Organic pollutants and heavy metals in samples associated with North Hungarian Chemical Works Ltd. (EMV Kft.) Sajobabony, Hungary

Iryna Labunska, David Santillo, Kevin Brigden
& Ruth Stringer

Greenpeace Research Laboratories
Technical Note 02/2002

June 2002

Table of contents

Publ: Greenpeace Research Laboratories, Department of Biological Sciences, University of Exeter, Exeter EX4 4PS, UK

Summary

EMV Kft., now owned by US-based TRI (Trans Resources Inc.) Chemicals Rt., was established in 1997 through privatisation of the herbicide production facility of the formerly state-owned North Hungarian Chemical Works, Sajobabony. The plant produces a range of carbamate, thiocarbamate and chlorinated acetanilide herbicides, with a total production capacity of around 12 000 t/y. Wastewaters from the processes pass through an on-site treatment plant, operated by Kiss Kft., before discharge to the Babony stream.

Analysis of three samples collected by Greenpeace in February 2002 confirmed that current production processes are resulting in ongoing releases to the Babony stream of a number of herbicide active ingredients, including the thiocarbamates butylate and cycloate, the triazine derivatives terbutryne and prometryne and the chlorinated acetanilide acetochlor. Residues of three thiocarbomates were also detected in sediments from the Babony stream channel downstream from the site, while none were detectable immediately upstream from the site.

Apart from the potential high direct toxicity to freshwater plants from some releases, there might also be significant negative impacts on aquatic fauna and the river ecosystem as a whole. In addition, several of those pesticides identified in the water are quite resistant to environmental degradation and hence may accumulate in sediments and present problems for many years to come. Although those herbicides detected are not among those yet identified as persistent organic pollutants (POPs) under the Stockholm Convention (2001), their release to water in this manner, with the potential for more widespread environmental contamination, is clearly of concern. Clearly the treatment processes currently provided by the Kiss Kft. facility are unable to prevent the continued discharge of these harmful herbicide residues to the Babony stream and, hence the Sajo River.

Introduction

North Hungarian Chemical Works Ltd. is situated on a large industrial site in Sajobabony, where chemical production commenced in 1953. The state industry's section for munitions produced gunpowder, explosives and nickel-cadmium batteries, whilst the civil section produced medical agents and their intermediates, polyurethane foams, herbicides and other chemicals.

The operation of the various industrial activities within this complex resulted in severe pollution of the surrounding environment, including significant soil pollution (heavy metals and organic compounds) and wastewater releases into the Tisza water system through the Babony stream (do we have a reference for this?). A significant cut in chemical production led to an overall reduction in ongoing releases of contaminants in the 1990's, although the complex undoubtedly remains a significant point source. During the privatisation process the production workshops were purchased individually, and now almost 30 companies operate different sections of the site.

EMV Kft. was established in 1997 through the privatisation of the herbicide agent workshops of the state company. It was purchased by TRI Chemical Rt in 1998, a company established by the New York-based Trans Resources Inc. EMV Kft covers only a portion of the industrial site (ca. 20 hectares) and presently operates with approx. 150 employees.

The main fields of activity of the EMV Kft. plant are the production and trade of chemicals, intermediates, and herbicides. The most important herbicide products are listed in Table 1 below.

Most of the products are manufactured using phosgene, which is produced on the site.

Wastewater from EMV Kft. is treated on the industrial site by the Kiss Kft. company, which bought several parts of the earlier state-owned company, including the wastewater treatment plant (WWTP) and hazardous waste incinerator. The treated water is released

Type of herbicide	**Active ingredients**	**Production capacity (t/y)**
Carbamates	Diuron, Fluorometron	1 800 t/y
Thiocarbamates	Molinate, EPTC, Butilate, Cycloate and preparations	9 000 t/y
Chloride-acetanilides	Acetochloride, Alachloride, Propachloride and preparations	1 200 t/y

Table 1: herbicide production at the EMV (TRI) facility, North Hungary Chemical Works, Sajobabony

into the Babony stream, a tributary of the Sajo River, as it runs through the territory of the plant.

In order to evaluate the significance of current releases to the environment from the EMV plant, representatives of Greenpeace International visited the industrial site in Sajobabony in February 2002. During that visit, three samples were collected, one of water from the channel of the Babony stream, immediately downstream from its exit from the site boundary, one of sediment from the same location and the third from upstream of the site as a control sample. Details of the samples collected are presented in Table 2, and the location of the sampling points indicated in Figure 1.

All samples were returned to the Greenpeace Research Laboratories, University of Exeter (UK), for analysis of heavy metal and organic contaminants.

Materials and Methods

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.

At wastewater sampling location, a 1 litre sample was collected in a screw-cap bottle, as well as a separate 125 ml sample collected in an amber bottle with a ground-glass stopper. Sediment samples were collected in 100ml bottles.

Heavy metal concentrations were determined by ICP atomic emission spectrophotometry (AES), following acid digestion and using appropriate certified reference materials in addition to intra-laboratory standards. Organic compounds were isolated and identified as far as possible using gas chromatography and mass spectrometry, following liquid:solid extraction into a mixture of pentane and acetone for sediment samples or liquid:liquid extraction with pentane only for the water sample. In addition, the water sample was analysed for volatile organic compounds using gas chromatography and mass spectrometry. Full details of the methods for sample preparation and for metals and GC-MS screening analysis are given in Appendix 1.

Results and Discussion

A summary of the results from the qualitative organic screen analysis is presented in Table 3; groups of compounds reliably identified in each sample are listed in Table 4.

Sediment sample CEE02048, collected approximately 100m upstream from the point at which the Babony stream enters the EMV Kft. site, contained only linear aliphatic hydrocarbons. Although some influence from anthropogenic inputs of hydrocarbons (e.g. run-off from roads or urban areas) cannot be ruled out, the majority of compounds

Sample Number	Sample Description	Sample Location
CEE02048	Sediment	Babony stream, upstream from EMV Kft. (control sample)
CEE02049	Fresh water/ effluent	Babony stream, downstream from EMV Kft.
CEE02052	Sediment	Babony stream, downstream from EMV Kft. (same location as CEE02049)

Table 2. Description of samples collected around the EMV Kft. site in Sajobabony, Hungary.

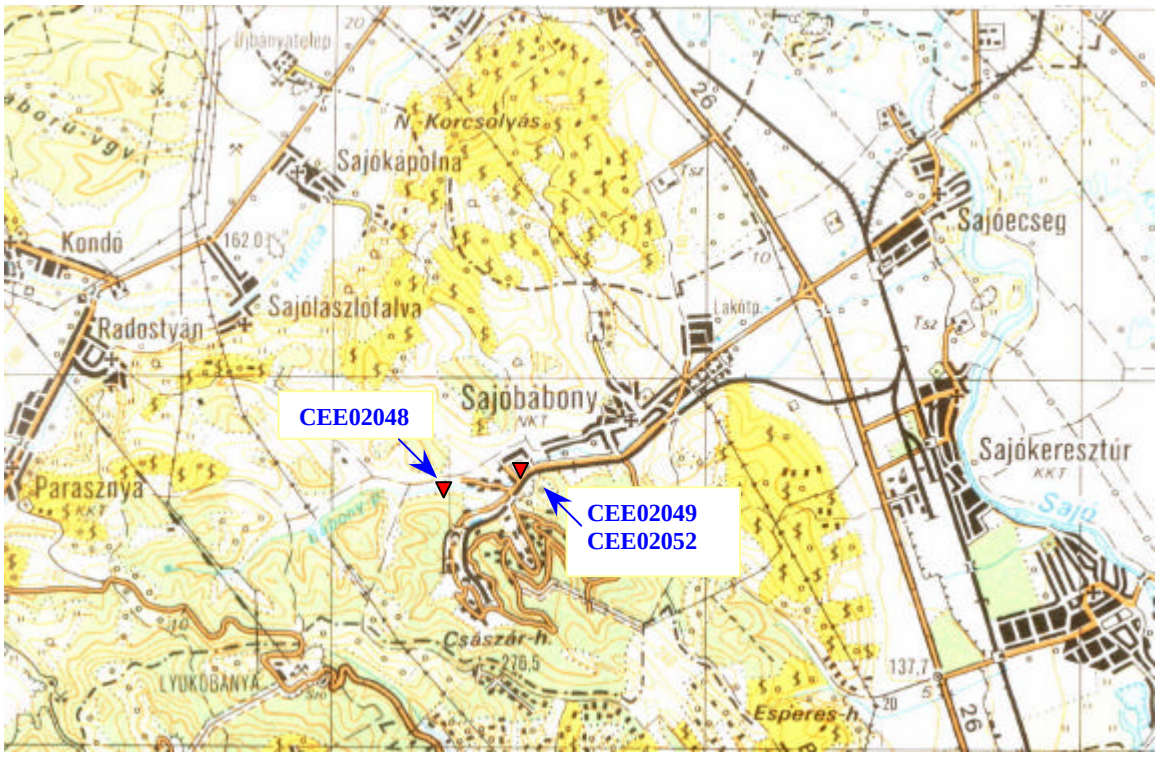

Figure 1. Location of samples collected around the EMV Kft. site, Sajobabony, Hungary.

identified could well be of natural origin. The levels of all metals found in this sample were in the range of concentrations typical for uncontaminated sediments (Salomons & Forstner 1984, Goldschmidt 1954, ATSDR 1997, Hamilton 1994).

In contrast, both samples collected from the Babony stream at the point where it leaves plant territory (water sample CEE02049 and sediment sample CEE02052) contained a

Sample code	Sample type	Number of compounds isolated	Number of compounds reliably identified	Number of compounds tentatively identified
CEE02048	sediment	35	19(54%)	13(37%)
CEE02049	Fresh water/ effluent	77	16(21%)	7(9%)
CEE02052	sediment	97	61(63%)	22(23%)

Table 3. Results of organic screening analysis for sediment samples CEE02048 and CEE02052 and fresh water/effluent sample CEE02049.

range of compounds clearly indicative of continued herbicide production and/or manufacture of other chlorinated organic compounds.

In total, six herbicides were reliably identified in the water sample CEE02049. Three of those, which were also detected in the sediment sample CEE02052, belong to the family of thiocarbamates:

- Carbamothioic acid, bis(2-methylpropyl)-, S-ethyl ester, also know as Butylate
- Carbamothioic acid, cyclohexylethyl-, S-ethyl ester, also know as Cycloate
- Carbamothioic acid, dipropyl-, S-ethyl ester, also know as EPTC.

These compounds are known selective systemic herbicides, absorbed by the roots of the plant, and are principally used to control annual grass weeds in a variety of crops. Once released into environment, these herbicides are subject to microbial degradation. However, whereas EPTC may be expected to degrade quite rapidly in aerobic soils, the degradation half-life of Cycloate in soil is between 4 and 8 weeks, while Butylate activity exhibits a half-life in the order of 4 months. Moreover, thiocarbamates are toxic to fish and, to a lesser degree, to mammals, such that releases to water and contamination of sediments could have immediate impacts, even if degradation of some active ingredients is relatively rapid.

Acute oral LD50s for Butylate have been determined as 4659 mg/kg for male rats, 5431 mg/kg for female rats and 1659 mg/kg for guinea pigs. Butylate is also a mild skin and eye irritant. Cycloate has an acute oral LD50 for male rats of 2000-3190 mg/kg and for female rats of 3160-4100mg/kg, while EPTC has an acute oral LD50 of 1652 mg/kg for male rats and 3160 mg/kg for malemics. At the same time, Butylate, Cycloate and EPTC show LC50s for rainbow trout of 4.2, 4.5 and 19 mg/l respectively (RSC 1987). Very little information exists regarding observed or potential chronic effects which may result from long-term exposure to lower doses of these chemicals. Significantly, however, a study of the toxicity of pesticides to human reproduction reported a higher risk of late spontaneous abortion (at 12-19 weeks) among women who had preconception exposure to thiocarbamate herbicides (Arbuckle *et al.* 2001).

Sample number	**CEE02048**	**CEE02049**	**CEE02052**
Description	Sediment	Fresh water/effluent	Sediment
Location	Babony stream, upstream of EMV Kft.	Babony stream, down-stream of EMV	Babony stream, down-stream of EMV
Metals	**mg/kg**	**ug/l**	**mg/kg**
Cadmium (Cd)	<1	<10	58
Chromium (Cr)	42	<20	44
Cobalt (Co)	8	<20	6
Copper (Cu)	11	<20	39
Lead (Pb)	17	<30	52
Manganese (Mn)	688	204	1050
Mercury (Hg)	0.1	<2	30.6
Nickel (Ni)	25	<20	44
Zinc (Zn)	70	41	185
HERBICIDES			
Cycloate		1	1
Butylate		1	1
EPTC		1	1
Terbutryne		1	
Prometryne		1	
Acetochlor		1	
ORGANOHALOGEN COMPOUNDS			
Ethane, 1,1-dichloro-2,2-diethoxy-		1	
Monochlorinated benzenamines		1	
Dichlorinated benzenamines			1
Dichlorobenzenes			*(2)
Trichlorobenzenes			2
Tetrachlorobenzenes			*(2)
Pentachlorobenzene			*
Hexachlorobenzene			*
Benzenemethanol, 4-chloro-, .alpha.-phenyl-			1
Benzophenone, 4-chloro-			1
PHTHALATE ESTERS			
DEHP			1
DBP			1
DiBP			1
POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)			
Anthracene			1
Benz[a]anthracene			1
Phenanthrene			1
9H-Fluorene			1
11H-Benzo[b]fluorene			1
Pyrene			1
Fluoranthene			1
OTHER AROMATIC COMPOUNDS			
Pyridine derivative		1	
Benzenamine derivatives		2	4
Alkylated benzenes			3
Phenolic compounds			2
ALIPHATIC HYDROCARBONS			
Cyclic			1
Linear	19	2	25
MISCELLANEOUS			
Fatty acid esters		2	1
Propanamine derivative		1	
Hexadecyloxirane			1

*Table 4. Organic chemicals and heavy metals identified in samples collected around EMV Kft, Sajobabony. For the groups of organic compounds: # signifies number of compounds reliably identified using general GC/MS screening method; * (#) signifies compounds identified only at trace levels using a selective ion monitoring (SIM) method, with the number of individual compounds in the identified group given in parentheses. Metal concentrations are mg/kg dry weight for solid samples and ug/l for liquid samples.*

Two other herbicides, which were detected in the water sample CEE02049 only, are representative of triazine-derived compounds:-

- 1,3,5-Triazine-2,4-diamine, N-(1,1-dimethylethyl)-N'-ethyl-6-(methylthio)-, also know as Terbutryne and
- 1,3,5-Triazine-2,4-diamine, N,N'-bis(1-methylethyl)-6-(methylthio)- , also known as Prometryne

Both compounds are also selective systemic herbicides, used in the control of most annual grasses and broad-leaved weeds in a variety of crops. Terbutryne is also used as an aquatic herbicide to control of submerged and free-floating weeds and algae in watercourses. Reported acute toxicities to mammals and fish are of the same order as for the thiocarbamate herbicides; acute oral LD50 for Prometryn in rats was 5235mg/kg, with LC50 for rainbow trout of 2.5mg/l; for Terbutryne, acute oral LD50 for rats is 2500mg/kg and LC50 for rainbow trout, 3mg/l (RSC 1986&1987). Prometryn has a relatively long half-life in soil (approximately 3 months).

One representative of the acetamide herbicides was also detected in the water sample CEE02049:

- Acetamide, 2-chloro-N-(ethoxymethyl)-N-(2-ethyl-6-methylphenyl)-, also known as Acetochlor

Acetochlor is commonly used in the control of annual grasses and some broad-leaved weeds. It is toxic to fish, bees and mammals, including humans (RSC 1986&1987). Hurley *et al.* (1998) reported that Acetochlor was among those pesticides which can produce significant incidences of thyroid, bone, granular stomach and nasal cavity tumours in rats, and of liver and lung tumours in mouse.

In addition to the herbicide residues identified above, a number of other compounds were reliably identified in the water sample CEE02049, including 1,2-dichloro-2,2-diethoxyethane, pyridine and propanamine derivatives and fatty acid esters. Very little or no information is available regarding the toxicity of these chemicals, although it is clear that some, if not all, are not of natural origin. Pyridine, for example, is primarily released to the environment from industries that make and use this chemical (ATSDR 1997).

Substituted benzenamines, also known as anilines, were also detected in the Babony stream which receives EMV Kft. wastewaters. Two alkylanilines and one chloroalkylaniline were identified in the water sample CEE02049, while sediment sample CEE02052 contained four alkylanilines and dichloroaniline. The presence of these compounds in the samples may result from their use as intermediates in the production of the nitrogen-containing herbicides.

None of the volatile organic compounds included in the current analysis (see Appendix 1) were found in water sample CEE02049 at the time of sampling.

Several chlorinated benzenes were reliably identified in the sediment sample CEE02052 including di-, tri-, tetra-, penta-, and hexachlorobenzene (although some were detected only at trace levels; see Table 3). The major current uses of chlorinated benzenes are as intermediates in organic chemical synthesis, particularly in the production of pesticides (dichlorobenzenes, trichlorobenzene, hexachlorobenzene) (Bryant 1993, Meek e*t al.* 1994, Budavari *et al.* 1989, Giddings *et al.* 1994a,b,c). They also may be formed as an unwanted by-products in the synthesis of other organochlorine compounds (Newhook & Meek 1994, Sala *et al.* 1999).

Chlorinated benzenes are among the most persistent organochlorine compounds, being highly resistant to microbial degradation. Also, di- and tri-chlorobenzenes may be formed as persistent end-products from the biodegradation of other organochlorines (Middeldorp *et al.* 1996). More information on chlorinated benzenes is present in Appendix II.

Note that chlorinated benzenes were not detected in the water sample (CEE02049) at the time of sampling. Their presence, nevertheless, in the sediment from the channel indicates past, or intermittent ongoing, release of these compounds into the Babony stream.

Seven polycyclic aromatic hydrocarbons (PAHs) were found in the sediment sample CEE02052 (collected downstream from the site), including anthracene, benz[a]antracene, phenanthrene, 9H-fluorene, 11H-benzp[b]fluorene, pyrene and fluoranthene. PAHs are a group of compounds found in coal, oil and certain chemical derivatives of such feedstocks, and are also formed as products of incomplete combustion, particularly of fossil fuels (ATSDR 1997). In addition, 25 linear aliphatic hydrocarbons were present in this sample together with three alkylated benzenes. The simultaneous occurrence of PAHs, hydrocarbons and alkylbenzenes in this sample may indicate contamination by oils or oil-based products used on site.

Like PAHs, alkylbenzenes are highly resistant to degradation and may accumulate in sediments (Preston & Raymundo 1993). Alkylbenzenes are useful markers (Chalaux *et al.* 1995) and due to their stability in sediments, they are very useful in tracing the transport of contaminants from their point sources. In terms of toxicity, acute exposure can cause central nervous system (CNS) depression. With impaired reaction times and impaired speech the two most commonly noted CNS effects (Klaassen *et al.* 1996). All alkylbenzenes can be irritating to the eyes and mucous membranes and can cause irritation and burning of the skin. All are narcotics at high concentrations (Budavari *et al.* 1989). More information on PAHs is presented in the Appendix II.

Additionally, three phthalate esters were reliably identified in the sediment sample CEE02052: diethylhexyl phthalate (DEHP), dibutyl phthalate (DBP) and diisobutyl phthalate (DiBP). These chemicals are well known environmental contaminants that have been widely used as plasticisers (Kemi 1994; Jobling *et al.* 1995). Numerous non-plasticizer uses of DEHP and DBP have also been reported, including use as a solvent in

erasable ink, in cosmetics, in vacuum pump oil, as a component of dielectric fluids in electrical capacitors, as a concrete additive, as an insect repellent, and as a solvent for perfume oils (ATSDR 1997; Jobling *et al*. 1995). It is not possible to determine from which application phthalates have arisen in this sample.

The only metal found at elevated level in the water sample CEE02049, was manganese, with the concentration about 20 times higher than background levels for uncontaminated fresh water (Bowen 1966). This is probably of little significance and is not discussed further here. Sediment sample CEE02052, however, contained several metals at concentrations exceeding background levels for sediments, in particular cadmium and mercury (about 58 and 80 times higher than background respectively) (Salomon & Forstner 1984). Elevated cadmium in the sediments of the Babony stream may reflect historical contamination from earlier manufacture of nickel-cadmium batteries. Heavy metals tend to bind to suspended material and finally accumulate in the bottom sediments for a long time (ATSDR 1997, Bryan & Langston 1992).

Heavy metals exert a broad range of toxic effects on humans, terrestrial and aquatic life and plants. A number of these metals also have the potential to bioaccumulate, including cadmium, lead, mercury and zinc (ATSDR 1997, MINDEC 1995). In addition, certain forms of cadmium have carcinogenic properties (US DHHS 2000). More information on cadmium and mercury toxicity could be found in Appendix II.

References

Arbuckle, T.E., Lin, Z. & Mery, L.S. (2001) An exploratory analysis of the effects of pesticide exposure on risk of spontaneous abortion in an Ontario farm population. Environmental Health Perspectives 109(8):851-857

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

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

Bowen, H.J.M. (1966) Trace Elements in Biochemistry. Academic press, London and New York.

Bryan, G.W. and 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

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

Chalaux N., Takada H., Bayona J.M. (1995). Molecular markers in Tokyo Bay sediments – sources and distribution. Marine Environmental Research 40 (1) : 77-92

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

Goldschmidt, V.M. (1954) Geochemistry. Oxford University Press.

Hamilton, E.I. (1998) The geobiochemistry of cobalt. The Science of the Total Environment 150: 7-39.

Howard, P. H. (1991). Handbook of environmental fate and exposure for organic chemicals. Volume I: Large production and priority pollutants. Lewis Publishers, Inc., USA

Hurley, P.M., Hill, R.N. & Whiting, R.J. (1998) Mode of carcinogenic action of pesticides inducing thyroid follicular cell tumors in rodents. Environmental Health Perspectives 106(8): 437-446

Jobling, S., Reynolds, T., White, R., Parker, M.G. & Sumpter, J.P. (1995) A variety of environmentally persistent chemicals, including some phthalate plasticizers, are weakly estrogenic. Environmental Health Perspectives 103(6): 582-587

Kemi (1994) Phthalic acid esters used as plastic additives. Publ: Swedish National Chemicals Inspectorate; report 12/94, ISSN 0248-1185

Klaassen, C.D, Amur, M.O. and Doull, J. [Eds](1996). Casarett and Doull's Toxicology: The basic science of poisons. 5th Edition, McGraw-Hill Companies Inc. IBSN 0-07-105476-6

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

Middeldorp, P.J.M., M. Jaspers, A.J.B. Zehnder and G. Schraa (1996). Biotransformation of alpha-hexachlorocyclohexane, beta-hexachlorocyclohexane, gamma-hexachlorocyclohexane, and delta-hexachlorocyclohexane under methanogenic conditions. Environmental Science & Technology 30(7): 2345-2349

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

Preston, M.R. & Raymundo, C.C. (1993) The associations of linear alkyl benzenes with the bulk properties of sediments from the River Mersey estuary. Environmental Pollution 81: 7-13

RSC (1986) Herbicides. European Directory of Agrochemical Products. Vol.2. [Ed] H. Kidd, D. Hartley & J.M. Kennedy. The Royal Society of Chemistry, ISBN 0-85186-623-9, 600pp.

RSC (1987) The Agrochemicals Handbook. Second Edition. [Ed] Hartly, D. & Kidd H. The Royal Society of Chemistry, ISBN 0-85186-416-3

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

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

US DHHS (2000) 9TH Report on Carcinogens.

Appendix I: Analytical methodology

A1.1 Organic analysis

A1.1.1 Preparation of samples for organic screen analysis

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

A1.1.1.1 Solid Samples

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

Extracts were decanted, filtered through a pre-cleaned hydrophobic phase separator filter and collected in reagent tubes. 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 concentrated extract and the mixture shaken for 1 min. After shaking, 20ml of deionised water was added to reagent tube and the phases were allowed to separate. Finally, the organic layer was transferred with a Pasteur pipette into a pentane pre-washed Florisil column. The compounds were eluted with a 95:5 mixture of pentane: toluene, and the elluent 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.

A1.1.1.2 Aqueous Samples

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

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

A1.1.2 Chromatographic Analysis

Organic compounds were identified qualitatively using Gas Chromatography Mass Spectrometry (GC-MS). Instrumentation was a Agilent 6890 Series gas chromatograph, interfaced with a Agilent Enhanced Chem-Station data system and linked to a Agilent 5973 Mass Selective Detector operated in SCAN mode. The identification of compounds was carried out by computer matching against Agilent Wiley7N and Pesticides Libraries of over 390,000 mass spectra combined with expert interpretation. Also all extracts were analysed using selective ion monitoring (SIM) method against two standard solutions. The lists of compounds containing in Standard I and Standard II are presented below. All individual standards were obtained from Sigma Aldrich Co. Ltd., Supelco, UK.

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
Atrazine	200, 215, 202, 217
beta-HCH	181, 183, 219, 217
gamma-HCH	181, 183, 219, 217
delta-HCH	181, 183, 219, 217
o,p'-DDE	246, 248, 318, 176
p,p'-DDE	246, 318, 246, 316
o,p'-DDD	235, 237, 165, 199
p,p'-DDD	235, 237, 165, 199
o,p'-DDT	235, 237, 165, 199
p,p'-DDT	235, 237, 165, 199

Table 1. List of compounds in the Standard I used for SIM analysis

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

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

PCB-156	360, 362, 290, 358
PCB-157	360, 362, 290, 358
PCB-180	396, 394, 324, 162
PCB-169	360, 362, 358, 145
PCB-170	396, 394, 324, 326
PCB-189	396, 394, 398, 324

Table 2 List of compounds in the Standard II used for SIM analysis

A1.1.3 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, if required. 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

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

A1.2. Heavy Metal Analysis

A1.2.1. Preparation of samples for heavy metal analysis

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

A1.2.1.1. Solid Samples

Samples were air dried until weighing readings became constant (approx. 5 days). They were then crushed using a pestle and mortar until homogenous and sieved through a 2-mm mesh.

0.5 g of sample was weighed into a glass 100 ml boiling tube and to this 10 ml of deionised water was added, followed by 7.5 ml of concentrated hydrochloric acid and 2.5 ml of concentrated nitric acid. The samples were digested at room temperature overnight prior to being placed onto a Gerhardt Kjeldatherm digestion block (40 space) connected to a Gerhardt Turbosog scrubber unit (filled with 10% w/v sodium hydroxide). The samples were then refluxed at 130°C for four hours.

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

A1.2.1.2. Aqueous samples

On arrival, 100ml of sample was transferred to a clean glass bottle and acidified with nitric acid (10% v/v). 50 ml of this solution was subsequently transferred to a 100ml boiling tube, placed onto the Gerhardt Kjeldatherm digestion block, and refluxed at 130°C for four hours. After cooling to ambient temperature, the digests were filtered into volumetric flasks, diluted with deionised water, made up to a volume of 50 ml and mixed.

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

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

reagent stocks. Any sample exceeding the calibration range was diluted accordingly, in duplicate, and re-analysed.

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

Appendix II: Toxicological outlines for key contaminants identified

A2.1 Polycyclic aromatic hydrocarbons (PAHs)

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

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

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

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

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

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

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

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

References

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

Williams G.M., Weisburger J.H. (1986). Chemical carcinogens. In Toxicology: the basic science of poisons. Klaasen C.D., Ambur M.O. and Doull J. [Eds], MacMillan Publishing Co., New York: 99-173.

A2.2 Phthalate esters

Phthalate esters are more often referred to as the phthalates. They are used in every major product category (Kemi 1994). 90% of all plasticizers is used in the production of soft PVC (Cadogan *et al.* 1993) but they are also used in inks and dyes (Jobling *et al.* 1995), which is of particular relevance here. Diethylhexyl phthalate (DEHP), di-n-butyl phthalate (DnBP or DBP) and diisobutyl phthalate (DiBP) were detected in this survey. Ten to fifteen years ago DEHP and DnBP were the phthalates produced in the greatest quantities (Menzert & Nelson 1986).

Phthalates are persistent in the environment and are the most abundant man-made chemicals in the environment (Jobling *et al.* 1995). They can also bioaccumulate to some degree, predominantly from food. The phthalates exhibit a wide range of toxic effects in laboratory animals. The summary below illustrates the scope of the toxicological problems associated with these phthalates, although no data were available for DiBP.

DEHP can cause liver cancer in laboratory animals and has been classified as possibly carcinogenic to humans by the IARC; as a probable human carcinogen by the USEPA and the US Department of Health and Human Services (DHHS) has determined that DEHP may reasonably be anticipated to be a carcinogen (ATSDR 1997). In its scientific opinion expressed in November 1998, the European Commission's Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE 1998) noted that the most

sensitive effect of DEHP may be damage to the development of the testes, based on tests involving exposure of rats to relatively low concentrations both in the womb and for the first three weeks after birth. The Committee also judged that such testicular toxicity may have greater relevance for humans than carcinogenic effects.

More recently concern has been raised about the ability of DEHP and some other phthalates to interact with hormone receptors in animals. Jobling and coworkers (1995) demonstrated that DEHP was able to bind to the human estrogen receptor, although it showed no significant estrogenic activity. Its potential to interfere with other aspects of the hormone system has not been fully investigated.

DEHP and DnBP can both damage the male and female reproductive systems (Chan & Meek 1994, ATSDR 1997). Both can damage sperm production (ATSDR 1997, Wine *et al.* 1997), impair reproductive success (Chan & Meek 1994, Ema *et al.* 1995, ATSDR 1997, Wine *et al.* 1997) and cause teratogenicity (malformation of the offspring)(Chan & Meek 1994, Ema *et al.* 1993; Ema *et al.* 1995, ATSDR 1997).

The liver and kidneys can be affected by DnBP (Chan & Meek 1994; ATSDR 1997) and DEHP (ATSDR 1997).

A group of phthalate esters including DnBP and DEHP has been found to have both acute (Adams *et al.* 1995) and chronic (Rhodes *et al.* 1995) toxicity to the representatives of freshwater and marine species, although toxicity may have been limited to some degree by the poor water solubility of these compounds. There was a general trend for the lower-molecular- weight phthalate esters (C-1 to C-4 alkyl chain lengths) to become more toxic with decreasing water solubility for all species tested.

Because of their recognised toxicity and widespread distribution, these two phthalates (DBP and DEHP) are included on the OSPAR List of Chemicals for Priority Action (Annex 2 to the OSPAR Strategy with Regard to Hazardous Substances, OSPAR 1998).

References

Adams, W.J., Biddinger, G.R., Robillard, K.A. & Gorsuch, J.W. (1995) A summary of the acute toxicity of 14 phthalate esters to representative aquatic organisms. Environmental toxicology and chemistry 14(9): 1569-1574

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

Cadogan, D.F., Papez, M., Poppe, A.C., Pugh, D.M. & Scheubel, J. (1993) An assessment of release, occurrence and possible effects of plasticisers in the environment. IN: PVC 93 The Future. Proceedings of Conference held on 27-29 April 1993, Brighton Metropole Hotel, Brighton, UK. Published by the Institute of Materials, pp260-274

Chan, P.K.L. & Meek, M.E. (1994) Di-n-butyl phthalate: Evaluation of risks to health from environmental exposure in Canada. Environ. Carcino. & Ecotox. Revs. C12(2): 257-268

CSTEE (1998) Opinion on phthalate migration from soft PVC toys and child-care articles - data made available since the 16th June 1998. European Commission Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE), opinion expressed at the 6th CSTEE plenary meeting, Brussels, 26/27 November 1998.

Ema, M., Itami, T. & Kawasaki, H. (1993) Teratogenic phase specificity of butyl benzyl phthalate in rats. Toxicology 79: 11-19

Ema, M., Kurosaka, R., Amano, H. & Ogawa, Y. (1995) Comparative developmental toxicity of n-butyl benzyl phthalate and di-n-butyl phthalate in rats. Arch Environ. Contam. Toxicol. 28: 223-228

Jobling, S., Reynolds, T., White, R., Parker, M.G. & Sumpter, J.P. (1995) A variety of environmentally persistent chemicals, including some phthalate plasticizers, are weakly estrogenic. Environmental Health Perspectives 103(6): 582-587

Kemi (1994) Phthalic acid esters used as plastic additives. Publ: Swedish National Chemicals Inspectorate; report 12/94, ISSN 0248-1185

Menzert, R.E. & Nelson, J.O. (1986) Water and soil pollutants. In Toxicology: the basic science of poisons. Klaasen C.D., Ambur M.O. and Doull J. [Eds], MacMillan Publishing Co., New York: 825-856.

OSPAR (1998) OSPAR Stategy with Regard to Hazardous Substances. OSPAR 98/14/1 Annex 34. OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic.

Rhodes, J.E., Adams, W.J., Biddinger, G.R., Robillard, K.A. & Gorsuch, J.W. (1995) Chronic toxicity of 14 phthalate esters to Daphnia-Magna and Rainbow-Trout (*Oncorhynchus-Mykiss*). Environmental toxicology and chemistry 14(11): 1967-1976

Wine, R.N., Li, L-H., Barnes, L.H., Gulati, D.K., & Chapin, R.E. (1997) Reproductive toxicology of di-n-butylphthalate in a continuous breeding protocol in Sprague-Dawley rats. Environmental Health Perspectives 105(1): 102-107

A2.3 Chlorinated benzenes

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

12 chlorinated benzenes are possible, with substitution patterns as follows:

1 chlorine monochlorobenzene,
2 chlorines 1,2-di-, 1,3-di- and 1,4-dichlorobenzenes
3 chlorines 1,2,3-tri-, 1,2,4-tri- and 1,3,5-trichlorobenzenes
4 chlorines 1,2,3,4-tetra-, 1,2,3,5,-tetra- and 1,2,4,5-tetrachlorobenzenes
5 chlorines pentachlorobenzene
6 chlorines hexachlorobenzene.

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

Mono- and di-chlorobenzenes

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

One of the earliest uses of chlorobenzene was as an intermediate for the explosive picric acid during the first World War (CEC 1986). It is used as a solvent and as an intermediate in chemical synthesis. In the US in the 1980s, the predominant use was for the production of ortho- and para-chlorobenzenes. Theses are used as intermediates for rubber chemicals, antioxidants, dyes and pigments, pharmaceuticals and agricultural chemicals. The fungicide benomyl, and carbofuran and the parathion group of insecticides are all derived from chlorobenzene. One previously important use was in the manufacture of DDT. Chlorobenzene production has fallen due to the development of other routes to aniline and phenol and the restriction of DDT use. By various routes, chlorobenzene is also used for the manufacture of specialty silicones, Grignard reagents and catalysts (Bryant 1993). Release to the environment is expected to derive from its use as a solvent, either through fugitive emissions or volatilisation from pesticides for which it is used as a carrier. Thus, inhalation is thought to be a major route of exposure for humans since it is rarely if ever found in food. It bioaccumulates in algae, fish and aquatic invertebrates. Mammalian metabolites are reported to be p-chlorophenol, p-chlorocatechol and p-chlorophenyl mercapturic acid. Human exposure causes CNS depression and respiratory tract irritation and animal studies have reported liver necrosis, renal toxicity and effects on the pancreas, blood and lymph and adrenal glands (Ware 1988a, Meek *et al.* 1994a). Canada has derived a TDI of 8.1ug/kg body weight/day; estimated exposures (0.05-0.14ug/kg/day) are considerably lower than this (Meek *et al.* 1994a).

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

dichlorobenzenes are bioaccumulative in algae, aquatic invertebrates and fish (Ware 1988b). All three have also been reportedly found in blood (Ware 1988b).

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

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

1,3-Dichlorobenzene is growing in importance as a starting product in the manufacture of dyes, pesticides and pharmaceuticals. However, this has not yet reached commercial importance. There are some other small, specialised uses, but larger markets have not been developed, mainly because 1,3-dichlorobenzene only occurs as a minor constituent (approx 1%) of the technical dichlorobenzene reaction mix, and to produce it by other routes is expensive (Bryant 1993). Mammalian (and human) metabolism is as for 1,2-dichorobenzene above, but generally little is known about this 1,3-dichlorobenzene in comparison to the more commercially important dichlorobenzenes.

1,4-Dichlorobenzene (p-dichlorobenzene) is used largely in the production of deodorant blocks and room deodorants. It is also used as a moth control agent, as an insecticide and an intermediate for production of insecticides and dyes. An emerging market is in the manufacture of poly(phenylene sulphide) resin (PPS), and minor uses are as a germicide, fungicide and extreme pressure lubricant (Bryant 1993, CEC 1986). 1,4-dichlorobenzene is not spontaneously combustible and does not assist fire, but it is flammable nevertheless. It may be absorbed both through the inhalation of vapours, through the skin and though consumption of contaminated food. Human symptoms include damage to the liver, kidneys and lungs. Accidental poisoning of children, presumably who have eaten moth repellent was widespread in the 1970s (CEC 1986). Once absorbed, 1,4-dichlorobenzene is stored in the adipose tissue, and has been detected in human samples (CEC 1986, Ware 1988b). The metabolism of 1,4-dichlorobenzene by mammals varies from that of the other two isomers in that mercapturic acids are not formed. 1,4-

dichlorobenzene causes abnormal mitosis in higher plants. 1,4-Dichlorobenzene has been reported in human adipose tissue, as well as in blood (Ware 1988b).

Trichlorobenzenes

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

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

The general human population would probably receive their greatest exposure to trichlorobenzenes through inhalation. The toxicity of all three appear similar; they damage the liver, kidney and thyroid. There is some indication of slight fetotoxicity at high doses. There is little evidence of mutagenicity and too few data are available for the trichlorobenzenes to given a carcinogenicity classification (Giddings *et al.* 1994a). All three isomers are toxic to phytoplankton (Sicko-Goad *et al.* 1989a-d, Sicko-Goad & Andresen 1993a & b).

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

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

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

Tetrachlorobenzenes

Giddings *et al.* (1994b) reviewed toxicity and exposure data for the tetrachlorobenzenes. They are no longer used or produced in Canada and releases come only from dielectric fluid spills and long-range transport. 1,2,4,5-Tetrachlorobenzene used to be used in the production of 2,4,5-trichlorophenol on a large scale, but this use has now been largely discontinued. There are not expected to be large differences between the behaviour of the isomers. Uptake of 1,2,4,5-tetrachlorobenzene was studied in rainbow trout. It is not volatile enough to evaporate from water easily, and is accumulated by the fish, through its gills. Bioaccumulation depended upon the rate of activity and oxygen uptake of the fish, and only the low water solubility prevented significant toxicity occurring (Brauner *et al.* 1994).

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

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

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

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

Pentachlorobenzene

Giddings *et al.* (1994c) found that though no longer manufactured or used in Canada, pentachlorobenzene could still enter the environment through spillage of dielectric fluids or atmospheric transport. Animal studies demonstrate weight loss and effects on the liver, thymus, kidney, adrenal glands and digestive tract. Anaemia and malformation of sperm also occurred. There is some indication of fetotoxicity and developmental toxicity. The thyroid was impacted, with thyroid hormone (free and total thyroxin) concentrations reduced. Pentachlorobenzene cannot be assigned a carcinogenicity classification because of lack of data. Pentachlorbenzene accumulates in, and is toxic to algae (Sicko-Goad *et al.* 1989d).

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

Hexachlorobenzene

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

HCB is toxic to aquatic life, land plants, land animals, and humans. It is listed by the IARC as a Group 2B carcinogen, i.e. possible carcinogen to humans and also appears to be a tumour promoter. Hexachlorobenzene may damage the developing foetus, liver, immune system, thyroid and kidneys and CNS. The liver and nervous system are the most sensitive to its effects. Porphyria is a common symptom of HCB toxicity. High or repeated exposure may damage the nervous system and can cause irritability, difficulty with walking and co-ordination, muscle weakness, tremor and/or a feeling of pins and needles on the skin. Repeated exposure, especially when skin effects occur, can lead to permanent skin changes, such as changes in pigmentation, tight, thickened skin, easy wrinkling, skin scarring, fragile skin, and increased hair growth, especially on the face and forearms (ATSDR 1997, Newhook & Meek 1994, van Birgelen 1998). Recent research (van Birgelen 1988) suggests that HCB has dioxin-like toxicity and that, based on a preliminary toxic equivalence factor (TEF) of 0.0001, HCB could contribute significantly to the dioxin-type toxicity of human milk based on PCB/PCDD/PCDF toxicity equivalents. In many countries, this could mean an increase of 10% - 60%, but in countries with high HCB exposure levels, the effects could be even greater. In Spain and the Czech Republic, inclusion of HCB in total breastmilk TEQ estimates could lead to totals 6 times higher than based only on PCBs and PCDFs. Slovakia and India also have very high HCB levels; other countries (eg Austria) high levels in previous decades. It has been suggested that more epidemiological studies should be undertaken, especially in the most highly contaminated countries.

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

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

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

HCB is one of twelve priority POPs intended for global action by the UN Environment Programme (UNEP) Governing Council. It is intended that HCB will be phased out worldwide under the proposed POPs Convention (UNEP 1995, 1997), which is expected to be signed in Stockholm in May 2001. Furthermore, HCB is included on Annex I of the Draft UNECE POPs Protocol under the Convention on Long-Range Transboundary Air Pollution (LRTAP)(UNECE 1998).

Within the EC, discharges of HCB are controlled as stipulated by EC Council Directive 86/280/EEC, which amends Directive 76/464/EEC, regarding pollution caused by certain dangerous substances discharged into the aquatic environment (EC 1986, 1976).

HCB is also included in the list of priority hazardous substances agreed by the Third and Fourth North Sea Conferences (MINDEC 1990 & 1995), where continuous reduction of all hazardous substances was agreed with the ultimate aim of reducing environmental concentrations of hazardous substances to near background levels (synthetic substances to zero) within the next 25 years. The 1998 Ministerial Meeting of the OSPAR Commission (OSPAR 1998a) further reinforced these objectives. HCB is included on the OSPAR 1998 List of Candidate Substances, Annex 3 of the OSPAR Strategy with regard to Hazardous Substances (OSPAR 1998b). In addition, HCB is regulated under the 1995 Barcelona Convention, the Rotterdam (PIC) Convention and the International Joint 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.)(1988) Chlorobenzene. Rev. Environ. Contam.Toxicol. 106: 37-49

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

A2.4 Cadmium

A2.4.1 Environmental Contamination and Behaviour

Cadmium is more mobile in aquatic environments than most other metals. It is also bioaccumulative and persistent in the environment ($t^{1/2}$ of 10-30 years) (USPHS 1997). It is found in surface and groundwater as either the +2 hydrated ion, or as an ionic complex with other inorganic or organic substances. While soluble forms may migrate in water, cadmium in insoluble complexes or adsorbed to sediments is relatively immobile. Similarly, cadmium in soil may exist in soluble form in soil water, or in insoluble complexes with inorganic and organic soil constituents (USPHS 1997, WHO 1992). Furthermore, cadmium is readily available for uptake in grain, rice and vegetables, and there is a clear association between the cadmium concentration in soil and the plants grown on that soil (Elinder and Jarup 1996, Cabrera et *al.* 1994, WHO 1992).

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

A4.1.2. Toxicity

Cadmium has no biochemical or nutritional function, and it is highly toxic to both plants and animals (USPHS 1997, WHO 1992, Alloway 1990). In humans and animals, there is strong evidence that the kidney is the main target organ of cadmium toxicity, following extended exposure (USPHS 1997, Elinder and Jarup 1996, Goyer 1996, Roels *et al.* 1993, Iwata *et al.* 1993, WHO 1992, Mueller *et al.* 1992). Renal damage includes tubular proteinuria (the excretion of low molecular weight proteins) and a decrease in the glomerular filtration rate. The latter results in a depressed re-sorption of enzymes, amino acids, glucose, calcium, copper, and inorganic phosphate. Furthermore, studies have shown that even when cadmium exposure ceases, proteinuria does not decrease, and renal tubular dysfunction and reduced glomerular filtration increase in severity (USPHS 1997, Jarup *et al.* 1997, Elinder and Jarup 1996, Goyer 1996, Iwata *et al.* 1993, WHO 1992, Nriagu 1988).

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

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

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

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

Regarding plant toxicity, adverse effects on plant growth and yield have been reported. Alloway (1990) reported stunted growth and toxic signs on leaves of lettuce, cabbage, carrot and radish plants, (which resulted from a cadmium content of around 20 mg/kg in

the upper parts of the plants). Other studies have shown reductions in the rates of photosynthesis and transpiration (WHO 1992).

Regarding the toxicity of cadmium to aquatic organisms, numerous findings have been reported. For example, some species of phytoplankton are very sensitive to cadmium, with inhibition of growth observed at concentrations as low as 1 ug/l (Bryan and Langston 1992). Deleterious effects have also been reported in limpets, where correlations between increased levels of cadmium and reduced ability to utilise glucose were found. Reductions in reproduction rates and population numbers in copepods and isopods have been shown at concentrations as low as 5 ug/l, and exposure to similar levels has resulted in changes in the immune function in some fish, and depressed growth in juvenile fish and invertebrates (Bryan and Langston 1992, Thuvander 1989). Furthermore, the toxicity of low sediment-cadmium concentrations has also been suggested following observations in San Francisco Bay. Here the condition of certain species of clam declined as cadmium concentrations rose from 0.1 to 0.4 mg/kg (Bryan and Langston 1992).

References

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

Cabrera, C., Ortega, E., Gallego, C., Lopez, M.C., Lorenzo, M.L. and Asensio, C. (1994). Cadmium concentration in farmlands in southern Spain: possible sources of contamination. The Science of the Total Environment 153: 261-265

Elinder, C.G. and Jarup, L. (1996). Cadmium exposure and health risks: recent findings. Ambio 25, 5: 370-373

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

IARC (1998). Cadmium and certain cadmium compounds. In: IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Chemicals, industrial processes and industries associated with cancer in humans. IARC monographs, Vol. 1 to 29

Iwata, K., Saito, H., Moriyama, M. and Nakano, A. (1993). Renal tubular function after reduction of environmental cadmium exposure: a ten year follow-up. Archives of Environmental Health 48, 3: 157-163

Jarup, L., Persson, B. and Elinder, C.G. (1997). Blood cadmium as an indicator of dose in a long-term follow-up of workers previously exposed to cadmium. Scandinavian Journal of Work Environment and Health 23, 1: 31-36

Mueller, P.W., Paschal, D.C., Hammel, R.R., Klincewicz, S.L. and MacNeil, M.L. (1992). Chronic renal effects in three studies of men and women occupationally exposed to cadmium. Arch. Environ. Contam. Toxicol. 23: 125-136

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

Roels, H., Bernard, A.M., Cardenas, A., Buchet, J.P., Lauwerys, R.R., Hotter, G., Ramis, I., Mutti, A., Franchini, I., Bundshuh, I., Stolte, H., De Broe, M.E., Nuyts, G.D., Taylor, S.A. and Price, R.G. (1993). Markers of early renal changes induced by industrial pollutants. III. Application to workers exposed to cadmium. British Journal of Industrial Medicine 50: 37-48

Thuvander, A. (1989). Cadmium exposure of rainbow trout, Salmo gairdneri Richardson: effects on immune functions. J. Fish Biol. 35: 521-529

USPHS (1997). Toxicological profile for cadmium on CD-ROM. Agency for Toxic Substances and Disease Registry

USPHS (1998). 8th Report on Carcinogens 1998 Summary

World Health Organisation (1992). Cadmium. Environmental Health Criteria 135. ISBN 9241571357

A2.5 Mercury

A2.5.1. Environmental Contamination and Behaviour

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

In the aquatic environment, mercury is most commonly found in the mercuric (II) state, and its fate, once released, is dominated by rapid adsorption to soluble and particulate organic material; followed by flocculation, precipitation and final accumulation in the bottom sediment. Because of the strength with which mercury is bound to sediment, exchange back to the water column is generally slight, although it can be accelerated in saline waters, and in the presence of high concentrations of sulphide (anoxic conditions) (USPHS 1997, Bryan and 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 and Langston 1992).

Inorganic mercury can be methylated by micro-organisms, indigenous to soils, fresh water and marine sediments. The most common form of organic mercury is methylmercury (MeHg), which is soluble, mobile, and quick to enter the aquatic food chain. The selective retention of MeHg at each step in the food chain, relative to inorganic mercury, is related to its high lipid solubility, its long biological half-life, and the increased longevity of top predators (Bryan and Langston 1992). As a result, MeHg provides one of the rare examples of metal biomagnification in food chains (USPHS 1997, WHO 1989). For example, concentrations in carnivorous fish at the top of freshwater and salt water food chains (e.g., pike, tuna, and swordfish) are biomagnified 10,000-100,000 times the concentrations found in ambient waters (USPHS 1997). The significance of this bioaccumulation is that it is generally the most important source of human, non-occupational mercury exposure (USPHS 1997, WHO 1989).

A2.5.2. Toxicity

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

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

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

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

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

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

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

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

References

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

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. and 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

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

Naimo, T.J. (1995). A review of the effects of heavy metals on freshwater mussels. Ecotoxicology 4: 341-362

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

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

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

Filename:	EMV technical note.doc
Directory:	I:\GRL\CEE sampling tour\Reports
Template:	C:\WINDOWS\Application Data\Microsoft\Templates\Normal.dot
Title:	Sample number
Subject:	
Author:	Greenpeace Research Labs
Keywords:	
Comments:	
Creation Date:	11/06/02 09:11
Change Number:	2
Last Saved On:	11/06/02 09:11
Last Saved By:	Exeter Office
Total Editing Time:	1 Minute
Last Printed On:	11/06/02 09:27

As of Last Complete Printing

Number of Pages:	36
Number of Words:	13,031 (approx.)
Number of Characters:	74,280 (approx.)