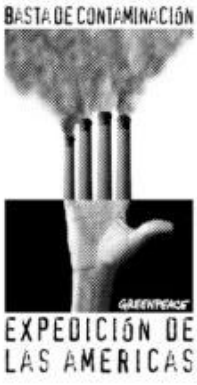


INFORME

Organochlorine and heavy metal contaminants in the environment around the Complejo Petroquimicos Paharitos, Coatzacoalcos, Mexico

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

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

March 2001

Technical Note: 05/2000

GREENPEACE

EXECUTIVE SUMMARY

The Complejo Petroquimico Pajaritos is located close to the town of Coatzacoalcos in Vera Cruz State, Mexico. The companies located in and around the complex process petroleum hydrocarbons and manufacture chemicals such as chlorine, 1,2-dichloroethane (EDC), vinyl chloride (VCM), sodium hydroxide, ethylene and phosphoric and sulphuric acids.

A sampling survey was carried out during September 2000 and March 2001. Water and sediment samples were collected from the discharge pipes of the facilty and the rivers to which they were discharged, the Arroyo Nuevo Teapa and the Rio Coatzacoalcos. They were analysed at the Greenpeace Research Laboratories at Exeter University in the UK. Deteminations carried out were: quantitative analyses of heavy metals and screening to identify organic contaminants on all samples and quantitative analysis of volatile organochlorines in all water samples.

These analyses revealed some degree of contamination in all samples, though the control sample collected from the Arroyo Nuevo Teapa only contained a small number of hydrocarbons. Hydrocarbon pollution was ubiquitous, as was pollution with 1,4-dichlorobenzene which is used as a toilet deodorant and is therefore most commonly asociated with sewage discharges.

Mercury is a far less common pollutant, but this was found in two of the effluents and the evidence from the sediments suggests that it has been or is being emitted from two other points. Mercury is an extremely hazardous heavy metal which is highly toxic and can accumulate in the food chain. Of the potential sources of mercury in the environment, use as a catalyst and use in mercury cells which are employed to make chlorine gas are the most likely. The presence of chloroform, which can be formed as a byproduct of chlorination of water, suggests that it might be the latter source, though some uncertainty remains.

Two of the effluents contained significant quantities of organochlorines and the analysis of the sediment samples indicates there may be a third discharge. The mixture of volatile organochlorines (especially EDC and VCM) and semivolatile organochlorines is typical for EDC/VCM manufacturing. The presence of copper, which is used as a catalyst in this process, also supports this conclusion. Concentrations of the volatile organochlorines in these effluents exceeded several of the limits set in US Federal regulations on the plastics industry. In the worst case, the effluent entering the Rio Coalzacoalcos contained over 100 times the US limit. Given that the production of EDC inevitably generates dioxin as an unwanted byproduct, it is highly likely that the environment around the Complex has also been contaminated with dioxins.

Other pollutants found in excessive concentrations around the Complex included zinc, chromium, lead, nickel and manganese. The lead is probably present from manufacturing of the petrol additive tetraethyl lead at the complex (now believed to have ceased), with a contribution from emissions from passing traffic. The other metals have so many uses that without more information it is not possible to draw any conclusions about their sources.

1 INTRODUCTION

The Complejo Petroquimico Pajaritos is located on the eastern bank of the Rio Coatzacoalcos. It contains of a number of different factories operating under different owners and manufacturing different products. There is some degree of integration, with some companies buying their raw products from their neighbours. There are also close inks with two other nearby petrochemical complexes, one at La Cangrejera to the east and Morelos to the north. Other raw materials can be shipped in via the Terminal Maritima at the Laguna de Pajaritos; products can also be shipped out from this port.

Some of the products believed to be manufactured at the complex include: chlorine, sodium hydroxide, salt, ethylene, ethylene oxide, hydrochloric acid, propane, refrigerants, 1,2-dichlorethane (EDC), vinyl chloride (VCM), sulphuric acid, phosphoric acid, tetraethyl lead (though production of this chemical is believed to have ceased), sodium tripolyphosphates, toluene diisocyanate.

At this time, no official information about the pollution monitoring from the complex is available. However, a number of pipes were observable pouring wastewaters into the Arroyo Nuevo Teapa, to the south of the complex, and the Rio Coatzacoalcos, to its west. To provide some basic information on the pollution status of the environment around the complex, a series of water and effluent and sediment samples were collected in September 2000 and March 2001.

2 SAMPLING PROGRAM

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 metal and organic residues. Sediment samples were collected in 100ml bottles. Water samples were collected in 1-litre or 125ml bottles. All samples were kept cool and returned to the Greenpeace Research Laboratories for analysis. The 125ml samples were analysed for volatile organochlorines; the one-litre samples were also screened to identify as many organic contaminants as possible and a quantitative determination of a range of heavy metals was carried out. Sediment samples were analysed for heavy metals and screened for organic contaminants, but volatile organochlorines were not determined. Detailed description of sample preparation and analytical procedures are presented in Appendix 1.

Sample Number	Sample Type	Sample Information
AM0157	sediment	Arroyo Nuevo Teapa, upstream of all identifiable discharges from the Pajaritos complex
AM0159	effluent	Square concrete sump between the complex and the Arroyo Nuevo Teapa.
AM0155	effluent	Right-hand of two pipes discharging to the Arroyo Nuevo Teapa.
AM0156	effluent	Left-hand of two pipes discharging to the Arroyo Nuevo Teapa.
AM0161	sediment	Arroyo Nuevo Teapa, immediately downstream from samples AM0155 & AM0156
AM0160	sediment	Adjacent to suspected sewage outfall
AM0153	effluent	From single pipe discharging to the Arroyo Nuevo Teapa.
AM0154	sediment	Arroyo Nuevo Teapa, adjacent to AM0153
AM0151	effluent	right-hand of two large pipes discharging to the Arroyo Nuevo Teapa
AM0150	effluent	Left-hand of two large pipes discharging to the Arroyo Nuevo Teapa
AM0152	sediment	Arroyo Nuevo Teapa, by road bridge immediately downstream of AM0151 & AM0152
AM0162	sediment	Wetland lagoon near incinerators
AM0158	sediment	Arroyo Nuevo Teapa downstream from known discharges
AM0149	sediment	Arroyo Nuevo Teapa just before it joins the Rio Coatzacoalcos
AM01001	effluent	Pipe discharging into the Rio Coatzacoalcos
AM01002	sediment	Below the pipe where AM01001 was collected
AM01003	sediment	6m from the pipe where AM01001 was collected

Table 1. Description of samples collected from the vicinity of the Complejo Pajaritos. Samples are arranged according to location, starting with those furthest up the Arroyo Nuevo Teapa and finishing with those from the Rio Coatzacoalcos.

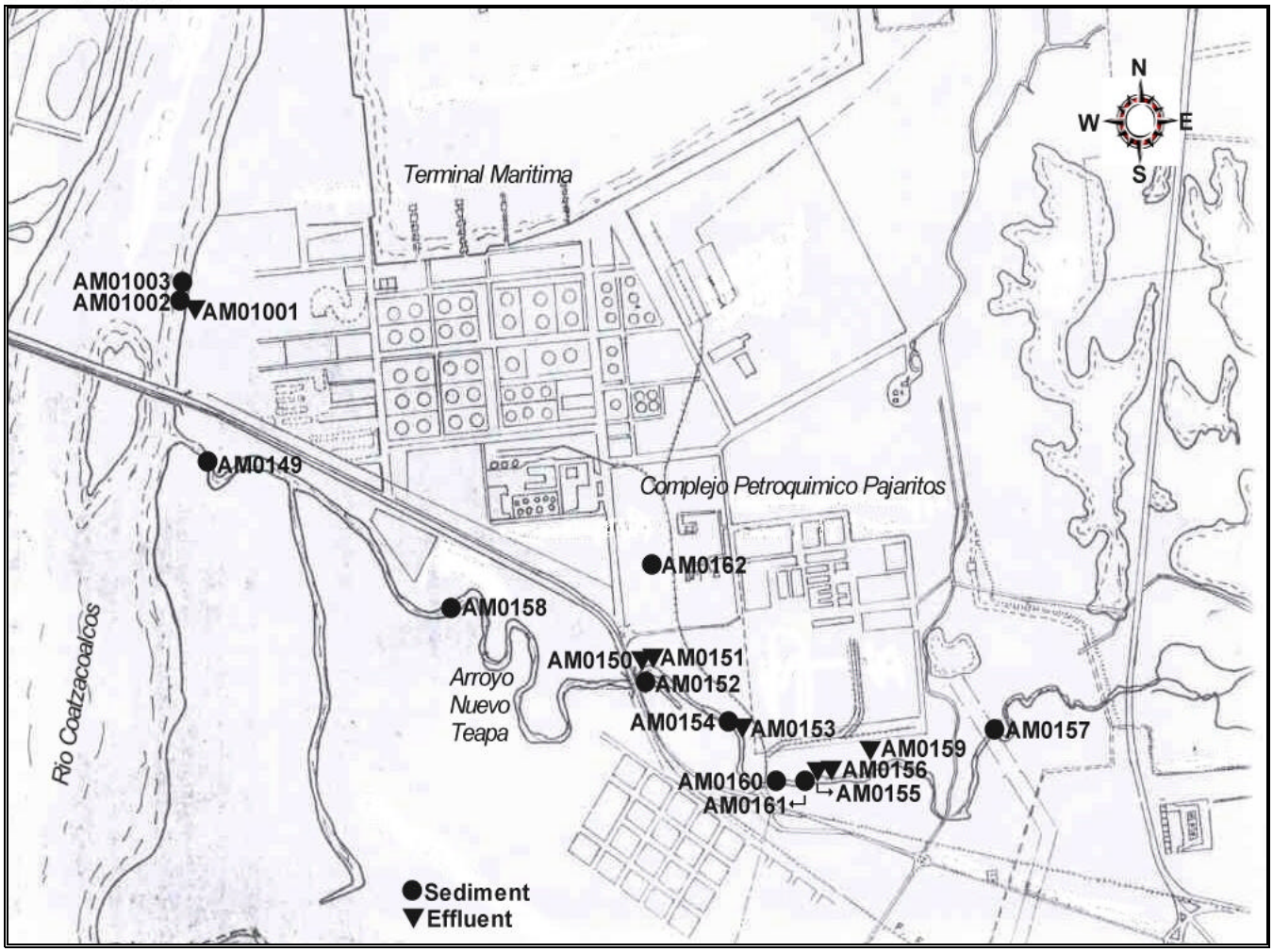


Figure 1. Map of sampling locations.

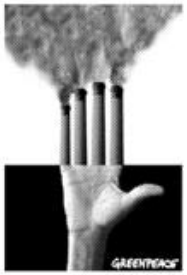
3 RESULTS AND DISCUSSION

The results of the organic screen analysis and heavy metals analysis of the are presented in Tables 2 and 3 including a breakdown of the groups of organic compounds reliably identified in the samples.

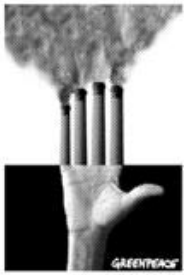
Analytical procedures used are described in Appendix 1. For more information on the common sources, environmental behavior and toxicological outlines for key pollutants detected during this study see Appendices 2 and 3.

Sample Number	AM 0157	AM 0159	AM 0155	AM 0156	AM 0161	AM 0160	AM 0153	AM 0154	AM 0151	AM 0150	AM 0152	AM 0162	AM 0158	AM 0149	AM 01001	AM 01002	AM 01003
Sample Type	sedi-ment	effl-uent	effl-uent	effl-uent	sedi-ment	sedi-ment	effl-uent	sedi-ment	effl-uent	effl-uent	sedi-ment	sedi-ment	sedi-ment	sedi-ment	effl-uent	sedi-ment	sedi-ment
METALS																	
Concentration	mg/kg	ug/l	ug/l	ug/l	mg/kg	mg/kg	ug/l	mg/kg	ug/l	ug/l	mg/kg	mg/kg	mg/kg	mg/kg	ug/l	mg/kg	mg/kg
Cadmium	<1	<10	<10	<10	<1	1	<10	<1	<10	<10	<1	<1	<1	<1	<10	<1	<1
Chromium	25	<20	<20	<20	184	75	<20	56	<20	<20	43	46	50	217	<20	14	17
Cobalt	6	<20	<20	<20	9	6	<20	8	<20	<20	6	5	8	13	<20	2	3
Copper	19	<20	<20	50	110	131	<20	43	<20	110	42	59	57	113	4919	53	75
Lead	11	<30	<30	<30	97	213	<30	18	47	<30	78	68	34	101	<30	33	9
Manganese	93	146	291	241	162	288	339	356	714	936	279	106	250	304	<10	79	120
Mercury	0.10	<1	196	9	8.35	60.83	<1	5.90	<1	<1	4.74	0.44	2.15	19.75	<1	0.20	0.18
Nickel	17	<20	<20	<20	30	44	<20	32	<20	<20	57	19	28	58	154	8	9
Zinc	97	41	205	1494	332	1743	95	477	33	5048	602	41	243	309	840	58	47
VOLATILE ORGANIC CHEMICALS																	
Concentration	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l	ug/l
Chloroform	N/A	<10	110	230	N/A	N/A	<10	N/A	<10	130	N/A	N/A	N/A	N/A	2700	N/A	N/A
Carbon tetrachloride	N/A	<5	<5	<5	N/A	N/A	<5	N/A	<5	40	N/A	N/A	N/A	N/A	180	N/A	N/A
Vinyl chloride	N/A	<10	<10	<10	N/A	N/A	<10	N/A	<10	40	N/A	N/A	N/A	N/A	390	N/A	N/A
1,1-Dichloroethene	N/A	<5	<5	<5	N/A	N/A	<5	N/A	<5	30	N/A	N/A	N/A	N/A	150	N/A	N/A
cis-1,2-Dichloroethene	N/A	<5	<5	<5	N/A	N/A	<5	N/A	<5	<5	N/A	N/A	N/A	N/A	255	N/A	N/A
trans-1,2-Dichloroethene	N/A	<5	<5	<5	N/A	N/A	<5	N/A	<5	<5	N/A	N/A	N/A	N/A	35	N/A	N/A
Trichloroethene	N/A	<5	<5	<5	N/A	N/A	<5	N/A	<5	<5	N/A	N/A	N/A	N/A	55	N/A	N/A
Tetrachloroethene	N/A	<5	<5	<5	N/A	N/A	<5	N/A	<5	60	N/A	N/A	N/A	N/A	110	N/A	N/A
1,1-Dichloroethane	N/A	<5	<5	<5	N/A	N/A	<5	N/A	<5	<5	N/A	N/A	N/A	N/A	900	N/A	N/A
1,2-Dichloroethane	N/A	<10	<10	<10	N/A	N/A	<10	N/A	170	7500	N/A	N/A	N/A	N/A	58500	N/A	N/A

Table 2. Quantitative analyses of samples from the Complejo Petroquimico Pajaritos. Concentrations are given in mg/kg dry weight for sediment samples and ug/l for water samples. Sediment samples are highlighted.



Sample Number	AM 0157	AM 0159	AM 0155	AM 0156	AM 0161	AM 0160	AM 0153	AM 0154	AM 0151	AM 0150	AM 0152	AM 0162	AM 0158	AM 0149	AM 01001	AM 01002	AM 01003
Sample Type	**sediment**	**effluent**	**effluent**	**effluent**	**sediment**	**sediment**	**effluent**	**sediment**	**effluent**	**effluent**	**sediment**	**sediment**	**sediment**	**sediment**	**effluent**	**sediment**	**sediment**
No. of organic compounds isolated	64	3	10	5	40	107	6	50	176	194	136	18	64	21	32	199	247
No. of compounds reliably identified	19 (30%)	1 (33%)	5 (50%)	2 (40%)	11 (28%)	36 (34%)	4 (67%)	15 (30%)	54 (31%)	27 (14%)	28 (21%)	8 (44%)	19 (30%)	10 (48%)	22 (69%)	62 (32%)	44 (19%)
ORGANOHALOGEN COMPOUNDS																	
Benzene, 1,2-dichloro-			1			*		1	*		*		*	*			1
Benzene, 1,3-dichloro-								*			*	*	*		*	1	1
Benzene, 1,4-dichloro-		*	*	*	*	*	*	1	*	1	1	*	*	*	*	1	1
Benzene, 1,2,3-trichloro-						*	*								*		
Benzene, 1,2,4-trichloro-						*	*				1				*	*	1
Benzene, 1,3,5-trichloro-									*		1					1	1
Benzene, 1,2,3,5-tetrachloro-						*					1				*	*	1
Benzene, 1,2,4,5-tetrachloro-						*					1				*	*	1
Benzene, 1,2,3,4-tetrachloro-						*	*								*	*	
Benzene, pentachloro-						*					*	*	*		*	*	1
Benzene, hexachloro-					*	1				*	1	1	1	*	*	1	1
Benzene, chloro(2-chloroethyl)-																	1
2-Butene, 1,4-dichloro-															1	1	
Naphthalene, dibromo-															1		
Naphthalene, chloro-																	1
Toluene, monochloro-																	2
PCB-138						*							*				
PCB-153						*							*				
PCB-180						*							*				
1,3-Butadiene, hexachloro-										1	*		*			1	1
Ethane, 1,1,2,2-tetrachloro-										1		1					
Ethane, 1,1'-oxybis[2-chloro-										1							
Styrene, 2,5-dichloro-																	1
Styrene, octachloro-											1						
PAHs																	
Naphthalene and/or its derivatives	2								15	2					5	15	9
Anthracene and/or its derivatives									1	1						1	2
Acenaphthene	1								1								



Sample Number	AM 0157	AM 0159	AM 0155	AM 0156	AM 0161	AM 0160	AM 0153	AM 0154	AM 0151	AM 0150	AM 0152	AM 0162	AM 0158	AM 0149	AM 01001	AM 01002	AM 01003
Sample Type	**sediment**	**effluent**	**effluent**	**effluent**	**sediment**	**sediment**	**effluent**	**sediment**	**effluent**	**effluent**	**sediment**	**sediment**	**sediment**	**sediment**	**effluent**	**sediment**	**sediment**
PAHs (continued)																	
Acenaphthylene									1	1							
Phenanthrene and/or its derivatives						1		1	1	1	1					3	
Pyrene and/or its derivatives	1					1		1		1	1					1	
9H-Fluorene and/or its derivatives									2	3					1	4	1
Fluoranthene	1					1				1						1	
Benz[a]anthracene and/or derivatives						1											
11H-Benzo[b]fluorene										1							
OTHER AROMATIC COMPOUNDS																	
DEP			1														
BHT											1						
Ter- and quaterphenyls																	
Alkylated benzenes	3							1	6						3	6	8
Biphenyl and/or its derivatives									1						2	6	1
Diphenyl ether			1														
Ethanone, 1-(2,5-dimethylphenyl)-																	1
1H-Indene and/or its derivatives						1			8								
Benzocycloheptene									1								
Benzocycloheptatriene									1								
Methyl naphthyl ketone			1														
ALIPHATIC HYDROCARBONS																	
Linear	11				9	19		8	12	11	14	3	10	7		13	7
Cyclic								1									
MISCELLANEOUS																	
Carbazole, 4-methyl-									1								
Isobornyl acetate				1													

*Table 3. Organic chemicals and heavy metals identified in samples from the Complejo Petroquimico Pajaritos. Numbers denote the number of compounds within each group identified using general GC/MS screening method. * Signifies compounds identified only at trace levels using a selective ion monitoring (SIM) method. Sediment samples are highlighted.*

Sediment sample AM0157 was collected upstream of all of the identifiable discharges from the complex and was intended to act as a control sample. It does contain some hydrocarbon pollutants. This is not surprising given the proximity of major petroleum processing operations, which could contaminate the river via runoff or atmospheric deposition. Other than this, though, the sample appears uncontaminated. No organochlorines were detected and metals were within concentrations expected in unpolluted freshwater systems.

AM0159 was collected from a square sump between the river and the perimeter wall of the complex. It was not highly contaminated. Of the metals, only zinc and manganese were present at above detection limits and only one organic compound, 1,4-dichlorobenzene, was identified. It is possible that the sump contains predominantly rainwater rather than industrial effluent.

Further downstream, two effluent samples were collected (AM0155 and AM0156). Both contained chloroform and 1,4-dichlorobenzene and one (AM0155) also contained 1,2-dichlorobenzene and diethyl phthalate. In addition, mercury was being discharged from both pipes. Copper was also present in effluent AM0156 and zinc, which was detected in all samples, was present at higher concentrations than normal. Close to where AM0155 and AM0156 were collected, river sediment AM0161 was obtained. This also contained few organic pollutants (1,4-dichlorobenzene, hexachlorobenzene and 9 aliphatic hydrocarbons). Concentrations of mercury were elevated to approximately 30-40 times that which would be expected in uncontaminated freshwater sediments (Salomons & Forstner 1984). This is clearly the consequence of the mercury from the two nearby pipes. Zinc was also elevated, presumably for the same reason. The concentrations of chromium and lead in the sediment were significantly higher that expected in uncontaminated sediment, although this metal was not detected in the effluents.

The next pipe downstream discharged what appeared to be sewage. This was not sampled, but sediment was collected from close downstream to establish whether the discharge did in fact alter the pollution status of the river. This sediment sample, AM0160 was significantly more contaminated than those from further upstream, presumably as a result of pollutants released via this pipe in addition to any sewage. At over 60mg/kg dry weight, the mercury concentration was extremely high; moreover it was the highest measured in this survey. Concentrations of copper, lead and zinc were higher than expected in uncontaminated freshwater sediments. AM0160 also contained far more organic compounds than the samples collected further upstream, with a total of 107 isolated. Of these, 36 could be identified with a high degree of certainty, including 9 chlorobenzenes, three PCB congeners and a number of hydrocarbons, mostly linear aliphatic compounds.

The next location downstream sampled was a pipe discharging into the arroyo close to the Cydsa facility. It is not know which company or companies use this pipe. Here, effluent AM0153 and sediment AM0154 were collected. Both samples contained chlorobenzenes, though the only one common to both was the ubiquitous 1,4-dichlorobenzene. Concentrations of most metals were unremarkable, though zinc was elevated. Mercury was not detected in the effluent and its concentration in the sediment, though still indicative of significant contamination, was less than a tenth of the concentration in the sediment AM0160 from slightly further upstream.

The next sampling site was close to where the road from Coatzacoalcos crosses the Arroyo Nuevo Teapa. Here, two large pipes discharge. They provided samples AM0150 and AM0151. Both of these contained significantly more organic contaminants that the effluents collected upstream; the screening analysis isolated 194 and 176 compounds respectively. In addition, quantitative analysis showed 1,2-dichlorethane (also known as EDC) in both samples and 5 other volatile organochlorines in AM0150. The organochlorines identified to a high degree of certainty in the screening analysis included chlorobenzenes (both samples), and hexachlorobutadiene, 1,1,2,2-tetrachloroethane and 1,1'-oxybis[2-chloroethane] (AM0150). Both effluents also contained numerous hydrocarbons and whereas AM0150 contained more organochlorines, AM0151 contained more hydrocarbons. AM0152, which came from immediately downstream of these two pipes, showed a similar profile of organochlorines in the screening analysis, with the addition of octachlorostyrene. However, the sediment contained fewer hydrocarbons than the wastewaters. Copper and high concentrations of zinc were present in the effluent AM0150 and concentrations of zinc and lead were above normal in the sediment. Mercury was not found in the effluents and the concentration in the sediments continued to decline.

Close to this sampling site, but north of the river, are wetlands which are close enough to the complex to be at risk of receiving its runoff. Sediment AM0162 from this wetland contained chlorobenzenes and 1,1,2,2-tetrachloroethane, but only three hydrocarbons were identified, fewer than in any other sediment analysed during this survey. The concentration of lead was above normal for soils and sediments.

Returning to the Arroyo Nuevo Teapa, two further sediments were collected in the lower reaches before it joins the Rio Coaztacoalcos. The first of these, AM0158, contained 5 chlorobenzenes, three PCBs and hexachlorobenzene. The other, AM0149 contained only three chlorobenzenes. The only hydrocarbon identified in these samples were linear aliphatic hydrocarbons, similar to those present along the length of the arroyo. However, AM0149 contained noticeably higher concentrations of heavy metals than AM0158. In particular, the concentration of mercury in AM0149, 19.75 mg/kg, was some nine times higher than in AM0158. Zinc was elevated in both samples; copper and lead were elevated in AM0149.

The Rio Coatzacoalcos flows north into the Gulf of Mexico. A little way downstream from where the Arroyo Nuevo Teapa joins it, and north of the bridge across the river, was the last discharge pipe sampled during this survey. Here, effluent AM01001 was collected directly from the pipe and sediment AM01002 from underneath it. Sediment AM01003 was collected approximately 6 metres away. This effluent was by far the most contaminated. The effluent contained 20 identifiable organochlorines, including 9 chlorobenzenes and 10 volatile compounds. There was also a moderate degree of hydrocarbon contamination. Concentrations of all of the volatile organochlorines were significantly higher than any other effluent analysed during this survey. The pollutants present in the greatest quantities were chloroform at 2700 ug/l and 1,2-dichloroethane (EDC) at 58500 ug/l. The effluent also contained a significant loading of copper as well as nickel and zinc. No mercury was detected.

The two sediments (AM01002 and AM01003) both contained numerous organic pollutants, both chlorinated and non-chlorinated. Both contained similar numbers of chlorobenzenes as the effluent, as

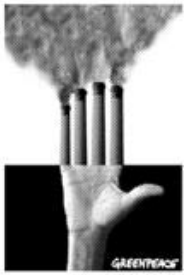
well as other organochlorines. Concentrations of metals in the sediments were not above background levels for freshwater sediments.

It is not possible to attribute all the pollutants detected around the Complejo Paharitos with specific production processes, but some conclusions can be drawn with some confidence. Although road traffic will release some oil and other hydrocarbons, the enormous amounts of petroleum products being processed at the complex must be the primary contributor to the oil contamination in the river.

Effluents AM1050 (discharged to the Arroyo Nuevo Teapa) and AM01001 (discharged to the Rio Coatzacoalcos) both contained high concentrations of 1,2-dichloroethane (EDC) which is known to be manufactured at the complex. The primary use of EDC is in the manufacturing of vinyl chloride (VCM) which was also present in both wasewaters. VCM is used to produce the common plastic, PVC. Both these effluents also contained chlorobenzenes and other organochlorines, as did the sediments collected neraby. In addition to these sites, nine chlorobenzenes were found in sediment sample AM0160, further up the river. The wastewater discharge immediately upstream from this location was not sampled so it is not known whether volatile organochlorines like EDC and VCM are being disharged here, but the presence of so many chlorobenzenes suggests that this site may have been contaminated by a similar source.

The mixture of organochlorine pollutants in the samples described above is also closely similar to that found in effluents from the PVC industry in Europe (Johnston *et al*. 1996). These effluents typically contain chlorinated methanes and ethanes, chlorinated benzenes, and chlorinated substituted benzenes, such as toluenes and styrenes. Moreover, both the effluents in question (AM0150 and AM01001) contained copper. This is used as a catalyst in the oxychlorination reaction which is used to produce EDC (ICI 1994). Copper is also controlled by the OSPAR Convention in Europe as one of the pollutants emitted by the EDC/VCM producers (see Table 4 below). This is further evidence that EDC/VCM production is the source of this pollution.

US federal regulations for this industrial sector are found under Title 40, part 414 (US GPO 2000). The limit values applicable to each discharge are dependent upon factors such as whether the effluent is biologically treated, whether discharges are direct or indirect and whether measurements are daily maxima or monthly averages. The effluent samples AM0150 and AM01001 exceeded one or more of these limts for eleven separate parameters: copper, zinc and nine volatile organochlorines. These are summarised in Table 4. For eight pollutants, the concentrations in one or other sample exceeds all the specified limits. AM0150 exceeded all limits for two parameters, AM01001 for six. In the worst case, AM01001 contains slightly over 100 times the highest limit for EDC. There are, in addition, several parameters in the Federal regulations which were detected in the screening analysis, but which were not quantified. These include four chlorobenzenes, hexachlorobutadiene and some aromatic hydrocarbons.



	Direct discharge point sources that use end-of-pipe biological treatment		Direct discharge point sources that do not use end-of-pipe biological treatment.		Toxic pollutant standards for indirect discharge point sources.		AM0150	AM01001
All concentrations are in ug/l	Maximum for any one day	Maximum for any monthly average	Maximum for any one day	Maximum for monthly average	Maximum for any one day	Maximum for any monthly average	results of current survey	results of current survey
Total Copper	3380	1450	3380	1450			110	4919**
Total Zinc	2610	1050	2610	1050	2610	1050	5048**	840
Chloroform	46	21	325	111	325	111	130*	2700**
Carbon Tetrachloride	38	18	380	142	380	142	40*	180*
Vinyl Chloride	268	104	172	97	172	97	40	390**
1,1-Dichloroethene	25	16	60	22	60	22	30*	150**
trans-1,2-Dichloroethene	54	21	66	25	66	25	<5	35*
Trichloroethene	54	21	69	26	69	26	<5	55*
Tetrachloroethene	56	22	164	52	164	52	60	110*
1,1-Dichloroethane	59	22	59	22	59	22	<5	900**
1,2-Dichloroethane	211	68	574	180	574	180	7500**	58500**

*Table 4: Comparison of analytical data of effluents from Coatzacoalcos with US Federal regulations for the plastics industry. All concentrations are in ug/l. *Results which exceed one or more of the limit values; **results which exceed all of the limit values.*

The European legislation pertaining to the production of EDC and VCM covers a different range of pollutants and does not provide specific effluent concentration limits. Rather, they specify maximum quantities that can be released on the basis of production capacity, and covers a smaller number of pollutants (Table 5). However, the European regulations do cover another critical parameter for emissions from this industry: the polychlorinated dibenzo-p-dioxins and dibenzofurans (dioxins or PCDD/Fs). High concentrations of PCDD/Fs can be found in the environment around EDC/VCM production plants and the "heavy ends" generated by the purification of EDC can exhibit extreme contamination (see eg ICI 1994, EA 1997, Stringer *et al*. 1995). Improper disposal of these wastes could have serious repercussions. Analysis of dioxins was beyond the scope of this survey, but given the nature of the organochlorine contamination found, it is inevitable that they will also be present.

Substance	**Emission to:**	**Limit value**
VCM	air	5 mg Nm^{-3}
EDC	air	5 mg Nm^{-3}
dioxins	air	0.1 ng ITEQ Nm^{-3}
chlorinated hydrocarbons	Water before secondary treatment)	0.7 g per tonne of EDC purification capacity
copper	water (after final treatment)	0.5 g per tonne oxychlorination capacity for plants with fixed bed reactors, 1.0 g per tonne oxychlorination capacity for plants with fluidised bed reactors
dioxins	water (after final treatment)	1 ug TEQ per tonne of oxychlorination capacity
COD	water (after final treatment)	250 mg/l effluent

Table 5: Limit values for emissions from plants manufacturing ethylene dichloride and/or vinyl chloride monomer as set by the OSPAR Convention.

There are clearly several locations at which mercury is being discharged to the Arroyo Nuevo Teapa. At one location, two direct discharges AM0155 and AM0156 were detected. The long-term record in the sediment shows contamination at this site and also at two further sites downstream (AM0160 and AM0149) though the source of the pollution at these last two locations is not so clear. Mercury is one of the most environmentally damaging of the heavy metals. It is widely used in the manufacturing of batteries and electronic components. Applications within the chemical industry include as a catalyst and as the cathode in the electrolysis of sodium chloride solution to make chlorine (ATSDR 1997, Curlin *et al*. 1993). Chlorine is among the chemicals manufactured at the Complejo Paharitos so this is one of the most likely sources of the mercury at this site. However, there are two methods of manucturing chlorine without use of mercury- the asbestos cell and the membrane process. Therefore, without more information about the processes employed at the complex, it is not possible to be certain of the exact source of the mercury. However, the two effluent samples which contained mercury also contained notable levels of chloroform. This is a byproduct of chlorination of water (IPCS 1994), so would be expected to be present in the wastewaters of chlorine production plants.

A small amount of lead (47ug/l) was found in only one of the effluents (AM0151). However, lead was present at above expected concentrations in most of the sediment samples. Emissions from traffic on the

nearby road may have comtributed to this. However, it is believed that the petrol additive, tetraethyl lead was manufactured at the complex. This would potentially have resulted in much larger inputs of lead to the river.

Trace levels of PCBs were detected in the river sediments at two locations (AM0160 & AM0158). They were not detected in any effluents, but the PCBs' poor water solubility mean thay would not be present in water samples at detectable levels. PCBs were used in applications from plastics additives to hydraulics fluids from the 1930s onwards. However, their environmental hazards meant that have been being phased out since the 1970s. In May 2001, a global convention will be signed which will lead to a global ban on PCB manufacturing. Despite this, PCBs have remained in place in old equipment such as electrical transformers in many places (see eg Stringer & Johnston 2001). Unless a facility is actually manufacturing PCBs, the largest reservoir of PCBs at a site such as the Complejo Paharitos would be transormers at electrical substations. However, on the current evidence it is not possible to come to any conclusions about the source of the PCBs; indeed their extreme environmental persistence means that they may have been discharged years in the past.

1,4-Dichlorobenzene was present in all samples except the upstream control sample. It is an extremely common pollutant because of its use in toilet deodorants and disinfectants. Consequently, though its presence in wastes from EDC/VCM production could explain its presence in most of the samples, in most of them its primary source is likely to be due to sewage discharges.

Lastly, there are several metals at elevated concentrations in samples from this survey. Chromium was present at higher than normal concentrations in two sediments (AM0161 and AM0149) although it was not present in any of the discharges. Nickel was found in effluent sample AM01001, though none of the concentrations in the sediments were above normal levels. Zinc, a common metal, was present in all samples, but at high concentrations in effluents AM0156, AM0150 and AM01001 and in sediments AM0161, AM0160, AM0154, AM0152, AM0158 and AM0149. Manganese was elevated in effluents AM0155, AM0156, AM0153 and AM0150 and in sediments AM0161, AM0160, AM0152, AM0158 and AM0149. Each of these metals has a wide range of uses in manufacturing and chemical processing. They are often used as catalysts and nickel, chromium and manganese are components of steel which may have been released as the steel corroded. More information is needed to draw any conclusions about the precise sources of these metals to the environment.

4 CONCLUSIONS

The environment in the vicinity of the Complejo Petroquimico Paharitos is subjected to pollution from several different discharges. These have been shown to be releasing hydrocarbons, heavy metals including mercury, and a wide range of volatile and semivolatile organochlorines. The Arroyo Nuevo Teapa, which receives most of the identified dicharges is, heavily polluted. In most cases the contamination in the sediments can be attributed directly to the effluents sampled, though the evidence available suggests there are further release points in addition to those sampled during this survey.

The hydrocarbon pollution is thought to be primarily due to the releases of petroleum hydrocarbons being processed at the complex. Sources of several heavy metals cannot be explained because of the number of possible applications within a plant such as this. However, lead pollution is probably due to the former manufacturing of tetraethyl lead for sale as a petrol additive, with the possibility of a contribution from road runoff. Mercury is not likely to be present as a consequence of its use as a catalyst or in the mercury cells which are one possible method of producing chlorine.

The extensive organochlorine contamination consists of volatile and semivolatile compounds in the effluents. Sediments were not analysed for volatile organic compounds, but contained similar mixtures of semivolatile organochlorines. These mixtures closely match the pollutants associated with the manufacturing of EDC and VCM which is polymerised to make the common plastic, PVC. Concentrations of several of the volatile organochlorines were present in concentrations above those that would be allowable under US federal regulations for the plastics industry. In particular, two discharges contained significant concentrations of EDC, making it almost certain that the production of this chemical is responsible for the observed pollution. Since the production of EDC is known to also generate dioxins and furans, there is also likely to be dioxin contamination at these locations. The disposal of the highly contaminated organic wastes from the purification of the EDC is also a cause for concern.

5 REFERENCES

ATSDR (1997) ATSDR's toxicological profiles on CD-ROM, CRC Press, Boca Raton, FL.

EA (1997) Regulation of dioxin releases from the Runcorn operations of ICI and EVC. UK Environment Agency Information Report.

Curlin, L.C., Bommaraju, T.V. & Hansson, C.B. (1993) Alkali and chlorine products. In: Kroschwitz, J.I. & Howe-Grant, M. (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition. Publ: Wiley-Interscience, N.Y. Volume 1 pp 938-1025

ICI (1994) Report to the Chief Inspector HMIP authorisation AK6039 Improvement Condition part 8, Table 8.1, item 2: Formation of dioxins in oxychlorination, significance for human health and monitoring proposals. ICI Chemicals and Polymers report NWJP/BMTD, 27th April 1994, 16pp

IPCS (1994) Chloroform. Ionternational Programme on Chemical Safety (IPCS) Environmental Health Criteria 163. Publ: World Health Organisation. ISBN 92 4 157 163 2, 174pp.

Johnston, P.A., Stringer, R.L., Clayton, R. & Swindlehurst, R.L. (1996) Regulation of toxic chemicals in the North Sea: the need for an adequate control strategy. In: Proceedings of the Scientific Symposium on the North Sea Quality Status Report 1993, 18-21 April 1994, Ebeltoft, Denmark. Publ: Danish Environmental Protection Agency, ISBN 87-7810-699-0, pp 269-274

Salomons, W. & Forstner, U. (1984). Metals in the hydrocycle. Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, ISBN 3540127550

Stringer, R.L., Costner, P. & Johnston, P.A. (1995) PVC manufacture as a source of PCDD/Fs. Organohalogen Compounds 24: 119-123

Stringer, R. & Johnston, P. (2001) Chlorine and the environment: an overview of the chlorine industry. Publ: Kluwer Academic Publishers, ISBN 0-7923-6797-9, 430pp

USGPO (2000) Title 40- Protection of the Environment. Chapter 1, Part 414- Organic chemicals, plastics and synthetic fibers. Publ: US Government Printing Office, http://www.access.gpo.gov/nara/cfr/waisidx_00/40cfr414_00.html

APPENDIX 1 ANALYTICAL METHODOLOGY

A1.1 Organic analysis

Preparation of samples for organic screen analysis

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

Solid Samples

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

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

Aqueous Samples

Prior to the extraction, samples were spiked with deuterated naphthalene (an internal standard) at a concentration of 10mg/l. 20ml of pentane 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 a 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.

Chromatographic Analysis

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

List of compounds in the Standard I used for SIM 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. Extracts were also analysed using selective ion monitoring (SIM) method against two standard solutions. The compounds contained in Standard I and Standard II are listed below. All individual standards were obtained from Sigma Aldrich Co. Ltd., Supelco, UK.

Results are reported as either reliably or tentatively identified. Match qualities of 90% or greater against 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

List of compounds in the Standard II used for SIM analysis

Volatile Organic Compounds (VOCs) analysis

For volatile organic compound analysis, no sample preparation was required. The original sample was sub-sampled immediately after opening. Three portions of 10ml each were transferred into 20ml headspace vials and sealed with Teflon-lined vial caps. One sub-sample was used for the organic screen analysis to evaluate the whole range of volatile compounds in the sample. The second sub-sample was analysed using Selective Ion Monitoring (SIM) method to detect the VOCs listed in the Table below. The third sub-sample was used for quantification of the detected compounds with an external standard using SIM method. All standard compounds were obtained from Sigma-Aldrich Co. Ltd./Supelco UK.

Name of compound	Target ion	Qualifying ions
1,1,1-Trichloroethane	97	61, 26, 117
1,1-Dichloroethane	63	27, 83, 98
1,1-Dichloroethene	61	96, 26, 35
Carbon tetrachloride	117	35, 47, 82
Chlorobenzene	112	77, 51, 38
Chloroform	83	47, 35, 118
cis-1,2-Dichloroethene	61	96, 26, 35
1,2-Dichloroethane	62	27, 49, 98
Hexachlorobutadiene	225	260, 190, 118
m- & p-Xylene	91	106, 77, 51
o-Xylene	91	106, 77, 51
Tetrachloroethene	166	129, 94, 47
Toluene	91	39, 65, 51
trans-1,2-Dichloroethene	61	96, 26, 37
Trichloroethene	130	95, 60, 35
Vinyl chloride	27	62, 37, 47

List of volatile organic compounds and appropriate ions that were monitored during GC/MS analysis using SIM method.

A1.2. Heavy Metal Analysis

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.

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.

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.

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 for solid samples, and 10% v/v nitric acid for aqueous samples). 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 for solid samples, and 10% v/v nitric acid for aqueous samples). 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

1,2-Dichloroethane (EDC)

Ethylene dichloride (EDC) is more properly called 1,2-dichloroethane. It is a pleasant-smelling, colourless, volatile liquid, which does not persist long in the environment but is both hazardous and toxic. It is highly flammable and may pose an explosion hazard. Violent reactions can take place with aluminium, nitric acid or ammonia. Its primary use is in the manufacture of vinyl chloride, which accounts for 96% of its consumption in the USA (Reed 1993).

1,2-Dichloroethane (EDC) is produced by chlorination or oxychlorination of ethene. It is one of the leading bulk chemical products and production nearly trebled in the decade to 1987. The 1988 EDC production capacity was about 31 million t y^{-1} (9 000 kt in North America, 10 000 kt in Western Europe and 3 000 kt in Japan) (Snedecor 1993). 96% of US production is used in the production of vinyl chloride monomer (VCM) used in the manufacture of PVC (Reed 1993). Other applications include solvent manufacture, ethyleneamine preparation and addition to fuel anti-knock mixtures (Snedecor 1993). EDC is regulated by the USEPA because of its contribution to the formation of tropospheric ozone (Sullivan 1997).

The production of EDC results in the generation of chlorinated toxic distillation residues called heavy ends. These are contaminated with dioxin (EA 1997, ICI 1994) and concentrations can reach part per million levels (Stringer *et al*. 1995).

Because of its volatility, the most usual route of exposure is via inhalation, though it can also be absorbed through the skin or gastro-intestinal tract (IPCS 1995). It is one of the more toxic chlorinated solvents and can be toxic at concentrations too low to be detected by smell (Snedecor 1993). Inhalation or ingestion of large amounts by humans can impact the central nervous system, liver, kidneys, lungs and cardiovascular system (IPCS 1995). It can irritate the eyes, nose and throat, upset the nervous and gastrointestinal systems, cause dizziness, nausea and vomiting, and may damage the liver, kidney and adrenal gland. It is distributed to all tissues of the body and can pass both the blood/brain barrier and the placenta. EDC is classified by the IARC in Group 2B (possibly carcinogenic to humans) and the US Department of Health and Human Services classifies it as reasonably anticipated to be a human carcinogen (USDHHS 2000).

References

EA (1997) Regulation of dioxin releases from the Runcorn operations of ICI and EVC. UK Environment Agency Information Report.

ICI (1994) Report to the Chief Inspector HMIP authorisation AK6039 Improvement Condition part 8, Table 8.1, item 2: Formation of dioxins in oxychlorination, significance for human health and monitoring proposals. ICI Chemicals and Polymers report NWJP/BMTD, 27th April 1994, 16pp

IPCS (1995) 1,2-Dichloroethane (second edition). International Programme on Chemical Safety Environmental Health Criteria 176, Publ: World Health Organisation, ISBN 92 4 157176 4, 148pp.

Reed D.J. (1993) Chlorocarbons, -hydrocarbons (survey). In: Kroschwitz, J.I. & Howe-Grant, M. (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition. Publ. Wiley-Interscience, N.Y. Volume 5, pp 1017-1028

Snedecor, G (1993) Other chloroethanes. In: Kroschwitz, J.I. & Howe-Grant, M. (Eds), The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition. Publ: Wiley-Interscience, N.Y., Vol. 6, pp.11-36

Stringer, R.L., Costner, P. & Johnston, P.A. (1995) PVC manufacture as a source of PCDD/Fs. Organohalogen Compounds 24: 119-123

Sullivan, D.A. (1997) Solvents, industrial. In: Kroschwitz, J.I. & Howe-Grant, M. (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition. Publ. Wiley-Interscience, N.Y. Vol. 22, pp 529-571

USDHHS (2000) 9th report on carcinogens. Publ: US Department of Health and Human Services, 334pp.

Vinyl chloride

Vinyl chloride (also known as vinyl chloride monomer or VCM) is a colourless, sweet smelling gas under normal conditions. Vinyl chloride manufacture accounts for 36% of global chlorine consumption (Chemical Week 1998). In the US in 1992, 98% of vinyl chloride was used to make PVC (ATSDR 1997). Approximately 95% of the VCM produced globally has an end use in polymer or copolymer applications, of which by far the largest is PVC production. Non-polymeric uses occur in the synthesis of a variety of industrial compounds and in the manufacture of vinylidene chloride and tri- and tetra-chloroethene. Total world capacity of VCM in 1996 was approximately 25 500 kt y^{-1}, of which about 54% was concentrated in the United States and Western Europe (Cowfer & Gorensek 1997).

In the wider environment, VCM is not persistent. It is degraded in a few hours by the action of light and reactions with the atmospheric OH radical. If released to water, vinyl chloride volatilises rapidly to the air. However, in soil, vinyl chloride migrates rapidly to groundwater. Here it is far more persistent, with a maximum estimated degradation half-life approaching eight years (Howard *et al.* 1991).

Vinyl chloride is an extremely hazardous substance. Mixed with air, it can be explosive and it causes a wide variety of toxic effects in humans and animals. It has been known for over 20 years that it causes cancer in humans. It was classified as known to be a human carcinogen by the US government in 1980. The cancer most strongly associated with vinyl chloride is angiosarcoma of the liver in the occupationally exposed and, although the evidence is less strong, other studies have shown elevated levels of cancers of the brain and nervous system, lung and respiratory tract and the lymphatic/haemapoietic system. There are less well supported indications of cancers at still further sites (ATSDR 1997). Retrospective analysis of histories of individuals with angiosarcomas at different sites suggest that exposure to vinyl chloride, PVC and other polymeric materials may have been a factor (Rhomberg 1998).

The most usual route of exposure to vinyl chloride is through inhalation. Vinyl chloride appears to cause emphysema, dyspnea, pulmonary lesions and a number of other lung problems (ATSDR 1997). Exposure to high levels of vinyl chloride can cause Raynaud's phenomenon, where the blood circulation in workers' fingers is damaged so that they become white and painful in cold conditions. This illness is

sometimes followed by resorption of the bones in the tips of the fingers or lesions on bones in other parts of the body (ATSDR 1997).

Workers have also been reported to die more frequently from cardiovascular and cerebrovascular disease (e.g. heart attacks and strokes). Vinyl chloride can also reduce the blood's ability to clot normally (ATSDR 1997).

Vinyl chloride is narcotic and inhalation can cause dizziness, headaches, drowsiness or unconsciousness, euphoria, memory loss, visual and/or hearing disturbances, sleep disturbance, nausea, irritability and nervousness. Damage to the nervous system manifests itself in peripheral neuropathy with tingling, pain or numbness in the fingers (ATSDR 1997). Also seen are toxic effects on the immune systems, livers, spleens, thyroid function, eyes and skin of workers. Anorexia (weight loss) has been reported and there are some indications that vinyl chloride can affect the reproductive systems of both men and women (ATSDR 1997).

To protect workers, many countries have set maximum limits for vinyl chloride in air. These range from 1ppm (2.6mg/m^3) in the USA to 7ppm (18.2 mg/m^3) in Finland, Germany and the UK. These limits are below odour thresholds (IPCS 1999).

The high toxicity of vinyl chloride has lead to it being banned for domestic use in both the USA and the European Community. The US ban dates from 1974 (USDHHS 2000). EC directive 76/769/EEC controls the sales and use of dangerous substances within the European Community. When this directive was brought in in 1976, it banned the use of vinyl chloride as an aerosol propellant on the basis of its hazards to human health (EEC 1976). Vinyl chloride was one of the first three substances controlled under this legislation, a measure of the hazard is represents.

VCM can be produced by the hydrochlorination of ethyne (acetylene), but currently over 90% of world production is carried out by the thermal cracking of 1,2-dichloroethane (EDC) (Cowfer & Gorensek 1997). It is then polymerised to PVC by the suspension, emulsion or mass process.

Substance	Emission to:	Limit value
VCM	air	80 g per tonne of s-PVC produced
VCM	water (after effluent stripper, before secondary treatment)	1 mg VCM per litre 5 g VCM per tonne s-PVC produced
COD	water (at outlet of effluent water treatment plant)	125 mg per litre for single plants 250 mg per litre for combined plants*
suspended solids	water (at outlet of effluent water treatment plant)	30 mg suspended solids per litre

*Limit values for suspension polymerisation PVC plants as set by the OSPAR Convention. *single plants manufacture only suspension-polymerisation PVC (s-PVC); combined plants are those where s-PVC is produced as part of an industrial site, where other chemical processes are being carried out.*

Emission limits for the ethylene dichloride/vinyl chloride production and PVC polymerisation using the suspension process are given in OSPAR decisions 98/4 and 98/5 respectively. Plants built after February 1999 would have to adhere to these limits immediately, but plants already in existence would have until the beginning of 2006 to meet the EDC/VCM limits and until the beginning of 2006 to meet the polymerisation plant limits. The European Commission has also put forward a proposed Decision which would incorporate these limits into EC law (EC 1999).

In the USA, federal legislation has been designed to control the emissions of vinyl chloride from plastics manufacturers. Title 40, Part 414 specifies a range of limits depending on the type of discharge and measurement period (USGPO 2000). These are summarised below.

Type of discharge	Maximum concentration for any one day	Maximum for any monthly average
Direct discharge point sources that use end-of-pipe biological treatment	268 ug/l	104 ug/l
Direct discharge point sources that do not use end-of-pipe biological treatment	172 ug/l	97 ug/l
Indirect source point discharges	172 ug/l	97 ug/l

References

ATSDR (1997) ATSDR's toxicological profiles on CD-ROM, CRC Press, Boca Raton, FL.

Chemical Week (1998) Polyvinyl chloride. Chemical Week 160(42): 36

Cowfer, J.A. & Gorensek, M.B. (1997) Vinyl chloride. In: Kroschwitz, J.I. & Howe-Grant, M. (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition. Publ. Wiley-Interscience, N.Y. Volume 24, pp 851-882

EC (1999) Proposal for a Council Decision concerning the approval, on behalf of the Community, of OSPAR Decision 98/4 on emission and discharge limit values for the manufacture of vinyl chloride monomer and of OSPAR Decision 98/5 on emission and discharge limit values for the vinyl chloride sector (1999/C 158/04) COM(1999) 190 final – 99/0098 (CNS). OJ C 158, 4.6.1999: 19-32

EEC (1976) Council Directive 76/769/EEC of 27 July 1976 on the approximation of the laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations. OJ L 262, 27.9.76: 201-203

Howard, P.H., Boethling, R.S., Jarvis, W.F., Meylan, W.M. & Michalenko, E.M. (1991) Handbook of environmental degradation rates. Publ: Lewis Publishers, ISBN 0-87371-358-3, 725pp

IPCS (1999)Vinyl Chloride. Environmental Health Criteria 215. Publ: WHO, ISBN 92 4 157215 9, 356pp.

Rhomberg, W. (1998) Exposure to polymeric materials in vascular soft-tissue sarcomas. International Archives of Occupational and Environmental Health 71: 343-347

USDHHS (2000) 9th report on carcinogens. Publ: US Department of Health and Human Services, 334pp.

USGPO (2000) Title 40- Protection of the Environment. Chapter 1, Part 414- Organic chemicals, plastics and synthetic fibers. Publ: US Government Printing Office, http://www.access.gpo.gov/nara/cfr/waisidx_00/40cfr414_00.html

Chloroform

Chloroform is a heavy, colourless, non-flammable liquid. It has a characteristic pleasant, sweet odour and a sweetish burning taste (CEC 1986).

It has been extensively used in the past as an anaesthetic (Snyder & Andews 1996). Currently the largest use of chloroform is to make HCFC-22, an ozone-depleting refrigerant (Holbrook 1993). The Montreal Protocol, the international legislation which protects the ozone layer, has set targets for reducing the use of HCFC-22, but it will not be totally phased out until 2030 (UNEP 1997).

Chloroform is the most abundant of the trihalomethanes (THMs) which are generated as by-products during water disinfection using chlorine-containing compounds (Oxenford 1996, ATSDR 1997, Health Canada 1996). Additionally it can be formed in washing machines into which chlorinated bleach has been added (Shepherd & Corsi 1996), in the natural waters where chlorine-containing effluents have been discharged (Mills *et al.* 1998). Exposure to chloroform may occur when breathing contaminated air, drinking contaminated water or through skin contact (Weisel & Chen 1994; Weisel & Jo 1996). Water is possibly now the major source of environmental exposure to chloroform.

Chloroform has been specified by the International Agency for Research on Cancer in the Group 2B as possibly carcinogenic to humans (IARC 1998). Investigations on animals have shown that the main target organs for carcinogenicity from chloroform are liver, kidney and/or intestine (Dunnick & Melnik 1993, Snyder & Andews 1996, Chiu *et al.* 1996). A guideline value of 200ug/l was calculated to correspond to an excess lifetime cancer risk of 10^{-5} by the World Health Organisation (WHO 1993). There are four contaminants included in this group: chloroform, bromodichloromethane, dibromochloromethane and bromoform (Oxenford 1996).

It is not known whether chloroform causes reproductive effects or birth defects in people, but animal studies have shown that miscarriages occurred in rats and mice that breathed air containing 30–300 ppm chloroform during pregnancy and also in rats that ate chloroform during pregnancy. Offspring of rats and mice that breathed chloroform during pregnancy had birth defects. Abnormal sperm were found in mice that breathed air containing 400 ppm chloroform for a few days (ATSDR 1997).

The levels of chloroform found in treated drinking water depends upon water treatment practice, age of the water, water temperature (Health Canada 1996) and can vary in the range from less than 1ug/l to 200ug/l (Wallace 1997, Health Canada 1996). Levels less than 10ug/l were found in US rural ground water (Wallace 1997). A mean value of 84ug/l was reported for surface waters (if detected) in the same survey.

Chloroform evaporates easily into the air. Most of the chloroform in air breaks down eventually, but it is a slow process. The breakdown products in air include phosgene and hydrogen chloride, which are both toxic (ATSDR 1997). It is poorly absorbed to soil and can travel through soil to groundwater where it can persist for years. Chloroform dissolves easily in water and some of it may break down to other chemicals (ATSDR 1997).

The presence of chloroform (as an organohalogen compound) in groundwater is controlled by European Community Environmental Legislation. Article 3 of EC Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances (EEC

1979) and amended later (EEC 1991) says that Member States shall take necessary steps to prevent the introduction into groundwater of substances in List I and organohalogen compounds are among the groups of the compounds listed there.

The quality objective of 12ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters and territorial waters) is set for chloroform by the EC Council Directive 86/280/EEC (EEC 1986) and amended in 1988 (EEC 1988).

The EC Council Directive 76/769/EEC (EEC 1976) which was last amended in 1996 (EEC 1996) restricts marketing and use of chloroform. Chloroform may not be used in concentrations equal to or greater than 0.1% by weight in substances and preparation placed on the market for sale to the general public and/or in diffusive applications such as in surface cleaning and cleaning of fabrics.

In the USA, federal legislation has been designed to control the emissions from plastics manufacturers. The Environmental Protection Agency Code of Federal Regulations, Title 40, Part 414 specifies a range of limits depending on the type of discharge and measurement period (USGPO 2000). These are summarised below.

Type of discharge	Maximum concent-ration for any one day	Maximum for any monthly average
Direct discharge point sources that use end-of-pipe biological treatment	46 ug/l	21 ug/l
Direct discharge point sources that do not use end-of-pipe biological treatment	325 ug/l	111 ug/l
Indirect source point discharges	325 ug/l	111 ug/l

References

ATSDR (1997) ATSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

CEC (1986) Chloroform. In: Organochlorine solvents: health risk to workers. Publ: Commission of the European Communities, ISBN 0-85186-078-8: 17-53

Chiu, N., Orme-Zavaleta, J., Chiu, A., Chen, C., DeAngelo, A., Brattin, W. & Blancato, J. (1996) Characterization of cancer risk associated with exposure to chloroform. Environmental Carcinogenesis & Ecotoxicology Reviews. Journal of Environmental Science and Health C14(2): 81-104

Dunnick, J.K & Melnik, R.L. (1993) Assessment of the carcinogenic potential of chlorinated water: experimental studies of chlorine, chloramine and trihalomethanes. Journal of the National Cancer Institute 85(10): 818-823

EEC (1976) Council Directive 76/769/EEC of 27 July 1976 on the approximation of laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations. OJ L 262: 201-203

EEC (1979) Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances. OJ L 020: 43-48

EEC (1986) Council Directive 86/280/EEC of 12 June 1986 on limit values and quality objectives for discharges of certain dangerous substances included in List I of the Annex to Directive 76/464/EEC. OJ L 181: 16-27

EEC (1988) Council Directive 88/347/EEC of 16 June 1988 on limit values and quality objectives for discharges of certain dangerous substances included in List I of the Annex to Directive 76/464/EEC. OJ L 158: 35-41

EEC (1991) Council Directive 91/692/EEC of 23 December 1991 standardizing and rationalizing reports on the implementation of certain Directives relating to the environment. OJ L 377: 48-54

EEC (1996) Commission Directive 96/55/EEC of 4 September 1996 adapting to technical progress for the 2nd time Annex I to Council directive 76/769/EEC of 27 July 1976 on the approximation of lows, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations (chlorinated solvents). OJ L 231: 20-21

Health Canada (1996) A one-year survey of halogenated disinfection by-products in the distribution system of treatment plant using three different disinfection processes. Published by authority of the Minister of Health, Canada. ISBN 0-662-25172-5, 59p.

Holbrook, M.T. (1993) Chloroform. IN: Kroschwitz, J.I. & Howe-Grant, (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition .Publ. Wiley-Interscience, N.Y. Volume 5, pp 1051-1062

IARC (1998) Overall evaluation of carcinogenicity: an updating of IARC Monographs Volumes 1 to 42. Chloroform. Supplement 7, p.152

Mills, W.B., Lew, C.S. & Loh, J.Y. (1998) Predictions of potential human health and ecological risks from power plant discharges of total residual chlorine and chloroform into rivers. Environmental Science and Technology 32(14): 2162-2171

Oxenford, J.L. (1996) Disinfection by-products: current practices and future directions. In: Disinfection by-products in water treatment: the chemistry of their formation and control. Minear, R.A. & Amy G.A. [Ed] CRC Press, Inc. ISBN 1-56670-136-8: 3-16

Shepherd, J.L. & Corsi, R.L. (1996) Chloroform in indoor air and wastewater: the role of residential washing machines. Journal of Air and Waste Management Association 46: 631-642

Snyder, R. & Andews, L.S. (1996) Toxic effects of solvents and vapours. In: Casarett and Doull's Toxicology: The Basic Science of Poisons. Fifth Edition. Klaasen, C.D, Amdur, M.O. & Doull, J. [Ed]. Mc-Graw-Hill, ISBN 0-07-105476-6, pp.737-772

USGPO (2000) Title 40- Protection of the Environment. Chapter 1, Part 414- Organic chemicals, plastics and synthetic fibers. Publ: US Government Printing Office, http://www.access.gpo.gov/nara/cfr/waisidx_00/40cfr414_00.html

UNEP (1997) The 1987 Montreal Protocol on Substances that Deplete the Ozone Layer, as adjusted and amended by the second Meeting of the Parties (London, 27-29 June, 1990) and by the fourth Meeting of the parties (Copenhagen, 23-25 November 1992) and further adjusted by the seventh Meeting of the Parties (Vienna, 5-7 December 1995) and further adjusted and amended by the ninth Meeting of the Parties (Montreal, 15-17 September 1997)

Wallace, L.A. (1997) Human exposure and body burden for chloroform and other trihalomethanes. Critical Reviews in Environmental Science and Technology 27(2): 113-194

Weisel, C.P. & Chen, W.J. (1994) Exposure to chlorination by-products from hot water uses. Risk Analysis 14(1): 101-107

Weisel, C.P. & Jo W-K. (1996) Ingestion, inhalation and dermal exposures to chloroform and trichloroethene from tap water. Environmental Health Perspectives 104(1): 48-51

WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

Hexachloro-1,3-butadiene

Hexachloro-1,3-butadiene (HCBD) is a colourless liquid with a turpentine-like odour. This compound is not found naturally in the environment. Hexachlorobutadiene was first prepared in 1877 by the chlorination of hexyl oxide (ATSDR 1997). HCBD either commercially manufactured or is known to be a by-product of the manufacture of chlorinated hydrocarbons such as tetrachloroethene, trichloroethene, and carbon tetrachloride (ATSDR 1997, US EPA, 1986, Johnston *et al.* 1994, Botta *et al.* 1996). It is present in relatively small quantities (up to 5% and sometimes over hexachlorobutadiene in the chlorolysis process of 1,2-dichloroethane for the production of carbon tetrachloride and tetrachloroethene), but, because of the huge production of volatile chlorinated solvents, the amounts of hexachlorobutadiene from the different processes are relevant (Botta *et al.* 1996). It is also reported as a

contaminant in technical formulations of pentachlorophenol, used widely as a wood preservative (Goodrichmahoney *et al.* 1993).

Hexachlorobutadiene is used as a chemical intermediate in the manufacture of rubber compounds (ATSDR 1997). Lesser quantities of hexachlorobutadiene are used as a solvent, a fluid for gyroscopes, a heat transfer liquid, hydraulic fluid, and as a chemical intermediate in the production of chlorofluorocarbons and lubricants. Small quantities are also used as a laboratory reagent. In the international market, Russia is reported to be one of the major users of hexachlorobutadiene, where it is used as a fumigant on grape crops (ATSDR 1997).

Hexachlorobutadiene is a wide spread environmental contaminant. It can exist in the atmosphere as a vapour or adsorbed to airborne particulate matter. HCBD has been found in wastewater from the chlorine industry, leachate from landfills and hazardous waste sites and also in air, soils, surface water and sediments (ATSDR 1997, Santillo & Labounskaia 1997a&b, Choudhary 1995). It has also been detected in fly ash from the incineration of HCBD-containing hazardous wastes (Choudhary 1995).

Hexachlorobutadiene is toxic compound. Acute toxic effects may include the death of animals, birds, or fish, and death or low growth rate in plants. Acute effects are seen two to four days after animals or plants come in contact with it (US EPA 1986, Choudhary 1995). Chronic toxic effects may include shortened lifespan, reproductive problems, lower fertility, and changes in appearance or behaviour. Hexachlorobutadiene has high acute and chronic toxicity to aquatic life (US EPA 1986).

The kidney was found to be an important target organ for HCBD (Jonker *et al.* 1996, Choudhary 1995). If ingested, HCBD concentrates in the kidney, interferes with fundamental processes of cell respiration and can, as a result of conjugation with other compounds in the body, react with DNA resulting in cell death or the development of tumours (Choudhary 1995, ATSDR 1997). Short and longer-term exposure to very low doses via food induced kidney and liver damage in laboratory animals, with juveniles more at risk than adults. It was shown that human exposures to HCBD were associated with highly significant increases in a number of individual and summed bile acid measures in the study of the possible hepatic effects of different chlorinated compounds including HCBD (Driscoll *et al.* 1992)

The International Agency for Research on Cancer concluded that there was limited evidence of HCBD carcinogenicity in rats and classified HCBD as compound not classifiable as to human carcinogenicity (Group 3) (IARC 1999).

References

ATSDR (1997) ARSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

Botta, D. Dancelli, E. & Mantica, E. (1996) A case history of contamination by polychloro-1,3-butadiene. Environmental Science and Technology 30(2): 453-462

Choudhary, G. (1995) Human health perspecives on environmental exposure to hexachlorobutadiene: a review. Environ. Carcino. & Ecotox. Revs. C13(2): 179-203

Driscoll, T.R., Hamdam, H.H., Wang, G., Wright, P.F.A. & Stacey, N.H. (1992) Concentrations of individual serum or plasma bile acids in workers exposed to chlorinated aliphatic hydrocarbons. British Journal of Industrial Medicine 49: 700-705

Goodrichmahoney, J.W., I.P. Murarka, L.J. Hocombe & M.E. Horn (1993). Pentachlorophenol-treated wood poles and crossarms - toxicity characteristic leaching procedure (tclp) results. Environment International 19(6): 535-542.

IARC (1999). 1,1,2,3,4,4-Hexachlorobutadiene. IARC Monographs. International Agency for Research on Cancer. Geneva. Vol. 73, p. 277

Johnston, P.A., R.L. Stringer, R. Clayton & R.J. Swindlehurst (1994). Regulation of toxic chemicals in the North Sea: The need for an adequate control strategy. North Sea Monitor June 1994: 9-16.

Jonker, D., Woutersen, R.A. & Feron, V.J. (1996) Toxicity of mixutes of nephrotoxicants with similar or dissimilar mode of action. Food and Chemical Toxicology 34: 1075-1082

Santillo, D. & Labounskaia, I. (1997a) Petkim: Organic Analytical report 1: Analysis of marine sediments and seawater from locations adjacent to the main wastewater discharges of the Petkim chemical complex, Aliaga, Turkey. Greenpeace Research Laboratories Technical note 08/97

Santillo, D. & Labounskaia, I. (1997b) Petkim: Organic analytical report 2: Analysis of wastes generated by integrated chemical production processes, including the manufacture of VCM and chlorinated organic solvents, at the Petkim chemical complex, Aliaga, Turkey. Greenpeace Research Laboratories Technical Note 09/97

US EPA (1986) Factsheets for regulated chemicals: Hexachlorobutadiene. Office of Environmental Health Hazard Assessment.

Carbon tetrachloride

Carbon tetrachloride is a manufactured compound that does not occur naturally (US EPA 1988). It is a clear, colourless, non-flammable liquid, which is heavier than water, and it is moderately soluble in water. Carbon tetrachloride itself does not burn but poisonous gases are produced in fire, including phosgene and hydrogen chloride. It is used as solvent for oils, fats, lacquers, varnishes, rubber waxes and resins. Carbon tetrachloride was formerly used as a dry cleaning agent and fire extinguisher. Because of its harmful and ozone depleting effects these uses are now banned and it is only used in some industrial applications. Principally it was used in the production of chlorofluorocarbon (CFC) refrigerants (Budavari *et al*. 1986, WHO 1993) but this use of carbon tetrachloride would have stopped in 1996 when CFC-11 and CFC-12 were banned (UNEP 1997).

Carbon tetrachloride is reasonably anticipated to be a human carcinogen by the US Department of Health and Human Services (USDHHS 2000) and has been classified as Group 2B carcinogen (possibly carcinogenic to humans) by International Agency for Research on Cancer (IARC 1999). Carbon tetrachloride induces hepatic cell prolifiration and DNA synthesis. It also has a mutagenic effect and induces aneuploidy in several in-vitro systems (IARC 1999). High exposure to carbon tetrachloride can cause liver, kidney, and central nervous system damage. The liver swells and cells are damaged or destroyed. Kidneys are also damaged, causing a build-up of wastes in the blood. If exposure is low and then stops, the liver and kidneys can repair the damaged cells and function normally again (ATSDR 1997). If exposure is very high, the nervous system, including the brain, is affected. People may feel intoxicated and experience headaches, dizziness, sleepiness, and nausea and vomiting. These effects may subside if exposure is stopped, but in severe cases, coma and even death can occur (ATSDR 1997).

Carbon tetrachloride may enter the environment from industrial effluents, municipal treatment plant discharges, or spills (Menzer *et al*. 1986, US EPA 1988). It has been found in river waters in areas influenced by chlorinated organic solvent plants (Amaral *et al.* 1996). Carbon tetrachloride is relatively stable in the environment. If carbon tetrachloride released to land, it does not sorb onto soil, but migrates readily to ground water and is believed to remain in ground water for month to years (US EPA 1988). Under anaerobic conditions carbon tetrachloride can be biotransformed producing hazardous intermediates such as chloroform and methylene chloride (Hashsham *et al.* 1995) and carbon disulphide under sulphate reducing conditions (Delvin & Muller 1999, Hashsham *et al.* 1995).

Carbon tetrachloride has been detected in drinking water (Abernathy 1994). In the EPA Ground Water Supply Survey on drinking water supplies that used groundwater as a source (Cotruvo *et al.* 1986) carbon tetrachloride was among six most frequently occuring in the samples analysed with the maximum concentration of 16ug/l. In the study carried out in different cities in Galicia, Spain (Freiria-Gandara *et al*. 1992) it was found that concentration of carbon tetrachloride in treated drinking water (if detected) was in the range between 39.5 and 1.5ug/l. In a similar investigation of drinking water in Barcelona, Spain (Amaral *et al*. 1996) the levels of carbon tetrachloride were less than 0.1ug/l.

The maximum level of this compound in drinking water stipulated by the World Health Organization is 2ug/l (WHO 1993). The Environmental Protection Agency (US EPA 1998) has set a limit for carbon tetrachloride of 5ug/l. The EPA recommends that drinking water levels which are “safe” for short-term exposures for a 10kg child consuming 1 litre of water per day: a one-day exposure of 4mg/l; a ten day exposure to 0.2mg/l; and up to 7 days’ exposure to 0.07mg/l (US EPA 1998).

Carbon tetrachloride belongs to the organohalogen compounds whose presence in groundwater are controlled by the European Community Environmental Legislation. Article 3 of EC Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances (EEC 1979) and amended later (EEC 1991) says that Member States shall take necessary steps to prevent the introduction into groundwater of substances in List I and organohalogen compounds are among the groups of the compounds listed there.

The quality objective of 12ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters, and territorial waters) is set for carbon tetrachloride by the EC Council Directive 86/280/EEC (EEC 1986) and amended in 1988 (EEC 1988).

The EC Council Directive 76/769/EEC (EEC 1976) which last was amended in 1996 (EEC 1996) restricts marketing and use of carbon tetrachloride. Carbon tetrachloride may not be used in concentrations equal to or greater than 0.1% by weight in substances and preparation placed on the market for sale to the general public and/or in diffusive applications such as in surface cleaning and cleaning of fabrics.

Carbon tetrachloride is included in Group II of Annex B of controlled substances of the Montreal Protocol (UNEP 1997) as an ozone depleting compound. Article 2D on carbon tetrachloride says that consumption and production of this substance should not exceed zero from 1 January 1996. However,

the developing countries are entitled to delay implementation for ten years in order to meet their basic domestic needs, as specified in the Article 5.

References

Abernathy, C.O. (1994) A retrospective on drinking water. In: Water contamination and health. Wang, R.G.M. [Ed], Marcel Dekker, Inc., New York: 1-14

Amaral, O.C., Otero, R., Grimalt, J.O. & Albaiges, J. (1996) Volatile and semi-volatile organochlorine compounds in tap and riverine waters in the area of influence of a chlorinated organic solvent factory. Water Resources. 30(8): 1876-1884

ATSDR (1997) ARSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

Budavari, S.M., O'Neil, J., Smith A., & Heckleman P.E. [Eds] (1989) The Merck index: an encyclopaedia of chemicals, drugs and biologicals. 11th Edn Merck and Co, Inc., New Jersey, USA

Cotruvo, J., Goldhaber, S. & Vogt, C. (1986) Regulatory significance of organic contamination in the decade of the 1980s. In: Organic carcinogens in drinking water. Ram, N.M., Calabrese, E.J. & Christman, R.F. [Ed]. John Wiley and Sons, New York: 511-530

Delvin, J.F. & Muller, D. (1999) Field and laboratory studies of carbon tetrachloride transformation in sandy aquifer under sulfate reducing conditions. Environmental Science and Technology 33(7): 1021-1027

EEC (1976) Council Directive 76/769/EEC of 27 July 1976 on the approximation of lows, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations. OJ L 262: 201-203

EEC (1979) Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances. OJ L 020: 43-48

EEC (1986) Council Directive 86/280/EEC of 12 June 1986 on limit values and quality objectives for discharges of certain dangerous substances included in List I of the Annex to Directive 76/464/EEC. OJ L 181: 16-27

EEC (1988) Council Directive 88/347/EEC of 16 June 1988 on limit values and quality objectives for discharges of certain dangerous substances included in List I of the Annex to Directive 76/464/EEC. OJ L 158: 35-41

EEC (1991) Council Directive 91/692/EEC of 23 December 1991 standardizing and rationalizing reports on the implementation of certain Directives relating to the environment. OJ L 377: 48-54

EEC (1996) Commission Directive 96/55/EC of 4 September 1996 adapting to technical progress for the 2nd time Annex I to Council Directive 76/769/EEC of 27 July 1976 on the approximation of lows, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations (chlorinated solvents). OJ L 231: 20-21

Freiria-Gandara, M.J., Lorenzo-Ferreira, R.A., Alvarez-Devesa, A. & Bermejo, F. (1992) Occurance of halogenated hydrocarbons in the water supply of different cities of Galicia (Spain). Environmental Technology 13:437-447

Hashsham, S.A., Scholze, R. & freedman, D.L (1995) Cobalamin-enhanced anaerobic biotransformation of carbon tetrachloride. Environmental Science and Technology 29(11): 2856-2863

IARC (1999) Carbon tetrachloride. IARC Monographs Vol.71, p.401

Menzer, R.E. & Nelson, J.O (1986) Water and soil pollutants. In: Casarett and Doull's Toxicology: The Basic Science of Poisons. Third Edition. Klaasen, C.D, Emdur, M.O. & Doll, J. [Ed]. Macmillan Publishing Company. ISBN 0-02-364650-0, pp.825-856

UNEP (1997) The 1987 Montreal Protocol on Substances that Deplete the Ozone Layer, as adjusted and amended by the second Meeting of the Parties (London, 27-29 June, 1990), and by the fourth Meeting of the parties (Copenhagen, 23-25 November 1992), and further adjusted by the seventh Meeting of the Parties (Vienna, 5-7 December 1995), and further adjusted and amended by the ninth Meeting of the Parties (Montreal, 15-17 September 1997)

USDHHS (2000) 9th report on carcinogens. Publ: US Department of Health and Human Services, 334pp.

US EPA (1988) Reviews of environmental contamination and toxicology. Vol. 106, Environmental Protection Agency, Office of Drinking Water Health Advisories, Springer-Verlag New York Inc. ISSN 0179-5953: 21-35

US EPA (1998) National Primary Drinking Water Regulations. Technical Factsheet on Carbon tetrachloride. Environmental Protection Agency, Office of Groundwater and Drinking Water

WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

Trichloroethene

Trichloroethene is a clear, colourless, heavy liquid with a pleasant, sweetish, chloroform-like odour, and a sweet burning taste. It easily evaporates at room temperature. Tetrachloroethene is non-flammable but it can decompose at high temperature in the air producing hydrochloric acid, phosgene and other compounds (CEC 1986). Other names for trichloroethene are trichloroethylene and ethylene trichloride.

Trichloroethene is a chlorinated solvent which was produced since the 1920s in many countries by chlorination of ethylene or acetylene (IARC 1995). Another method of producing trichloroethylene is by direct chlorination of ethylene dichloride to form trichloroethylene and tetrachloroethylene (ATSDR 1997). Oxychlorination of chlorinated wastes from PVC manufacturing (EDC tars) can be used to make chlorinated solvents including trichloroethene, but this method also results in the generation of large quantities of dioxins (EA 1997, ICI 1994). It has been used in vapour degreasing in the 1920s, later it was introduced for use in dry cleaning but its use in this industry has declined sharply since the 1950s. In the 1990s, 80-90% of trichloroethene worldwide was used for degreasing metals (IARC 1995). Use for all applications in Western Europe, Japan and the United States in 1990 was about 225 thousand tonnes. Other uses include processes requiring strong solvent action to dissolve rubbers and resins and in the manufacture of paints, lacquers and adhesives and oils from animal and vegetable matter. The textile industry also uses trichloroethylene as a solvent in waterless dying and finishing operations (ASTDR 1997). It is also used as a chain terminator in the production of polyvinyl chloride, has also been used in fire extinguishing and fire retarding applications (CEC 1986).

Trichloroethene is a photochemically reactive compound and it can decompose in the presence of free radicals. Stabilisers such as epoxides (including the carcinogen epichlorohydrin) or combinations of epoxides, esters and amines are added to commercial trichloroethene to prevent it becoming acidic towards equipment and degreased materials (CEC 1986).

Trichloroethylene is not thought to occur naturally in the environment. However, it is present in most underground water sources and many surface waters as a result of the manufacture, use, and disposal of the chemical (Hughes *et al.* 1994, WHO 1993, ATSDR 1997).

Trichloroethene was found at different levels in drinking water supplies: in Galicia (Spain) in a range of concentrations between 1 and 11.6ug/l (Freiria-Gandara *et al.* 1992), in drinking water samples from Zagreb, Croatia, betwen 0.69 to 35.90 ug/l (ATSDR 1997), and up to 212 ug/l in the drinking water from two villages in Finland (Vartiainen *et al* 1993). It was also detected in the breath of people after inhalation and dermal exposure to tap water contaminated with trichloroethene (Weisel & Jo 1996). The World Health Organisation guideline value for trichloroethene in drinking water is 70ug/l, assuming that this route provides 10% of exposure (WHO 1993). The maximum contaminant level (MCL) for trichloroethylene in drinking water set by US Environmental Protection Agency is 5 ug/l (US EPA 1999). Some surface waters have been found to contain more than 400ug/l of this contaminant (CEC 1986).

Trichloroethylene dissolves slightly in water. Under anaerobic conditions (for example in ground water) trichloroethene may degrade to more toxic compounds including vinyl chloride (Klier *et al.* 1999, Su & Puls 1999, WHO 1993). Trichloroethene itself could be formed as a degradation product of another chlorinated volatile compound – 1,1,2,2-tetrachloroethane (Loran & Olsen 1999).

Trichloroethylene may absorb to particles in water, which will cause it to eventually settle to the bottom sediment (ATSDR 1997). Soil contamination by trichloroethene has been reported with concentration ranging from below 1mg/kg to approximately 1500mg/kg (Ho *et al.* 1999). Trichloroethylene evaporates less easily from the soil than from water and may remain in soil for a long time.

Among the most heavily trichloroethene-exposed people are those working in the degreasing of metals, who are exposed by inhalation. Breathing large amounts of trichloroethylene may cause impaired heart function, coma, and death. Breathing it for long periods may cause nerve, lung, kidney and liver damage. Inhaling small amounts for short periods of time may cause headaches, lung irritation, dizziness, poor co-ordination, and difficulty concentrating. Drinking large amounts of trichloroethylene may cause nausea, liver and kidney damage, convulsions, impaired heart function, coma, or death. Drinking small amounts of trichloroethylene for long periods may cause liver and kidney damage, nervous system effects, impaired immune system function and impaired foetal development in pregnant women, although the extent of some of these effects is not yet clear. Skin contact with trichloroethylene for short periods may cause skin rashes (ATSDR 1997).

Trichloroethene has been classified by International Agency for Research on Cancer in Group 2A (probably carcinogenic to humans) (IARC 1995) and the US authorities reasonably anticipate it to be a human carcinogen (USDHHS 2000). The most important observations are the elevated risk for cancer of the liver and biliary tract and the modestly elevated risk for non-Hodgkin's lymphoma. Trichloroethylene-contaminated groundwater may marginally increase a risk for non-Hodgkin's lymphoma. It has been shown to induce lung and liver tumours in various strains of mice (Fisher & Allen 1993, WHO 1993). It is also a weakly active mutagen in bacteria and yeast (WHO 1993).

Trichloroethene is in the first list of priority substances of the Commission Regulation (EC) No 1179/94 (EC1994) which is a part of the Council Regulation (EEC) No 793/93 on the evaluation and control of the risks of existing substances (EEC 1993).

Discharges of trichloroethene during several industrial processes (including production of tetrachloroethene and trichloroethene) and usage of trichloroethene for degreasing of metals are controlled by the European Community Legislation and special provisions are set relating to trichloroethene in the Council Directive 90/415/EEC (EEC 1990). The quality objective of 10ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters, and territorial waters) is set for trichloroethene in the same Directive.

Article 5(6) of the recent document (EC 1999) concerning limitation of emissions of volatile organic compounds due to the use of organic solvents in certain activities and installations says that substances

or preparations which, because of their content of volatile organic compounds classified as carcinogens, mutagens, or toxic to reproduction, shall be replaced as far as possible by less harmful substances or preparations within the shortest possible time.

References

ATSDR (1997) ATSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

CEC (1986) Trichloroethylene. In: Organochlorine solvents: health risk to workers. Publ: Commission of the European Communities. ISBN 0-85186-078-8: 93-130

EA (1997) Regulation of dioxin releases from the Runcorn operations of ICI and EVC. Information Report. Environmental Agency Licence GD03177G0003. Pub. By Crown. 36pp.

EC (1994) Commission Regulation (EC) No 1179/94 of 25 May 1994 concerning the first list of priority substances as foreseen under Council Regulation (EEC) No 793/93. OJ L 131: 3-4

EC (1999) Council Directive 1999/13/EC of 11 March 1999 on the limitation of emission of volatile organic compounds due to the use of organic solvents in certain activities and installations. OJ L 85: 1-22

EEC (1990) Council Directive of 27 July 1990 amending Annex II to Directive 86/280/EEC on limit values and quality objectives for discharges of certain dangerous substances included in list I of the Annex to Directive 76/464/EEC. OJ L 219:49-57

EEC (1991) Council Directive 91/692/EEC of 23 December 1991 standardizing and rationalizing reports on the implementation of certain Directives relating to the environment. OJ L 377: 48-54

EEC (1993) Council Regulation (EEC) No 793/93 of 23 March 1993 on the evaluation and control of the risks of existing substances. OJ L 84: 1-75

Fisher, J.W. & Allen, B.C. (1993) Evaluating the risk of liver cancer in humans exposed to trichloroethylene using physiological models. Risk Analysis 13(1): 87-95

Freiria-Gandara, M.J., Lorenzo-Ferreira, R.A., Alvarez-Devesa, A. & Bermejo, F. (1992) Occurance of halogenated hydrocarbons in the water supply of different cities of Galicia (Spain). Environmental Technology 13: 437-447

Ho, S.V., Athmer, C., Sheridan, P.W., Hughes, B.M., Orth, R., McKenzie, D., Brodsky, P.H., Shapiro, A.M., Sivavec, T.M., Salvo, J., Schultz, D., Landis, R., Griffith, R. & Shoemaker, S. (1999) The Lasagna technology for *in situ* soil remediation. 2. Large field test. Environmental Science and Technology 33(7): 1092-1099

Hughes, K., Meek, M.E. & Windle, W. (1994) Trichloroethylene: Evaluation of risks to health from environmental exposure in Canada. Environmental Carcinogenesis and Ecotoxicology Reviews. Journal of Environmental Science and Health 12(2): 527-543

IARC (1995) Trichloroethylene. IARC Monographs Vol.63, p.75

ICI (1994) Report of the Chief Inspector HMIP authorisation AK6039 Improvement Condition part 8, table 8.1, item 2: Formation of dioxines in oxychlorination, significance for human health and monitoring proposals. ICI Chemicals and Polymers report NWJP/BMTD, 27th April, 1994, 16pp.

Klier, N.J., West, R.J. & Donberg, P.A. (1999) Aerobic biodegradation of dichloroethylenes in surface and subsurface soils. Chemosphere 38(5): 1175-1188

Loran, M.M. & Olsen, L. (1999) Degradation of 1,1,2,2-tetrachloroethane in a freshwater tidal wetland: field and laboratory evidence. Environmental Science and Technology 33(2): 227-234

Su, C. & Puls, R.W. (1999) Kinetics of trichloroethene reduction by zerovalent iron and tin: pretreatment effect, apparent activation energy, and intermediate products. Environmental Science and Technology 33(1): 163-168

USDHHS (2000) 9th report on carcinogens. Publ: US Department of Health and Human Services, 334pp.

US EPA (1999) Current drinking water standards. National primary and secondary drinking water regulations. Office of Ground Water and Drinking Water. US Environmental Protection Agency

Vartiainen, T., Pukkala, E., Rienoja, T., Strandman, T., Kaksonen, K. (1993) Population exposure to trichloroethene and tetrachloroethene and cancer risk - 2 cases of drinking-water pollution. Chemosphere 27(7): 1171-1181

Weisel, C.P. & Jo, W-K (1996) Ingestin, inhalation, and dermal exposures to chloroform and trichloroethene from tap water. Environmental Health Perspectives 104(1): 48-51

WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

Tetrachloroethene

Tetrachloroethene is a clear liquid, which is heavier than water, with a sweet chloroform-like odour. Tetrachloroethene does not burn but it can produce poisonous gases in fire including hydrogen chloride and phosgene (US EPA 1989). Other names for tetrachloroethene include perchloroethylene and tetrachloroethylene.

Tetrachloroethene was first prepared in 1821 by Faraday by thermal decomposition of hexachloroethane (Hickman 1993). Tetrachloroethene is one of the most important chlorinated solvents worldwide and it has been produced commercially since the early 1900s. About 513 thousand tonnes were used in all applications in Western Europe, Japan and the United States in 1990 (IARC 1995). Tetrachloroethene is typically produced as a co-product with either trichloroethene or carbon tetrachloride from hydrocarbons, partially chlorinated hydrocarbons, and chlorine (Hickman 1993). Oxychlorination of chlorinated wastes from PVC manufacturing (EDC tars) can be used to make chlorinated solvents including tetrachloroethene, but this method also results in the generation of large quantities of dioxins (EA 1997, ICI 1994). Most of the tetrachloroethene produced was used for the dry cleaning garments and smaller amounts were used for degreasing and in the production of chlorofluorocarbons (CFCs) (IARC 1995). However, this latter application will have been reduced since the Montreal Protocol banned CFC-11 and CFC-12 over most of the world (UNEP 1997). Tetrachloroethene was used in the textile industry for processing, finishing and sizing (US EPA 1998). Other uses include: insulating/cooling fluid in electric transformers; in typewriter correction fluids, as veterinary medication against worms, and it was once used as a grain fumigant (US EPA 1998).

Tetrachloroethene is well known environmental contaminant. It has been detected in air, lakes, rainwater, seawater, rivers, soil, food and human tissues (ATSDR 1997, Bauer 1990, CEC 1986). It has also been found in drinking water at concentration in the range from 10 ug/l to 180 ug/l (Bauer 1990, Freiria-Gandara *et al.* 1992, Vartiainen *et al.* 1993, CEC 1986). Contamination of well water with the concentration of 375ug/l was recorded at a waste disposal site due to tetrachloroethene leaching through soil (CEC 1986). The World Health Organisation guideline value for tetrachloroethene in drinking water is 40ug/l assuming that 10% of exposure comes from this source (WHO 1993). The maximum contaminant level (MCL) for tetrachloroethene in drinking water set by US Environmental Protection Agency is 5 ug/L (US EPA 1999). Tetrachloroethene has also been detected in the effluents from industrial plants and refineries and in sewage treatment plant effluents before and after chlorination (Santillo *et al.* 1997, US EPA 1989, CEC 1986).

The majority of the produced tetrachloroethene (80-85%) is lost in the atmosphere as a result of evaporation during production, storage and use (US EPA 1994, CEC 1986) and only 1% is released to water. Releases of tetrachloroethene to the environment are primarily from alkali and chlorine industries (US EPA 1998). In 1992, more than 12.3 million pounds (5584.2 tonnes) were released to the atmosphere, 10 thousand pounds (4.54 tonnes) to surface water, 13 thousand pounds (5.9 tonnes) to

underground injection sites and 9 thousand pounds (4.07 tonnes) to land from U.S. facilities (US EPA 1994). Once released into the environment tetrachloroethene can undergo transformation. The degradation of tetrachloroethene through biotic mechanism includes the formation of lesser chlorinated compounds including trichloroethene, cis- and trans-1,2-dichloroethene, and vinyl chloride (Klier *et al.* 1999). In the air a photochemical degradation occurs with trichloroacethyl chloride as a major degradation product and phosgene a lesser one (CEC 1986).

The major route of human exposure to tetrachloroethene is from inhalation of contaminated urban air, especially near point sources such as dry cleaners, drinking contaminated water from contaminated aquifers (US EPA 1998), drinking water distributed in pipelines with vinyl liners (Webler & Brown 1993), and inhalation of contaminated occupational atmospheres in metal degreasing and dry cleaning industries (US EPA 1998).

Exposure to very high concentrations of tetrachloroethene can cause dizziness, headaches, sleepiness, confusion, nausea, difficulty in speaking and walking, and unconsciousness (ATSDR 1997). Prolonged and frequently repeated dermal exposure can cause irritation, dryness, and dermatitis due to defatting (US EPA 1994). Tetrachloroethene is classified as Group 2A carcinogen (probably carcinogenic to humans) by the International Agency for Research on Cancer (IARC 1995) and reasonably anticipated to be a human carcinogen by the US Department of Health and Human Services (USDHHS 2000). This compound induces leukemia in rats and increases risk for oesophageal cancer, non-Hodgkin's lymphoma and cervical cancer (IARC 1995). Tetrachloroethene has been shown to cause liver tumours in mice and kidney tumours in male rats (ASTDR 1997). Exposure to tetrachloroethene-contaminated drinking water was associated with an increased risk of leukemia and bladder cancer. The risk was dose related (Aschengrau *et al.* 1993).

Specific provisions are set by the European Community Legislation relating to tetrachloroethene in the Council Directive 90/415/EEC (EEC 1990) which controls discharges of tetrachloroethene during several industrial processes (including production of tetrachloroethene, trichloroethene, carbon tetrachloride and chlorofluorocarbons) and usage of tetrachloroethene for degreasing of metals. The quality objective of 10ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters, and territorial waters) is set for tetrachloroethene in the same Directive.

Article 5(6) of the recent document (EC 1999) concerning limitation of emissions of volatile organic compounds due to the use of organic solvents in certain activities and installations says that substances or preparations which, because of their content of volatile organic compounds classified as carcinogens, mutagens, or toxic to reproduction, shall be replaced as far as possible by less harmful substances or preparations within the shortest possible time.

Tetrachloroethene is in the first list of priority substances of the Commission Regulation (EC) No 1179/94 (EC1994) which is a part of the Council Regulation (EEC) No 793/93 on the evaluation and control of the risks of existing substances (EEC 1993).

References

Aschengrau, A., Ozonoff, D., Paulu, C., Coogan, P., Vezina, R., Heeren, T. & Zhang, Y. (1993) Cancer risk and tetrachloroethylene-contaminated drinking water in Massachusetts. Archives of Environmental Health 48(5): 284-292

ATSDR (1997) ATSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

Bauer, U. (1990) Occurrence of tetrachloroethylene in the FRG. In: Organohalogen compounds. Vol.2: Dioxin'90 – EPRI-Seminar. Hutzinger, O. & Fiedler, H. [Ed] published by Ecoinforma Press, Bayreuth, FRG. pp385-388

CEC (1986) Perchloroethylene. In: Organochlorine solvents: health risk to workers. Publ: Commission of the European Communities, ISBN 0-85186-078-8: 191-224

EA (1997) Regulation of dioxin releases from the Runcorn operations of ICI and EVC. Information Report. Environmental Agency Licence GD03177G0003. Pub. By Crown. 36pp.

EC (1994) Commission Regulation (EC) No 1179/94 of 25 May 1994 concerning the first list of priority substances as foreseen under Council Regulation (EEC) No 793/93. OJ L 131: 3-4

EC (1999) Council Directive 1999/13/EC of 11 March 1999 on the limitation of emission of volatile organic comp[ouinds due to the use of organic solvents in certain activities and installations. OJ L 85: 1-22

EEC (1990) Council Directive of 27 July 1990 amending Annex II to Directive 86/280/EEC on limit values and quality objectives for discharges of certain dangerous substances included in list I of the Annex to Directive 76/464/EEC. OJ L 219: 49-57

EEC (1993) Council Regulation (EEC) No 793/93 of 23 March 1993 on the evaluation and control of the risks of existing substances. OJ L 84: 1-75

Freiria-Gandara, M.J., Lorenzo-Ferreira, R.A., Alvarez-Devesa, A. & Bermejo, F. (1992) Occurrence of halogenated hydrocarbons in the water supply of different cities of Galicia (Spain). Environmental Technology, 13: 437-447

Hickman, J.C. (1993) Tetrachloroehylene. Chlorocarbons, -hydrocarbons ($CHCl=CCl_2$). In: Kirk-Othmer Encyclopedia of Chemical Technology. [Eds] Jacqueline I. Kroschwits; Mary Howe-Grant. Fourth edition, Vol. 6. John Wiley & Sons, Inc. pp.50-59

IARC (1995) Tetrachloroethylene. IARC Monographs Vol.63, p.159

ICI (1994) Report of the Chief Inspector HMIP authorisation AK6039 Improvement Condition part 8, table 8.1, item 2: Formation of dioxines in oxychlorination, significance for human health and monitoring proposals. ICI Chemicals and Polymers report NWJP/BMTD, 27th April, 1994, 16pp.

Klier, N.J., West, R.J. & Donberg, P.A. (1999) Aerobic biodegradation of dichloroethylenes in surface and subsurface soils. Chemosphere 38(5): 1175-1188

Santillo, D., Labounskaia, I., Stringer, R.L. & Johnston, P. A. (1997) Report on the analysis of industrial wastewaters from the Frutarom VCM/PVC plant near Haifa, Israel and adjacent shoreline sediments for organic contaminants. Greenpeace Research Laboratories Technical Note 03/97

UNEP (1997) The 1987 Montreal Protocol on Substances that Deplete the Ozone Layer, as adjusted and amended by the second Meeting of the Parties (London, 27-29 June, 1990), and by the fourth Meeting of the parties (Copenhagen, 23-25 November 1992), and further adjusted by the seventh Meeting of the Parties (Vienna, 5-7 December 1995), and further adjusted and amended by the ninth Meeting of the Parties (Montreal, 15-17 September 1997)

USDHHS (2000) 9th report on carcinogens. Publ: US Department of Health and Human Services, 334pp.

US EPA (1989) Tetrachloroethylene. US Environmental Protection Agency factsheets for regulated chemicals. Aquire Database, ERL-Duluth, US Environmental Protection Agency

US EPA (1994) Chemical summary for perchloroethylene. Office of Pollution Prevention and Toxics. US Environmental Agency

US EPA (1998) Technical factsheet on: Tetrachloroethylene. National primary drinking water regulations. Drinking water and health. Office of Ground Water and Drinking Water, US Environmental Protection Agency

US EPA (1999) Current drinking water standards. National primary and secondary drinking water regulations. Office of Ground Water and Drinking Water. US Environmental Protection Agency

Vartiainen, T., Pukkala, E., Rienoja, T., Strandman, T., Kaksonen, K. (1993) Population exposure to trichloroethene and tetrachloroethene and cancer risk - 2 cases of drinking-water pollution. Chemosphere 27(7): 1171-1181

Webler, T. & Brown, H.S. (1993) Exposure to tetrachloroethylene via contaminated drinking pipes in Massachusetts: a predictive model. Archives of Environmental Health 48(5): 293-297
WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

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, at 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) had 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 Comission on the Great Lakes (IJC) has called for all uses to be eliminated.

References

ASTDR (1997) ATSDR's toxicological profiles on CD-ROM. U.S. Department of Health and Human Services, Public Health Service, CRC Press Inc

Bahnick, D.A. & Doucette, W.J. (1988) Use of molecular connectivity indices to estimate soil sorption coefficients for organic chemicals. Chemosphere 17: 1703-1715 .

Brauner, C.J., Randall, D.J., Neuman, J.F. & Thurston, R.V. (1994) The effect of exposure to 1,2,4,5-tetrachlorobenzene and the relationship between toxicant and oxygen uptake in rainbow trout (*Oncorhynchus mykiss)* during exercise. Environ. Toxic. Chem. 13(11): 1813-1820

Bryant, J.G. (1993) Chlorinated benzenes. IN: Kroschwitz, J.I. & Howe-Grant, (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition .Publ. Wiley-Interscience, N.Y. Volume 6: 87-100

Budavari, S.M., O'Neil, J., Smith A. & Heckleman P.E. [Eds] (1989) The Merck index: an encyclopaedia of chemicals, drugs and biologicals. 11th Edn, Merck and Co, Inc., New Jersey, USA

CEC (1986) p-dichlorobenzene. IN: Organo-chlorine solvents: Health risks to workers. Publ: Commission of the European Communities, ISBN 0-85186-078-8, pp1-16

EEC (1976) Council Directive 76/464/EEC of 4 May 1976 on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. OJ L 129: 23-29 as amended.

EEC (1986) Council Directive 86/280/EEC of 12 June 1986 on limit values and quality objectives for discharges of certain dangerous substances included in List 1 of the Annex to Directive 76/464/EEC.

Giddings, M., Meek, M.E. & Gomes, R. (1994a) Trichlorobenzenes: Evaluation of risks to health from environment exposure in Canada. Environ. Carcin. Ecotox. Revs. C12(2): 517-525

Giddings, M., Meek, M.E. & Gomes, R. (1994b) Tetrachlorobenzenes: Evaluation of risks to health from environment exposure in Canada. Environ. Carcin. Ecotox. Revs. C12(2): 473-481

Giddings, M., Meek, M.E. & Gomes, R. (1994c) Pentachlorobenzene: Evaluation of risks to health from environment exposure in Canada. Environ. Carcin. Ecotox. Revs. C12(2): 435-441

Grimalt, J.O., Sunyer, J., Moreno, V., Amaral, O.C., Sala, M., Rosell, A., Anto, J.M. & Albaiges, J. (1994) Risk excess of soft-tissue sarcoma and thyroid cancer in a community exposed to airborne organochlorinated compounds with a high hexachlorobenzene content. International Journal of Cancer 56: 200-203

Harper, D.J., Ridgeway, I.M. & Leatherland, T.M. (1992) Concentrations of hexachlorobenzene, trichlorobenzenes and chloroform in the waters of the Forth estuary, Scotland. Mar. Poll. Bull. 24(5): 244-24

Hermanson, M.H., Monosmith, C.L. & Donnelly-Kelleher, M.T. (1997) Seasonal and spatial trends of certain chlorobenzene isomers in the Michigan atmosphere. Atmos. Environ. 31(4): 567-573

Johnston, P.A., Stringer, R.L., Clayton, R. & Swindlehurst, R.L. (1993) Regulation of toxic chemicals in the North Sea: the need for an adequate control strategy. Proceedings of the Scientific Symposium on the North Sea Quality Status Report 1993, 18-21 April 1994, Ebeltoft, Denmark: 269-274

Meek, M.E., Giddings, M. & Gomes, R. (1994a) Monochlorobenzene: evaluation of risks to health from environmental exposure in Canada. Environ. Carcin. Ecotox. Revs. 12(2): 409-415

Meek, M.E., Giddings, M. & Gomes, R. (1994b) 1,2-Dichlorobenzene: evaluation of risks to health from environmental exposure in Canada. Environ. Carcin. Ecotox. Revs. 12(2): 269-275

MINDEC (1990) Final Declaration of the Third Ministerial Conference on the Protection of the North Sea. Ministry of Transport Conference on the Protection of the North Sea. Ministry of Transport and Public Works, The Haugue.

MINDEC (1995) Ministerial Declaration of the Fourth International Conference on the Protection of the North Sea. 8-9 June 1995, Esjberg, Denmark.

Newhook, R. & Meek, M.E. (1994) Hexachlorobenzene: Evaluation of risks to health from Environmental Exposure in Canada. Environ. Carcin. Ecotox. Revs. C12(2): 345-360

OSPAR (1998a) OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Sintra Statement, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998.

OSPAR (1998b) OSPAR Convention for the Protection of the Marine Environment of the North East Atlantic. OSPAR Strategy with regard to Hazardous Substances, Reference no. 1998-16, OSPAR 1998/14/1/Annex 34, Ministerial Meeting of the OSPAR Commission, Sintra b22-23 July 1998.

Sala, M., Sunyer, O., Otero, R., Santiago-Silva, M., Ozalla, D., Herrero, C., To-Figueras, J., Kogevinas, M., Anto, J., Camps, C. & Grimalt, J. (1999) Health effects of chronic high exposure to hexachlorobenzene in a general population sample. Arch. Environ. Health 54(2): 102-109

Sicko-Goad, L. & Andersen, N.A. (1993a) Effect of lipid composition on the toxicity of trichlorobenzene isomers to diatoms. I. Short-term effects of 1,3,5-trichlorobenzene. Arch. Environ. Contam. Toxicol. 24: 236-242

Sicko-Goad, L. & Andersen, N.A. (1993b) Effect of lipid composition on the toxicity of trichlorobenzene isomers to diatoms. II. Long-term effects of 1,2,3-trichlorobenzene. Arch. Environ. Contam. Toxicol. 24: 243-248

Sicko-Goad, L., Evans, M.S., Lazinsky, D., Hall, J. & Simmons, M.S. (1989d) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. IV. Pentachlorobenzene and comparison with trichlorobenzene isomers. Arch. Environ. Contam. Toxicol. 18: 656-668

Sicko-Goad, L., Hall, J., Lazinsky, D. & Simmons, M.S. (1989b) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. II. 1,3,5-Trichlorobenzene. Arch. Environ. Contam. Toxicol. 18: 638-646

Sicko-Goad, L., Hall, J., Lazinsky, D. & Simmons, M.S. (1989c) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. III. 1,2,3-Trichlorobenzene. Arch. Environ. Contam. Toxicol. 18: 647-655

Sicko-Goad, L., Lazinsky, D., Hall, J. & Simmons, M.S. (1989a) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. I. 1,2,4-Trichlorobenzene. Arch. Environ. Contam. Toxicol. 18: 629-637

UNECE (1998) Protocol to the 1979 Convention on long-range transboundary air pollution on persistent organic pollutants. Publ UNECE, 61pp.

UNEP (1995) Decision 18/32 of the UNEP Governing Council: Persistent Organic Pollutants. UNEP Governing Council, 25th May 1995.

UNEP (1997) Decisions adopted by the Governing Council at its nineteenth session: 13c. International action to protect human health and the environment through measures which will reduce and/or eliminate emissions and discharges of persistent organic pollutatns, including the development of an international legally binding instrument. UNEP Governing Council, 7th February 1997.

US EPA (1989). EPA Factsheets for Regulated Chemicals. Hexachlorobenzene. Washington, DC.

Van Birgelen, A.P.J.M. (1998) Hexachlorobenzene as a possible major contributor to the dioxin activity of human milk. Environ. Health Persp. 106(11): 683-688

Ware, G.W. (Ed.)(1988a) Chlorobenzene. Rev. Environ. Contam.Toxicol. 106: 37-49

Ware, G.W. (Ed.)(1988b) Ortho-, meta- and para- dichlorobenzene. Rev. Environ. Contam.Toxicol. 106: 51-56

APPENDIX 3 TOXICOLOGICAL OUTLINES FOR HEAVY METALS

Mercury (Hg)

Mercury is a naturally occurring metal, which can exist in several forms. Metallic mercury is a shiny, silver-white, odorless liquid, which forms a colorless, odorless gas if heated. Mercury combines with other elements such as chlorine, sulfur, or oxygen to form inorganic mercury compounds or salts, which are usually white solids. Mercury also combines with carbon to make organic mercury compounds, the most common of which is methylmercury.

Mercury is a very rare metal, found in the earth's crust at concentrations frequently below 0.03 mg/kg (Alloway 1990). It is estimated that the total amount of mercury released to the atmosphere from natural sources is of the order of 2700-6000 tonnes/year, mainly from volcanic activity and biogenic processes. This compares with an estimated anthropogenic load of 3000 tonnes/year (WHO 1991). The major sources of air emissions of inorganic mercury (metallic mercury and inorganic mercury compounds) are from mining ore deposits, burning coal and waste and from manufacturing plants.

Uses of mercury are extensive, due to its unique properties of fluidity, its high surface tension and its ability to alloy with other metals. It is primarily used in the electrical industry in alkaline batteries, electric lamps and wiring and switching devices, such as thermostats and cathode tubes. It is also used in the chemical industry as a catalyst; for example to form compounds such as vinyl chloride (VCM) via the acetylene process (Matthews 1996), and as the cathode in the chlor-alkali electrolytic separation of brine to produce chlorine and sodium hydroxide (caustic soda). At present, there is an estimated 12 000 tonnes of pure mercury in use in mercury cells within the EU alone.

Metallic mercury is used in dental restorations, and in medical equipment, such as thermometers and manometers (ATSDR 1997).

Mercuric oxide and mercuric sulphide are used as pigments in paints; and gold mining operations utilise mercury to extract gold from ores through amalgamation. Until 30 years ago, mercury compounds were used extensively as pharmaceuticals and agrochemicals, e.g. as components of antiseptics, diuretics, skin lightening creams, laxatives, anti-syphilitic drugs, fungicides, bactericides, wood and felt preservatives. However, due to the high toxicity of mercury, most of these applications are banned in most parts of the World (ATSDR 1997).

Mercury is found at very low concentrations in many aquatic and terrestrial ecosystems (see Table 1). Due to the fact that mercury is the only metal that can exist as both a liquid and a vapour at ambient temperatures, its environmental behaviour differs from that of most other toxic elements (ATSDR 1997, WHO 1989). Mercury can exist in three valence states, Hg (0) metallic, Hg (I) mercurous and Hg (II) mercuric. In the atmosphere, elemental mercury is by far the most common form, and, as a vapour, it is responsible for the long-range, global cycling of mercury. In addition, though to a far lesser degree,

mercury may be associated with particulates, which can be moved by dry or wet deposition (ATSDR 1997, WHO 1991).

Environmental Matrix	Concentration	Reference
Seawater (open ocean)	0.001-0.004 ug/l 0.02 ug/l	Bryan & Langston 1992, WHO 1989 ATSDR 1997
Freshwater	<0.005 ug/l	ATSDR 1997
Marine sediment	0.02-0.1mg/kg	WHO 1989
Freshwater sediment	0.2-0.35 mg/kg	Salomons & Forstner 1984
Soil	0.02-0.625 mg/kg	Alloway 1990, WHO 1989
Fish	<0.2 mg/kg	ATSDR 1997

Table 1. Background concentrations of mercury found in water, sediments, soil and fish.

In the aquatic environment, mercury is most commonly found in the mercuric (II) state. Once released, its fate is dominated by rapid adsorption to soluble and particulate organic material; followed by flocculation, precipitation and final accumulation in the bottom sediment. Because of the strength with which mercury is bound to sediment, exchange back to the water column is generally slight, although it can be accelerated in saline waters, and in the presence of high concentrations of sulphide (anoxic conditions) (ATSDR 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 converted to an organic form by micro-organisms indigenous to soils, fresh water and marine sediments. The most common form of organic mercury is methylmercury (MeHg), which is soluble, mobile and quick to enter the aquatic food chain. Increased levels of mercury in the environment can increase the amount of methylmercury that these bacteria produce. 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 and Langston 1992). As a result, MeHg provides one of the rare examples of metal biomagnification in food chains (ATSDR 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 (ATSDR 197). The significance of this bioaccumulation is that it is generally the most important source of human, non-occupational mercury exposure (ATSDR 1997, WHO 1989).

Exposure to mercury generally occurs from breathing contaminated air, ingesting contaminated water and food and having dental and medical treatments.

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 inhalation of high levels of mercury vapour may cause nausea, vomiting, diarrhoea, increases in blood pressure or heart rate, skin rashes, eye irritation, corrosive bronchitis and pneumonitis, and may be associated with central nervous system (CNS) effects such as tremor or increased excitability (ATSDR 1997, Goyer 1996). With chronic exposure, the major effects are on the CNS (tremor, spasms, loss of memory, increased excitability, severe depression, personality changes, even delirium and hallucination), although renal damage, associated with chronically exposed workers, has also been shown (Ratcliffe *et al*. 1996, Goyer 1996). These effects have also been reported in animal studies (ATSDR 1997).

Acute exposure to high levels of mercury salts, or chronic low-dose exposure, is directly toxic to the kidney (Zalups & 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 (ATSDR 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 (ATSDR 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). 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).

There are inadequate human cancer data available for all forms of mercury. Mercuric chloride has caused increases in several types of tumors in rats and mice, while methylmercury increased kidney tumors in male mice. The USEPA has determined that mercuric chloride and methyl mercury are possible human carcinogens (ATSDR 1997)

Studies on the aquatic toxicity of mercury are numerous, and again show that MeHg is more toxic than any of the inorganic forms. (WHO 1989)

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 mercury 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 elevated levels, including prevention of oocyte 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). 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 (Bryan and Langston 1992).

Council Directive 80/778/EEC relating to the quality of water intended for human consumption. A maximum permissible concentration of 1 ug/l is set (EEC 1980). The US Environment Protection Agency (USEPA) and Food and Drug Administration (FDA) set a maximum permissible level for drinking water of 2 ug/l (ATSDR 1997). The Food and Drug Administration (FDA) has set a maximum permissible level of 1 ppm of methylmercury in seafood (ATSDR 1997).

Relating to mercury emissions, the international body which controls the pollution of the North-east Atlantic has recommended that existing mercury cell chlor-alkali plants be phased out as soon as practicable, and that they should be phased out completely by 2010 (PARCOM 1990). This has been reinforced in the Sintra Statement at the 1998 Ministerial Meeting of the OSPAR Commission (OSPAR 1998a) in which mercury has been selected for priority action, and included in Annex 2 of the OSPAR Strategy with regard to Hazardous Substances (OSPAR 1998b). This agreement states that environmental concentrations of naturally occurring hazardous substances should be reduced to near background levels within the next 25 years.

References

Akagi, H., Malm, O., Kinjo, Y., Harada, M., Branches, F.J.P, Pfeiffer, W.C. & Kato, H. (1995) Methylmercury pollution in the Amazon, Brazil. The Science of the Total environment 175: 85-95

Alloway, B.J. (1990) Heavy metals in soils. John Wiley and Sons, Inc. New York, ISBN 0470215984

ATSDR (1997) Toxicological profile for Mercury on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

Barbosa, A.C. (1997) Mercury in Brazil: present or future risks? Journal of the Brazilian Association for the Advancement of Science 49,1/2: 111-116

Bryan, G.W. & Langston, W.J. (1992) Bioavailability, accumulation and effects of heavy metals in sediments with special reference to United Kingdom estuaries: a review. Environmental Pollution 76: 89-131

D'Itri, F.M. (1991) Mercury contamination: what we have learned since Minamata. Environmental Monitoring and Assessment 19: 165-182

EC (1999) Integrated Pollution Prevention and Control (IPPC) Draft best available techniques reference document on the chlor-akali industry. February 1999, 160pp

EEC (1980) Council Directive 80/778/EEC of 15 July 1980 relating to the quality of water intended for human consumption OJ L 30.8.1980, 229:11

Goyer, R.A. (1996) Toxic effects of metals. In Casarett & Doull's Toxicology. The Basic Science of Poisons, Fifth Edition, Klaassen, C.D. [Ed]. McGraw-Hill Health Professions Division, ISBN 0071054766

Harada, M. (1997) Neurotoxicity of methylmercury; Minamata and the Amazon. In Mineral and Metal Neurotoxicology. Yasui, M., Strong, M.J., Ota, K. and Verity, M.A.[Eds]. CRC Press Inc., ISBN 0849376645

Matthews, G. (1996) PVC: Production, properties and uses. Publ: The Institute of Materials, London

OSPAR (1998a) OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Sintra Statement, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998

OSPAR (1998b) OSPAR Convention for the Protection of the Marine Environment of the North East Atlantic. OSPAR Strategy with regard to Hazardous Substances, Reference no. 1998-16, OSPAR 1998/14/1/Annex 34, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998

PARCOM (1990) PARCOM decision 90/3 of 14 June 1990 on reducing atmospheric emissions from existing chlor-alkali plants

Ratcliffe, H.E., Swanson, G.M. & Fischer, L.J. (1996) Human exposure to mercury: a critical assessment of the evidence of adverse health effects. Journal of Toxicology and Environmental Health 49: 221-270

Salomons, W. & Forstner, U. (1984) Metals in the hydrocycle. Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, ISBN 3540127550

World Health Organisation (1989) Mercury. Environmental Health Criteria 86. ISBN9241542861

World Health Organisation (1991) Inorganic mercury. Environment Health Criteria 118. ISBN9241571187

World Health Organisation (1993) Guidelines for drinking water quality. Volume 1: Recommendations. ISBN 9241544600

Zalups, R.K. & Lash, L.H. (1994) Advances in understanding the renal transport and toxicity of mercury. Journal of Toxicology and Environmental Health 42: 1-44

Lead (Pb)

Lead is one of the most ubiquitous toxic metals. It is chiefly obtained from the sulphide ore galena, by a roasting process; and is currently mined in 47 countries, making it one of the most widespread metals in terms of primary production. In addition to this, secondary smelters, processing lead metal products, are located in 43 countries, reflecting widespread recycling of lead in electric storage batteries (Dudka & Adriano 1997).

Natural sources of atmospheric lead include wind-borne soil particles and volcanic emissions, forest fires, biogenic processes and sea salt sprays. The total amount of lead released to the atmosphere from natural sources is estimated at 12,000 tonnes / year, compared with an estimated anthropogenic load of 332,000 tonnes /year (Nriagu 1990).

Uses of lead and its compounds are extensive. As a metal, it is often used as pipe-work for water distribution, or as containers for storing corrosive liquids (e.g. sulphuric acid). Its alloys are used in welding, printing and as anti-friction metals; and great quantities, both of the metal and its dioxide, are used in electric storage batteries. Other uses include cable coverings, ammunition and in the manufacture of tetraethyl lead, used as an anti-knock compound in petrol. Compounds of lead are used as paint pigments, PVC stabilisers, pesticides, varnishes, lubricants, as glazes for pottery and porcelain and in leaded glass crystal (Budavari *et al.* 1989, ATSDR 1997).

Lead is present in uncontaminated aquatic and terrestrial ecosystems at relatively low levels. However, as anthropogenic emissions far exceed those from natural sources, elevations above these natural, background concentrations are often found. Lead tends to accumulate in soils and sediments, where it can remain accessible to the food chain and to human metabolism for many years (Sauve *et al.* 1997, ATSDR 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).

Plants and animals can accumulate lead from water, soil and sediment, with organic forms being more easily absorbed than inorganic. In general, the highest lead concentrations are found in aquatic and terrestrial organisms that live near to lead mining, smelting and refining facilities; storage battery recycling plants; areas affected by high automobile and truck traffic; sewage sludge and spoil disposal areas; sites where dredging has occurred; areas of heavy hunting (spent lead shot); and in urban and industrialised areas (ATSDR 1997).

Lead has no known, nutrition, biochemical or physiological function (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. Health impacts 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, ATSDR 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 10 ug/dl (for both adults and children) (ATSDR 1997). However, it has been suggested that there may be no level of blood-lead that does not produce a toxic effect, particularly in the developing central nervous system (ATSDR 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, ATSDR 1997, Collivignarelli *et al.* 1986). In addition, birds feeding on contaminated prey or ingesting lead shot into their gizzards can be exposed to severe levels of lead. This can result in high kidney, liver and bone concentrations, reduced growth and development, behavioural abnormalities and sometimes death (Mateo *et al.* 1997, WHO 1989).

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).

European legislation concerned with water quality and permissible environmental levels does not generally treat lead as a priority pollutant. However, anthropogenic discharges of lead into the aquatic environment still need to cease and therefore lead is included on the majority of subsidiary and secondary lists.

Council Directive 80/778/EEC relating to the quality of water intended for human consumption set a maximum permissible concentration of 50 ug/l. Other drinking water legislation includes that set by the Bureau of Indian Standards (1995), which currently set a maximum permissible concentration for lead of 50 ug/l; the USEPA, which limits the concentration of lead to 15 ug/l (ATSDR 1997); and the WHO, which currently recommends a limit of 10 ug/l (WHO 1993).

The Water Research Centre in the UK recommends the following Environmental Quality Standards for lead: protection of freshwater salmonid fish 4-20 ug/l; protection of freshwater coarse fish 50-250 ug/l; protection of other freshwater life and associated non-aquatic organisms 5-60 ug/l; protection of saltwater fish, shellfish, other salt water life and associated non-aquatic organisms 25 ug/l (Brown *et al.* 1984).

Regarding soil contamination, the UK Department of the Environment (ICRCL) regards a level of 0-500 mg/kg as being typical of uncontamination. Anything above this is regarded as being contaminated and as such, restrictions on recreational and agricultural uses apply (Alloway 1990). In terms of permissible sewage sludge levels, acceptable concentrations for lead range from 300-1200 mg/kg. However resulting soil concentrations should not exceed 50-100 mg/kg (recommended EC, UK, France, Germany) (Alloway 1990).

Finally, lead is included in the list of priority hazardous substances agreed by the Third North Sea Conference (MINDEC 1990), Annex 1A to the Hague Declaration and confirmed at the Fourth Conference in Esjberg, Denmark, in 1995 (MINDEC 1995). Here it was agreed that environmental concentrations of hazardous substances should be reduced to near background level within the next 25 years. An objective further reinforced in the Sintra Statement at the 1998 Ministerial Meeting of the OSPAR Commission (OSPAR 1998a). Lead and organic lead compounds were selected for priority action and consequently is included in Annex 2 of the OSPAR Strategy with regard to Hazardous Substances (OSPAR 1998b).

Denmark has imposed a wide ranging ban on lead. Apart from a limited number of exceptions, such as second-hand products which met Danish standards when they were first sold, the import and marketing of products containing lead was banned on the 1st March 2001 (Danish EPA 2000).

References

Alloway, B.J. (1990) Heavy metals in soils. John Wiley & Sons, Inc. New York, ISBN 0470215984

ATSDR (1997) Toxicological profile for lead on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

Bernard, A.M., Vyskocil, A., Kriz, J., Kodl, M. & Lauwerys, R. (1995) Renal effects of children living in the vicinity of a lead smelter. Environmental Research 68: 91-95

Blus, L.J., Henny, C.J., Hoffman, D.J. & Grove, R.A. (1991) Lead toxicosis in tundra swans near a mining and smelting complex in Northern Idaho. Arch. Environ. Contam. Toxicol. 21: 549-555

Brown, V.M, Gardiner, J. & Yates, J. (1984) Proposed environmental quality standards for List II substances in water. Inorganic lead. Water Research Centre Technical Report TR208

Bryan, G.W. & Langston, W.J. (1992) Bioavailability, accumulation and effects of heavy metals in sediments with special reference to United Kingdom estuaries: a review. Environmental Pollution 76: 89-131

Budavari, S.M., O'Neil, J., Smith, A. & Heckleman, P.E. [Eds] (1989) The Merck Index: an encyclopaedia of chemicals, drugs and biologicals. 11TH Edn Merck & Co, Inc., New Jersey, USA

Bureau of Indian Standards (1995) Drinking water specifications. Second Edition, UDC 628.1.033

Collivignarelli, C., Riganti, V. & Urbini, G. (1986) Battery lead recycling and environmental pollution hazards. Conservation and Recycling 9(1): 111-125

Danish EPA (2000) Statutory order on prohibition of import and marketing of products containing lead.

Dudka, S. & Adriano, D.C. (1997) Environmental impacts of metal ore mining and processing: a review. J. Environ. Qual. 26: 590-602

EEC (1980) Council Directive 80/778/EEC of 15 July 1980 relating to the quality of water intended for human consumption (OL J 229, 30.8.1980, p. 11)

Forsyth, D.S., Dabeka, R.W. & Cleroux, C. (1991) Organic and total lead in selected fresh and canned seafood products. Food Additives and Contaminants 8, 4: 477-484

Goyer, R.A. (1993) Lead toxicity: current concerns. Environmental Health Perspectives 100: 177-187

Goyer, R.A. (1996) Toxic effects of metals. In Casarett & Doull's Toxicology. The Basic Science of Poisons, Fifth Edition, Klaassen, C.D. [Ed]. McGraw-Hill Health Professions Division, ISBN 0071054766

Henny, C.J., Blus, L.J., Hoffman, D.J., Grove, R.A. & Hatfield, J.S. (1991) Lead accumulation and osprey production near a mining site on the Coeur d'Alene River, Idaho. Arch. Environ. Contam. Toxicol. 21: 415-424

Mateo, R., Martinez-Vilalta, A. & Guitart, R. (1997) Lead shot pellets in the Ebro Delta, Spain: densities in sediments and prevalence of exposure in waterfowl. Environmental Pollution 96, 3: 335-341

MINDEC (1990) Final Declaration of the Third Ministerial Conference on the Protection of the North Sea. Ministry of Transport Conference on the Protection of the North Sea. Ministry of Transport and Public Works, The Hague.

MINDEC (1995) Ministerial Declaration of the Fourth International Conference on the Protection of the North Sea. 8-9 June 1995, Esjberg, Denmark.

Nriagu, J.O. (1988) A silent epidemic of environmental metal poisoning. Environmental Pollution 50: 139-161

Nriagu, J.O. (1990) Global metal pollution. Poisoning the biosphere? Environment 32, 7: 7-33

OSPAR (1998a) OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Sintra Statement, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998.

OSPAR (1998b) OSPAR Convention for the Protection of the Marine Environment of the North East Atlantic. OSPAR Strategy with regard to Hazardous Substances, Reference no. 1998-16, OSPAR 1998/14/1/Annex 34, Ministerial Meeting of the OSPAR Commission, Sintra b22-23 July 1998.

Pirkle, J.L., Kaufman, R.B., Brody, D.J., Hickman, T., Gunter, E.W. & Paschal, D.C. (1998) Exposure of the U.S. population to lead, 1991-1994. Environmental Health Perspectives 106, 11: 745-750

Sadiq, M. (1992) Toxic metal chemistry in marine environments. Marcel Dekker Inc., New York, Basel, Hong Kong. ISBN 0824786475

Sauve, S., McBride, M.B. & Hendershot, W.H. (1997) Speciation of lead in contaminated soils. Environmental Pollution 98, 2: 149-155

WHO (1989) Lead- environmental aspects. Environmental Health Criteria 85. ISBN 9241542853

WHO (1993) Guidelines for drinking-water quality. Volume 1: Recommendations. ISBN 9241544600, 188pp.

Copper (Cu)

Abundance of copper in the Earth's crust is reported as ranging from 24-55 mg/kg (Alloway 1990), although higher levels are associated with some shales and clays (Thornton 1995). Copper can occur in the elemental state, however it is found more commonly as a sulphide (copper pyrite), oxide (cuprite) or carbonate (malachite). The largest deposits of copper are found in the USA, Chile, Canada, the Commonwealth of Independent States, Zambia and Peru (Dudka & Adriano 1997, Alloway 1990).

Volcanic activity is the major source of copper released to the atmosphere, followed by emissions from wind-borne soil particles, forest fires, sea salt spray and biogenic processes. It is estimated that the total amount of copper released to the atmosphere from natural sources is 28,000 tonnes / year, compared with an estimated anthropogenic load of 35,000 tonnes / year (Nriagu 1990).

After the copper ore has been mined, crushed, enriched and concentrated, it is roasted at temperatures in excess of 1200°C, sintered and smelted. Alternatively copper can be recovered from secondary sources (i.e. scrap). This process is far less energy intensive and therefore is playing an increasingly important role in terms of global copper production (UNEP 1993).

Copper is a highly malleable and ductile metal, as well as being an excellent conductor of heat and electricity. Its principal use is as an electrical conductor (copper cables and wires), however it is also widely employed in coinage alloys, in traditional alloys such as bronze (copper and tin), brass (copper and zinc) and Monel (copper and nickel), in corrosive-resistant and decorative plating, in munitions and in dental alloys. Its compounds are used as chemical catalysts, wood preservatives, algicides, fungicides, anti-fouling paints, disinfectants, nutritional supplements in fertilisers and feeds, in petroleum refining and as printing inks and dyes, (ATSDR 1997, UNEP 1993).

Copper is a relatively abundant "trace" metal, found at varying concentrations in nearly all uncontaminated aquatic and terrestrial ecosystems. However, as anthropogenic emissions of copper exceed those from natural sources, elevations above these natural, background concentrations are often found.

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 (ATSDR 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 (ATSDR 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 (ATSDR 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 (ATSDR 1997).

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 (ATSDR 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 (ATSDR 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. Resulting in liver and kidney damage and haemolytic anaemia (ATSDR 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 (ATSDR 1997).

Copper is not a priority pollutant but is included on the majority of subsidiary and secondary pollutant lists. Examples of guidelines and permissible environmental levels include the following:

European Council Directive 75/440/EEC concerning the quality required of surface water intended for the abstraction of drinking water in the Member States. Water containing more that 50 ug/l must be subjected to physical and chemical treatment prior to use (EEC 1975).

European Council Directive 78/659/EEC on the quality of fresh waters needing protection or improvement in order to support fish life. An Environmental Quality Standard of 40 ug/l is set (EEC 1978).

European Council Directive 80/778/EEC relating to the quality of water intended for human consumption. Guide levels of 100 ug/l (for outlets of pumping and/or treatment works) and 3000 ug/l (after water has been standing for 12 hours in the piping/made available to the customer) are set (EEC

1980). Other drinking water standards include those set by the Bureau of Indian Standards (1995) (50ug/l), the USEPA (1300 ug/l) (ATSDR 1997) and the WHO (2000 ug/l)(1993).

References

Alloway, B.J. (1990) Heavy metals in soils. John Wiley & Sons, Inc. New York, ISBN 0470215984

ATSDR (1997) Toxicological profile for copper on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

Bureau of Indian Standards (1995) Drinking water specifications. Second Edition, UDC 628.1.033

Dudka, S. & Adriano, D.C. (1997) Environmental impacts of metal ore mining and Processing: a review. J. Environ. Qual. 26: 590-602

EEC (1975) Council Directive 75/440/EEC of 16 June 1979 concerning the quality required of surface water intended for the abstraction of drinking water in the Member States (OJ L 194, 25.7.1975, p. 26)

EEC (1978) Council Directive 78/659/EEC of 18 July 1978 on the quality of fresh waters needing protection or improvement in order to support fish life (OJ L 222, 14.8.1978, p. 1)

EEC (1980) Council Directive 80/778/EEC of 15 July 1980 relating to the quality of water intended for human consumption (OJ L 229, 30.8.1980, p. 11)

Goyer, R.A. (1996) Toxic effects of metals. In Casarett & Doull's Toxicology. The Basic Science of Poisons, Fifth Edition, Klaassen, C.D. [Ed]. McGraw-Hill Health Professions Division, ISBN 0071054766

Mance, G., Brown, V.M. & Yates, J. (1984) Proposed environmental quality standards for List II substances in water. Copper. Water Research Centre Technical Report TR210

Nriagu, J.O. (1990) Global metal pollution. Poisoning the biosphere? Environment 32, 7: 7-11;28-33

Thornton, I. (1995) Metals in the global environment: facts and misconceptions. A publication by the International Council on Metals and the Environment (ICME). ISBN 1895720095

UNEP (1993) Preliminary assessment of the state of pollution of the Mediterranean Sea by zinc, copper and their compounds and proposed measures. Mediterranean Action Plan UNEP (OCA)/MED/WG.66/Inf.3, Athens 3-7 May 1993

WHO (1993) Guidelines for drinking-water quality. Volume 1: Recommendations. ISBN 9241544600, 188pp.

Zinc (Zn)

Zinc is a relatively common metal, found in the Earth's crust at an average concentration of 80 mg/kg, although some clay sediments and shales may contain higher concentrations (Alloway 1990, Salomons & Forstner 1984). It is not found naturally in its pure form (as a lustrous, blue-white metal) but as a mineral (most commonly sphalerite, zinc sulphide), often associated with the ores of other metals (e.g. copper, lead and cadmium) (Kroschwitz & Howe-Grant 1995).

Natural sources of atmospheric zinc include wind-borne soil particles, emissions from forest fires and volcanoes, biogenic emissions and sea-salt sprays. The total amount of Zinc released to the atmosphere from natural sources is estimated at 45,000 tonnes / year, compared with an estimated anthropogenic load of 132,000 tonnes / year (Nriagu 1990).

Zinc is one of the most extensively utilised "trace" metals (Nriagu 1990). It is most commonly employed as a protective coating for other metals e.g. galvanised steel, or as a component of bronze, brass and die-casting alloys. In addition, zinc salts are widely employed as wood preservatives, herbicides, catalysts, analytical reagents, vulcanisation accelerators for rubber and stabilisers in PVC.

They can also be found in ceramics, textiles, fertilisers, paints, pigments, batteries and dental, medical and household products (ATSDR 1997, Annema & Ros 1994, UNEP 1993, Budavari *et al.* 1989).

Zinc is an essential element, present in the tissues of animals and plants at normal, ambient concentrations. However, if plants and animals are exposed to high concentrations of bioavailable zinc, significant bioaccumulation can result, with possible toxic effects (ATSDR 1997).

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

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 (ATSDR 1997). Humans taking supplements at higher than recommended doses (400-500 mg/day) suffered severe gastro-enteritis (Abernathy & Poirier 1997); and humans who drank water from galvanised pipes, over a prolonged period, suffered irritability, muscular stiffness and pain, loss of appetite and nausea (UNEP 1993).

With regard to industrial exposure, metal fume fever resulting from the inhalation of zinc oxide fumes presents the most significant effect. Attacks usually begin after 4-8 hours of exposure and last between 24-48 hours. Symptoms include chills and fever, profuse sweating and weakness (ATSDR 1997, Goyer 1996).

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).

Zinc is not a priority pollutant, but it is included on the majority of subsidiary and secondary pollutant lists. Examples of guidelines and permissible environmental levels include the following:

European Council Directive 78/659/EEC on the quality of fresh waters needing protection or improvement in order to support fish life. An Environmental Quality Standard of 0.3 mg/l is set (EEC 1978).

European Council Directive 80/778/EEC relating to the quality of water intended for human consumption sets guide levels of 100 ug/l (for outlets of pumping and / or treatment works) and 5000 ug/l (after water has been standing for 12 hours in the piping / made available to the consumer)(EEC 1980). The USEPA recommends a maximum permissible concentration for drinking water of 5 mg/l (ATSDR 1997) as does the Bureau of Indian Standards (1995).

The UK Department of the Environment (ICRCL) classifies a soil level of 0-250 mg/kg as being typical of uncontamination. Anything above this is classified as contaminated and as such, restrictions on recreational and agricultural uses apply (Alloway 1990). In terms of permissible sewage sludge levels, acceptable concentrations for zinc range from 1000-10,000 mg/kg, however resulting soil concentrations should not exceed 560 mg/kg (UK) or 300 mg/kg (EC, France, Germany) (Alloway 1990).

References

Abernathy, C.O. & Poirier, K.A. (1997) Uncertainties in the risk assessment of essential trace elements: the case of zinc. Human and Ecological Risk Assessment 3(4): 627-633

Aggett, P.J. & Comerford, J.G. (1995) Zinc and human health. Nutrition Reviews 53, 9: S16-S22

Alloway, B.J. (1990) Heavy metals in soils. John Wiley and Sons, Inc. New York, ISBN 0470215984

Annema, J.A. & Ros, J. (1994) Management of the zinc chain. Resources, Conservation and Recycling 12: 209-219

ATSDR (1997) Toxicological profile for zinc on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

Bagshaw, J.C, Rafiee, P., Matthews, C.O. & Macrae, T.H. (1986) Cadmium and zinc reversibly arrest development of Artemia larvae. Bull. Environ. Contam. Toxicol. 37: 289-296

Budavari, S.M., O'Neil, J., Smith, A. & Heckleman, P.E. [Eds] (1989) The Merck Index: an encyclopaedia of chemicals, drugs and biologicals. 11TH Edn Merck and Co, Inc., New Jersey, USA

Bureau of Indian Standards (1995) Drinking water specifications. Second Edition, UDC 628.1.033

EEC (1978) Council Directive 78/659/EEC of 18 July 1978 on the quality of fresh waters needing protection or improvement in order to support fish life (OJ L 222, 14.8.1978, p. 1)

EEC (1980) Council Directive 80/778/EEC of 15 July 1980 relating to the quality of water intended for human consumption (OJ L 229, 30.8.1980, p. 11)

Goyer, R.A. (1996) Toxic effects of metals. In Casarett & Doull's Toxicology. The Basic Science of Poisons, Fifth Edition, Klaassen, C.D. [Ed]. McGraw-Hill Health Professions Division, ISBN 0071054766

Kroschwitz, J.I & Howe-Grant, M. [Eds] (1995) Kirk-Othmer Encyclopaedia of Chemical Technology Volume 7. Fourth Edition. Zinc and zinc alloys. John Wiley & Sons, New York, Chichester, Brisbane, Toronto and Singapore

Nriagu, J.O. (1990) Global metal pollution. Poisoning the biosphere? Environment 32, 7: 7-11;28-33

Salomons, W. & Forstner, U. (1984) Metals in the hydrocycle. Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, ISBN 3540127550

UNEP (1993) Preliminary assessment of the state of pollution of the Mediterranean Sea by zinc, copper and their compounds and proposed measures. Mediterranean Action Plan UNEP (OCA)/MED/WG.66/Inf.3, Athens 3-7 May 1993