STOP POLLUTION

AMERICAS TOUR

REPORT

Identification of organic pollutants and heavy metal contaminants in sediments and wastewaters from the Bahia Blanca Petrochemical Complex, Argentina 2000

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

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

December 2000

Technical Note: 16/00

GREENPEACE

TABLE OF CONTENTS

Table of Contents 1
Executive summary 2
1 Introduction 3
2 Sampling Program 5
3 Results and discussion 5
3.1.1 The main effluent canal 8
3.1.2 Runoff/drainage system 10
4 Conclusions 11
5 References 12
Appendix 1 Analytical methodology 14
Appendix 2 Toxicological outlines for key organic compounds 19
Appendix 3 Toxicological outlines for heavy metals 34

EXECUTIVE SUMMARY

The Petrochemical Complex at Bahia Blanca is home to a number of environmentally hazardous industries. One of these, Solvay-Indupa, produces PVC, which entails using and manufacturing dangerous chemicals such as mercury, chlorine and vinyl chloride. Between 1996 to 2000, three surveys have been carried out to ascertain the extent of pollution emanating from this plant.

The most recent samples show that toxic pollutants are being released not only via the main effluent canal, but also via the system of runoff channels that drain rainwater from the site. Toxicants identified include volatile pollutants such as vinyl chloride which evaporate readily and are easily carried from the plant in the air; persistent compounds such as hexachlorobenzene, which are under international scrutiny as being among the worst organic pollutants known; and mercury, one of the most toxic metals, which can build up in concentration as it passes up the food chain.

It can be concluded, therefore, that significant releases are continuing and there is also potential for some of the most toxic pollutants to be emitted through routes that it was not possible to investigate at this time.

1 INTRODUCTION

Bahia Blanca, in the south of Buenos Aries province, is home to one of the largest petrochemical plants in Argentina. The industries at this location manufacture products including chlorine, PVC and polyethylene.

The manufacture of PVC is a particularly polluting process, utilising as it does a number of extremely hazardous chemicals. The process is illustrated in Figure 1 below.

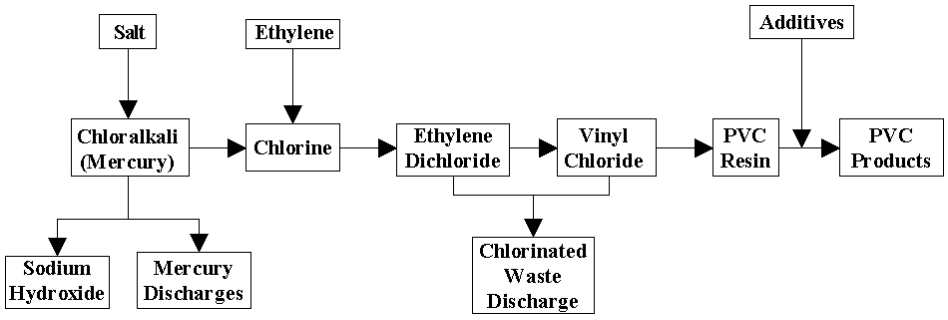

Figure 1: simplified diagram of PVC production.

The first stage is the electrolytic production of chlorine. There are three types of electrolytic cell in use today. Of these the mercury cell has the greatest potential to create environmental pollution. Further serious environmental risks result from the production, liquefaction, transport and storage of chlorine as well as the other substances produced.

Vinyl chloride momoner (vinyl chloride, VCM) for the production of PVC is almost always derived from 1,2-dichloroethane (ethylene dichloride or EDC). EDC can be produced by oxychlorination or by direct chlorination. In the direct chlorination method, ethene is reacted with chlorine to produce EDC (reaction a). Oxychlorination produces EDC by reacting ethene with dry hydrogen chloride and oxygen (reaction b). Vinyl chloride (VC) is subsequently produced by subjecting EDC to high temperatures and pressures in a process called "thermal cracking". This yields not only vinyl chloride monomer but also hydrogen chloride (reaction c). The hydrogen chloride is often fed back into the oxychlorination process to make further ethylene dichloride in what is known as the balanced process (OSPAR 1996, Sittig 1978, Snedecor 1993).

a $CH_2=CH_2 + Cl_2 \rightarrow CH_2ClCH_2Cl$

b $2CH_2=CH_2 + 4HCl + O_2 \rightarrow 2CH_2ClCH_2Cl + H_2O$

c $CH_2ClCH_2Cl \rightarrow CH_2=CHCl + HCl$

In addition to the reaction products themselves, these processes result in the generation of various wastes and byproducts containing toxic and persistent organochlorines.

Sample Number	Year	Sample Information
		Main effluent channel
AM0028	**2000**	**Sediment from main discharge canal, immediately before it passes under the road.**
AM0029	***2000***	***Effluent from main discharge canal, immediately before it passes under the road.***
AM0030	**2000**	**Sediment from discharge channel, 3m down from the end of underground tunnel.**
LA8041	*1998*	*Effluent from main discharge channel, downstream (unlined) section.*
LA8042	1998	Sediment collected main discharge channel, downstream (unlined) section, close to the Bay.
LA8053	*1998*	*Effluent from main discharge channel, downstream (unlined) section.*
LA8057	*1998*	*Effluent from main discharge channel, downstream (unlined) section.*
LA8058	1998	Sediment from main discharge channel, downstream (unlined) section.
LA8061	1998	Sediment from main discharge channel, downstream (unlined) section.
MI6067	1996	Sediment from main discharge channel, downstream (unlined) section.
		Runoff location 1
AM0021	**2000**	**Sediment from run-off channel 1.9 km from ethylene pipes.**
AM0022	***2000***	***Water from run-off channel 1.9 km from ethylene pipes.***
LA8062	1998	Sediment collected from runoff/drainage channel running from the chlorine plant (1.9 km from ethylene pipes)
MI6068	1996	Sediment collected from runoff/drainage channel running from the chlorine plant
		Runoff location 2
AM0026	**2000**	**Sediment from run-off channel from Solvay 1.3 km from ethylene pipes, same location as LA8056**
AM0027	***2000***	***Water from run-off channel from Solvay 1.3 km from ethylene pipes.***
LA8055	*1998*	*Effluent collected from runoff/drainage channel running from the PVC plant (1.3 km from ethylene pipes)*
LA8056	1998	Sediment collected from a runoff/drainage channel running from the PVC plant (1.3 km from ethylene pipes)
MI6063	*1996*	*Effluent collected from runoff/drainage channel running from the PVC plant*
MI6070	1996	Sediment collected from runoff/drainage channel running through the PVC plant
		Runoff location 3
AM0025	**2000**	**Sediment from side-arm of main runoff channel, 1.0 km from ethylene pipes.**
		Runoff location 4
AM0024	***2000***	***Water from run-off channel, 0.75 km from ethylene pipes.***
AM0023	**2000**	**Sediment from run-off channel 0.75 km from ethylene pipes.**
LA8059	1998	Sediment from runoff channel (0.75 km from ethylene pipes)
LA8060	*1998*	*Water from runoff channel(0.75 km from ethylene pipes)*
MI6069	1996	Sediment from runoff/drainage channel running from the EDC/VCM plant
		Combined runoff ditch
LA8054	1998	Sediment from runoff/drainage channel (1.6 km from ethylene pipes)
LA8044	1998	Sediment from runoff/drainage channel, at point of confluence with the Bay
LA8045	1998	Sediment from runoff/drainage channel, at point of confluence with the Bay (Puerto Galvan)
MI6066	1996	Sediment from runoff/drainage channel, at point of confluence with Bahia Blanca Bay
		Dredge spoil dump site
MI6062	*1996*	*Water from an area of dredged soils, in front of PVC, EDC, VCM and chlorine plants*
MI6064	1996	Sediment from an area of dredged soils (see MI6062)
MI6065	1996	Soil dredged from the estuary, and stored on land, in front of the PVC, EDC, VCM and chlorine plants

Table 1. Summary of samples collected around the Solvay site since 1996. Samples are grouped according to the discharge point or channel from which they were collected. Samples from the year 2000 are in bold type, water/effluent samples are in italics.

2 SAMPLING PROGRAM

In order to investigate the extent of existing contamination, in June 2000, ten samples of sediment, industrial wastewater and runoff were collected from sites in and around the complex, with the aim of identifying organic pollutants and quantifying heavy metal contaminants. Three of the samples were collected from a wide, open waste canal running through the middle of the site and into a channel leading to the bay. The other samples were collected from a number of runoff/drainage channels running through and around the complex, close to, or leading from, the Solvay-Indupa chlorine, VCM and PVC plants. This effectively continues work carried out in 1996, when nine samples from the same and similar sites were collected, and 1998, when twelve samples were collected. Table 1 summarises al the samples collected since 1996, grouping them according to the channel or discharge point from which they were collected.

3 RESULTS AND DISCUSSION

The results of the organic and heavy metals analyses are presented in Table 2, including a breakdown of the groups of organic compounds reliably identified in the samples. As in Table 1, the samples have been grouped according to location.

Metals concentrations are given in parts per million (mg/kg) on a dry weight basis for sediments and parts per billion (ug/l) for water samples. Organic contaminants detected by GC/MS scanning methods are indicated with a tick (✓); those detected only at low concentration using SIM methods are indicated with an asterisk (*).

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

Producing the chemical precursors needed to manufacture PVC produces complex wastes containing many toxic organochlorines. These are known to include dioxin (eg USEPA 2000, Stringer & Temuge 1998, Stringer *et al.* 1995, ICI 1994) as well as dozens of other organochlorines (Stringer & Temuge 1998, Johnston *et al.* 1996). Many hydrocarbon pollutants can also be detected at most sites (Johnston *et al.* 1996). Copper is used as a catalyst in the process (ICI 1994), so may be expected to be released to the environment. If the chlorine necessary for the process is manufactured on site, the much more toxic metal, mercury, may also be present. This is as a consequence of mercury being used as a component of the electrolytic cells that produce the chlorine.

Sample Number	**AM0028**	**AM0029**	**AM0030**	**AM0021**	**AM0022**	**AM0026**	**AM0027**	**AM0025**	**AM0023**	**AM0024**
Sample location	Main discharge channel			Runoff location 1		Runoff location 2		Runoff 3	Runoff location 4	
Sample type	sediment	water	sediment	sediment	water	sediment	water	sediment	sediment	water
Sampling date	02.06.00	02.06.00	03.06.00	02.06.00	02.06.00	02.06.00	02.06.00	02.06.00	02.06.00	02.06.00
Sample information	where channel passes under the road	where channel passes under the road	unlined effluent channel	1.9 km from ethylene pipes	1.9 km from ethylene pipes	1.3 km from ethylene pipes	1.3 km from ethylene pipes	1.0 km from ethylene pipes	0.75 km from ethylene pipes	0.75 km from ethylene pipes
METALS	mg/kg dry wt	ug/l	mg/kg dry wt	mg/kg dry wt	ug/l	mg/kg dry wt	ug/l	mg/kg dry wt	mg/kg dry wt	ug/l
Cadmium	2	<20	4	<2	<20	<2	<20	<2	<2	<20
Chromium	41	<20	840	59	<20	31	<20	14	20	<20
Cobalt	7	<20	8	13	<20	11	<20	9	10	<20
Copper	173	42	210	43	41	20	<20	17	18	24
Lead	20	<30	38	29	<30	21	<30	3	14	<30
Manganese	296	100	380	585	400	535	78	348	402	56
Mercury	7.95	2.7	14.98	21.29	2	0.36	1.8	0.2	0.19	<1
Nickel	24	<20	33	36	<20	17	<20	8	13	<20
Zinc	758	126	3848	164	<10	63	69	47	60	<10
ORGANOHALOGENS										
Chlorobenzene						*				
Benzene, 1,2-dichloro-			✓			*				
Benzene, 1,3-dichloro-			✓			*				
Benzene, 1,4-dichloro-	✓	*	✓		*	*	*	*		*
Benzene, 1,2,3-trichloro-						*				
Benzene, 1,2,4-trichloro-		*	*			*				
Benzene, 1,3,5-trichloro-			✓							
Benzene, 1,2,3,5-tetrachloro-						*				
Benzene, 1,2,4,5-tetrachloro-						*				
Benzene, 1,2,3,4-tetrachloro-						*				
Benzene, pentachloro-	*	*	*			*		*		
Benzene, hexachloro-	✓	✓	✓			*		*		*
PCB-138		*								
PCB-153		*								
Chloroform	*	*		*			*			
Carbon tetrachloride		*								
Tetrachloroethene	*			*		*	*	*		
Trichloroethene	*									
1,1-Dichloroethene	*	*								
1,2-Dichloroethane (EDC)	✓	✓	✓							
Vinyl chloride (VCM)	*	*	*							

Sample Number	**AM0028**	**AM0029**	**AM0030**	**AM0021**	**AM0022**	**AM0026**	**AM0027**	**AM0025**	**AM0023**	**AM0024**
Sample location	Main discharge channel			Runoff location 1		Runoff location 2		Runoff 3	Runoff location 4	
Sample type	sediment	water	sediment	sediment	water	sediment	water	sediment	sediment	water
POLYNUCLEAR AROMATIC HYDROCARBONS										
Naphthalene and/or its derivatives	✓	✓	✓							
Anthracene and/or its derivatives	✓		✓							
Acenaphthene	✓	✓	✓							
Acenaphthylene	✓	✓	✓							
Phenanthrene and/or its derivatives	✓	✓	✓							
Pyrene and/or its derivatives		✓	✓							
9H-Fluorene and/or its derivatives	✓	✓	✓							
Fluoranthene			✓							
Benz[a]anthracene and/or its derivatives			✓							
Chrysene and/or its derivatives			✓							
PHENOLIC COMPOUNDS										
BHT	✓	✓	✓		✓	✓				
Phenol, 4-methyl-										
Phenol, 2,6-bis(1,1-dimethylethyl)-					✓					
Phenol, 2,4-bis(1,1-dimethylethyl)-						✓				
OTHER AROMATIC COMPOUNDS										
Alkylated benzenes	✓	✓	✓							
Biphenyl and/or its derivatives	✓	✓	✓							
Diphenyl ether	✓	✓								
ALIPHATIC HYDROCARBONS										
Linear	✓		✓	✓		✓		✓	✓	
Cyclic	✓		✓						✓	

Table 2. Analytical results for the samples collected around the Solvay plant in 2000. See text for further details.

3.1.1 The main effluent canal

In the current study, the most contaminated set of samples was that associated with the main effluent canal running through the complex. This is an open, concrete channel, which runs through both the site and the adjacent polyethylene manufacturing facility. As it leaves the Bahia Blanca petrochemical complex, it passes under the road and flows through an underground tunnel for approximately 100 metres. It emerges via two pipes into an unlined channel that runs into the bay at Puerto Galvan. Three samples from this channel were collected in 2000; one water/effluent sample (AM0029) and two sediments. Of the sediments, one (AM0028) was collected at the same site as the wastewater sample, before the effluent channel left the industrial complex. The other (AM0030) was collected further downstream, where the concrete tunnel ends and the effluents pass into the unlined channel that carries the wastewaters to the bay.

All three of these samples contained notable concentrations of toxic metals and numerous chlorinated and non-chlorinated samples were identifiable.

The organochlorines detected ranged from extremely volatile compounds such as vinyl chloride (VCM) and chloroform to polychlorinated biphenyls (PCBs) and chlorobenzenes, which are semivolatile compounds that can be highly persistent in the environment and build up in the food chain.

Both vinyl chloride (VCM) and 1,2-dichloroethane, also known as ethylene dichloride or EDC, were detected in all three samples from this main discharge channel, though the vinyl chloride was only detectable at trace concentrations. These compounds are the feedstocks for the manufacture of PVC, as discussed above.

Both EDC and vinyl chloride are highly toxic, with vinyl chloride being classified as a class 1 carcinogen by the International Agency for Research on Cancer (IARC). They are volatile compounds, which evaporate easily and do not usually persist for very long periods in the environment. However, under the right conditions, they can remain in sediments, soils or aquifers for extended periods.

Other volatile organochlorines, 1,1-dichloroethene, trichloroethene, tetrachloroethene, chloroform and carbon tetrachloride were also present in the effluent sample or the sediment collected closest to the industrial complex, in each case at trace levels. However, the fact that most of the volatile pollutants were only present at trace concentrations does not mean that they are not being emitted in significant quantities. As mentioned above, all these compounds evaporate quickly and the presence of even trace quantities in wastewaters and sediments may be indicative of substantial releases to air.

Some of these chemicals, though toxic, are still manufactured in large quantities. For example, tri- and tetrachloroethene are used as solvents in applications such as industrial degreasing or dry-cleaning. However, the uses of the others have fallen over the years, mostly for environmental and health reasons. Carbon tetrachloride is an ozone depleter

and production has been banned in most of the world under the terms of the Montreal Protocol, which has set a date of 2006 for completion of a global phase-out (UNEP 1997).

Semivolatile compounds identified included the chlorobenzenes, seven of which were found in one or more of the three samples from the effluent channel. There are twelve different chemicals in this group, of which only three (chlorobenzene, 1,2-dichlorobenzene and 1,4-dichlorobenzene) are still produced in large quantities. Hexachlorobenzene, which was present at above trace concentrations in all three samples, has been recognised as one of the most dangerous persistent pollutants and is thought to be able to act on the in the same way as the polychlorinated dioxins and furans (PCDD/Fs)(van Birgelen 1998).

The downstream sediment contained a wider range of chlorobenzenes than that collected upstream. The most likely explanation for this is the difference in physical characteristics between the two locations. The upstream sample came from the concrete-lined effluent channel, whereas the downstream sample came from the ditch into which this channel empties. This ditch is unlined and the mud/sediment in it would have been accumulating pollutants from the discharge over a period of years.

Although some of the organochlorines found in the samples from the effluent channel are still manufactured intentionally, all of the volatile compounds, except 1,1-dichloroethene, and most of the chlorobenzenes have been detected in the effluents from PVC industry facilities at other sites (Johnston *et al.* 1996). In the absence of evidence that any of these chemicals are being manufactured or used in significant quantities in the industrial complex, it is reasonable to assume that they are present as byproducts of the PVC manufacturing industry.

In the sample of water from the effluent channel (AM0029), however, two isomers of PCBs (PCB 138 and PCB 153) were detected at trace concentrations. This is a notable result since, although it is known that PCBs can be produced as byproducts of manufacturing PVC precursors, it is highly unlikely that they would be detectable in an aqueous effluent. Moreover, since they are poorly soluble as well as highly persistent, regular discharges of PCBs in wastewaters might be expected to lead to the accumulation of detectable concentrations in the sediments of the channel, and this is not the case. Perhaps more likely a source is a brief release of "technical PCBs" which in the past were used in a wide range of applications, from paper additives to insulating fluids in electrical transformers. Although PCB manufacturing has almost totally ceased, the possibility remains that PCBs could be released from older equipment that was made before their phaseout. If any equipment on the site does still contain PCBs, it should be identified in order that the PCBs can be destroyed and not released to the environment.

Numerous non-chlorinated pollutants were also present in the effluent channel. All three samples contained polynuclear aromatic hydrocarbons (PAHs), aromatic compounds and aliphatic hydrocarbons. Similar hydrocarbon pollutants have been identified in the effluents from the PVC industry in Europe (Johnston *et al.* 1996), but these are common pollutants, usually derived from mineral oils, and are therefore ubiquitous pollutants at petrochemical plants. In this case it is not possible to draw firm conclusions about their source.

Nine different metals were detected in the sediments from the effluent channel, though not all were detectable in the wastewater. The most significant elevations in concentration were observed for mercury, zinc, chromium and copper. In each case, the concentrations were higher in the downstream sediment sample. As with the chlorobenzenes, this is probably indicative of greater accumulation of pollutants in the natural sediments at this site compared to the concrete effluent channel.

As discussed earlier, both mercury and copper are likely to be released from facilities producing chlorine and vinyl chloride. Mercury is one of the most toxic metals and can biomagnify significantly as it passes up the food chain. Chlorine production is one of the few uses of mercury remaining in the modern chemical industry, so there is little doubt about its source. Copper, however, as well as the other metals found at elevated concentrations, chromium and zinc, still have numerous industrial sources and so it is more difficult to say with certainty whether they are discharged from the Solvay site or from another facility.

This discharge canal site was previously sampled in 1996 and 1998 (Stephenson & Labunska 1998). Some caution is needed in comparing directly the results from the effluent channel since those collected in 1996/8 were from sites further downstream from those sampled in 2000. Nevertheless, the samples collected in 2000 show a similar pollution profile to these earlier samples. The samples of sediments collected from this channel during 1996, 1998 all show mercury contamination. The maximum mercury concentration in 2000 was almost 15 mg/kg, whereas at an uncontaminated site the concentration would be expected to be less than 1 mg/kg. A greater variety of organochlorines was also found in the 2000 samples. This is in part because the analytical procedures used in 2000 are more extensive than those applied in 1996/8 and have allowed the identification of toxic chemicals too volatile to have been detected before. These include the extremely hazardous chemicals vinyl chloride and 1,2-dichloroethane, which are the primary products of the Solvay facility, and which have undoubtedly been routinely discharged since production commenced.

3.1.2 Runoff/drainage system

The other samples collected during the year 2000 survey were all associated with a system of pipes and channels which carry rainwater and other runoff from the site into a ditch running between the road and the industrial complex's perimeter fence. In Tables 1 and 2, these samples are arranged in groups according to the part of this system from which they were collected. The sites are arbitrarily numbered 1-4, beginning with that furthest from the location where the pipelines carrying the ethylene needed for the production of PE and PVC cross the road and which serves as a useful reference point. Water discharged from these locations join a ditch and when volumes are great enough they flow from the direction of location 1 to location 4 and thereafter into the bay.

Location 1 is a runoff discharge pipe adjacent to area of the plant where the chlorine is produced and storage facilities are located, approximately 1.9 km from the place where the ethylene pipes cross the road. Location 2 is a runoff discharge pipe about 1.3km form the

ethylene pipes; location 3 is a channel joining the runoff ditch about 1km from the ethylene pipes. Location 4 is a runoff discharge pipe about 1km from the ethylene pipes and close to the section of the plant where EDC/VCM are reported to be manufactured.

Overall, the samples from the runoff/drainage system showed a similar range of pollutants to the main discharge canal. Some hydrocarbon pollution was present, though it was less extensive.

Both the water and sediment samples collected at location 1 exhibit contamination by copper and mercury. The copper contamination is only slight, but the mercury sediment contamination is the highest measured during this survey, being over twenty times the concentration expected in a typical uncontaminated river sediment (Salomons & Forstner 1984). The only organochlorine contaminant identified in the wastewater was a trace amount of 1,4-dichlorobenzene, which is an extremely common pollutant since it is used in lavatory cleaning products. Two other organochlorines, chloroform and tetrachloroethene, were identified in the sediment at trace concentrations.

All three of these organochlorines were also detected at trace concentrations in the water sample collected from location 2. The sediment here exhibited trace contamination with a greater range of organochlorines: tetrachloroethene and no fewer than eleven chlorobenzenes. In fact, the only member of the chlorobenzene family that was not detected was 1,3,5-trichlorobenzene.

Only a sediment sample was collected from location 3. Metals concentrations in this sample were not notably elevated, but there were trace levels of four different organochlorines: tetrachloroethene, 1,4-dichlorobenzene, pentachlorobenzene and hexachlorobenzene. This location was not sampled during the earlier surveys.

The water sample from location 4 contained two organochlorines, 1,4-dichlorobenzene and hexachlorobenzene, at trace levels. Metals were present only low or non-detectable concentrations. The sediment was also showed little evidence of contamination, with no notable elevations in the concentrations of metals present and linear and cyclic hydrocarbons present, but no detectable organochlorines.

In comparison, the earlier surveys of this runoff/drainage system (Stephenson & Labunska 1998) found a closely similar range of metal, organochlorine and hydrocarbon pollutants.

4 CONCLUSIONS

The current confirms that there is continued pollution of the environment emanating from the petrochemical complex at Bahia Blanca. The most environmentally harmful pollutants, the organochlorines and mercury, are almost certainly derived from the manufacturing of chlorine and PVC-related chemicals by Solvay. Not only is it clear that organochlorine and metallic toxicants continue to be released through the aqueous effluents, but there are also expected to be emissions to air and, moreover, there are organic waste streams produced in this process. These organic wastes are generated

during the purification of EDC and VCM by distillation. The products that are more volatile than the chemical being purified distill off first, and are called light ends. Those that are less volatile remain after the desired product has evaporated off are called heavy ends. These wastes consist primarily of a mixture of organochlorine pollutant, which can include the polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs). In particular, the heavy ends can contain part per million concentrations of PCDD/Fs (Stringer *et al.* 1995, ICI 1994), making them one of the most dioxin contaminated wastes produced by the modern petrochemical industry. These wastes may be recycled (though this can increase the dioxin content even further (ICI 1994)), dumped, or incinerated. It is not known how the Solvay facility disposes of its organic wastes, and no dioxin analyses have been carried out during the current surveys. Until this has been addressed by the company, the potential for serious dioxin contamination cannot be ruled out.

5 REFERENCES

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

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

OSPAR (1996) Best available techniques for the vinyl chloride industry. Publ: Oslo and Paris Commissions.

Sittig, M. (1978) Vinyl Chloride and PVC manufacture: process and environmental aspects. Publ: Noyes Data Corporation, New Jersey, USA

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. Volume 6 pp 11-36

Stephenson, A. & Labunska, I. (1998) Identification of organic pollutants and heavy metal contaminants found in sediments and wastewaters collected from the Bahia Blanca Petrochemical Complex, Argentina 1996 and 1998. Greenpeace Research Laboratories Technical note 11/98, 27pp.

Stringer, R. & Temuge, T. (1998) PVC: The dark side of Petkim: PVC. Publ. Greenpeace Mediterranean, 31pp

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

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)

USEPA (2000) Exposure and Human Health Reassessment of 2,3,7,8-tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Part I: Estimating Exposure to Dioxin-Like Compounds. Volume 2: Sources of Dioxin-Like Compounds in the United States. DRAFT EPA/600/P-00/001Ab. March 2000, Draft Final, www.epa.gov/ncea

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

APPENDIX 1 ANALYTICAL METHODOLOGY

A1.1 Organic analysis

Samples underwent two separate analyses for organic pollutants. They were analysed by a headspace technique for volatile organic compounds (VOCs) and also screened to identify semivolatile chemicals.

A1.1.1 Preparation of samples for organic analysis

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

A1.1.2 Volatile Organic Compounds (VOCs) analysis

For volatile organic compound analysis, no sample preparation was required. The original sample was sub-sampled immediately after opening. Two 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 the compounds in the sample. The second sub-sample was used for identification of target compounds (see table A1.1) at trace concentrations with an external standard using a Selective Ion Monitoring (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
Ethylene chloride	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

Table A1.1. List of volatile organic compounds and appropriate ions that were monitored during GC/MS analysis using SIM method.

A1.1.3 Solid Samples

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

Extracts were decanted, filtered through a pre-cleaned hydrophobic phase separator filter and collected in reagent tubes. They were then acidified to pH 2 with 10% nitric acid. Following this, a second portion of 20ml pentane was added and the extraction procedure repeated. Finally, both extracts obtained for each sample were combined and evaporated to a volume of approximately 3ml. The concentrated extract was cleaned through a Florisil column, eluted with a 95:5 mixture of pentane: toluene, and evaporated down to a volume of 2 ml under a stream of analytical grade nitrogen. 1-Bromonaphthalene was then added at concentration 10mg/l to provide an indication of GC/MS performance.

A1.1.4 Aqueous Samples

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

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

A1.1.4 Chromatographic Analysis

Organic compounds were identified qualitatively and quantitatively using Gas Chromatography Mass Spectrometry (GC-MS).

Instrumentation was a Hewlett Packard (HP) 5890 Series II gas chromatograph, interfaced with a HP Chem-Station data system and linked to a HP 5972 Mass Selective Detector operated in scan mode. The identification of compounds was carried out by computer matching against a HP Wiley 275 library of 275,000 mass spectra combined with expert interpretation. Also all extracts were analysed using selective ion monitoring (SIM) method against two standard solutions. The lists of compounds containing in Standard I and Standard II are presented below. All individual standards were obtained from Sigma Aldrich Co. Ltd., Supelco, UK.

All organic contaminants discussed in this report were identified with a 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.

Compound	Ions to monitor
Benzene, 1,3-dichloro-	146, 148, 111, 75
Benzene, 1,4-dichloro-	146, 148, 111, 75
Benzene, 1,2-dichloro-	146, 148, 111, 75
Benzene, 1,3,5-trichloro-	180, 182, 145, 74
Phenol, 2,4-dichloro-	162, 164, 63, 98
Benzene, 1,2,4-trichloro-	180, 182, 145, 109
Benzene, 1,2,3-trichloro-	180, 182, 145, 109
Dichlorvos	109, 185, 79, 47
Benzene, 1,2,3,5-tetrachloro-	216, 214, 218, 179
Benzene, 1,2,4,5-tetrachloro-	216, 214, 218, 179
Benzene, 1,2,3,4-tetrachloro-	216, 214, 218, 179
Benzene, pentachloro-	250, 252, 248, 215
alpha-HCH	181, 183, 219, 217
Benzene, hexachloro-	284, 286, 282, 249
Simazine	200, 215, 202, 173
beta-HCH	181, 183, 219, 217
gamma-HCH	181, 183, 219, 217
delta-HCH	181, 183, 219, 217
o,p'-DDE	246, 248, 318, 176
p,p'-DDE	246, 318, 246, 316
o,p'-DDD	235, 237, 165, 199
p,p'-DDD	235, 237, 165, 199
o,p'-DDT	235, 237, 165, 199
p,p'-DDT	235, 237, 165, 199

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

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

A.1.3 List of compounds in the Standard II used for SIM analysis

A1.2 Heavy Metal Analysis

A1.2.1 Preparation of samples for heavy metal analysis

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

A1.2.2 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.

A.2.1.3 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.

A2.1.4 Inductively Coupled Plasma Atomic Emission Spectrometry (ICP-AES)

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

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

APPENDIX 2 TOXICOLOGICAL OUTLINES FOR KEY ORGANIC COMPOUNDS

A2.1 Vinyl chloride

Vinyl chloride is a colourless, sweet smelling gas under normal conditions. 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. In soil, vinyl chloride migrates rapidly to groundwater.

However, vinyl chloride remains 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 is a known human carcinogen (Group 1 as assessed by the IARC). Numerous studies demonstrate that it causes 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).

References

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

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

A2.2 EDC (1,2-dichloroethane, ethylene dichloride)

EDC 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.

Because of its volatility, the most usual route of exposure is via inhalation, when it is one of the more toxic chlorinated solvents. However, it can also cause harm through skin or eye contact. 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 can be toxic at concentrations too low to be detected by smell (Snedecor 1993).

References

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. Volume 6 pp 11-36

A2.3 Chlorinated benzenes

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 (Bryant 1993, CEC 1986).

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.

A2.3.1 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 (Bryant 1993).

Chlorobenzene bioaccumulate in algae, fish and aquatic invertebrates. 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 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). 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 (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 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 repellant 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).

A2.3.2 Trichlorobenzenes

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

The 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) and sediment (Labunska *et al.* 1998), 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. 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 (Hermanson *et al.* 1997).

Giddings *et al.* (1994a) report 1,3,5-trichlorobenzene air, drinking water, food and 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).

A2.3.3 Tetrachlorobenzenes

Giddings *et al.* (1994b) reviewed toxicity and exposure data for the tetrachlorobenzenes. 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 fish, through their gills. Bioaccumulation depended upon the rate of activity and oxygen uptake of the fish, and only the low water solubility prevented significant toxicity occurring (Brauner *et al.* 1994).

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

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

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

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

A2.3.4 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 and thyroid hormone (free and total thyroxin) concentrations reduced. Pentachlorobenzene cannot be assigned a carcinogenicity classification because of lack of data. Pentachlorbenzene accumulates in, and is toxic to algae (Sicko-Goad *et al.* 1989d).

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.

A2.3.5 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). Recent research (van Birgelen 1998) suggests that HCB has dioxin-like toxicity and more epidemiological studies should be undertaken especially concerning infants fed breast milk in countries with HCB exposure levels.

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 both in workers at an electrochemical plant at Flix in Spain and the local residents. The authors of the 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 absorb to soil materials and almost no desorption take place (Bahnick & Doucette 1988). It is bioaccumulative and biomagnifies. It can be measured in ambient air, drinking water, soil, food and breast milk (Newhook & 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 a convention currently being drawn up (UNEP 1995, 1997). Furthermore, HCB is included on Annex I of the Draft UNECE POPs Protocol under the Convention on Long-Range Transboundary Air Pollution (LRTAP)(UNECE 1998). Within the EC, discharges of HCB are controlled as stipulated by Council Directive 86/280/EEC, which amends Directive 76/464/EEC, regarding pollution caused by certain dangerous substances discharged into the aquatic environment (EEC 1986, 1976).

HCB is also included in the list of priority hazardous substances agreed by the Third and Fourth North Sea Conferences (MINDEC 1990, 1995), where continuous reduction of all hazardous substances was agreed with the ultimate aim of reducing environmental concentrations of hazardous substances to near background levels (synthetic substances to zero) within the next 25 years. The 1998 Ministerial Meeting of the OSPAR Commission (OSPAR 1998a) further reinforced these objectives. HCB is included on the OSPAR 1998 List of Candidate Substances, Annex 3 of the OSPAR Strategy with regard to Hazardous Substances (OSPAR 1998b).

References

ASTDR (1997) Toxicological profiles. 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. and 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. and 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. European Community Environmental Legislation Volume 7: Water.

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. European Community Environmental Legislation Volume 7: Water.

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

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. Atmosph. Environ. 31(4): 567-573

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. & Mek, M.E. (1994) Hexachlorobenzene: Evaluation of risks to health from Environmental Exposure in Canada. Environ. Carcino. & 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. Of 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.

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: 51-6

A2.4 Chloroform

Chloroform is a heavy, colourless, non-flammable liquid. It has a characteristic pleasant, sweet, ethereal odour and a sweetish burning taste. The odour is non-irritant (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). Investigation 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). The Maximum Contaminant Level (MCL) of 100ug/l in drinking water which is delivered to any user of a public water system is set by EPA for the total THMs (US EPA 1999). 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 depend 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 have been found in the US rural ground water (Wallace 1997), a mean value of 84ug/l has been reported for the 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) as 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.

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

A2.5 Tetrachloroethene

Tetrachloroethene is a clear liquid, which is heavier than water, with a sweet chloroform-like odour. Tetrachloroethene itself 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 has been 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 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 10ug/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) of perchloroethylene 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 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). This compound induces leukaemia 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). It has been found that exposure to tetrachloroethene-contaminated drinking water was associated with an increased risk of leukemia and bladder cancer and that 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

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 (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

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)

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.

APPENDIX 3 TOXICOLOGICAL OUTLINES FOR HEAVY METALS

A3.1 Mercury

Mercury is an extremely toxic, non-essential trace metal, having no biochemical or nutritional function. Biological mechanisms for its removal are poor and mercury is known to biomagnify i.e. progressively accumulate though the food chain.

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, pneumonitis and may affect the central nervous system, causing 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).

Inorganic mercury can be methylated by micro-organisms, indigenous to soils, fresh water and marine sediments. The most common form of organic mercury is methyl mercury, which is soluble, mobile and quick to enter the aquatic food chain.

Exposure to methyl mercury has resulted in permanent damage to the CNS, kidneys, and the developing foetus. The worst incident of methyl mercury poisoning occurred in the Japanese fishing town of Minamata during the 1950s and 1960s.

Over 100 people died (Kudo *et al.* 1998, Kudo & Miyahara 1991), but estimates of the total number of people affected vary. Rogan (1995) gives the number of confirmed cases in 1975 as 800, with 2 800 seeking compensation. Hosokawa (1993) reports that 2 000 had been recognised as affected, but according to Davies (1991), as many as 20 000 people were thought to have been affected by 1989. Symptoms included progressive numbness of fingers, lips and tongue, paralysis, cerebellar ataxia, concentric constriction of visual fields and dysarthria (Fujiki & Tajima 1992, Kudo 1992, Kudo & Miyahara 1991). Congenital effects were also noted, with symptoms including mental retardation and sensory disturbance (Grandjean *et al.* 1996); the effects were also described as similar to those of cerebral palsy (Rogan 1995). Seventeen children with symptoms similar to those of cerebral palsy had been born by 1962. Not all of these children survived and post-mortem examination of those that died showed mercury-induced lesions of the cerebral cortex (Rogan 1995). A wide range of persistent chronic symptoms have been identified (Davies 1991) despite mercury in red blood cells having declined to a level comparable to those of similar coastal communities in Japan (Sakamoto *et al.* 1991).

Because of the inevitable loss of mercury from the process, 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. The objective is that they should be phased out completely by 2010" (PARCOM 1990).

References

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

Davies, F.C.W. (1991) Minamata disease – a 1989 update on the mercury poisoning epidemic in Japan. Environmental Geochemistry and Health 13(1): 35-38

Fujiki, M. & Tajima, S. (1992) The pollution of Minamata Bay by mercury. Water Science and Technology 25(11): 133-140

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

Grandjean, P., White, R.F. & Weihe, P. (1996) Neurobehavioural epidemiology: application in risk assessment. Environmental Health Perspectives 104(Suppl. 2): 397-400

Hosokawa, Y. (1993) Remediation work for mercury contaminated bay- experiences of Minamata Bay project, Japan. Water Science & Technology 28(8-9): 339-348

Kudo, A. & Miyahara, S. (1991) A case history; Minamata mercury pollution in Japan- from loss of human lives to decontamination. Water Science & Technology 23(1-3): 283-290

Kudo, A. (1992) Natural and artificial mercury decontamination- Ottawa River and Minamata Bay (Yatsushiro Sea). Water Science & Technology 26(1-2): 217-226

Kudo, A., Fujikawa, Y., Miyahara, S., Zheng, J., Takigami, H., Sugahara, M. & Muramatsu, T. (1998) Lessons from Minamata mercury pollution, Japan- after a continuous 22 years of observation. Water Science & Technology 38(7): 187-193

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. and 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

Rogan, W.J. (1995) Environmental poisoning of children - lessons from the past. Environmental Health Perspectives 103(Suppl. 6): 19- 23

Sakamoto, M., Nakano, A., Kinjo, Y., Higashi, H. & Futatsuka, M. (1991) Present mercury levels in red blood cells of nearby inhabitants about 30 years after the outbreak of Minamata disease. Ecotoxicology and Environmental Safety 22: 58-66