample AM0097 was generally similar to that found in sediment sample AM0101 (see Figure 2), but with contaminants at lower concentrations. This provides evidence that heavy metal contamination in this section of the Sarapui is solely due to inputs from the Bayer treatment plant because, as discussed in section 3.1.1.1, all heavy metals analysed in this study were present at background levels upstream of Bayer plant.

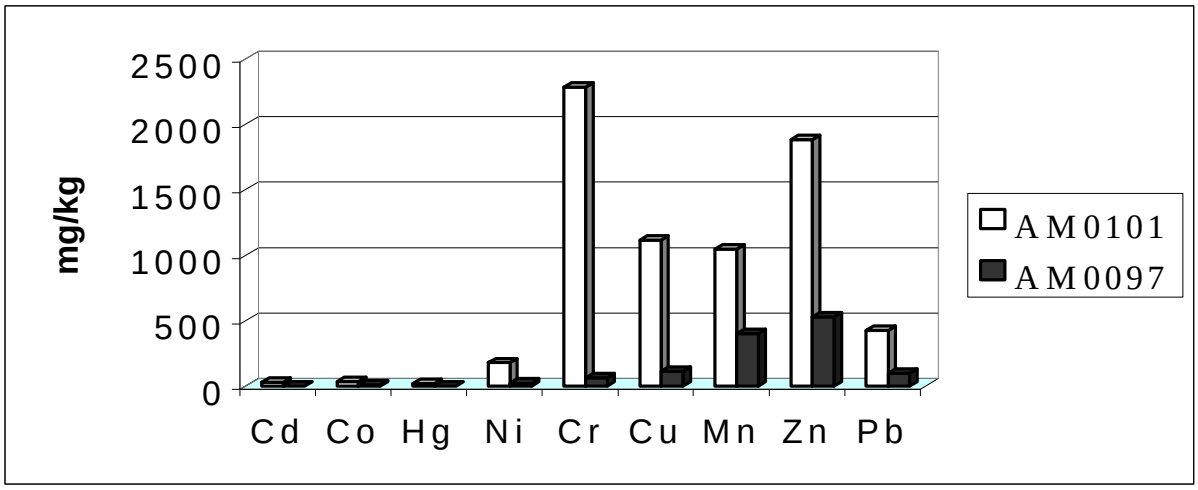


Figure 2. Distribution of heavy metals in the sediment samples collected from the Rio Sarapui, Brasil. AM0101 - sample collected by the discharge pipe from the Bayer treatment plant, AM0097 - sample collected downstream of the Bayer plant.

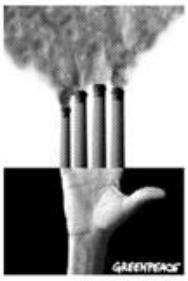
3.1.2 The Landfill

Two samples of solid waste (AM0104 and AM0105), which were collected from the Bayer landfill, show very different composition of both organic contaminants and heavy metals.

Sample AM0104 was a reddish sludge and contained almost all of the organic compounds which were detected in the samples which were collected by the discharge near the treatment plant (effluent AM0100 and sediment AM0101). 118 organic compounds were isolated from the sample AM0104 and, of these, 74 were reliably identified. These included 21 congeners of polychlorinated biphenyls, chlorinated benzenes, halogenated benzenamines, beta-HCH, several PAHs, alkylated benzenes, diphenyl ether, linear alkanes and tetracyclohexyltin (see Table 2). More isomers of chlorinated benzenes and PAHs were found in this sample than in sediment sample AM0101. However, DDT derivatives, which were found in sediment sample AM0101, were not detected in the waste sample AM0104.

Waste sample AM0104 had the highest level of cadmium, copper, lead, nickel and zinc among all the samples considered in this study. The similarity between the samples from these two sites suggests that AM0104 might represent sludges from the wastewater treatment plant. Further, the chlorinated benzenes and PCBs might be present as a result of imperfectly combusted PCBs from the incinerator being trapped by the air pollution control devices and passed to the wastewater treatment plant in the effluents. However, there is presently not enough data to establish whether this is in fact the case.

The other waste sample collected from the Bayer landfill (AM0105) was clearly different to AM0104. It consisted of small cylinders of a grey/yellow/green colour. It is possible that they were spent catalysts or other similar materials.



The most important finding concerning waste sample AM0105 was extremely high levels of mercury present in this sample - 244 mg/kg (parts per million). The concentration of mercury in sample AM0105 exceed background levels for uncontaminated soil from 300 to 12 000 times (Alloway 1990, WHO 1989).

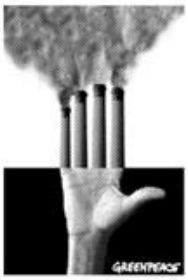
A much lower number of organic compounds was isolated and reliably identified than in AM0104 (see Table 2). Of eleven reliably identified organic compounds, nine were linear aliphatic hydrocarbons. 1,4-Dichlorobenzene was detected only at trace levels. Possible sources of these compounds have been discussed in previous sections. Diisobutyl phthalate (DiBP) was also detected in the sample AM0105. No data were available on toxicological problems associated with this phthalate. However, its close relative, di-n-butyl phthalate (DnBP), which was produced in large quantities ten to fifteen years ego (Menzert & Nelson 1986), exhibits a wide range of toxic effects in laboratory animals (Chan & Meek 1994, ATSDR 1997, Wine *et al.* 1997, Ema *et al.* 1995).

4 CONCLUSIONS

A wide range of organic pollutants and elevated levels of heavy metals were found in the effluent sample from the Bayer treatment plant, discharging into the Sarapui river, and in the sediment sample by the point of discharge. The effluent sample contained PAHs, halogenated benzenamine, benzene and benzamide, it also showed the presence of the flame retardant Fyrol PCF, which was the most abundant compound in this sample. The sediment sample contained chlorinated benzenes, PCBs, DDT derivatives, PAHs and HCH isomers, and indicates that this discharge is a significant source of pollution to the river. It is also plausible that some of the pollutants collected here, such as the PCBs, originated in wastes sent for incineration at this site. A sediment sample from the Sarapui river, collected further downstream of Bayer site, showed similar contaminants, but at lower levels.

Analysis of effluent from the pipe near the landfill showed that neither organic compounds nor heavy metals were at levels as high as the samples from the discharge point near the treatment plant. Trace amounts of DDT derivatives and beta-HCH found in the sediment sample by the pipe near the landfill may either have been discharged from Bayer at this point, or may have been transported from upstream attached to suspended particles. Local use as an insecticide is also a possibility.

One waste sample from the Bayer landfill was highly contaminated with heavy metals and contained wide range of organic pollutants including PCBs, chlorinated benzenes, halogenated benzenamines, PAHs and beta-HCH. This sample was a reddish sludge, which may have come from a wastewater treatment plant. Assuming that this sample comes from the Bayer wastewater treatment plant, the presence of PCBs and other chloro-organic compounds might indicate incomplete combustion of hazardous wastes by the incinerator.



Another waste sample from Bayer landfill contained mercury as a main pollutant at very high concentration indicating that landfill receives a range of hazardous wastes.

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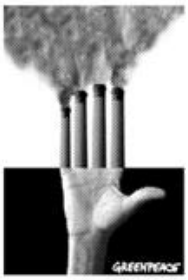

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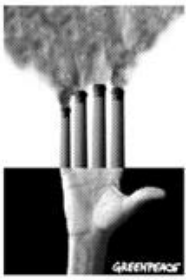


APPENDIX 1
ANALYTICAL METHODOLOGY

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.

A1.1.1.1 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. They 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. The concentrated extract was cleaned through a Florisil column, eluted with a 95:5 mixture of pentane: toluene, and evaporated down to a volume of 2 ml under a stream of analytical grade nitrogen. 1-Bromonaphthalene was then added at concentration 10mg/l to provide an indication of GC/MS performance.

A1.1.1.2 Aqueous Samples

Prior to the extraction, samples were spiked with deuterated naphthalene (an internal standard) at a concentration of 10mg/l. 20ml of pentane were 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 hydrophobic phase separator filter and collected in 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 a Hewlett Packard (HP) 5890 Series II gas chromatograph, interfaced with a HP Chem-Station data system and linked to a HP 5972 Mass Selective Detector operated in scan mode. The identification of compounds was carried out by computer matching against a HP Wiley 275 library of 275,000 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. Additionally, samples were analysed using SIM method against PCBs standard mixture Aroclor 1254, obtained from Chem Service Inc., UK.

Compound	**Ions to monitor**
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
Simazine	200, 215, 202, 173
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 A.1.1 List of compounds in the Standard I used for SIM analysis

Results are reported as either reliably or tentatively identified. Match qualities of 90% or greater against HP Wiley 275 library 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 HP Wiley 275 library only. Analytes yielding match qualities of 50% or less are assumed to be unidentified.

Compound	**Ions to monitor**
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, 2,5-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, 2,3,5-trichloro-	196, 198, 160, 97
Phenol, 2,4,5-trichloro-	196, 198, 97, 132
Phenol, 3,5-dichloro-	162, 164, 99, 63
Phenol, 2,3,6-trichloro-	196, 198, 97, 132
Phenol, 3,4-dichloro-	162, 164, 99, 63
Atrazine	200, 215, 202, 173
Phenol, pentachloro-	266, 268, 264, 165
Chlordane I	373, 375, 272, 237
Chlordane II	373, 375, 272, 237
PCB-153	360, 362, 290, 218
PCB-138	360, 362, 290, 292
PCB-180	394, 396, 324, 252

Table A.1.2 List of compounds in the Standard II used for SIM analysis

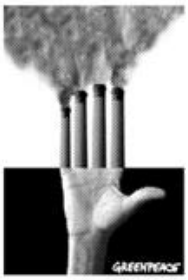
A1.2. Heavy Metal Analysis

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.

A1.2.1.1. 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.



0.5 g of sample was 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.

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.

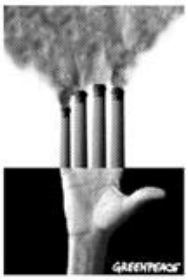
A1.2.1.2. Aqueous samples

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.

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

Following preparation, samples were analysed by ICP-AES, using a Varian Liberty-100 Sequential Spectrometer. The following metals were quantified directly: manganese, chromium, zinc, copper, lead, nickel, cobalt and cadmium. 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. Hg (II) was reduced to Hg (0) i.e. a vapour, following reduction of the samples with sodium borohydride (0.6% w/v), sodium hydroxide (0.5% w/v) and hydrochloric acid (10 molar). The vapour was carried in a stream of argon into the spectrometer. Two calibration standards were prepared, at 10 ug/l and 100 ug/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 (80 ug/l), prepared internally from different reagent stock. Any sample exceeding the calibration range was diluted accordingly, in duplicate, and re-analysed.



APPENDIX 2
TOXICOLOGICAL OUTLINES FOR KEY ORGANIC COMPOUNDS

A2.1 Polychlorinated biphenyls (PCBs)

Polychlorinated biphenyls are a group of synthetic organic chemicals that contain 209 individual compounds (known as congeners) with varying harmful effects. There are no known natural sources of polychlorinated biphenyls in the environment. PCBs are either oily liquids or solids, and are colourless to light yellow in colour. PCBs enter the environment as mixtures containing a variety of individual components and impurities.

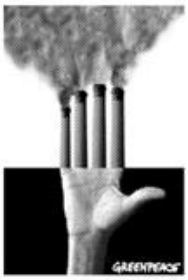
The polychlorinated biphenyls have been used in a wide variety of applications, including transformer oils, hydraulic fluids, plasticisers, 'kiss-proof' lipsticks and carbonless copy papers. They have also been used in capacitor dielectrics, heat transfer fluids, lubricating and cutting oils, and in paints and printing inks (ATSDR 1997).

PCBs have always been sold as technical mixes rather than individual chemicals. de Voogt & Brinkman (1989) list some 46 trade names used for PCBs and PCB-containing products. Of these, the Aroclor range manufactured by the American company Monsanto was probably the most widely used. The most important PCB applications in tonnage terms were transformer oils and capacitors (de Voogt & Brinkman 1989). In transformer oils, the PCBs were mixed with chlorobenzenes (mainly trichlorobenzenes and tetrachlorobenzenes) as solvents (Swami *et al*. 1992, de Voogt & Brinkman 1989). PCBs are also synthesised as by-products in processes ranging from incinerators (USEPA 1998, Ballschmiter *et al.* 1989, Alcock *et al.* 1998) to metallurgical processing (Knutzen & Oehme 1989, Alcock *et al.* 1998, Thiesen *et al.* 1993) to dye manufacturing (USEPA 1998).

PCBs can be absorbed through the skin as well as through ingestion and inhalation. For the general population today, food is the primary source, though dermal exposure may be dominant amongst those directly handling PCBs or PCB-contaminated materials (Lees *et al*. 1987).

Kidney cancer has been reported in workers with known exposure to PCBs although insufficient data are available for statistical analysis and more research is called for (Shalat *et al*. 1989). In a review of epidemiological PCB research, cancer of the kidney and skin were marginally significant but the reviewers regarded the overall picture as inconclusive (Longnecker *et al*. 1997). Exposure of “clean” PCBs in an occupational setting exerts effects on the human CNS, with symptoms such as headaches, lassitude and slowed nerve signals (Rogan & Gladen 1992).

In a review of PCB toxicity, Safe (1984) lists the following symptoms of PCB toxicity: enzyme induction, decreased vitamin A levels, lymphoid involution, thymic and splenic



atrophy, immunosuppression, chloracne, alopecia, oedema, hyperkeratosis, blepharitis, hyperplasia of the epithelial lining of the extrahepatic bile duct, the gall bladder and urinary tract, hepatomegaly and liver damage including necrosis, haemorrhage, hepatotoxicity (altered porphyrin metabolism), tumour promotion, altered levels of steroid and thyroid hormones, reproductive toxicity including menstrual irregularities, reduced conception, early abortion, excessive menstrual and postconceptual haemorrhage, anovulation, testicular atrophy, decreased spermatogenesis, teratogenesis and developmental toxicity. In addition, low levels of PCBs have caused behavioural impairment in monkeys (Rice 1999).

Aroclors 1221, 1254 and 1268 all reduced in vitro fertilisation rates in mice, with PCB 1254 being the most potent mixture (Kholkute *et al*. 1994). Aroclor 1254 also compromised the immune response of earthworms (Roch & Copper 1991).

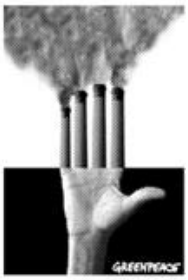
Although much of the toxicological research relates to technical mixtures of PCBs, individual congeners have different effects and act through several different mechanisms. Certain of the PCBs are called coplanar since the molecules can take of a flattened shape and these can act in the same was as the dioxins.

Some congeners, or their metabolites, exhibit endocrine disruption, including both oestrogenicity and anti-oestrogenicity. In general, ortho-substituted PCBs are oestrogenic whereas coplanar PCBs are anti-oestrogenic, as is 2,3,7,8-TCDD (Li *et al*. 1994). According to a recent review (Brouwer *et al*. 1999), PCBs may affect not only the oestrogen system, but also the androgen system, the thyroid hormone system, the retinoid system, the corticosteroid system and several other endocrine pathways. In addition, effects on the thyroid system on wild populations of fish-eating birds and captive seals have been correlated with PCB exposure (Brouwer *et al*. 1999).

Ortho-substituted (non-dioxin-like) PCBs have been found to have the greatest effects on neurochemical function. They were found to reduce dopamine synthesis and it was further established that the effects were caused by the congeners rather than their metabolites. 2,2'-dichlorobiphenyl (PCB 4) was the most potent congener (Seegal & Shain 1992).

The dioxin-like PCB 77 (3,3',4,4'-TeCB) also caused long-term changes in behavioural and neurochemical changes in laboratory animals, including alterations in dopamine function. This congener, however, did not accumulate in brain tissue in the same way as some ortho-substituted congeners, indicating that it operates via a second mechanism, or that it is a metabolite which is the active agent (Seegal & Shain 1992).

The extensive body of information concerning the global cycling of PCBs has been accumulated in response to concerns about the environmental impact of these chemicals. PCBs are highly persistent. Although there is evidence of biodegradation in contaminated sediments (see: Brown & Wagner 1989) and some marine mammals appear to be able to selectively degrade some of the lower chlorinated congeners (Boon *et al*. 1987), the



detoxification potential of these processes would appear to be rather limited. Indeed, Cummins (1988) has suggested that unless further escape of PCBs is prevented then the eventual extinction of marine mammals is a very real possibility.

Levels of PCBs in biological material may be several orders of magnitude higher than ambient. PCBs are bioconcentrated to a factor of 6 000 for fish and 47 000 for invertebrates (Jones *et al*. 1988). Train (1979) reports bioconcentration factors of between 2 500 and 100 000.

The effects of chronic exposure to PCBs in marine mammals has been found to include physical deformity and impairment of reproductive success (Reijnders 1986). More recently, they have been implicated in the outbreaks of disease amongst populations of seals and dolphins (see review by Gilbertson 1989) suggesting that they may have a disruptive influence on immune capability.

The PCBs are controlled under most of the international legal instruments relating to organochlorines, *inter alia*, the Barcelona, Helsinki, Basel, Bamako, Rotterdam OSPAR and LRTAP Conventions and the International Joint Commission on the Great Lakes. In addition, PCBs are targeted for global production ban under the UNEP POPs Convention, which will be signed in Stockholm in May 2001. Within the EC, applications for the PCBs were first restricted by directive 76/769/EEC, which deals with the marketing and use of dangerous substances and preparations (EC 1976). This directive, and its amendment (EC 1991), restricted the applications of PCBs and their replacements, the polychlorinated terphenyls (PCTs).

EC regulations on disposal of PCBs, as set out in a 1996 Directive, dictate that the PCB phaseout should be completed by 2010. Further, national enabling legislation should have been emplaced by March 1998. Several countries have missed this deadline and in mid 1999, the EC initiated action through the European Court of Justice against Germany, Greece, Spain, Portugal and UK for failing to implement the directive (ENDS 1999).

The US Toxics Substances Control Act (TOSCA) designates wastes containing greater than 50ppm PCBs are designated as hazardous (Rogan 1995).

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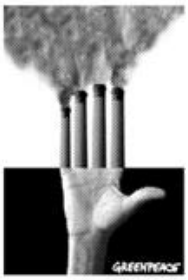

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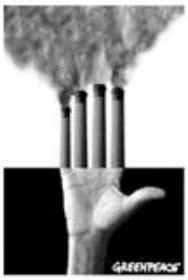

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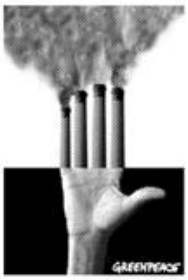
A2.2 Hexachlorocyclohexane (HCH)

Mixtures of hexachlorocyclohexanes are produced by the photochemical reaction between chlorine and benzene (Safe 1993). Technical grade hexachlorocyclohexane (HCH) is comprised of different isomeric forms. The approximate isomer content is alpha-HCH (60-70%), beta-HCH (7-10%), gamma-HCH (14-15%), delta-HCH (7%), and epsilon-HCH (1-2%). Lindane is the gamma isomer of hexachlorocyclohexane and it is commercially produced by purification of the technical HCH (Safe 1993). This compound has been produced worldwide for use as an insecticide to control grasshoppers, cotton and rice pests, wireworms and other soil pests. Lindane has been used for protection of seeds, for treatment of poultry and livestock and for control of household insects. It is also still used as a scabicide and pediculocide, usually in lotions, creams, and shampoos.

Alpha-, beta-, and gamma-HCH are the most important isomers in terms of environmental impact. The relatively high stability and lipophilicity of HCH and its global use pattern has resulted in significant environmental contamination by this chlorinated hydrocarbon. Once introduced into environment HCH may persist for many years (Martijn & Schreuder 1993). The beta-isomer is more persistent than others (ATSDR 1997).

Human intake of HCH compounds is largely through food consumption (Toppari *et al.* 1995). Alpha-, beta- and gamma-HCH have been recorded in human breast-milk with the beta-isomer being the most ubiquitous (Waliszewski *et al.* 1996, Safe 1993). The generally less widespread nature of the alpha- and gamma-isomers in comparison to beta-HCH is due to the more rapid clearance of these isomers from the body. Like many persistent organochlorines, HCH levels in the body have been found to increase with age (ASTDR 1997).

Hexachlorocyclohexane isomers have been detected in air, surface and ground water, soil and sediments (El-Gendy *et al.* 1991, Safe 1993, Xu 1994, Tan & Vijayaletchumy 1994, Skark & Zullei-Seibert 1995, Ramesh *et al.* 1991), plants (Xu 1994), birds, fish and mammals (Smith 1991, Xu 1994, Abd-Allah 1994, Norstrom & Muir 1994). In humans lindane mostly concentrates in adipose tissue (Safe 1993). It has been reported that lindane and other organochlorine compounds can be transferred through the pathway



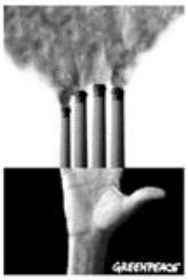
soil→earthworm→bird/mammal (Hernandez, *et al.* 1992, Romijn *et al.* 1994) thereby causing secondary poisoning.

Lindane, the gamma-isomer of hexachlorocyclohexane, is toxic to animals, humans, and aquatic species. Acute animal poisoning by lindane causes increased respiratory rate, restlessness accompanied by increased frequency of urination, 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 (Smith 1991).

Chronic health effects can occur at some time after exposure to lindane and can last for months or years. Lindane has been shown to cause liver, lung, endocrine gland and certain other types of cancer in animals (Smith 1991). Repeated overexposure may damage the liver. Chronic toxic effects may also include shortened lifespan, reproductive problems, lower fertility, and changes in appearance or behaviour. The differential actions of hexachlorocyclohexane isomers may produce variable effects on different regions of the nervous systems and in different species of animals (Nagata *et al.* 1996).

Hexachlorocyclohexane may be introduced to the environment from industrial discharges, insecticide applications or spills, and may 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). The insecticide load in surface waters does not ordinarily reach concentrations acutely toxic to aquatic fauna. However, lindane has high chronic toxicity to aquatic life. 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 macroinvertebrates 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.

Hexachlorocyclohexane, as a toxic, persistent and bioaccumulative chemical, is a subject to European Community legislation. The limit values and quality objectives for discharges of hexachlorocyclohexane are set by Council Directive 84/491/EEC (EEC 1984) as amended. The uses of hexachlorocyclohexane (including lindane) were severely restricted under the LRTAP 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 is 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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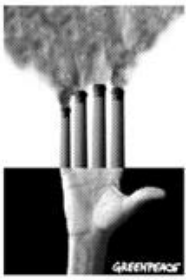

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A2.3 Tris(monochloroisopropyl) phosphates (TMCPPs)

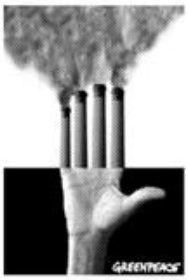
Tris(monochloropropyl) phosphates (TMCPPs) are four isomers having the molecular formula $C_9H_{18}Cl_3O_4P$ which belong to the class of chlorinated alkyl phosphate esters. The most abundant isomer in commercial products is the completely branched isomer, tris(1-chloro-2-propyl) phosphate and the least abundant form is the completely liner isomer, tris(2-chloropropyl) phosphate. Variations in manufacturing methods result in commercial formulations that contain different proportions of four isomers. Although tris(1-chloro-2-propyl) phosphate is the most abundant isomer, companies have tended to refer to their product by the name tris(2-chloropropyl) phosphate.

TMCPPs are not known to occur naturally but are manufactured from propylene oxide and phosphorous oxychloride (IPCS 1998). TMCPPs are used as a flame retardants in rigid and flexible polyurethane foams, although they may also be used for textile (non-apparel) finishes (NRC 2000). According to the International Programme on Chemical Safety, (IPCS 1998), the annual worldwide demand for TMCPPs exceeded 40,000 metric tonnes in 1997. TMCPP mixtures are sold under various trade names including Antiblaze 80, Amgard TMCP, Fyrol PCF, and Hostaflam PO 820.

Tris(1-chloro-2-propyl) phosphate is not readily biodegraded in sewage sludge inocula though it is rapidly metabolised by fish (IPCS 1998). These compounds are relatively stable and undergo only slow hydrolysis under weak alkaline or acid conditions. There is little or no further information on the degradation products of TMCPPs other than under combustion conditions, which leads to the production of hydrochloric acid and chlorinated hydrocarbons (Akzo Nobel 1999).

Traces of tris(1-chloro-2-propyl) phosphate have been detected in industrial and domestic effluents, but not in surface waters and sediments (IPCS 1998). Also three isomers of TMCPP have been detected in air samples from some common indoor work environment, i.e., an office building, a day care center, and a school building (Carlsson *et al.* 1997).

Tris(1-chloro-2-propyl) phosphate is of low to moderate acute toxicity by oral (LD_{50} in rats is 101-4200 mg/kg body weight), dermal (LD_{50} in rats and rabbits is > 5000 mg/kg body weight) and inhalation routes (LD_{50} in rats is > 4.6 mg/litre) (IPCS 1998). Rabbit eye and skin irritant studies have indicated that tris(1-chloro-2-propyl) phosphate is either non-irritant or mildly irritant.



The reproductive toxicity, immunotoxicity and carcinogenic potential of tris(1-chloro-2-propyl) phosphate have not been investigated. There are no data on the subchronic or chronic toxicity of TMCPPs for dermal, inhalation, or oral routes of exposure (NRC 2000). The National Research Council (NRC) recommends that further studies need to be carried out into the effects of the release of TMCPPs vapours into air and into saline from treated fabric (NRC 2000). In addition, no studies of the effects of this flame retardant on humans are available. However, acute toxicity values for other organisms in the environment are available, with LD_{50} values ranging from 3.6 to 180 mg/litre. Aquatic toxicity information shows that changes occur in algae, daphnids and fish at concentrations of 6, 32, and 9.8 mg/litre, respectively (IPCS 1998).

Despite the lack of information on the carcinogenic potential of the TMCPPs identified, studies on a very similar compound, tris(2-chloroethyl) phosphate, showed that is possesses carcinogenic properties when tested on mice and rats (IPCS 1998).

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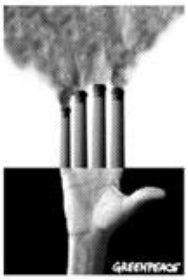
A2.4 Chlorinated benzenes

The production of chlorinated benzenes is a multiple product operation achieved by direct chlorination of benzene in the liquid phase using a ferric chloride 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.

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.



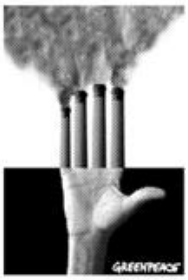
Both technological changes and environmental concerns have severely affected the production of chlorobenzenes; today only monochlorobenzene and 1,2- and 1,4-dichlorobenzenes are manufactured in large quantities. These are often produced together, with the economically optimised reaction yielding approximately 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).

Mono- and di-chlorobenzenes

Chlorobenzene, 1,2-dichlorobenzene and 1,3-dichlorobenzene are colourless liquids; 1,4-dichlorobenzene forms colourless crystals at room temperature (Ware 1988a & b).

One of the earliest uses of chlorobenzene 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). 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 is 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 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,



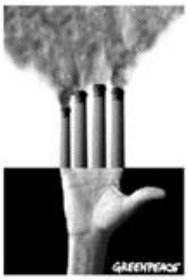
aquatic invertebrates and fish (Ware 1988b). All three have also been reportedly found in blood (Ware 1988b).

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-isocyantes, 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.

Exposed laboratory animals 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, which 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).

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). Mammalian (and human) metabolism 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 (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, fungicide and extreme pressure lubricant (Bryant 1993, CEC 1986). 1,4-dichlorobenzene is not spontaneously combustible and does not assist fire, but it is flammable nevertheless. It 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

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

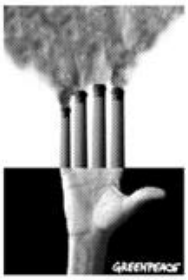
Environmental regulations have curbed the use and discharge of trichlorobenzenes to the environment, as least in Europe and the USA (Harper *et al.* 1992, Bryant 1993). Not surprisingly, therefore, little research appears to have been carried out in comparison with some other chlorobenzenes.

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. They are no longer used or produced in Canada and releases come only from dielectric fluid spills and long-range transport. 1,2,4,5-Tetrachlorobenzene used to be used in the production of 2,4,5-trichlorophenol on a large scale, but this use has now been largely discontinued. 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-tetrachlorobenzene being the most toxic. Not enough information was available to classify tetrachlorobenzenes as to carcinogenicity.

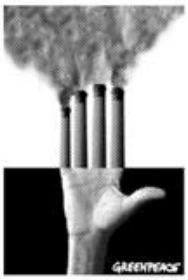
In addition to the effects noted above, 1,2,4,5-tetrachlorobenzene has also caused changes in the spleen, thymus, lymph nodes and haematological parameters in animals (Giddings *et al.* 1994b). An increase in chromosomal aberrations was seen in workers exposed to 1,2,4,5-tetrachlorphenol at a pesticide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

Giddings *et al.* (1994c) found that though no longer manufactured or used in Canada, pentachlorobenzene could still enter the environment through spillage of dielectric fluids or atmospheric transport. 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 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).



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.

Hexachlorobenzene

Hexachlorobenzene (HCB) is a manufactured chemical, which was used as a wood preservative, as a fungicide for treating seeds and as an intermediate in organic syntheses (Budavari *et al.* 1989). Additionally, hexachlorobenzene may be formed as an unwanted by-product in the synthesis of other organochlorine compounds high-temperature sources (Newhook & Meek 1994, Sala *et al.* 1999). The UNECE (1998) lists HCB alongside PCDD/Fs and PAHs as being the most important POPs emitted from stationary sources. HCB emissions from waste incineration, metallurgical industries and burning of chlorinated fuels are highlighted (UNECE 1998)(Annex V).

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 also 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 1997, Newhook & Meek 1994, van Birgelen 1998). Recent research (van Birgelen 1988) 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.

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).

Once introduced into environment, HCB strongly adsorbs 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 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 the proposed POPs Convention (UNEP 1995, 1997), which is expected to be signed in Stockholm in May 2001. 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 (EC 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 International Joint Commission on the Great Lakes (IJC) has called for all uses to be eliminated.

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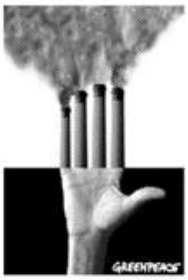

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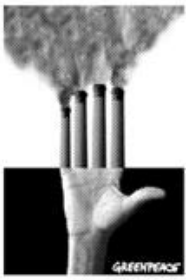

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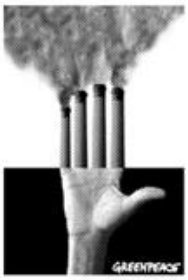
A2.5 DDT and metabolites

Technical DDT is made by condensing chloral hydrate with chlorobenzene in concentrated sulfuric acid. It was first synthesized in 1874, but only in 1939 Mueller and his coworkers discovered its insecticidal properties (ATSDR 1997). DDT is one of the most notorious environmental pollutants and has been banned or restricted in most western countries. Few DDT manufacturers are left. Hindustan Insecticides Ltd (India) currently manufactures DDT and is cited by several sources (Dinham 1993, FAO/UNEP 1991, RSC 1991). EniChem Synthesis S.p.A. (Italy) are listed by some sources (Dinham 1993, FAO/UNEP 1991), though production is believed to have ceased. Other manufacturers, for whom the current status is not certain are: P.T. Montrose Pesticido Nusantara (Indonesia) (Dinham 1993, FAO/UNEP 1991), and All-India Medical (RSC 1991). Unnamed producers are thought also to be operating in China, Mexico, Russia, South Korea and former Soviet Union States (WWF 1998).

DDT is an insecticide, which was first widely used during the Second World War to control disease-carrying insects. Such insects are known as vectors, and thus DDT is often described as being used for “vector control”. For a time it was also used in agriculture (see eg Carson 1962, Copper 1991), but because of its environmental impact this has been almost universally banned. Consequently, today it is again licensed almost exclusively for vector control.



However, it is thought that some of DDT manufactured for vector control is on fact illegally used in agriculture.

The term "DDT" refers to technical DDT, which is a mixture of several compounds and may not always have the same composition. The main component is p,p'-DDT, though it also contains a variable mix of other compounds. These are reported by different sources to include 15-20% of o,p'-DDT (ATSDR 1997, DHHS 1998), 4% p,p'-DDE (Smith 1991, DHHS 1998) and traces of other compounds (ATSDR 1997, DHHS 1998).

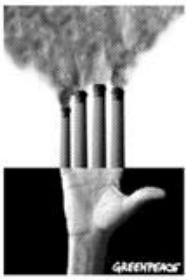
DDT is poorly absorbed through the skin, with powder forms being far less easily taken up than oil-based formulations. DDT is readily absorbed through the gastrointestinal tract, with increased absorption in the presence of fats (ASTDR 1997). Inhalation exposure of powders may also take place though the may in fact be trapped in the upper reaches of the respiratory tract and be ingested rather than through the lungs (ATSDR 1997, Smith 1991). In people who do not work with DDT, food is the greatest source of exposure.

DDT is bioaccumulative. The main ingredient, p,p'-DDT, is broken down in the environment or in the body to p,p'-DDE and smaller quantities of other chemicals. p,p'-DDE is more persistent both in the body and the environment than p,p-DDT (Smith 1991) and responsible for most of the observed toxic effects, unless there has been recent exposure to technical DDT.

DDT is moderately to slightly toxic to studied mammalian species via the oral route (RSC 1991, Meister 1992, ASTDR 1997). The primary target of DDT is the nervous system and high doses can cause trembling, increased susceptibility to cold and fear, with convulsions happening 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). It has caused chronic effects on the nervous system, liver, kidneys, and immune systems in experimental animals (ASTDR 1997, WHO 1979). There is evidence that DDT causes reproductive effects in test animals, including reduced fertility (ASTDR 1997).

Dose levels at which effects were observed in test animals are very much higher than those that may be typically encountered by humans (WHO 1979, Smith 1991). Human occupational and dietary exposure to DDT may differ both in dose and in chemical nature. Occupational exposure would be to technical DDT (predominantly p,p'-DDT) whereas dietary exposure, especially in those countries where DDT is no longer used, would be predominantly to p,p'-DDE, although there are several breakdown products to which individuals would also be exposed (Longnecker *et al*. 1997, ATSDR 1997).

Several of the DDT group are endocrine disruptors, exhibiting 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 highest concentrations in most humans, is an antiandrogen (Longnecker *et al*. 1997).



Acute effects likely in humans due to low to moderate exposure may include nausea, diarrhoea, increased liver enzyme activity, irritation (of the eyes, nose or throat), disturbed gait, malaise and excitability; at higher doses, tremors and convulsions are possible (ASTDR 1997).

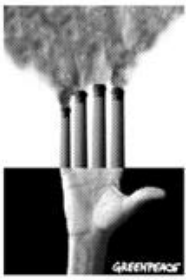
The IARC classified p,p'-DDT as possbly carcinogenic to humans (group 2B) and the US Department of Health and Human Services regards it as being "reasonably anticipated to be a human carcinogen" (DHHS 1998).

However, DDT's most severe impacts are on the environment. DDT, or rather, its metabolite, 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, Fry 1995, Copper 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).

DDT is controlled under numerous international legal instruments - notably the PIC Convention, the LRTAP POPs protocol, the Barcelona Convention, the Helsinki Convention, the IJC and the draft UNEP POPs Convention, which is due for signing in Stockholm in May 2001. It is also, of course, included under wider groupings of organochlorine pesticides or organohalogens under the various waste trade Conventions and the OSPAR Convention. Agricultural use of DDT is almost totally banned, but its use is frequently retained for public health purposes. According to FAO/UNEP (1991) DDT is banned in Chile, Cuba, the EC, Liechtenstein, Mexico, Panama, Republic of Korea, Singapore, Sri Lanka, Sweden, Togo and the USSR and has been withdrawn from sale in Canada and Poland. It is severely restricted in Argentina, Belize, China, Colombia, Dominica, Ecuador, Japan, Kenya, Mauritius, the USA,



Venezuela, and Yugoslavia. In many of these countries, use is only permitted for control of critical disease vectors and would be carried out only at the behest of the government health department. In addition, DDT is banned (except for drug use) in the countries which are party to the 1992 Helsinki Convention. Unfortunately, DDT is still diverted illegally from government health programmes to agricultural use on a regular basis. This is known or suspected to have happened in Bangladesh, Belize, Ecuador, India, Kenya, Madagascar, Mexico and Tanzania (WWF 1998).

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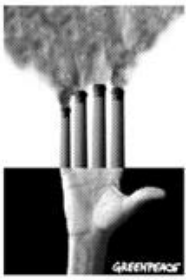


A2.6 Polycyclic aromatic hydrocarbons (PAHs)

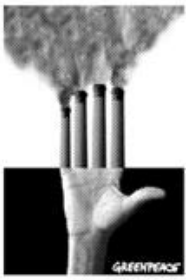
Polycyclic aromatic hydrocarbons occur in a variety of environmental products such as soot, coal, tar, tobacco smoke, petroleum, and cutting oil. They are commonly found as product of incomplete combustion. The commercial production of PAHs is not a significant source of these compounds in the environment. However, some of the PAHs - acenaphthene, acenaphthylene, and anthracene - are produced commercially (ATSDR 1997).

There is no known use for acenaphthylene, benz[a]anthracene, benzo[a]fluoranthene, benzo[e]pyrene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, benzo[a]pyrene, chrysene, dibenz[a,h]anthracene, indeno[1,2,3-c,d]pyrene, or pyrene except as research chemicals.

Anthracene is used as an intermediate in dye production, in the manufacture of synthetic fibers, and as a diluent for wood preservatives. It is also used in smoke screens, as scintillation counter crystals, and in organic semiconductor research. Anthracene is used to synthesize the chemotherapeutic agent, Amsacrine. Acenaphthene is used as a dye intermediate, in the manufacture of pharmaceuticals and plastics, and as an insecticide and fungicide. Fluorene is used as a chemical intermediate in many chemical processes, in the formation of polyradicals for resins, and in the manufacture of dyestuffs. Phenanthrene is used in the manufacture of dyestuffs and explosives and in biological research. Fluoranthene is used as a lining material to protect the interior of steel and ductile-iron drinking water pipes and storage tanks (ATSDR 1997).

The major products made from naphthalene are moth repellents, in the form of mothballs or crystals, and toilet deodorant blocks. It is also used for making dyes, resins, leather-tanning agents, and the insecticide, carbaryl (ATSDR 1997). The simplest alkyl derivatives of naphthalene, 1-methylnaphthalene and 2-methylnaphthalene are used to make other chemicals such as dyes, resins, and, for 2-methylnaphthalene, vitamin K. Along with naphthalene, they are present in cigarette smoke, wood smoke, tar, and asphalt, and at some hazardous waste sites (ATSDR 1997).

PAHs are found to cause harm to human health. Individuals exposed by breathing or skin contact for long period of time to mixtures of PAHs and other compounds can develop cancer (ATSDR 1997). Many of the carcinogenic polycyclic aromatic hydrocarbons are derived from an angular benz[a]anthracene skeleton. Anthracene itself is not carcinogenic, but benz[a]anthracene appears to have weak carcinogenicity. Addition of another benzene ring in select positions result in agents with powerful carcinogenicity such as dibenz[a,h]anthracene or benzo[a]pyrene. In addition, substitution of methyl groups on specific carbons of the ring also enhances carcinogenity. Thus, 7,12-dimethylbenz[a]anthracene (DMBA) is one of the most powerful synthetic, polycyclic aromatic hydrocarbon carcinogenes known (Williams 1986). Studies in laboratory animals have demonstrated the ability of benz[a]anthracene,



benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[a]pyrene, chrysene, dibenz[a,h,]anthracene, and indeno[1,2,3-c,d]pyrene to induce skin tumors (i.e., they are complete carcinogens) following intermediate dermal exposure. Anthracene, fluoranthene, fluorene, phenanthrene, and pyrene do not act as complete carcinogens (ATSDR 1997).

Pre- and post-natal exposure to PAHs could produce adverse reproductive and developmental effects in human fetuses. Most PAHs and their metabolites cross the placenta because of their lipid solubility (ATSDR 1997).

Exposure to a large amount of naphthalene may damage or destroy some of human red blood cells. People, particularly children, have developed this problem after eating naphthalene-containing mothballs or deodorant blocks. Anemia has also occurred in infants wearing diapers after storage in mothballs (ATSDR 1997).

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A2.7 Alkylbenzenes

Alkylbenzenes are single-ring aromatic compounds containing one or more aliphatic side chains. While there are theoretically thousands of alkylbenzenes, the major products of commence and, therefore, those to which humans are most likely to be exposed included toluene (methylbenzene), ethylbenzene, cumene (isopropylbenzene), and three xylenes (1,2-, 1,3-, and 1,4-dimethylbenzene).

The occurrence of these compounds in the environment is due to their presence in crude oil and petroleum products. Alkylbenzenes are also produced following the degradation of the linear alkylbenzene sulphonate (LAS) detergents. The alkylbenzenes are highly resistant to degradation and may accumulate in sediments (Preston & Raymundo 1993). Alkylbenzenes are useful sewage markers (Chalaux *et al*. 1995) and due to their stability in sediments, they are very useful in tracing the transport of contaminants from their point sources. Monoaromatic (benzene derivatives) and polyaromatic hydrocarbons (PAHs) are considered to be the most toxic, and are known to be present at the highest concentrations during the initial phase of a crude oil spill (Overton 1994).

The acute toxicity of inhaled alkylbenzenes is best described as central nervous system (CNS) depression (Andrews & Snyder, 1986). Acute toxicity does not vary very much within the group. In animal models, relatively similar concentrations of inhaled alkylbenzene vapours were found to be lethal. Impaired reaction times and impaired speech are the two most

commonly noted CNS effects (Klaassen *et al*. 1996). All alkylbenzenes mention above are irritating to the eyes and mucous membranes, can cause irritation and burning of the skin, and all are narcotics at high concentrations. Benzene itself is a known carcinogen. Chronic exposure can lead to bone marrow depression, which in a few cases, can progress to leukemia (Budavari *et al*. 1989).

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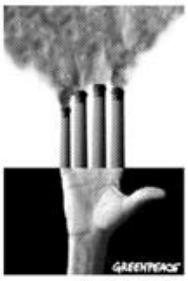


APPENDIX 3
TOXICOLOGICAL OUTLINES FOR HEAVY METALS

A3.1. Cadmium

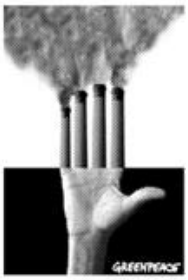
A3.1.1. Environmental Contamination and Behaviour

Cadmium is more mobile in aquatic environments than most other metals. It is also bioaccumulative and persistent in the environment ($t^{1/2}$ of 10-30 years) (USPHS 1997). It is found in surface and groundwater as either the +2 hydrated ion, or as an ionic complex 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 (USPHS 1997, WHO 1992). Furthermore, cadmium is readily available for uptake in grain, rice and vegetables, and there is a clear association between the cadmium concentration in soil and the plants grown on that soil (Elinder and Jarup 1996, Cabrera *et al.* 1994, WHO 1992).

When present in a bioavailable form, both aquatic and terrestrial organisms are known to bioaccumulate cadmium. Studies have shown accumulation in aquatic animals at concentrations hundreds to thousands of times higher than in the water (USPHS 1997). With reported bioconcentration factors ranging from 113 to 18,000 for invertebrates and from 3 to 2,213 for fish. Cadmium accumulation has also been reported in grasses and food crops, and in earthworms, poultry, cattle, horses, and wildlife (USPHS 1997, WHO 1992). Evidence for biomagnification is inconclusive. However, uptake of cadmium from soil by feed crops may result in high levels of cadmium in beef and poultry (especially in the liver and kidneys). This accumulation of cadmium in the food chain has important implications for human exposure, whether or not significant biomagnification occurs (USPHS 1997).

A3.1.2. Toxicity

Cadmium has no biochemical or nutritional function, and it is highly toxic to both plants and animals (USPHS 1997, WHO 1992, Alloway 1990). In humans and animals, there is strong evidence that the kidney is the main target organ of cadmium toxicity, following extended exposure (USPHS 1997, Elinder and Jarup 1996, Goyer 1996, Roels *et al.* 1993, Iwata *et al.* 1993, WHO 1992, Mueller *et al.* 1992). Renal damage includes tubular proteinuria (the excretion of low molecular weight proteins) and a decrease in the glomerular filtration rate. The latter results in a depressed re-sorption of enzymes, amino acids, glucose, calcium, copper, and inorganic phosphate. Furthermore, studies have shown that even when cadmium exposure ceases, proteinuria does not decrease, and renal tubular dysfunction and reduced glomerular



filtration increase in severity (USPHS 1997, Jarup *et al.* 1997, Elinder and Jarup 1996, Goyer 1996, Iwata *et al.* 1993, WHO 1992, Nriagu 1988).

Other toxic effects of cadmium, based on findings from occupation, animal, and epidemiological studies, can be summarised as follows:

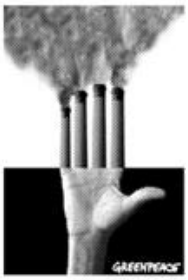
The inhalation of high levels of cadmium oxide fumes or dust is intensely irritating to respiratory tissue, and acute high-level exposures can be fatal. Typical non-fatal symptoms can include severe tracheobronchitis, pneumonitis, and pulmonary oedema, which can develop within hours of exposure (USPHS 1997, Goyer 1996, WHO 1992). At lower levels, lung inflammation have been known to cause emphysema (swelling of the lung air sacs resulting in breathlessness) and dyspnoea (difficult and laboured breathing) (USPHS 1997, Goyer 1996, WHO 1992). Animal studies have confirmed that inhalation exposure to cadmium leads to respiratory injury (USPHS 1997, WHO 1992).

There have been a number of epidemiological studies intended to determine a relationship between occupational (respiratory) exposure to cadmium and lung and prostatic cancer, and these along with animal studies have provided considerable support for the carcinogenic potential of cadmium (IARC 1998, Goyer 1996). Cadmium, and certain cadmium compounds, are therefore listed by the International Agency for Research on Cancer (IARC) as carcinogenic (IARC 1998). The US Department of Health and Human Services in its 8th Report on Carcinogens, lists cadmium and certain cadmium compounds as Reasonably Anticipated to be Human Carcinogens (USPHS 1998).

In addition to these toxic effects, it has also been suggested that cadmium may play a role in the development of hypertension (high blood pressure) and heart disease (USPHS 1997, Goyer 1996, Elinder and Jarup 1996). It is also known that severe oral exposure can result in severe irritation to the gastrointestinal epithelium, nausea, vomiting, salivation, abdominal pain, cramps and diarrhoea (USPHS 1997).

Regarding plant toxicity, adverse effects on plant growth and yield have been reported. Alloway (1990) reported stunted growth and toxic signs on leaves of lettuce, cabbage, carrot and radish plants, (which resulted from a cadmium content of around 20 mg/kg in the upper parts of the plants). Other studies have shown reductions in the rates of photosynthesis and transpiration (WHO 1992).

Regarding the toxicity of cadmium to aquatic organisms, numerous findings have been reported. For example, some species of phytoplankton are very sensitive to cadmium, with inhibition of growth observed at concentrations as low as 1 ug/l (Bryan & Langston 1992). Deleterious effects have also been reported in limpets, where correlations between increased levels of cadmium and reduced ability to utilise glucose were found. Reductions in reproduction rates and population numbers in copepods and isopods have been shown at concentrations as low as 5 ug/l, and exposure to similar levels has resulted in changes in the



immune function in some fish, and depressed growth in juvenile fish and invertebrates (Bryan & Langston 1992, Thuvander 1989). Furthermore, the toxicity of low sediment-cadmium concentrations has also been suggested following observations in San Francisco Bay. Here the condition of certain species of clam declined as cadmium concentrations rose from 0.1 to 0.4 mg/kg (Bryan & Langston 1992).

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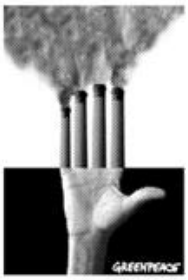
A3.2. Chromium

A3.2.1. Environmental Contamination and Behaviour

Although many different oxidation states of chromium exist in the environment, only the trivalent (III) and hexavalent (VI) forms are considered to be 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 intra-media transport, however chromium (VI) will eventually be converted to chromium (III), by reducing species such as organic



substances, hydrogen sulphide, sulphur, iron sulphide, ammonium and nitrite (USPHS 1997, Kimbrough *et al.* 1999). This trivalent form is generally not expected to migrate significantly in natural systems. Instead, it is rapidly precipitated and adsorbed onto suspended particles and bottom sediments. However, changes in the chemical and physical properties of an aquatic environment can result in changes to the chromium (III)-chromium (VI) equilibrium (Richard and Bourg 1991).

Chromium (III) and (VI) have been shown to accumulate in many aquatic species, especially in bottom-feeding fish, such as the brown bullhead (*Ictalujrus nebulosus*); and in bivalves, such as the oyster (*Crassostrea virginica*), the blue mussel (*Mytilus edulis*) and the soft shell clam (*Mya arenaria*) (Kimbrough *et al.* 1999).

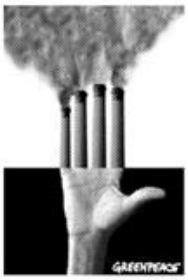
In soils, chromium (III) is relatively immobile due to its strong adsorption capacity onto soils. In contrast, chromium (VI) is highly unstable and mobile, since it is poorly adsorbed onto soils under natural conditions (Mukherjee 1998). Redox reactions (oxidation of chromium (III) to chromium (VI) and reduction of chromium (VI) to chromium (III)) are important processes affecting the speciation and hence the bioavailability and toxicity of chromium in soils. Oxidation can occur in the presence of oxides of manganese and iron, in fresh and moist (anaerobic) soils, and under slightly acidic conditions. Reduction can occur in the presence of sulphide and iron (II) (anaerobic conditions), and is accelerated by the presence of organic matter in the soil (Mukherjee 1998).

The importance of this lies in the fact that 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 (Mukherjee 1998, Outridge and Sheuhammer 1993, UNEP 1991, Richard and Bourg 1991).

A3.2.2. Toxicity and Essentiality

Chromium (III) is considered an essential trace nutrient, required for glucose, protein and fat metabolism in mammals. Signs of deficiency in humans include weight loss and the impairment of the body to remove glucose from the blood (USPHS 1997, Goyer 1996). The minimum human daily requirement of chromium (III) for optimal health is not known, but a daily ingestion of 50-200 ug/day has been estimated to be safe and adequate. However, although an essential food nutrient, very large doses may be harmful (USPHS 1997).

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 of exposed skin, perforations of respiratory surfaces and irritation of the gastrointestinal tract. Damage to the kidney and liver have also been reported



(USPHS 1997). In addition, the International Agency for Research on Cancer (IARC) classifies chromium (VI) compounds as known carcinogens (1998). Long-term occupational exposure to airborne levels of chromium higher than those in the natural environment has been associated with lung cancer. Individuals at most risk include those in chromate-production industries and chromium pigment manufacture and use; and similar risks may exist amongst chromium-alloy workers, stainless steel welders, and chrome-platers (Kimbrough 1999, USPHS 1998).

The aquatic toxicology of chromium is also dependant upon speciation, with chromium (III) far less biologically available and toxic than chromium (VI). This has been observed in barnacles, Balanus sp., and in the polychaete Neanthes arenaceodentata. Experiments have shown that the number of offspring produced by the Neanthes arenaceodentata was reduced by exposure to 39 ug/l of dissolved chromium (VI) (Bryan & Langston 1992).

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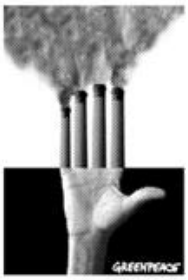
A3.3. Copper

A3.3.1. Environmental Contamination and Behaviour

Copper may exist in natural waters either in the dissolved form as the cupric (+2) ion or complexed with inorganic anions or organic ligands (e.g. carbonates, chlorides, humic and fulvic acids). It may also be present as an insoluble precipitate (e.g. a hydroxide, phosphate, or sulphide) or adsorbed onto 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, alkalinity, and the presence of organic ligands, inorganic anions and other metal ions. However, studies have frequently shown that the free +2 ion concentration is low, compared to the levels of copper associated with suspended and bottom sediments (USPHS 1997, Mance *et al*. 1984).

In soils, copper has a high affinity for sorption by organic and inorganic ligands (e.g. humic and fulvic acids, hydroxides of iron, aluminium and manganese). However, it can also exist as soluble ions and complexes. Copper in a soluble form 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, copper sulphate, or chloride, present in MSW incinerator ash or mine tailings, is far more bioavailable and migratory than the organically bound copper found in sewage sludge (USPHS 1997, Alloway 1990, Mance *et al*. 1984).

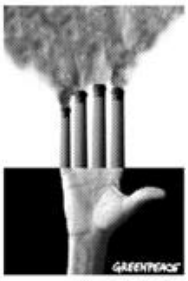
Copper is one of the most important, essential elements for plants and animals. However if plants and animals are exposed to elevated concentrations of bioavailable copper, bioaccumulation can result, with possible toxic effects (USPHS 1997).

A3.3.2. Toxicity and Essentiality

Copper is an essential nutrient that is incorporated into numerous plant and animal enzyme systems, e.g. in humans, those involved in haemoglobin formation, carbohydrate metabolism, melanin formation, and cross-linking of collagen, elastin and hair keratin (USPHS 1997). Human deficiency is characterised by anaemia, resulting from defective haemoglobin synthesis (Goyer 1996). However, at the other extreme, vomiting, hypotension, jaundice, coma and even death, can result from acute poisoning (USPHS 1997).

Therefore, even though copper is essential for good health, a very large single dose, or long term elevated exposure can be harmful. Inhalation of dust and vapours can irritate the nose, mouth and eyes, and cause headaches, dizziness, nausea and diarrhoea. Oral exposure to high levels can cause vomiting, diarrhoea, stomach cramps and nausea (USPHS 1997). Copper homeostasis plays an important role in the prevention of copper toxicity, in humans, terrestrial animals, and aquatic organisms. Copper is readily absorbed from the stomach and small intestine; and after requirements are met, there are several mechanisms that prevent copper overload e.g. bile excretion, increased storage in the liver or bone marrow (USPHS 1997). However, failure of this homeostatic mechanism can occur in humans and animals 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. This can result in liver and kidney damage and haemolytic anaemia (USPHS 1997).

In addition to these effects, developmental and reproductive damage, following exposure to high levels of copper, has been seen in animals. However, no such effects have been reported in humans (USPHS 1997).



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 ug/l (Bryan & Langston 1992). For example, studies have shown that at levels of 2 ug/l, the survival rate of young bay scallops was significantly affected; and in the embryos of oysters and mussels concentrations of 5 ug/l were seen to induce abnormalities. A similar concentration resulted in increased mortalities 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 and DePledge 1998, UNEP 1993), and embryonic sensitivity in fish exposed to levels of 25 ug/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. In the UK, such concentrations are exceeded in a number of estuaries, including the Fal and the Tamar. Here, many species of bivalves, including some mussels, clams and cockles are absent, and at best distribution is severely limited. The toxicity of the surface sediment containing over 2000 mg/kg of copper, towards juvenile bivalves appears to be the reason (Bryan & Langston 1992).

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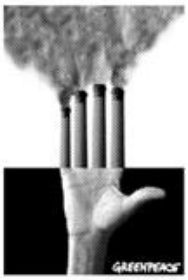
A3.4. Lead

A3.4.1. Environmental Contamination and Behaviour

When lead is released into the environment it has a long residence time compared with most pollutants. As a result, it tends to accumulate in soils and sediments. Where, due to low solubility, it can remain accessible to the food chain and to human metabolism far into the



future (Sauve *et al.* 1997, USPHS 1997, Alloway 1990). However, as with all metals, speciation is critical when assessing bioavailability and the potential threat to the environment.

Two oxidation states of lead, +2 and +4, are stable, but the environmental chemistry is dominated by the Pb^{+2} ion, its compounds, and complexes. In general the free +2 ion is more toxic than inorganic complexes, and therefore any factor which increases complexation and decreases the concentration of the free ion is bound to affect lead toxicity adversely. Toxic 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).

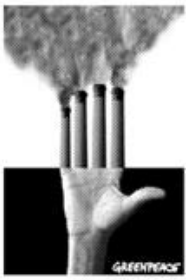
As mentioned, lead has a tendency to form compounds with anions having low solubility, such as hydroxides, carbonates, and phosphates. Thus the amount of lead remaining in solution in surface waters (also dependent upon pH and salinity) is often low. In addition to this, a significant fraction of insoluble lead may be incorporated in surface particulate matter from runoff, or as sorbed ions or surface coatings on sediment, or may be carried as a part of suspended living or nonliving organic matter (USPHS 1997).

In soils and sediments, the fate of lead is affected by similar processes, which often lead to the formation of relatively stable organic-metal complexes. Most of the lead is retained strongly, and very little is transported into surface water or groundwater. However, re-entry to surface waters as a result of erosion of lead-containing soil particulates; or through the conversion to the relatively soluble lead sulphate at the soil / sediment surface, can occur (USPHS 1997, Sadiq 1992, Alloway 1990). As can the downward movement of lead from soil to groundwater by leaching (USPHS 1997).

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

A3.4.2. Toxicity

Lead is one of the most ubiquitous toxic metals. It has no known, nutrition, biochemical or physiological function, and because there is no demonstrated biological need, and because it is toxic to most living things, the major concern of the moment is at what dose does lead become toxic (Goyer 1996). The toxic effects of lead are the same, irrespective of whether it is ingested or inhaled, and blood levels as low as <10-100 ug/dl in children, and 10-100 ug/dl in adults have been associated with a wide range of adverse effects. These 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 exposure on cognitive and behavioural development in children (Pirkle *et al.* 1998, USPHS 1997, Bernard *et al.* 1995, Goyer 1993, Nriagu 1988).



In 1975 the Centre for Disease control (CDC) in Atlanta recommended that the maximum permissible level of blood-lead be 30 ug/dl (for both adults and children). This levels was revised downward in 1985 to 25 ug/dl, and again in 1991, defining a blood-lead of 10 ug/l as an action or intervention level (USPHS 1997). Perhaps even more importantly is the now suggested recommendation that there may be no level of blood-lead that does not produce a toxic effect, particularly in the developing central nervous system (USPHS 1997, Goyer 1993).

Animals studies have reproduced many of the toxic effects listed above, and animals feeding close to smelting, mining and recycling facilities, have often ingested levels of lead that have resulted in poisoning and death (Henny *et al.* 1991, Blus *et al.* 1991, USPHS 1997, WHO 1989, Collivignarelli *et al.* 1986).

Lead is also toxic to all aquatic biota, and even though it is not considered one of the most environmentally mobile of metals, there is still appreciable evidence showing the bioavailability of sediment-bound lead to deposit feeding species (Bryan & Langston 1992). In addition, lead can be accumulated directly from sea and fresh waters, especially in organisms that utilise gill tissue as the major nutrient uptake route (Sadiq 1992). Toxicological studies have reported sub-lethal effects in fish including changes in morphology, metabolism and enzymatic activity. Avoidance behaviour has also been observed in adult fish exposed to levels ranging from 10-100 mg/l (WHO 1989). Studies involving invertebrates (oysters, sea urchins, snails, copepods and water fleas) often report a reduction in growth, fertility and reproduction suppression, and mortality, at ug/l (parts per billion) concentrations (WHO 1989).

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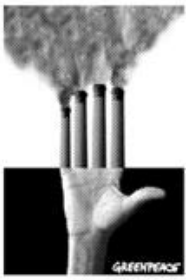

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A3.5. Manganese

Manganese is an essential trace metal, although human and animal exposure to high levels can cause serious illness. Workers chronically exposed to high levels of manganese in the air have suffered both mental and emotional disturbances, along with increased slowness and clumsiness of body movements. This combination of symptoms is a disease called manganism. The symptoms can be reduced by medical treatment, but due to the high levels of manganese accumulated in the brain, any brain injury is often permanent (ATSDR 1997). It is not certain whether eating or drinking elevated levels of manganese can cause manganism or not. Low-level manganese exposure in workers at two steel smelters has been linked with early signs of neurological impairment (Wennberg 1991). In one report, humans exposed to contaminated drinking water, developed symptoms similar to those seen in manganese miners or steel workers, but it is not certain if the effects were caused by the manganese alone. Another report found that people who drank water with above average levels of manganese seemed to have a slightly higher frequency of symptoms such as weakness, stiff muscles, and trembling of the hands. However, these symptoms are not specific for manganese, and might have been caused by other factors (ATSDR 1997).

Studies in animals have shown that very high levels of manganese in food or water can cause changes in the brain, suggesting that high levels might cause brain injury. In addition, animal studies have indicated that manganese may also be a reproductive toxicant, especially to males, injuring the testes and causing impotence.

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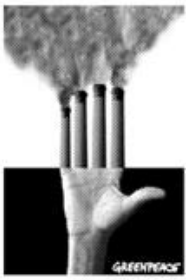


A3.6. Mercury

A3.6.1. Environmental Contamination and Behaviour

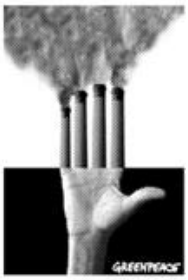
Due to the fact that mercury is the only metal that can exist as both a liquid and a vapour at ambient temperatures, its environmental behaviour differs from that of most other toxic elements (USPHS 1997, WHO 1989). Mercury can exist in three valence states, Hg (0), Hg (I) and Hg (II). In the atmosphere, elemental mercury is by far the most common form, and as a vapour it is responsible for the long-range, global cycling of mercury. In addition, to a far lesser degree, mercury may be associated with particulates, which are removed by dry or wet deposition. Atmospheric inputs may be more significant in areas where other sources, such as contaminated rivers, are less important or non-existent (USPHS 1997, WHO 1993).

In the aquatic environment, mercury is most commonly found in the mercuric (II) state, and its fate, once released, is dominated by rapid adsorption to soluble and particulate organic material; 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) (USPHS 1997, Bryan & Langston 1992). Dredging or re-suspension of bed materials may cause short-term release of mercury, although levels of dissolved metal quickly return to pre-disturbance values. 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 methylated 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. The selective retention of MeHg at each step in the food chain, relative to inorganic mercury, is related to its high lipid solubility, its long biological half-life, and the increased longevity of top predators (Bryan & Langston 1992). As a result, MeHg provides one of the rare examples of metal biomagnification in food chains (USPHS 1997, WHO 1989). For example, concentrations in carnivorous fish at the top of freshwater and salt water food chains (e.g., pike, tuna, and swordfish) are biomagnified 10,000-100,000 times the concentrations found in ambient waters (USPHS 1997). The significance of this bioaccumulation is that it is generally the most important source of human, non-occupational mercury exposure (USPHS 1997, WHO 1989).

A3.6.2. Toxicity

Mercury is an extremely toxic, non-essential trace metal, having no biochemical or nutritional function. Biological mechanisms for its removal are poor, and, as mentioned above, mercury



is the only metal known to biomagnify i.e. progressively accumulate though the food chain (WHO 1989).

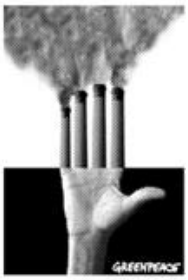
Acute exposure to high levels of mercury salts, or chronic low-dose exposure, is directly toxic to the kidney (Zalups and Lash 1994). In addition, nausea and diarrhoea may result after swallowing large amounts of inorganic mercury salts, and some nervous system effects have also been recorded (USPHS 1997, WHO 1989).

Exposure to MeHg has resulted in permanent damage to the CNS, kidneys, and the developing foetus. The levels of MeHg that result in these effects are not usually encountered by the general population, however they were encountered by the population of Minamata, in Japan, who were exposed to high levels of MeHg from eating contaminated fish and seafood collected from the Bay (USPHS 1997). Symptoms such as brain damage, numbness of extremities, and paralysis, along with the loss of hearing, speech and sight were reported (D'Itri 1991). However even today, the full range of neurological symptoms caused by the ingestion of MeHg in fish and shellfish has not been fully characterised, and the total number of Minamata Disease sufferers has not been determined (D'Itri 1991). The problem of methylation of past and present inorganic mercury discharges continues, and the long retention time of mercury by sediments delays the elimination of contamination for many years (Harada 1997, Barbosa 1997, Akagi *et al.* 1995, Bryan & Langston 1992, D'Itri 1991).

Studies on the aquatic toxicity of mercury are numerous, and again show that MeHg is more toxic than any of the inorganic forms. Invertebrate studies have reported significant reductions in the growth rate of the mussel *Mytilis edulis* at concentrations of 0.3 ug/l, with growth almost ceasing at 1.6 ug/l, and acute lethal effects observed at 25 ug/l (WHO 1989). In addition, changes in filtering activity, oxygen consumption, blood osmotic pressure, ciliary and valve activity have also been reported (Naimo 1995). In the American oyster *Crassostrea virginica* embryonic abnormalities were evident at concentrations of 5-10 ug/l. With survival rates of exposed clams and barnacles, copepods, shrimps and crustaceans all greatly affected by increased levels of mercury (Bryan & Langston 1992).

Inorganic mercury is toxic to fish at low concentrations. The 96-h LC_{50}s vary between 33-400 ug/l for freshwater fish and are higher for salt-water fish; with organic compounds being more toxic to both (Bryan & Langston 1992, WHO 1989). Studies have reported a wide range of adverse reproductive effects in fish exposed to increased levels including prevention of ocyte development in the ovary and spermatogenesis in the testis of freshwater fish. Reductions in embryo survival and hatching success of *Fundulus heteroclitus* has also been reported, along with reductions in growth and an increase in deformities in trout (WHO 1989). Lack of movement and reduced food consumption, blindness and reduced respiratory rate have also been found in rainbow trout, bass and roach exposed to high levels of mercury (WHO 1989).

High incidences of abnormalities have also been observed in seabirds, abnormalities that seem to correlate with mercury residues in tissues. Even at sites apparently remote from



contamination, elevated mercury concentrations have been determined in the liver and kidneys of fish eating seabirds, e.g. *Fulmarus glacialis*. Levels comparable with those suspected of producing sub-lethal effects, notably pathological changes to the kidney; and which have been shown to cause death in other species (Bryan & Langston 1992).

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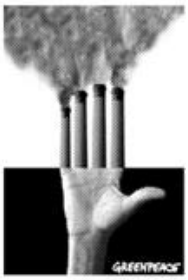
A3.7. Zinc

A3.7.1. Environmental Contamination and Behaviour

Zinc occurs in the environment primarily in the +2 oxidation state, either as the free (hydrated) zinc ion, or as dissolved and insoluble complexes and compounds (USPHS 1997). In soils, it often remains strongly sorbed, and in the aquatic environment it will predominantly bind to suspended material before finally accumulating in the sediment (USPHS 1997, Bryan & Langston 1992, Alloway 1990). However, re-solubilisation back into an aqueous, more bioavailable phase is possible under certain physical-chemical conditions, e.g. in the presence of soluble anions, the absence of organic matter, clay minerals and hydrous oxides of iron and manganese, low pH and increased salinity (USPHS 1997). Zinc in a soluble form (e.g. sulphate or chloride, present in incinerator ash, or mine tailings) 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) (USPHS 1997).

Zinc is an essential element, present in the tissues of animals and plants even at normal, ambient concentrations. However, if plants and animals are exposed to high concentrations of



bioavailable zinc, significant bioaccumulation can results, with possible toxic effects (USPHS 1997).

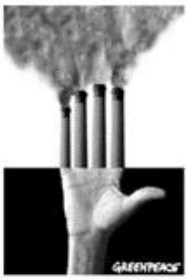
A3.7.2. Toxicity and Essentiality

Zinc is a nutritionally essential metal, having enzymatic, structural and regulatory roles in many biological systems (Goyer 1996, Aggett and Comerford 1995). Deficiency in humans can result in severe health consequences including growth retardation, anorexia, dermatitis, depression and neuropsychiatric symptoms (Aggett and Comerford 1995). At the other extreme, excessive dietary exposure, in both humans and animals, can cause gastrointestinal distress and diarrhoea, pancreatic damage and anaemia (USPHS 1997, Goyer 1996).

Due to the essentiality of zinc, dietary allowances of 15 mg/day for men, and 12 mg/day for women are recommended (USPHS 1997). However, eating food containing very large amounts of zinc can induce the symptoms listed above. 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; and rats that ate very large amounts of zinc became infertile (USPHS 1997). Humans taking supplements at higher than recommended doses (400-500 mg/day) suffered severe gastro-enteritis (Abernathy and 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).

Aquatic studies have shown that whilst zinc is not considered as being especially toxic to organisms, it is sometimes released into the aquatic environment in appreciable quantities. And in appreciable quantities, zinc can have a direct disrupting effect on the external cell membranes or cell walls of organisms, resulting in rapid mortality (UNEP 1993). However, many studies now report that zinc is not only harmful at high concentrations, but also at lower sub-lethal concentrations, especially after prolonged exposure. For example, studies have shown that at concentrations as low as 15 ug/l, carbon fixation rates in natural phytoplankton populations were depressed. Others observed that the growth of cultured diatoms was inhibited at 20 ug/l (Bryan & Langston 1992). Effects on fertilisation and embryonic development in Baltic spring-spawning herring at low salinity were detected at only 5 ug/l (UNEP 1993); and the fertility of successive generations of harpacticoid copepod *Tisbe holothuria* was reduced by continuous exposure to only 10 ug/l (Verriopoulos and Hardouvelis 1988).

At slightly higher concentrations, studies investigating the effects of zinc on the hatching of brine shrimp (*Artemia salina*), noted that although increased concentrations of zinc did not affect development before emergence, the hatching stage of development was highly sensitive to, and heavily disrupted by, zinc (Bagshaw *et al* 1986). In addition, the inhibition of larval development was observed in the echinoderm (e.g. sea urchins and starfish) *Paracentrotus lividus* at a zinc concentration of only 30 ug/l (UNEP 1993). Shell growth in the mussel *Mytillus edulis* was effected at a concentration of 200 ug/l. With oxygen uptake, feeding and



filtration rates were reduced at concentrations ranging between 750-2000 ug/l. Harmful effects on mollusc larva were seen to occur at levels as low as 40 ug/l (UNEP 1993).

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). It was the observed phytotoxicity of zinc in sewage-sludge amended soils, that led several countries to formulate guidelines for sludge usage (Alloway 1990).

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