RELATÓRIO

Pollution with organic tin compounds, organochlorines, hydrocarbons and metals in sediment samples from Guanabara Bay, Rio de Janeiro, Brazil.

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1 EXECUTIVE SUMMARY

During August 2000, fourteen samples of sediment were collected from the Baia de Guanabara and some of the rivers that feed into it. These were analysed for heavy metals and screened to identify as many organic contaminants as possible. In addition, six selected samples were analysed for organotin compounds.

The results demonstrate that the bay is subject to pollution by a wide range of toxic compounds from a variety of sources. Overall, the south and east of the bay are the worst affected areas, although the north and east are not entirely unaffected. All sites were polluted with hydrocarbons and 1,4-dichlorobenzene, often sound in sewage discharges, was detected at almost all sites.

Organotins wer detectable in all samples analysed, including the "pristine" north-east of the bay. The predominant source of these identified were the naval and docking facilities in the south of the bay, though some containation could result from normal shipping traffic. organotins have long been known to have a severe impact on aquatic ecosystems.

Some other metallic contaminants, particularly copper, lead and zinc, were also present at significant concentrations in the dock areas. Moreover, lead appears to be entering the bay in runoff from the city and there is evidence of riverine input of mercury and zinc. The mouth of the Rio Iguaçu exhibits elevations in chromium and nickel, which may be associated with petroleum refining.

Organochlorine pollution is evident at most of the sites sampled, with chlorinated benzenes, PCBs and pesticides or their derivatives accounting for almost all of the contamination. Again, the dock ares were among the most polluted, particularly with PCBs and chlorobenzenes. PCBs and organochlorine pesticides (DDT and HCH) may also be entering the bay via the rivers

2 INTRODUCTION

The Baia de Guanabara is the natural harbour by which the city of Rio de Janeiro is located. Situated within the harbour are a number of islands, the largest of which is the Ilha do Governador on the western side of the bay. Much of the bay is extremely shallow, with depths at high tide of only a metre or two. However, deeper water extends from the channel that connects the bay to the Atlantic Ocean, up to the east of the Ilha do Governador.

The northern and eastern coasts of the bay are sparsely populated and the north-eastern extreme is edged with mangroves. The city of Rio de Janeiro is sited on the western edge of the bay. The Baia de Guanabara is also home to most of the city's port and harbour facilities. These include Navy facilities at the Ilha das Cobras and (naval) shipyards, which are situated in the southern extreme of the bay.

The city discharges sewage to the bay, some of which is discharged untreated to the rivers that run through it. Rivers and sewers will also carry urban run-off into the bay and some of the rivers are known to receive industrial effluents. In addition, large-scale oil refining and transportation activities take place in and around the bay and have been responsible for significant spills in recent years.

3 SAMPLING PROGRAM

During August 2000, a series of fourteen sediment samples were collected from the bay and some of the rivers that feed into it. Locations of the sampling sites are shown in Figure 1 and brief descriptions of the samples are given in Table 1. Samples were collected from all sections of the bay, to ascertain the pollution status of the "pristine" mangrove-lined areas of the north-eastern bay as well as the areas impacted by the city and the input from the rivers and the docks.

Samples were returned to the Greenpeace Research Laboratories for analysis. There they were analysed for organic contaminants using GC/MS techniques and for heavy metals with ICP. Details of the analytical methods are given in Appendix 1. In addition to the analysis of these samples for heavy metals and screening them qualitatively for the presence of organic contaminants, a sub-set of six samples were selected for quantification of organic tin compounds (including the marine antifoulant tributyltin, TBT). These analyses were carried out by GALAB laboratories, Germany (see also Appendix 1).

These six samples for organotin analysis were selected in order to represent areas of the bay close to key areas of shipping and docking activities (to the south), areas more distant from such activities (to the north) and an intermediate area in the central part of the bay. The samples selected for TBT analysis are indicated with the symbol # in Table 1.

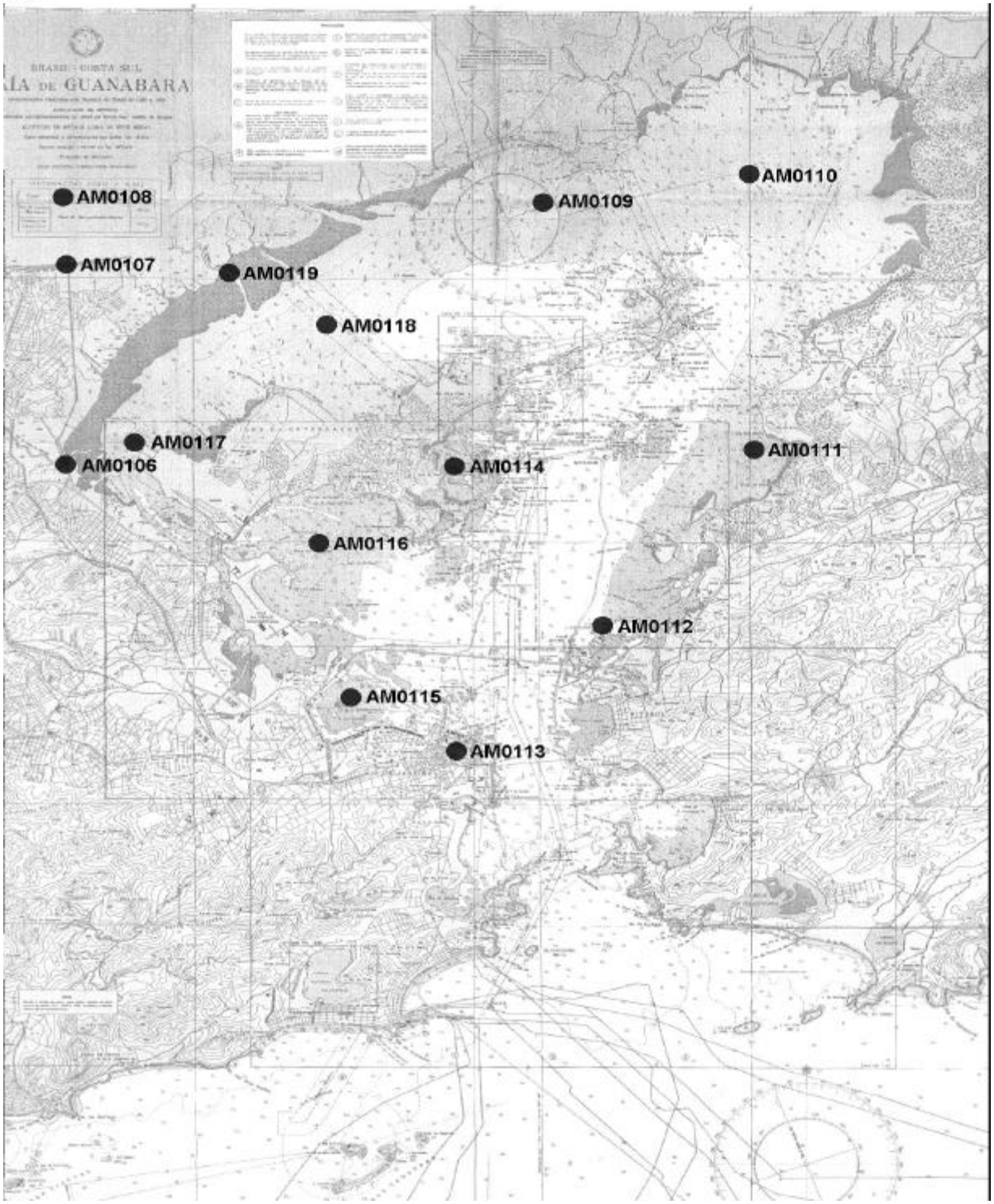


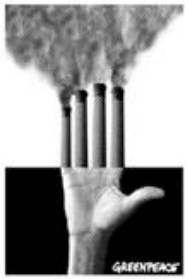
Figure 1. Location of samples collected from the Baia de Guanabara and some of th rivers that feed into it.

Sample number	Location	Coordinates
AM0106	west (Rio São João de Meriti)	S 22° 48.102’ W 43° 17.386’
AM0107	west (Rio Sarapui)	S 22° 44.739’ W 43° 17.278’
AM0108	north-west (Rio Iguaçu)	S 22° 43.474’ W 43° 17.334’
AM0109	north	S 22° 43.645’ W 43° 08.817’
AM0110#	north-east	S 22° 43.048’ W 43° 05.007’
AM0111	east	S 22° 48.301’ W 43° 04.998’
AM0112#	south east (near shipyards)	S 22° 51.651’ W 43° 07.665’
AM0113	south west (near Snake Island Naval dockyard)	S 22° 54.027’ W 43° 10.360’
AM0114	central (east of Ilha do Governador)	S 22° 48.536’ W 43° 10.281’
AM0115#	south west (near city centre)	S 22° 52.982’ W 43° 12.229’
AM0116#	central (south of Ihla do Governador)	S 22° 50.001’ W 43° 12.785’
AM0117	west (near mouth of Rio S. João de Meriti)	S 22° 48.197’ W 43° 16.088’
AM0118#	north west (near mouths of rivers inc Sarapui)	S 22° 46.021’ W 43° 12.602’
AM0119	north west (near mouth of Rio Iguaçu)	S 22° 44.906’ W 43° 14.333’

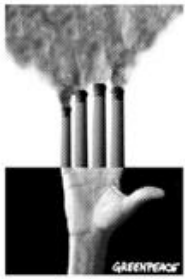
Table 1. Description of samples collected from the Baia de Guanabara in August 2000. All samples were analysed for metals and organic compounds. # denotes samples also analysed for organotin compounds.

4 RESULTS AND DISCUSSION

The results of the organic screen analysis and heavy metals analysis of the are presented in Table 2, including a breakdown of the groups of organic compounds reliably identified in the samples. For more information on the common sources, environmental behaviour and toxicological outlines for key pollutants detected during this study see Appendix 2.



Sample Number	AM0106	AM0107	AM0108	AM0109	AM0110	AM0111	AM0112	AM0113	AM0114	AM0115	AM0116	AM0117	AM0118	AM0119
Cadmium (mg/kg dry weight)	2	1	<1	<1	<1	<1	<1	<1	<1	2	<1	2	<1	1
Chromium (mg/kg dry weight)	109	157	54	195	45	51	48	66	31	83	115	96	154	256
Cobalt (mg/kg dry weight)	8	9	9	8	9	9	7	7	4	10	7	9	8	13
Copper (mg/kg dry weight)	132	85	9	103	24	36	163	120	18	98	32	158	64	95
Lead (mg/kg dry weight)	111	63	16	49	16	22	52	115	15	135	28	137	51	59
Manganese (mg/kg dry weight)	361	422	456	820	466	715	316	194	174	271	154	234	549	255
Mercury (mg/kg dry weight)	6.08	0.86	0.27	0.14	0.34	0.35	0.53	1.12	0.21	0.67	0.13	4.07	0.56	0.32
Nickel (mg/kg dry weight)	41	39	18	41	19	17	18	19	9	25	17	46	20	70
Zinc (mg/kg dry weight)	900	444	85	192	118	164	235	350	84	65	92	986	236	312
No. of organic compounds isolated	117	77	61	27	46	10	146	104	59	67	49	83	52	73
No. of organics reliably identified	35(30%)	12(16%)	18(30%)	13(48%)	9(20%)	4(40%)	64(44%)	42(36%)	9(15%)	13(19%)	15(31%)	12(14%)	4(8%)	10(14%)
Groups reliably identified														
ORGANOHALOGENS								*						
Benzene, 1,2-dichloro-							*				*			
Benzene, 1,4-dichloro-	*	*	*	1	*	*	*	*	*	*	*	1		1
Benzene, 1,2,3-trichloro-							*	*						
Benzene, 1,2,4-trichloro-							*	*						
Benzene, 1,3,5-trichloro-							*	*						
Benzene, 1,2,3,5-tetrachloro-							*	*						
Benzene, 1,2,3,4-tetrachloro-							*	*						
Benzene, hexachloro-											*			
Trichlorobiphenyl isomers	*(2)						*(3)	*(2)						
Tetrachlorobiphenyl isomers	*						*(6)	*						
Pentachlorobiphenyl isomers	*(7)						*(18)	*(7)						
Hexachlorobiphenyl isomers (other than 138&153)	*(8)						*(11)	*(4)						
Heptachlorobiphenyl isomers (other than 180)	*(2)						*(3)							
PCB-138	*						*	*						
PCB-153	*						*	*						



Sample Number	AM0106	AM0107	AM0108	AM0109	AM0110	AM0111	AM0112	AM0113	AM0114	AM0115	AM0116	AM0117	AM0118	AM0119
PCB-180	*						*	*						
1,1'-Biphenyl, 4,4'-dichloro-											1			
p,p'-DDE		*	*											
p,p'-DDD		*	*											
p,p'-DDT		*	*											
beta-HCH		1	*	1										
Phenol, 2,5-dichloro-								*						
PAHs														
Naphthalene and/or its derivatives	4			1								1		
Pyrene and/or its derivatives						1								
Fluoranthene						1								
PHENOLIC COMPOUNDS														
BHT							1		1	1		1		
PHTHALATE ESTERS														
DEHP				1										
DBP				1										
DiBP			1											
OTHER AROMATIC COMPOUNDS														
Alkyl benzenes				1				3			4			
Benzaldehyde			1	1										
Benzenemethanol			1											
ALIPHATIC HYDROCARBONS														
Linear	7	7	9	5	8	1	11	11	7	11	7	9	4	9
Cyclic			1	1			1							

*Table 2. Organic chemical and heavy metal results. For groups of organic compounds, the number given is the number of compounds reliably identified; *(x) signifies the number of compounds identified at trace levels using a selective ion monitoring (SIM) method.*

Data for all organotin analyses are included in Table 3. In all samples, tributyltin (TBT) was the most abundant organotin compound detected, followed by dibutyl and monobutyl tin (DBT and MBT respectively). Tetrabutyltin (TeBT) was present as a minor contributor to total butyltin concentrations in only three of the six samples (from the southern part of the bay), and triphenyltin (TPT) in only two. Residues of octyltins (MOT and DOT) and of tricyclohexyltin (TCHT) were below limits of detection (<1 ug/kg dry weight) in all samples.

Code	**AM0112**	**AM0113**	**AM0115**	**AM0118**	**AM0116**	**AM0110**
MBT	219	94.4	76.1	39.2	25.1	20.7
DBT	300	158	140	63.2	28.6	26.1
TBT	5100	1930	615	207	33.5	49.7
TeBT	36.6	17.4	8.8	<1	<1	<1
ÓBT	5656	2200	840	309	87	97
MOT	<1	<1	<1	<1	<1	<1
DOT	<1	<1	<1	<1	<1	<1
TCHT	<1	<1	<1	<1	<1	<1
TPT	28.7	35.7	<1	<1	<1	<1
ÓOT	5684	2235	840	309	87	97
Ratio TBT:DBT	17	12.2	4.4	3.3	1.2	1.9
Ratio TBT:MBT	23.3	20.4	8.1	5.3	1.3	2.4

Table 3. Organotins in selected samples. Concentration data are in ug/kg dry weight as tin cation.

Overall, the southern and western areas of the bay exhibit the greatest pollution. These sections of the bay are impacted by the city and the naval and dock areas as well as receiving riverine input, some of which shows signs of industrial pollutants. The central areas of the bay, being closer to the deep water where more rapid water exchange may occur, are less severely polluted. The north-eastern and eastern parts of the bay receive pollutants primarily by diffusion from other parts of the bay are also less affected. However, these more remote areas of the bay have not escaped impacts.

All samples collected contained petroleum hydrocarbons and all except one (AM0118) contained detectable concentrations of the organochlorine 1,4-dichlorobenzene. Moreover, the most remote location sampled (sample AM0110), in the north-east of the bay, contains almost 200 ug/kg of organotin compounds, which can have severe impacts upon the marine environment. The hydrocarbon contamination could come from the recent large-scale oil spills, ongoing emissions from nearby refineries and oil terminals and also from exhaust fumes from boats and road traffic. 1,4-dichlorobenzene is one of the few chlorobenzenes still manufactured in large quantities. Its major use is in lavatory and room deodorants and so it is to be expected as a component of sewage discharges. This is likely to be the primary source to the bay and demonstrates clearly the vulnerability of even the furthest corners of the bay to inputs of toxic chemicals.

In the extreme south of the bay, on either side of the channel connecting the bay with the ocean, the highest concentrations of TBT (5100 and 1930 ug/kg dw respectively) were found in sediments collected from the two harbour areas sampled (AM0012 - near a shipyard between I. da Conceição and I. do Viana and AM0013 - near the Naval base at to I. das Cobras). These high levels of contamination undoubtedly result from the use of TBT antifoulant formulations on ships, although the extent to which such contamination results

from docking activities or from hull maintenance operations (e.g. hosing, scraping, sandblasting, repainting) is clearly not possible to determine from these data. The possibility cannot be ruled out, however, that high values resulted from the presence of small paint flakes in the sediment samples.

Table 3 also gives ratios of concentrations of TBT:DBT and TBT:MBT for each of the samples. Both ratios were strongly related to TBT concentration, with higher ratios in the most contaminated sites. Given that DBT and MBT residues in marine sediments are likely to arise primarily from the degradation of TBT, the appearance of the highest TBT:DBT ratios in the most contaminated sites, closest to the sources of ongoing TBT releases, may well be expected.

Triphenyltin (TPT) was present at above limits of detection only at the two most contaminated stations (AM0112 and AM0113). The origin of these residues, at concentrations almost two orders of magnitude below those of TBT, is not clear, although TPT is known to have been used in some antifouling formulations.

In addition to containing such elevated concentrations of these antifouling agents, the concentrations of some of the heavy metals at these locations were amongst the highest recorded in this survey. The three metals present in excess of typical background concentrations were the metals copper, lead and zinc. Each may have come from a variety of practices typical in ship maintenance. In particular, the copper may have been used in antifouling paints, though it is less toxic than the organotins.

Further, the sediments at these two sites contained a greater range of organochlorine contaminants than anywhere else in the bay. Both sites contain numerous PCB congeners, with the pattern of isomers present being close to that present in Aroclor 1254, a commonly used PCB mixture produced in the past by Monsanto. Although the use of PCBs has mostly ceased, they were extensively used formerly in transformer oils, hydraulic fluids and lubricants, applications common in maintenance operations. Moreover, their extreme environmental persistence means that any released to the bay could remain in the sediments for decades.

Trace concentrations of chlorinated benzenes were detected in both samples AM00112 and AM00113, with seven isomers in AM00112 and six in AM0113. The chlorobenzenes are toxic and persistent, properties that have caused their industrial applications to be severely curtailed over recent years. The tri- and tetra-chlorobenzenes were mixed with PCBs in transformer oils (Swami *et al.* 1992) and this may explain their presence at these locations. As discussed earlier, 1,4-dichlorobenzene is a common pollutant since it is used in lavatory deodorants so its presence may result from sewage discharges.

The western sector of the bay demonstrates a somewhat different pollutant profile. Lower levels of contamination with organotins were apparent at sample station AM0015, just west of

AM0013 and near the Ilha da Pombeba (615 ug/kg dw). The lower levels might result from a lesser significance in this area of docking and/or hull maintenance activities. Clearly this could only be confirmed on the basis of further information regarding the nature and intensity of shipping and docking activities at each of the locations sampled.

Several of the samples in the western sectors of the bay contain metals at concentrations above typical background, in patterns suggestive of different routes of entry to the bay. Nine of the fourteen samples contained concentrations of lead above that expected in an uncontaminated sediment (20-30 mg/kg (ATSDR 1997)). Those closest to Rio de Janeiro (AM0106, AM0113, AM0115 and AM0117) contained over 100 mg/kg lead. Urban runoff contaminated by emissions from leaded fuel vehicles are a plausible explanation for this finding.

The same nine samples also show copper concentrations above 50 mg/kg. In this case, however, the highest concentrations are in the dock areas (discussed above) and in samples AM0106 and AM0117. This latter pair are associated particularly closely with the Rio São João de Meriti; sample AM0106 came from the river itself and AM0117 from the region of the bay between the mouth of the river and the Ilha do Governador. Consequently an up-river source of copper inputs is suggested.

Indeed, this river may be the source of other pollutants as well. The samples from this river (AM0106) and from the bay near its mouth (AM0117) also show the highest concentrations of zinc and of mercury determined during the survey. Finally, trace concentrations of PCBs are detectable in the river. Again, the isomers present are mostly those found in Aroclor 1254. The Rio São João de Meriti flows through the city of Rio de Janeiro for some distance and may be subject to inputs from a variety of sources both domestic and industrial. Metals may also come from natural sources. Further data on the potential sources of these pollutants to this river would be desirable to prevent further pollution taking place.

Similarly, organochlorine pesticides were found in the samples from the Rio Sarapui (AM0107) and the Rio Iguaçu (AM0108). These samples from the Sarapui contained beta-HCH and trace concentrations of three isomers of the DDT family, mostly at trace concentrations. These are often detectable in the environment many years after their use because they are highly persistent. Long distance transport can also occur. The DDT isomers represent the pesticide itself and two of its environmental breakdown products. The beta-isomer of HCH is not insecticidal but is a byproduct of the manufacturing of the insecticide gamma-HCH (lindane)(Safe 1993). This beta HCH is especially persistent (ATSDR 1997), which may account for its presence here. Thus, use in agricultural or health applications, as well as leakage from manufacturing or storage locations could have contaminated these rivers with these pesticides. More data would therefore be required to identify possible sources of these pollutants.

There is evidence of pollution entering the bay near the mouth of the Rio Iguaçu. Concentrations of nickel and chromium were below average background concentrations in

most almost all the samples analysed, though elevated levels were of both metals were found in sample AM0119, from the mouth of the Rio Iguaçu, a short distance downstream from the Petrobras oil refinery. Sample AM0108, collected further upstream did not exhibit elevated concentrations of either of these metals, implying that the refinery is a likely source of the contamination. Nickel is used in catalysts in cracking and other processes within the refining of crude oil. It has been reported that refineries can generate 4-5 tonnes of spent catalyst residues for every Mt of oil refined and that these sludges can contain almost 900 mg/kg of nickel (EC 2000). Chromium may be present in several of the waste streams generated by refineries, partly because of low part per million concentrations in certain crude oils, but also because it may be used as an anticorrosion agent in cooling water circuits (EC 2000).

5 CONCLUSIONS

The current survey has revealed numerous sources of pollution in the Baia de Guanabara. Hydrocarbon contamination is ubiquitous, and 1,4-dichlorobenzene almost ubiquitous.

The levels of TBT and other organotins recorded within the very broad range of concentrations reported for other ports and harbours (see eg CEFAS 2000). However, at 5 100 ug/kg dry weight, the level found in the sediment AM0112 (near shipyard, between I. da Conceição and I. do Viana) is among the higher values previously reported.

Moreover, TBT was substantially above background levels in sediments from all six sample stations in Guanabara Bay. This more widespread contamination probably results from a combination of ongoing inputs from shipping traffic across the bay and the longer-term redistribution of sediments from particularly heavily contaminated areas.

Toxic metals are present at above background concentrations in most of the samples in the south and west of the Bay. Peak concentrations of copper are located in the southern dock areas and samples associated with the Rio São João de Meriti. Two of the dock areas also contain significant elevations of lead and zinc, whereas the river also seems to be a source of mercury and zinc. Runoff from the city appears to be a source of lead, which would have come originally from the use of leaded additives in petrol. Chromium and nickel may be entering the bay at the mouth of the Rio Iguaçu.

Organochlorine pollutants, predominantly chlorinated benzenes, PCBs and pesticides, were identified with a high degree of certainty. PCBs and chlorinated benzenes detected in samples AM0112 and AM0113 may have been a result of spillages of PCB transformer oils, which would have contained not only PCBs but also chlorobenzenes.

Further PCBs were found at trace concentrations in the sample from the Rio São João de Meriti and generally low concentrations of the organochlorine pesticides HCH and DDT in the Rio Sarapui and the Rio Iguaçu.

6 REFERENCES

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APPENDIX 1 ANALYTICAL METHODOLOGY

A1.1 Organic analysis

A1.1.1 Preparation of samples for organic screen analysis

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

A1.1.1.1 Solid Samples

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

Extracts were decanted, filtered through a pre-cleaned hydrophobic phase separator filter and collected in reagent tubes. They were then acidified to pH 2 with 10% nitric acid. Following this, a second portion of 15ml of pentane was added, followed by 5ml of acetone and the extraction procedure repeated. Finally, both extracts obtained for each sample were combined and evaporated to a volume of approximately 3ml. The concentrated extract was cleaned through a Florisil column, eluted with a 95:5 mixture of pentane: toluene, and evaporated down to a volume of 2 ml under a stream of analytical grade nitrogen. 1-Bromonaphthalene was then added at concentration 10mg/l to provide an indication of GC/MS performance.

A1.1.1.2 Aqueous Samples

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

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

A1.1.2 Chromatographic Analysis

Organic compounds were identified qualitatively using Gas Chromatography Mass Spectrometry (GC-MS). Instrumentation was a Hewlett Packard (HP) 5890 Series II gas chromatograph, interfaced with a HP Chem-Station data system and linked to a HP 5972 Mass Selective Detector operated in scan mode. The identification of compounds was carried out by computer matching against a HP Wiley 275 library of 275,000 mass spectra combined with expert interpretation. Also all extracts were analysed using selective ion monitoring (SIM) method against two standard solutions. The lists of compounds containing in Standard I and Standard II are presented below. All individual standards were obtained from Sigma Aldrich Co. Ltd., Supelco, UK. Additionally, samples were analysed using SIM method against PCBs standard mixture Aroclor 1254, obtained from Chem Service Inc., UK.

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

Table A.1.1 List of compounds in the Standard I used for SIM analysis

Results are reported as either reliably or tentatively identified. Match qualities of 90% or greater against HP Wiley 275 library or identification confirmed against standard compounds (using retention times and mass-spectra obtained during calibration) are assumed to give reliable identifications. Tentative identification refers to qualities between 51% and 90% against HP Wiley 275 library only. Analytes yielding match qualities of 50% or less are assumed to be unidentified.

Compound	**Ions to monitor**
Phenol, 2-chloro-	128, 64, 92, 39
Phenol, 2-methyl-	108, 79, 90, 51
Phenol, 3-methyl- and 4-methyl-	108, 107, 79, 77
Phenol, 2,5-dichloro-	162, 164, 63, 99
Phenol, 2,3-dichloro-	162, 126, 63, 99
Phenol, 4-chloro-	128, 65, 130, 100
Phenol, 2,6-dichloro-	162, 164, 63, 98
Butadiene, hexachloro-	225, 190, 260, 118
Phenol, 2,3,5-trichloro-	196, 198, 160, 97
Phenol, 2,4,5-trichloro-	196, 198, 97, 132
Phenol, 3,5-dichloro-	162, 164, 99, 63
Phenol, 2,3,6-trichloro-	196, 198, 97, 132
Phenol, 3,4-dichloro-	162, 164, 99, 63
Atrazine	200, 215, 202, 173
Phenol, pentachloro-	266, 268, 264, 165
Chlordane I	373, 375, 272, 237
Chlordane II	373, 375, 272, 237
PCB-153	360, 362, 290, 218
PCB-138	360, 362, 290, 292
PCB-180	394, 396, 324, 252

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

A1.2. Heavy Metal Analysis

A1.2.1. Preparation of samples for heavy metal analysis

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

A1.2.1.1. Solid Samples

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

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

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

A1.2.1.2. Aqueous samples

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

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

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

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

A1.3. Organotin Analysis

Sub-samples (approx. 20g in each case) were forwarded from the Greenpeace Research Laboratories to the laboratories of GALAB, Geesthacht (Germany), for quantification of a range of organotin compounds including butyltins (mono-, di-, tri- and tetra-butyltin), octyltins (mono- and di-octyltin), tricyclohexyltin and triphenyltin. Samples were extracted using a mixture of methanol and hexane and analysed using gas chromatography – (GC/AED) according to an accredited methodology (DIN EN 17025, further details can be provided on request). Limits of detection were 1 ug tin cation/kg dry weight in each case.

APPENDIX 2 TOXICOLOGICAL OUTLINES FOR KEY COMPOUNDS

A2.1 Organotins

Organotin compounds were first developed as moth-proofing agents in the 1920s, and only later used more widely as bactericides and fungicides (Moore *et al.* 1991) and as stabilisers in PVC (Sadiki and Williams 1999, Matthews 1996). It has recently been reported that the major use of organotins is for the heat stabilisation of PVC which represents about two-thirds of the global consumption of these compounds (Sadiki & Williams 1999). Both butyltins and octyltins have been used. The latter group of compounds were specifically developed in an attempt to overcome toxicity problems of the generally toxic butyltins (Matthews 1996). Phenyltin compounds (Fentins) including not only triphenyltin but also triphenyltin acetate (TPTA), triphenyltin chloride and triphenyltin hydroxide are used as agricultural fungicides, herbicides, molluscicides, insect and rodent repellants, as well as cotoxicants with TBT compounds in marine antifouling paints (Cima *et al.* 1997, Staub *et al.* 1995). Tricyclohexyltin (TCT) and fenbutatinoxide (FBTO) are used as acaricides (Staub *et al.* 1995).

By far the most widely intensively researched aspect of organotin ecotoxicology is the impact of tributyltin (TBT) on the marine environment. Use of TBT in marine antifouling paints dates only from the 1960s (Balls 1987, ten-Hallers Tjabbes 1997) when it was used as a booster biocide in copper-based formulations. As a result of its superior effectiveness over copper as an antifoulant (Wade *et al.* 1988), the use of TBT paints by private and commercial users accelerated greatly in the 1970s, during which these formulations captured a major proportion of the antifouling market (Evans 2000).

Toxicity

Obviously, it was intended that TBT be toxic to marine organisms. Acute toxicity of TBT to a range of marine organisms is generally measured in the µg/l (parts per billion) range (Weis & Perlmutter 1987, Bushong *et al.* 1988). However, the sublethal consequences of more prolonged exposure to low doses were initially overlooked (Laughlin & Linden 1987). The most widespread environmental effect of TBT use is imposex (the development of male sexual structures in females). This can be initiated in some gastropod molluscs by TBT in the low ng/l (parts per trillion) range (Bryan *et al.* 1986, Alzieu 1998). Similar concentrations are also known to cause shell deformity and larval mortality in certain bivalve species (Alzieu *et al.* 1986).

Sterilisation of female dogwhelk *N. lapillus*, resulting from the occlusion of the oviduct by the developing penis in TBT-exposed individuals, occurs at TBT concentrations in the order of 1-2 ng/l (as tin; approximately 3-5 ng/l TBT) (Gibbs *et al.* 1988). Although there was some degree

of differential sensitivity between individuals, occlusive sterility affects almost all females once concentrations reaches 3-5 ng/l tin. More fundamental physiological changes occurred at concentrations above 10 ng/l tin, including the suppression of egg production and the initiation of spermatogenesis and testis development. *Ocinebrina aciculata* is perhaps the most sensitive species; imposex can occur at 0.1ng/l tin (Vos *et al.* 2000).

Overall, TBT is regarded as the most toxic organotin. However, triphenyltin (TPT) has similar effects on marine algae (Fargasova 1998) and these two compounds were also most effective at inducing imposex in *Thais clavigera* (Horiguchi *et al.* 1997). The di- and mono- substituted compounds are generally less toxic. Concern about the toxicity of triphenyltin in the environment is further heightened by evidence that it may biomagnify (Mensink *et al.* 1997).

Studies on laboratory rodents have shown that TBT is toxic to the immune system (see e.g. Kergosien & Rice 1998). Tests on fish cells indicated that DBT is in fact a more potent immunotoxin than TBT (O'Halloran *et al.* 1998). Triphenyl tin is also able to affect the immune systems of tunicates (marine invertebrates) (Cima *et al.* 1997).

Although controversy surrounds the significance of such ubiquitous contamination, correlative evidence is available to suggest that accumulation of butyltin residues in the tissues of top predators might have adverse effects on the immune system. Kannan *et al.* (1997) reported particularly high (µg/g) levels of TBT and its breakdown products (primarily DBT) in specimens of bottlenose dolphin associated with mortality events on the US Atlantic and Gulf coasts. Similar concerns have been raised in relation to Californian southern sea otters (*Enhydra lutris nereis*), in which elevated levels of butyltin residues were found in specimens exhibiting various infectious diseases (Kannan *et al.* 1998).

The potential for human intake of organotin residues from seafood, especially from fish farmed in TBT-treated cages, has long been recognised (Davies & McKie 1987, Ishizaka *et al.* 1989). It is only more recently, however, that the significance of such intake has been evaluated. In a study of seafood marketed in the US, Cardwell *et al.* (1999) concluded that, although residues were widely detectable, estimated intakes were significantly below levels judged to be of concern with respect to human health, even during periods of peak usage. However, Belfroid *et al.* (2000) concluded that average intakes of TBT through consumption of seafood could lead to excedence of tolerable daily intakes (TDIs, based on immunotoxicological end-points) for some products on sale in North America, Europe and Asia. These authors also stressed that for the majority of countries, no data were available on levels of butyltins in seafood.

Environmental impacts

The phenomenon of imposex was first described in detail by Smith (1971), based on examinations of populations of the American mud-snail (*Nassarius obsoletus*) in the vicinity of harbours on the East coast of the USA. At around the same time, Blaber (1970) recorded

the appearance of a penis in female dogwhelks in Plymouth Sound, UK, at much greater prevalence in close proximity to the harbour than at a more distant location. At that time, however, despite the increasing prevalence and severity of the phenomenon, the causal agent remained unclear. As analytical capabilities improved in the late 1970s and early 1980s, the connection with shipping was made (though indirectly at first) and the extent of the threat posed, and damage already caused, by TBT antifouling paints became widely recognised. Vos *et al.* (2000) estimate that imposex has now been documented in the wild for as many as 150 species of marine prosobranch snails worldwide.

TBT contamination can have severe economic impacts. Until the mid-1970s, Arcachon Bay in France had been an important area for the culture of oysters (*Crassostrea gigas*), with production in the range of 10-15 000 tonnes per year (Evans 2000). The bay was also popular with leisure craft, with numbers of vessels in the bay increasing from around 7500 in the mid-70s to a maximum of 15 000 at the start of the 80s. Inputs of TBT to the waters of the bay during this peak period have been estimated at around 8kg per day (Ruiz *et al.* 1996).

Imposex was first observed in some sections of the bay in 1970, affecting the predatory gastropod *Ocenebra erinacea* (oyster drill). Frequency of imposex in this species increased sharply in the first years of that decade leading to the virtual extinction of *O. erinacea* from all but the central part of the bay (Gibbs 1993). This was followed by a failure in the oyster stocks themselves. By 1981, annual oyster production had been reduced from a maximum of 10-15 000 tonnes in the 70s to only 3 000 tonnes (Ruiz *et al.* 1996). In addition to reproductive failure, adult oysters were rendered unsaleable as a result of thickening and deformation of the shell leading, in the most severe instances, to “ball-shaped” specimens. Shell thickening, first observed in Arcachon Bay in the late 1960s, is also a sensitive indicator of TBT exposure in oysters, occurring at concentrations in the low ng/l range (Alzieu *et al.* 1989).

Balls (1987) reported that the use of TBT paints as antifoulants on fish cages by the salmon farming industry had resulted in contamination of a Scottish sea loch, and noted the generally higher prevalence of imposex within sea lochs used for aquaculture operations.

Subsequent to the bans on TBT for small boats, there is evidence from several parts of Europe for the partial recovery of the most severely affected mollusc populations (Minchin 1995, Evans *et al.* (2000). However, Minchin *et al.* (1997) stressed that the recovery of dogwhelk populations had been somewhat slower than might have been expected. In other areas where a recovery was reported, imposex remained at high level and dogwhelks were still absent from the worst affected areas (Harding *et al.* 1997). In Canada, St-Jean *et al.* (1999) noted that tissue concentrations of butyltins in blue mussels (*Mytilus edulis*) from the Southern Gulf of St Lawrence remained remarkably high eight years after the introduction of controls for small vessels, in common with levels in the sediments. A recent analysis of concentrations in French coastal waters (Michel & Averty 1999) indicated that, despite initial declines from the very high levels of the early 1980s, dissolved concentrations of TBT had largely stabilised, and

remained at levels often well in excess of the 1 ng/l known to cause adverse effects in some species. Thus, in some areas of Arcachon Bay, TBT concentrations remained high enough to cause imposex in sensitive species 10 years after the introduction of the 1982 regulations (Ruiz *et al.* 1996). Similar trends and concerns were voiced for contamination in several UK estuarine and coastal locations, following the legislation in 1987 (Cleary 1991, Langston *et al* 1994).

The causes of continued contamination and failure of invertebrate populations fully to recover undoubtedly differ from one location to another. In some cases (e.g. Huet *et al.* 1996), isolated but significant illegal use of TBT formulations on small vessels has been implicated in the continuation of inputs to the water column. Other authors have stressed that marina sediments contaminated with TBT prior to restrictions can act as reservoirs of organotins for many years (Waldock *et al.* 1990, Langston *et al.* 1994). The high sensitivity of larval stages of some species to residual concentrations of TBT (Gibbs 1993) and the long life-histories of certain benthic species (Rees *et al.* 1999) undoubtedly also contribute to the relatively slow rate of recovery in some instances.

On a broad scale, however, potentially the most significant single contributor to the continued widespread presence of TBT and its effects in the marine environment (and currently the most fiercely debated) is the continued use of TBT formulations on larger vessels.

The national and regional prohibitions introduced in the 1980s applied only to retail sale of TBT antifoulants, their use on vessels <25m in length and on nets and cages used for aquaculture. At that time, these were perceived as the dominant threat to aquatic life. The use of TBT-based formulations on commercial and military ships was viewed as a lesser concern because, it was argued, these vessel spend most of their operational lives on the open seas (where contamination was expected to be diluted and dispersed).

Nevertheless, these ships use inshore waters and port facilities on a regular basis. Waldock *et al.* (1988) recorded TBT concentrations in wash water from a naval frigate in the mg/l range (approximately 1 million times higher than the lowest biologically effective concentrations) and estimated a total input from the cleaning of a single such vessel at 100g of freely available TBT. Inclusion of TBT still bound to copolymer formulations, which might act as a long-term reservoir of butyltins to the marine environment, led to estimates of almost 1kg of TBT per vessel per cleaning operation. Guidelines to control inputs from such operations have been in use in many countries for some time, although their effectiveness is often difficult to evaluate.

Significant inputs of organotins can occur simply as a result of the presence and normal transport operations of large vessels with TBT-containing coatings (Bailey & Davies 1988, Harding *et al.* 1997). Significant contributions from heavy commercial traffic have also been implicated in the persistence of organotin contamination levels in parts of the Gulf of St Lawrence (St-Jean *et al.* 1999). Indeed, there is a growing body of evidence supporting a correlative link between the density of shipping traffic and the incidence of biological effects

(predominantly imposex) in offshore waters, though the matter is still the subject of intense debate (ten Hallers-Tjabbes *et al.* 1994, Evans 2000, Cadee *et al.* 1995). Davies *et al.* (1998) estimated total annual inputs of TBT to the North Sea from shipping- related activities to be in the order of 68 tonnes, with the major contributions arising from continuous leaching of TBT from painted hulls when in port or underway.

Location	Areas/Activities	TBT (ug/kg d.w. as alkyltin cation)	Reference
France, Arcachon Bay	Open bay Harbours/marinas	<1 >600	Sarradin *et al*. 1994
Hong Kong, Hong Kong Harbour	Marinas/typhoon shelters Shipyards	*medians* <250-3000 <250-4500	Ko *et al*. 1995
USA, Maine	Portland Harbour Shipyards STP Veterans Bridge Boothbay Harbour (hull refinishing area) Point 1 Point 2	*means ± std error* 350±172 39±14 90±53 1400±230 2760±1840	Page *et al*. 1996
Thailand, various	Coastal mariculture Fishing boat piers Ports Far seas vessels harbours	4-81 7-93 9-880 3600-4500	Kan-Atireklap *et al*. 1997
Japan, Osaka	Osaka Bay Port of Osaka	nd-23 11-692	Harino *et al*. 1998
USA, San Francisco Bay	Adjacent to Mare Island Naval Yard and Carquinez Strait	0.3-8.1	Periera *et al*. 1999
USA, Oregon	Reference sites 1993 Shipyards 1993 Shipyards 1997	<2.9 456-2955 24-1196	Elgethun *et al*. 2000
N. Germany/Baltic	Marinas	80-17 000	Biselli *et al*. 2000
Spain, Andalucia	Saladillo Harbour	1.2-280	Gomez-Ariza *et al*. 2000

Summary of sediment TBT concentrations recently reported for marinas, ports and harbours from various parts of the world

Correlations between the intensity of commercial shipping traffic and incidence of imposex have also been reported within the Strait of Malacca, connecting the Bay of Bengal with the

South China Sea (Swennen *et al.* 1997, Hashimoto *et al.* 1998), and in relatively remote coastal regions of the Galician coast (Ruiz *et al.* 1998).

During the last decade, research has also highlighted the widespread accumulation of butyltin (and other organotin) residues in organisms higher in the food chain, including cetaceans. Iwata *et al.* (1995) were among the first to determine concentrations of butyltin residues in marine mammals and suggested that the high levels recorded (up to 10 µg/g wet weight in porpoise liver) resulted in part from a relatively low potential for metabolism of these compounds. Tanabe *et al.* (1998) later extended the data set to include a greater range of species from North Pacific and Asian coastal waters. Kannan *et al.* (1996) described butyltin accumulation in dolphin, tuna and sharks from the Mediterranean, noting markedly different speciation within the different taxa. First reports of butyltin residues in cetaceans and pinnipeds in the North East Atlantic region were published by Ariese *et al.* (1998) and Law *et al.* (1998). The occurrence of detectable residues of butyltins in pelagic cetacean species which primarily feed in remote offshore waters (Ariese *et al.* 1998, Law *et al.* 1999) provided further evidence of the widespread distribution of organotins in the marine environment. There is also concern that accumulations of butyltins in the tissues and eggs of the horseshoe crab (*Tachypleus tridentatus*) could affect their chances of survival (Kannan *et al.* 1995).

The co-occurrence of triphenyltin (TPT) in imposex-effected whelk populations has been documented by Mensink *et al.* (1997) for sections of the North Sea and Eastern Scheldt. TPT is used in some antifouling formulations, at concentrations up to 10% those of TBT, and is also a highly bioactive and endocrine disrupting substance.

Legislation

France was the first country to introduce legislation prohibiting the application of TBT-containing antifouling formulations to small (<25m) vessels, in 1982 (Michel & Averty 1999, Evans 2000). The UK Government introduced controls on the sale of TBT-based paints for use on small vessels in 1985 under the Control of Pollution Act (1974). Measures included regulations controlling retail sales, guidelines for handling of paints and the setting of an environmental quality target (EQT) for TBT of 20 ng/l (8 ng/l as tin), a level which was considered at that time to be sufficiently protective of marine biota (Waldock *et al.* 1987). However, it quickly became apparent that concentrations well below 20 ng/l could evoke severe biological responses. At the same time, TBT concentrations in many coastal waters consistently exceeded the EQT. Consequently, the UK Government considered its options for further restrictions on the sale of TBT antifouling paints (Side 1987) and in May 1987 set a total ban on the retail sale of paint formulations containing TBT (Waldock *et al.* 1987, 1988). The prohibition extended not only to sale for use on vessels under 25 m in length, but also for use on fish cages used in aquaculture. The 1985 EQT of 20 ng/l was replaced by an environmental quality standard (EQS) of only 2 ng/l (Cleary 1991).

The restrictive measures taken in the UK in 1987 were quickly followed by recognition by the Paris Commission (PARCOM, responsible for administering the Paris Convention, 1978, for the Prevention of Marine Pollution from Land-Based Sources in the North East Atlantic), that the use of TBT paints was causing "serious pollution in the inshore areas of the Convention waters" (PARCOM 1987). PARCOM Recommendation 87/1, of June 3rd 1987, called on Contracting Parties to the Convention to adopt a harmonised ban on the retail sale of organotin paints for application to pleasure boats and fish cages and, furthermore, to consider introducing restrictions on use for sea-going vessels and underwater structures.

Prohibitions on the use of TBT-based antifoulants on small vessels were adopted in the US in 1988. These were followed in 1989 by similar measures in Canada, New Zealand and Australia, and by European Community legislation in 1991 which harmonised existing controls through out the EU (Evans 2000).

International agreements and discussions on the need to address all uses of TBT (eg MINDEC 1995, ten Hallers-Tjabbes 1997, MEPC 1997, MEPC 1998) have culminated in the draft International Convention on the Control of Harmful Anti-fouling Systems (2003 for phase-out of application of organotin paints, 2008 for their presence on ship hulls), which were adopted under IMO Assembly Resolution A.895(21) in November 1999. Finalisation of the Convention text is now expected in 2001, prior to formal adoption later in the year.

Alternatives

In the search for replacements for TBT, it is clear that some of the antifouling mechanisms in use prior to the widespread introduction of TBT (in some cases based on mercury or arsenic compounds) would not represent sensible or acceptable alternatives. Copper-based systems are available, but often require additional booster biocides in order to be fully effective - biocides which themselves may present additional problems. A class of antifoulants commonly cited to illustrate the difficulties of biocide substitution are those containing the triazine herbicide Irgarol 1051, used extensively in some areas as an alternative to TBT antifoulants on leisure craft following the controls introduced in the 80s. The widespread occurrence of the active ingredient within certain estuaries (Scarlett *et al.* 1997), coupled with observations of direct effects on periphyton and macrophyte growth in the field (Dahl & Blanck 1996, Scarlett *et al.* 1999), in some ways mirror early findings with respect to TBT and are clearly cause for concern.

Clare (1998) reviews research underway into the potential exploitation of natural products which inhibit settlement, through their incorporation as alternative biocides in antifouling paints. However, commercial applications may remain some time away. Perhaps the most promising direction in antifouling technology, however, is provided by biocide-free, "non-stick" surface coatings which simply present a physical barrier to settlement. Silicone-based non-stick coatings have been commercially available for many years, and retain a significant share of the market with regard to leisure craft. Their applicability, economic viability and

performance on larger vessels remains, to some degree, under trial, although their commercial viability for relatively fast moving craft is becoming more generally accepted. While significant technical problems remain in some cases, there appear to be no fundamental reasons why such problems should not be resolved in the near future. Moreover, from a more general standpoint of the need to ensure protection of the marine environment, trends towards antifouling mechanisms which do not rely on the open release of bioactive substances to the sea would seem to desirable.

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A2.2 Polychlorinated biphenyls (PCBs)

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

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

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

1998) to metallurgical processing (Knutzen & Oehme 1989, Alcock *et al.* 1998, Thiesen *et al.* 1993) to dye manufacturing (USEPA 1998).

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

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

In a review of PCB toxicity, Safe (1984) lists the following symptoms of PCB toxicity: enzyme induction, decreased vitamin A levels, lymphoid involution, thymic and splenic atrophy, immunosuppression, chloracne, alopecia, oedema, hyperkeratosis, blepharitis, hyperplasia of the epithelial lining of the extrahepatic bile duct, the gall bladder and urinary tract, hepatomegaly and liver damage including necrosis, haemorrhage, hepatotoxicity (altered porphyrin metabolism), tumour promotion, altered levels of steroid and thyroid hormones, reproductive toxicity including menstrual irregularities, reduced conception, early abortion, excessive menstrual and postconceptual haemorrhage, anovulation, testicular atrophy, decreased spermatogenesis, teratogenesis and developmental toxicity. In addition, low levels of PCBs have caused behavioural impairment in monkeys (Rice 1999).

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

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

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

wild populations of fish-eating birds and captive seals have been correlated with PCB exposure (Brouwer *et al*. 1999).

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

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

The extensive body of information concerning the global cycling of PCBs has been accumulated in response to concerns about the environmental impact of these chemicals. PCBs are highly persistent. Although there is evidence of biodegradation in contaminated sediments (see: Brown & Wagner 1989) and some marine mammals appear to be able to selectively degrade some of the lower chlorinated congeners (Boon *et al*. 1987), the detoxification potential of these processes would appear to be rather limited. Indeed, Cummins (1988) has suggested that unless further escape of PCBs is prevented then the eventual extinction of marine mammals is a very real possibility.

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

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

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

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

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

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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.

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A2.4 DDT and metabolites

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

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

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

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

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

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

DDT is moderately to slightly toxic to studied mammalian species via the oral route (RSC 1991, Meister 1992, ASTDR 1997). The primary target of DDT is the nervous system and high doses can cause trembling, increased susceptibility to cold and fear, with convulsions happening at the highest doses. Death can occur through respiratory arrest, though animals that survive a day or more after the last dose usually recover completely (Smith 1991). It has caused chronic effects on the nervous system, liver, kidneys, and immune systems in experimental animals (ASTDR 1997, WHO 1979). There is evidence that DDT causes reproductive effects in test animals, including reduced fertility (ASTDR 1997).

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

Several of the DDT group are endocrine disruptors, exhibiting different modes of action. Several are weakly oestrogenic. Of these, o,p'-DDT is the most active. p,p'-DDE, the compound likely to be present at highest concentrations in most humans, is an antiandrogen (Longnecker *et al*. 1997).

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

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

However, DDT's most severe impacts are on the environment. DDT, or rather, its metabolite, p,p'-DDE, causes the thinning of bird's eggshells through perturbation of calcium metabolism. Eggshell thinning caused by p,p'-DDE results in crushed eggs, or, if the egg is not crushed, the embryo can die of dehydration as too much water is lost through the thinned shell (Hickey & Anderson 1968, Newton 1995, Provini & Galassi 1999). Tests on 15 different toxic pollutants found that only p,p'-DDE has the ability to thin shells over an extended period (Haegele & Tucker 1974, Peakall & Lincer 1996). Although DDT primarily causes population decline through reproductive failure, it may also kill highly exposed birds directly (Carson 1962, Fry 1995, Copper 1995, Newton *et al*. 1982, Garcelon & Thomas 1997). Analysis of kestrels and sparrowhawks in the 1960s and 1970s suggest that some were being killed directly by p,p'-DDE exposure (Newton *et al*. 1982).

Some bird populations which previously suffered from p,p'-DDE impacts of egg-shell thinning and egg breakage are no longer at such risk. Studies in the UK on the grey heron, Ardea cinerea L., (Newton *et al*. 1993) show that levels of DDE in herons or their eggs have significantly declined. A study on grey herons in France noted that levels of p,p'-DDE in eggs were lower than levels associated with reproductive effects reported in the wild or in laboratory studies (de Cruz *et al*.1997).

However, some effects of organochlorines in seabirds have been observed recently despite the general downward trend in many organochlorines. In the Arctic, present p,p'-DDE levels in Canadian tundra peregrines, Fennoscandian merlin and white-tailed sea eagle are still causing significant egg shell thinning (de Wit *et al*. 1997).

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

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

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A2.5 Polycyclic aromatic hydrocarbons (PAHs)

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

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

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

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

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

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

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

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

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A2.6 Alkylbenzenes

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

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

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

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

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A2.7 Phthalate esters

Phthalate esters are commonly referred to as the phthalates. They are used in every major product category (Kemi 1994). 90% of all plasticizers are used in the production of soft PVC (Cadogan *et al.* 1993) but they are also used in inks and dyes, in cosmetics, as a concrete additive, and as a solvent for perfume oils (ATSDR 1997, Jobling *et al.* 1995).

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.

DEHP is the most extensively research phthalate ester. It can cause liver cancer in laboratory animals. It has been classified as possibly carcinogenic to humans by the IARC and US Department of Health and Human Services has determined that DEHP may reasonably be anticipated to be a carcinogen (DHHS 2000). 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 (also called 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 also 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.

PVC plasticised with dibutyl phthalate (DBP), and used in the glazing systems of glasshouses, can be seriously phytotoxic. DBP is released at concentrations far below those regarded as potentially harmful to human health, but is toxic enough to young plants to cause significant damage to, or death of, entire crops. The food plants known to be directly affected include brassicas (particularly some cabbage cultivars), tomatoes and peppers. There is unconfirmed evidence that cucumbers, tobacco, begonias and hibiscus are also affected (Cole *et al.* 1984).

Phthalates are semivolatile and are found in the atmosphere of primary PVC processing plants at levels of 0.02 - 0.5mg m^{-3}. This results in a significant exposure of workers (Dirven *et al.* 1993). Exposure to workers recycling soft PVC is likely to occur at similar levels. Another suspected source of harm to people - particularly children- is from inhaling phthalates. Recent research has suggested that atmospheric DEHP from PVC floors and wallcoverings could have a role in asthma in children (Oie *et al.* 1997, Jaakola *et al.* 1999). The migration of DEHP from cling film into foods, especially fatty foods such as dairy produce, has led many manufacturers to offer non-PVC film, or to reduce the content of DEHP (MAFF 1987 & 1990).

Children's toys, many intended to chewed by young children, are amongst the products softened with phthalates. Some toys can contain as much as 40% phthalates by weight, as well as a range of other compounds such as the alkyl phenols (Stringer *et al.* 2000). The risk to health that these pose has been the subject of some debate over the last few years (CSTEE 1998 & 1999, Janssen *et al.* 1998, MacKenzie 1997) and resulted in an emergency ban being

imposed by the EC (EC 1999). This measure covers toys and child-care items which are intended to be placed in the mouths of children under the age of 3 years and which contain over 0.1% of DINP, DEHP, DnOP, DIDP, BBP or DBP. The EC's move follows restrictions being placed on various categories of phthalate-containing PVC toys by Austria, Denmark, Finland, France, Germany, Greece, Italy, Spain and Sweden. The three-month ban, which has been extended until March 2001 (EC 2000), is intended to allow the European Parliament and Council time to consider a permanent ban which would be implemented through amendment of Directive 76/769/EC on restrictions on the marketing and use of dangerous substances and preparations (EC 1999).

Because of their recognised toxicity and widespread distribution, 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).

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(DEHP), dibutyl phthalate (DBP), di-iso-decyl phthalate (DIDP), di-n-octyl phthalate (DNOP) and butylbenzyl phthalate (BBP). OJ L 315, 9.12.999: 46-49

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A2.8 Chromium

Environmental Contamination and Behaviour

Although many different oxidation states of chromium exist in the environment, only the trivalent (III) and hexavalent (VI) forms are considered to be of biological importance. In aquatic environments, chromium (VI) will be present predominantly in a soluble form. These

soluble forms may be stable enough to undergo intra-media transport, however chromium (VI) will eventually be converted to chromium (III), by reducing species such as organic substances, hydrogen sulphide, sulphur, iron sulphide, ammonium and nitrite (ATSDR 1997, Kimbrough *et al.* 1999). This trivalent form is generally not expected to migrate significantly in natural systems. Instead, it is rapidly precipitated and adsorbed onto suspended particles and bottom sediments. However, changes in the chemical and physical properties of an aquatic environment can result in changes to the chromium (III)-chromium (VI) equilibrium (Richard and Bourg 1991).

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

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

The importance of this lies in the fact that whilst chromium (III) is an essential trace element in animals, chromium (VI) is non-essential and toxic at low concentrations. Thus, because oxidation processes can result in the formation of chromium (VI), anthropogenic activities that release either chromium (III) or chromium (VI) are equally non-desirable. Even if chromium (III) is discharged into the environment, there is no guarantee that it will remain in this chemical state (Mukherjee 1998, Outridge and Sheuhammer 1993, UNEP 1991, Richard and Bourg 1991).

Toxicity and Essentiality

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

Chromium (VI) is non-essential and toxic. Compounds are corrosive, and allergic skin reactions readily occur following exposure, independent of dose. Short-term exposure to high

levels can result in ulceration of exposed skin, perforations of respiratory surfaces and irritation of the gastrointestinal tract. Damage to the kidney and liver have also been reported (ATSDR 1997). In addition, the International Agency for Research on Cancer (IARC) classifies chromium (VI) compounds as known carcinogens (1998). Long-term occupational exposure to airborne levels of chromium higher than those in the natural environment has been associated with lung cancer. Individuals at most risk include those in chromate-production industries and chromium pigment manufacture and use; and similar risks may exist amongst chromium-alloy workers, stainless steel welders, and chrome-platers (Kimbrough 1999, DHHS 1998).

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

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A2.9 Copper

Environmental Contamination and Behaviour

Copper may exist in natural waters either in the dissolved form as the cupric (+2) ion or complexed with inorganic anions or organic ligands (e.g. carbonates, chlorides, humic and fulvic acids). It may also be present as an insoluble precipitate (e.g. a hydroxide, phosphate, or sulphide) or adsorbed onto particulate matter. Alternatively, it can be adsorbed to bottom sediments or exist as settled particulates. The relative concentrations of each of these forms is dependant upon a number of chemical parameters, including pH, salinity, alkalinity, and the presence of organic ligands, inorganic anions and other metal ions. However, studies have frequently shown that the free +2 ion concentration is low, compared to the levels of copper associated with suspended and bottom sediments (ATSDR 1997, Mance *et al*. 1984).

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

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

Toxicity and Essentiality

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

Therefore, even though copper is essential for good health, a very large single dose, or long term elevated exposure can be harmful. Inhalation of dust and vapours can irritate the nose, mouth and eyes, and cause headaches, dizziness, nausea and diarrhoea. Oral exposure to high levels can cause vomiting, diarrhoea, stomach cramps and nausea (ATSDR 1997). Copper homeostasis plays an important role in the prevention of copper toxicity, in humans, terrestrial animals, and aquatic organisms. Copper is readily absorbed from the stomach and small

intestine; and after requirements are met, there are several mechanisms that prevent copper overload e.g. bile excretion, increased storage in the liver or bone marrow (ATSDR 1997). However, failure of this homeostatic mechanism can occur in humans and animals following exposure to high levels of copper. This rare disease, known as Wilson's disease, is characterised by the excessive retention of copper in the liver and impaired copper excretion in the bile. This can result in liver and kidney damage and haemolytic anaemia (ATSDR 1997).

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

Aquatic toxicity to copper is well studied, and there is experimental evidence that a considerable number of species are sensitive to dissolved concentrations as low as 1-10 ug/l (Bryan & Langston 1992). For example, studies have shown that at levels of 2 ug/l, the survival rate of young bay scallops was significantly affected; and in the embryos of oysters and mussels concentrations of 5 ug/l were seen to induce abnormalities. A similar concentration resulted in increased mortalities in populations of the isopod crustacean *Idothea baltica* (UNEP 1993, Bryan & Langston 1992, Giudici *et al*. 1989). Other studies have reported reductions in the survival, growth and fertility of amphipods and copepods (Conradi and DePledge 1998, UNEP 1993), and embryonic sensitivity in fish exposed to levels of 25 ug/l (UNEP 1993, Mance *et al*. 1984). Furthermore, a study of species diversity in benthic communities from Norwegian fjords, led to the conclusion that the most sensitive animals were missing from sites where sediment-copper levels exceeded 200 mg/kg. In the UK, such concentrations are exceeded in a number of estuaries, including the Fal and the Tamar. Here, many species of bivalves, including some mussels, clams and cockles are absent, and at best distribution is severely limited. The toxicity of the surface sediment containing over 2000 mg/kg of copper, towards juvenile bivalves appears to be the reason (Bryan & Langston 1992).

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A2.10 Lead

Environmental Contamination and Behaviour

When lead is released into the environment it has a long residence time compared with most pollutants. As a result, it tends to accumulate in soils and sediments. Where, due to low solubility, it can remain accessible to the food chain and to human metabolism far into the future (Sauve *et al.* 1997, ATSDR 1997, Alloway 1990). However, as with all metals, speciation is critical when assessing bioavailability and the potential threat to the environment.

Two oxidation states of lead, +2 and +4, are stable, but the environmental chemistry is dominated by the Pb^{+2} ion, its compounds, and complexes. In general the free +2 ion is more toxic than inorganic complexes, and therefore any factor which increases complexation and decreases the concentration of the free ion is bound to affect lead toxicity adversely. Toxic organic forms of lead are also present in the environment. From direct inputs (manufacture, transport and storage of leaded petrol and consequent car exhaust emissions) and the possible chemical / biological methylation of inorganic lead in anaerobic sediments (Sadiq 1992, Forsyth *et al.* 1991).

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

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

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

Toxicity

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

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

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

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

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A2.11 Mercury

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 (ATSDR 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 (ATSDR 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) (ATSDR 1997, Bryan & Langston 1992). Dredging or re-suspension of bed materials may cause short-term release of

mercury, although levels of dissolved metal quickly return to pre-disturbance values. Mercury accumulation from sediments may therefore be a dominant pathway for uptake in aquatic organisms and accounts for relatively high concentrations in deposit feeders, in both freshwater and marine systems (Bryan & Langston 1992).

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

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 (ATSDR 1997, WHO 1989).

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

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

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

High incidences of abnormalities have also been observed in seabirds, abnormalities that seem to correlate with mercury residues in tissues. Even at sites apparently remote from contamination, elevated mercury concentrations have been determined in the liver and kidneys of fish eating seabirds, e.g. *Fulmarus glacialis*. Levels comparable with those suspected of producing sub-lethal effects, notably pathological changes to the kidney; and which have been shown to cause death in other species (Bryan & Langston 1992).

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A2.12 Zinc

Environmental Contamination and Behaviour

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

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

Toxicity and Essentiality

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

Due to the essentiality of zinc, dietary allowances of 15 mg/day for men, and 12 mg/day for women are recommended (ATSDR 1997). However, eating food containing very large amounts of zinc can induce the symptoms listed above. For example, animal studies involving doses 1,000 times higher than the RDA, taken over a period of a month, resulted in anaemia and injury to the pancreas and kidney; and rats that ate very large amounts of zinc became infertile (ATSDR 1997). Humans taking supplements at higher than recommended doses (400-500 mg/day) suffered severe gastro-enteritis (Abernathy and Poirier 1997); and humans who drank water from galvanised pipes, over a prolonged period, suffered irritability, muscular stiffness and pain, loss of appetite and nausea (UNEP 1993).

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

At slightly higher concentrations, studies investigating the effects of zinc on the hatching of brine shrimp (*Artemia salina*), noted that although increased concentrations of zinc did not affect development before emergence, the hatching stage of development was highly sensitive to, and heavily disrupted by, zinc (Bagshaw *et al* 1986). In addition, the inhibition of larval development was observed in the echinoderm (e.g. sea urchins and starfish) *Paracentrotus lividus* at a zinc concentration of only 30 ug/l (UNEP 1993). Shell growth in the mussel *Mytillus edulis* was effected at a concentration of 200 ug/l. With oxygen uptake, feeding and filtration rates were reduced at concentrations ranging between 750-2000 ug/l. Harmful effects on mollusc larva were seen to occur at levels as low as 40 ug/l (UNEP 1993).

Plant studies have shown that although an essential element for higher plants, in elevated concentrations zinc is considered phytotoxic, directly affecting crop yield and soil fertility. Soil concentrations ranging from 70-400 mg/kg are classified as critical, above which toxicity is considered likely (Alloway 1990). It was the observed phytotoxicity of zinc in sewage-sludge amended soils, that led several countries to formulate guidelines for sludge usage (Alloway 1990).

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A2.13 Nickel

Nickel is the 24th most abundant element in the Earth's crust, with an average concentration of 75 mg/kg. However in some igneous rocks, clays and shales, higher concentrations can be found (Alloway 1990). Its commercially important ores are of two types, laterites, which are oxide and silicate ores, and sulphides (e.g. pentlandite) often associated with precious metals, copper and cobalt. The largest deposits of nickel are found in Canada, Cuba, Australia, CIS, and South Africa, with the most important single deposit (supplying over a quarter of the world's nickel), found in Canada, at Sudbury Basin (Greenwood & Earnshaw 1984).

Volcanic activity is the largest natural source of atmospheric nickel, followed by emissions from wind-borne soil particles, forest fires, sea salt spray and biogenic processes. It is estimated that the total amount of nickel released to the atmosphere from natural sources is 29,000 tonnes / year, compared with an estimated anthropogenic load of 52,000 tonnes / year (Nriagu 1990).

Production, Use and Anthropogenic Sources

Primary nickel is recovered from mined ore, which is first crushed, enriched and concentrated, prior to roasting and smelting operations; secondary nickel can also be recovered, from scrap metal. Alternatively, reduced nickel oxide ores can be electrolysed in the presence of nickel sulphate or chloride, to yield metal of 99% purity (Greenwood & Earnshaw 1984).

Nickel is a white-silver metal, hard but brittle, polishable, and a good conductor of both heat and electricity. It is most commonly used to form stainless and heat resistant steels, high nickel heat- and corrosion resistant alloys, alloy steels, super-alloys and cast irons. It is extensively used in electroplating, in the petroleum industry, in ceramics, in nickel-cadmium batteries and as an industrial catalyst, used for the hydrogenation of fats and methanation of fuel gases (ATSDR 1997, Alloway 1990, Greenwood & Earnshaw 1984).

Environmental Levels, Contamination and Behaviour

Nickel is found in varying concentrations in nearly all uncontaminated aquatic and terrestrial ecosystems (see Table 4). However, as anthropogenic emissions of nickel far exceed those

from natural sources, elevations above these natural, background concentrations, are often found (see Table 5).

Environmental Matrix	Concentration	Reference
Seawater	0.1-0.5 ug/l	ATSDR 1997, Law *et al.* 1994
Freshwater	<10-20 ug/l	ATSDR 1997, Mance and Yates 1984
Freshwater sediment	45-65 mg/kg	Salomons and Forstner 1984
Soil	5-500 mg/kg (average 50 mg/kg) 40 mg/kg	ATSDR 1997 Alloway 1990

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

Nickel persists in water with an estimated residence time of 23 000 years in deep oceans and 19 years in near shore waters (Nriagu 1980). Its behaviour in the aquatic environment is governed by reactions with both soluble species and particulate matter. Complexes may be formed, with a variety of soluble organic and inorganic species. In addition, interactions with solid phases may occur. For example, direct adsorption onto particles such as clays; adsorption to or co-precipitation with hydroxides of iron and manganese, complexation with natural organic particles or direct precipitation. Studies have shown that nickel is a fairly mobile metal in natural waters, especially soluble at higher pH values. However generally speaking, concentrations of soluble nickel are low compared with that associated with suspended and bottom sediments (ATSDR 1997, Mance & Yates 1984).

Site Description	Concentration	Reference
Drinking water, near a large, open-pit mine, USA	200 ug/l	ATSDR 1997
Seawater, coastal and estuarine sites of industrial and domestic discharges, UK	0.23-4.9 ug/l	Law *et al*. 1994
Sediment, Elsburgspruit-Natalspruit Rivers, South Africa (mining discharges and sewage)	54.5-890 mg/kg	Steenkamp *et al*. 1995
Soil, Sudbury Basin / Coniston, Canada (nickel mining and smelting)	100-3 000 mg/kg 160-12 300 mg/kg	Freedman & Hutchinson 1980 Hazlett *et al*. 1983
Soil, Upper Silesia, Poland (mining and smelting)	5-2 150 mg/kg	Dudka *et al*. 1995
MSW incinerator ash, UK	45-2 204 mg/kg	Mitchell *et al*. 1992

Table 3. Nickel concentrations associated with anthropogenic contamination and waste

Nickel is significantly bioaccumulated in some, but not all, aquatic organisms. Typical bioconcentration factors for significant bioaccumulators include marine phytoplankton <20-2 000, seaweeds 550-2 000 and algae 2 000-40 000 (ATSDR 1997).

In soils, the average residence time of nickel is estimated to be 2400-3500 years (Nriagu 1980), and although it is extremely persistent in soil, it is reasonably mobile and has the potential to leach through soil and subsequently enter groundwater (ATSDR 1997, Alloway 1990).

Toxicity and Essentiality

Very small amounts of nickel have been shown to be essential for normal growth and reproduction in some species of animals, plants and micro-organisms. It is therefore assumed that small amounts may also be essential to humans, although the precise function of nickel is unclear (ATSDR 1997, Alloway 1990). However, at the other extreme, there is sufficient evidence for the carcinogenicity of nickel and certain nickel compounds e.g. oxide, subsulphide, carbonate, acetate, carbonyl and hydroxide. The US Department of Health and Human Services, in its 8th Report on Carcinogens, therefore lists nickel and these compounds as Reasonably Anticipated to be Human Carcinogens (DHHS 1998). Metallic nickel and its alloys are listed as possible human carcinogens (Group 2B) by the International Agency for Research on Cancer (1998).

Nickel is a respiratory tract carcinogen in workers in the nickel refining and processing industries. Here, individuals are frequently exposed to atmospheric levels in excess of 1 mg of nickel per cubic meter of air (ATSDR 1997, Goyer 1996). Other serious consequences of long term exposure to nickel may include chronic bronchitis and reduced lung function (ATSDR 1997). Whilst other studies have reported pregnancy complications in nickel-exposed workers, i.e. an increased rate of spontaneous abortion, and a higher incidence of birth malformations, including cardiovascular and musculoskeletal defects (Chashschin *et al*. 1994).

Allergic contact dermatitis is the most prevalent adverse effect of nickel in the general population (2-5% may be nickel sensitive). Here, people become sensitive to nickel when jewellery or other nickel- containing objects are in direct contact with the skin. Once a person is sensitised to nickel, any further contact will produce a reaction. A rash at the site of contact is visible, and in some cases eczema may develop. Therefore, although non-sensitised individuals would have to ingest or inhale a large amount of nickel to suffer adverse health effects, sensitised individuals react adversely to far lower concentrations (ATSDR 1997).

Few studies on the aquatic toxicity of nickel are available. However one toxicity study, carried out using temperate marine diatoms (*Nitzschia closterium*), juvenile banana prawns (*Penaeus merguiensis*), leader prawns (*Penaeus monodon*) and gastropods (*Nerita chamaeleon*), did find that survival and growth rates were effected by increased concentrations of nickel (Florence *et al.* 1994).

Legislation

Unlike mercury, cadmium and lead, nickel and its compounds are not included on National and International lists of priority pollutants. However, whilst the reduction of anthropogenic emissions does not require priority action, pollution of terrestrial and aquatic environments by nickel still needs to cease. Nickel is therefore included on the majority of subsidiary and secondary pollutant lists. Examples of guidelines and permissible environmental levels include the following:

European Council Directive 76/464/EEC on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. Nickel is included in List II and consequently, water pollution caused by its presence must be reduced in keeping with National Environmental Quality Standards. European Community Council Directive 80/778/EEC relating to the quality of water intended for human consumption. A Maximum Permissible Limit of 50 ug/l is set.

The Water Research Centre in the UK recommends the following Environmental Quality Standards for nickel: protection of freshwater fish 50-200 ug/l; protection of other freshwater life and associated non-aquatic organisms 8-100 ug/l; protection of saltwater fish, shellfish, other saltwater life and associated non-aquatic organisms 30 ug/l (Mance & Yates 1984).

Regarding soil contamination, the UK Department of the Environment (ICRCL) classifies a level of 0-20 mg/kg as being typical of uncontamination. Anything above this is classified as contaminated, and as such, restrictions on recreational and agricultural uses apply (Alloway 1990). In terms of permissible sewage sludge levels, acceptable concentrations for nickel range from 30-500 mg/kg. However soil concentrations should not exceed 30-50 mg/kg (EC, France, Germany) (Alloway 1990).

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