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 the 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 was added, and the sample agitated for 2 hours on a bottle roller to maximize 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 an Agilent 6890 Series gas chromatograph, interfaced with an Agilent Enhanced Chem-Station data system and linked to an Agilent 5973 Mass Selective Detector operated in scan mode. The identification of compounds was

carried out by computer matching against an Agilent Wiley7N and Pesticides Libraries of over 390,000 mass spectra combined with expert interpretation. In addition, all extracts were analysed using a selective ion monitoring (SIM) method against two standard solutions. The lists of compounds contained in Standard I and Standard II are presented below. All individual standards were obtained from Sigma Aldrich Co. Ltd., Supelco, UK.

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

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

Table A1.1. List of compounds in the Standard I used for SIM analysis

Compound	**Ions to monitor**
Phenol	94, 66, 65, 95
Phenol, 2-chloro-	128, 64, 92, 39
Phenol, 2-methyl-	108, 79, 90, 51
Phenol, 3-methyl- and 4-methyl-	108, 107, 79, 77
Phenol, 2-nitro-	139, 65, 81, 109
Phenol, 2,5-dichloro-	162, 164, 63, 99
Phenol, 2,3-dichloro-	162, 126, 63, 99

Phenol, 4-chloro-	128, 65, 130, 100
Phenol, 2,6-dichloro-	162, 164, 63, 98
Butadiene, hexachloro-	225, 190, 260, 118
Phenol, 4-chloro-3-methyl-	107, 142, 77, 144
Phenol, 2,3,5-trichloro-	196, 198, 160, 97
Phenol, 2,4,6-trichloro-	196, 198, 97, 132
Phenol, 2,4,5-trichloro-	196, 198, 97, 132
Phenol, 2,3,4-trichloro-	196, 198, 97, 160
Phenol, 2,3,6-trichloro-	196, 198, 97, 132
Phenol, 3,5-dichloro-	162, 164, 99, 63
Phenol, 3,4-dichloro-	162, 164, 99, 63
Phenol, 2,3,5,6-tetrachloro-	232, 234, 230, 131
Phenol, 2,3,4,6-tetrachloro-	232, 234, 230, 131
Phenol, pentachloro-	266, 268, 264, 165
Dinoseb	211, 163, 147, 117
PCB-28	256, 258, 186, 150
Heptachlor	100, 272, 274, 137
PCB-52	292, 220, 290, 222
Aldrin	66, 263, 265, 261
Octachlorostyrene	308, 310, 380, 378
Chlordane I	373, 375, 272, 237
PCB-101	326, 324, 254, 328
Chlordane II	373, 375, 272, 237
PCB-81	292, 290, 294, 220
Dieldrin	79, 81, 263, 265
PCB-77	292, 290, 294, 220
Endrin	67, 317, 319, 345
PCB-123	326, 324, 254, 328
PCB-118	326, 324, 256, 328
PCB-114	326, 324, 256, 328
PCB-153	360, 362, 290, 358
PCB-105	326, 324, 254, 328
PCB-138	360, 362, 290, 358
PCB-126	326, 324, 254, 328
PCB-167	360, 362, 290, 358
PCB-156	360, 362, 290, 358
PCB-157	360, 362, 290, 358
PCB-180	396, 394, 324, 162
PCB-169	360, 362, 358, 145
PCB-170	396, 394, 324, 326
PCB-189	396, 394, 398, 324

Table A1.2. 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 used 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 at 105°C.

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. Approximately 0.5 g of sample was accurately 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. One sample (AT03056) was analysed in duplicate to assess the reproducibility of the method. Furthermore, two standard reference materials, and a blank sample, were prepared in an identical manner with the batch of samples. The standard reference materials analysed were; 7004 (Loam with elevated analyte levels) certified by the Czech Metrological Institute, and GBW8301 (River Sediment) certified by the State Bureau of Metrology, The People's Republic of China. Recovery data for the standard reference materials and duplicate analysis data is presented below in Table A1.3 and Table A1.4

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 Quantitative analysis

Following preparation, samples were analysed by Inductively Coupled Plasma Atomic Emission Spectrometry (ICP-AES), using a Varian Liberty-100 Sequential Spectrometer. The following metals were quantified directly: arsenic (As), cadmium (Cd), cobalt (Co), copper (Cu), chromium (Cr), lead (Pb), manganese (Mn), nickel (Ni) and zinc (Zn). 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. Ionic mercury, Hg (II), was reduced to elemental mercury, Hg (0), following reduction of the samples with sodium borohydride (0.6% w/v), sodium hydroxide (0.5% w/v) and hydrochloric acid (10 molar). The elemental mercury vapour was carried in a stream of argon into the spectrometer. Two calibration standards were prepared, at 10 µg/l and 100 µg/l, matrix matched to the samples (i.e. in 15% v/v hydrochloric acid and 5% v/v nitric acid for solid samples or 10% v/v nitric acid for aqueous samples). The calibration was validated using a quality control standard (80 µg/l), prepared internally from different reagent stock. Any sample exceeding the calibration range was diluted accordingly, in duplicate, and re-analysed.

A1.2.3 Quality control data

Recovery data for the standard reference materials analysed is presented below in Table A1.3. Duplicate analysis data fro sample AT03056 is presented below in Table A1.4.

Reference material	As (%)	Cd (%)	Cr (%)	Co (%)	Cu (%)	Hg (%)	Mn (%)	Ni (%)	Pb (%)	Zn (%)
CRM7004	88	104	58	102	90	82	80	95	85	89
GBW8301	---	70	56	86	86	---	84	85	78	92

Table A1.3. Percentage recovery data for the standard reference materials analysed

Metal	AT03056a	AT03056b	Difference (%)
Arsenic	<40	<40	0
Cadmium	<1	<1	0
Chromium	118	122	3
Cobalt	16	16	1
Copper	113	105	7
Lead	48	46	4
Manganese	731	735	1
Mercury	0.3	0.3	6
Nickel	96	92	4
Zinc	980	978	0

Table A1.5. Duplicate analysis data for sample AT03056

APPENDIX 2

LIST OF COMPOUNDS RELIABLY IDENTIFIED AND COMPOUNDS TENTATIVELY IDENTIFIED IN THE SAMPLES. * - COMPOUNDS IDENTIFIED ONLY AT TRACE LEVELS USING SELECTIVE ION MONITORING (SIM) METHOD

Sample Number: MI01013	Sample Number: MI01014	Sample Number: MI01015	Sample Number: MI01016	Sample Number: MI01017
Number of compounds isolated: 41	Number of compounds isolated: 21	Number of Compounds isolated: 32	Number of compounds isolated: 53	Number of compounds isolated: 4
COMPOUNDS RELIABLY IDENTIFIED: 1-Decene 1-Dodecene 1-Hexadecene 1-Octadecene 5-Eicosene, (e)- Benzene, (1,1-dimethylbutyl)- Benzene, 1,3-dimethyl- Benzene, 1,4-dichloro- * Benzene, ethyl- Cyclohexane, 1,4-dimethyl-, trans- Dodecane Eicosane Heptadecane Hexadecane Octacosane Octadecane Pentadecane Tetradecanol COMPOUNDS TENTATIVELY IDENTIFIED : 1-Docosene 3-Ethyl-3-hexene Cyclohexane, 1,4-dimethyl-Decane, 2-methyl- Docosane Dodecane, 2-methyl-6-propyl- Heptadecane, 2,6,10,15-tetramethyl- Hexadecane, 2-methyl- Octane Tetracosane Tricosane Tridecane, 3-methyl- Undecane, 3,7-dimethyl-Undecane, 5-methyl-	COMPOUNDS RELIABLY IDENTIFIED: 1-Docosene 1-Dodecene 1-Hexadecene 1-Octadecene 5-Eicosene, (E)- Benzene, 1,3-dimethyl- Benzene, 1,4-dichloro- * Benzene, ethyl- Cyclotetradecane COMPOUNDS TENTATIVELY IDENTIFIED : 1-Decene Benzene, (1,1-dimethylbutyl)- Cyclohexane, 1,4-dimethyl-, cis- Hexadecane Octadecane Octane	COMPOUNDS RELIABLY IDENTIFIED: 1-Docosene 1-Dodecene 1-Octadecene 1-Tetradecene 1-Tricosanol Benzene, 1,4-dichloro- * Benzene, ethyl-E- 14-Hexadecenal Octane, 4-methyl- Tricosane COMPOUNDS TENTATIVELY IDENTIFIED: 1-Decene 1-Octanol, 2,2-dimethyl- Benzene, 1,3-dimethyl- Cyclohexane, 1,4-dimethyl-Decane, 2-methyl- Decane, 3,8-dimethyl- Eicosane Heneicosane Heptacosane Heptadecane, 2-methyl- Heptane, 2,4-dimethyl-Hexadecane Hexadecane, 7,9-dimethyl-Nonane, 3,7-dimethyl- Octadecane Octane Pentacosane Pentadecane, 8-hexyl- Tetracosane Tetradecane	COMPOUNDS RELIABLY IDENTIFIED: 1-Dodecene 1-Heptadecene 1-Pentadecene 1-Tetradecene 9-Nonadecene Benzene, 1,2,4,5-tetrachloro- * Benzene, 1,2,4-trichloro- * Benzene, 1,3,5-trichloro- * Benzene, 1,3-dichloro- * Benzene, 1,4-dichloro- * Benzene, ethyl- Benzene, hexachloro- * Benzene, pentachloro- * Cyclohexane, 1,4-dimethyl-Eicosane Octadecane Octane, 4-methyl- Pentadecane, 2-methyl-Tetradecane COMPOUNDS TENTATIVELY IDENTIFIED : 1-Decene 1-Octene, 4-methyl- Benzene, 1,3-dimethyl- Decane, 2,2,5-trimethyl- Decane, 2,4,6-trimethyl- Decane, 2-methyl- Decane, 3,6-dimethyl- Decane, 3,8-dimethyl- Docosane Dodecane Heptane, 2,4-dimethyl-Hexadecane, 2,6,10,14-tetramethyl- Hexane, 3,3-dimethyl- Hexatriacontane Nonacosane Octane Octane, 2,2-dimethyl-	COMPOUNDS RELIABLY IDENTIFIED: Benzene, 1,2-dichloro- * Benzene, 1,3-dichloro- * Benzene, 1,4-dichloro- * COMPOUNDS TENTATIVELY IDENTIFIED : Tributylamine

AT03049	AT03050	AT03051	AT03052	AT03053	AT03054	AT03055	AT03056	AT03057	AT03058
No. isolated: 71	No. isolated: 19	No. isolated: 18	No. isolated: 19	No. isolated: 38	No. isolated: 12	No. isolated: 10	No. isolated: 15	No. isolated: 40	No. isolated: 43
COMPOUNDS RELIABLY IDENTIFIED: 1-Methylpenta-1,3-dienyl)benzene 1(2H)-Naphthalenone, 3,4 dihydro-3-methyl- 1(2H)-Naphthalenone, 3,4 dihydro-5-methyl- 1(2H)-Naphthalenone,3,4-dihydro-5,7-dimethyl- 1(2H)-Naphthalenone, 3,4 dihydro-5,8-dimethyl- Benzene, 1,3,5-trimethyl-2 (1,2-propadienyl)- Ethanol, 2-butoxy-, phosphate (3:1) Naphthalene, 1,2,3,4 tetrahydro-1,4,6-trimethyl- Naphthalene, 1,2-dihydro 3,5,8-trimethyl- Phenol, 2-nitro- * Phenol, 2,4,6-trichloro-* COMPOUNDS GROUPS TENTATIVELY IDENTIFIED Naphthalenone derivatives Indanone derivatives Phenanthrene derivatives Aliphatic hydrocarbons	COMPOUNDS RELIABLY IDENTIFIED: 1,2-Benzenedicarboxylic acid, bis(2-ethylhexyl) ester Benzenemethanol Benzene, 1,2-dichloro- * Benzene, 1,3-dichloro- * Benzene, 1,4-dichloro- * Benzene, 1,2,4-trichloro- * Naphthalene Nonadecane Tetracosane, 2,6,10,15,19,23 hexamethyl- COMPOUNDS GROUPS TENTATIVELY IDENTIFIED Aliphatic hydrocarbons	COMPOUNDS RELIABLY IDENTIFIED: 1,2-Benzenedicarboxylic acid, bis(2-ethylhexyl) ester Benzenemethanol Benzene, 1,2-dichloro- * Benzene, 1,3-dichloro- * Benzene, 1,4-dichloro- * Benzene, 1,2,4 trichloro- * COMPOUNDS GROUPS TENTATIVELY IDENTIFIED None	COMPOUNDS RELIABLY IDENTIFIED: 1,2-Benzenedicarboxylic acid, bis(2-ethylhexyl) ester 1,2-Benzenedicarboxylic acid, dibutyl ester Benzene, 1,2-dichloro- * Benzene, 1,4-dichloro- * Benzene, 1,2,4-trichloro- * COMPOUNDS GROUPS TENTATIVELY IDENTIFIED None	COMPOUNDS RELIABLY IDENTIFIED: (10.beta.h)-des-a-Lupane 1,2-Benzenedicarboxylic acid, bis(2-ethylhexyl) ester 1,2-Benzenedicarboxylic acid, dibutyl ester 4B, 8-dimethyl-2-isopropylphenanthrene, 4b,5,6,7,8,8a,9,10-octahydro- Benzene, 1,2-dichloro- * Benzene, 1,4-dichloro- * Benzene, 4-(1,1 dimethylethyl)-1,2 dimethyl- des-a-Lup-5(10)ene Hexadecane, 2,6,10,14 tetramethyl- Pentadecane, 2,6,10,14 tetramethyl- Phenol, 2,6-bis(1,1 dimethylethyl)-5-methyl- Sulfur, mol. (S8) COMPOUNDS GROUPS TENTATIVELY IDENTIFIED Oxygenated hydrocarbons Alkyl benzenes Aliphatic hydrocarbons	COMPOUNDS RELIABLY IDENTIFIED: 1,2-Benzenedicarboxylic acid, bis(2-ethylhexyl) ester 1,2-Benzenedicarboxylic acid, dibutyl ester Benzene-methanol Benzene, 1,2-dichloro- * Benzene, 1,4-dichloro- * Benzene, 1,2,4-trichloro- * COMPOUNDS GROUPS TENTATIVELY IDENTIFIED None	COMPOUNDS RELIABLY IDENTIFIED: 1,2-Benzenedicarboxylic acid, bis(2-ethylhexyl) ester Heptadecane COMPOUNDS GROUPS TENTATIVELY IDENTIFIED Aliphatic hydrocarbons Oxygenated hydrocarbons	COMPOUNDS RELIABLY IDENTIFIED: 1,2-Benzenedicarboxylic acid, bis(2-ethylhexyl) ester Benzene, 1,4-dichloro- * Phenol, nonyl-, mixture of isomers COMPOUNDS GROUPS TENTATIVELY IDENTIFIED Aliphatic hydrocarbons	COMPOUNDS RELIABLY IDENTIFIED: 2,4,7,9-Tetramethyl-5 decyne-4,7-diol Benzothiazole, 2 (methylthio)- Carbamodithioic acid, dimethyl-, methyl ester Benzene, 1,2,3-trichloro- * Benzene, 1,2,4-trichloro- * Ethanol, 2-butoxy-, phosphate (3:1) Phenol, nonyl-, mixture of isomers Phenol, 2,6-dichloro-3 methyl- Phenol, 2,4-dichloro- * Phenol, 2,6-dichloro- * Phenol, 2,4,6-trichloro- * Phenol, 4-(1,1,3,3 tetramethylbuty)- COMPOUNDS GROUPS TENTATIVELY IDENTIFIED Oxygenated hydrocarbons Alkyl benzenes Phenol derivatives	COMPOUNDS RELIABLY IDENTIFIED: 1,2-Benzenedicarboxy-lic acid, bis(2-ethylhexyl) ester 1,2-Benzenedicarboxy-lic acid, dimethyl ester Benzene, 1,2,3-trichloro-4 methyl- Benzene, 1,2,4-trichloro-3 methyl- Benzene, 1,2-dichloro-* Benzene, 1,3-dichloro-* Benzene, 1,4-dichloro-* Benzene, 1,2,3-trichloro* Benzene, 1,2,4-trichloro* Benzene, 1,3,5-trichloro* Benzene, 1,2,3,4-tetrachloro- * Benzene, 1,2,3,5 tetrachloro- * Benzene, 1,2,4,5 tetrachloro- * Benzene, pentachloro- Benzene, hexachloro- * Benzene, trichloro (chloromethyl)- Benzenemethanol Ethanone, 1-phenyl- Butadiene, hexachloro-* Phenol, 4-(1,1,3,3 tetramethylbuty)- Phenol, 4-methyl- Phenol, nonyl-, mixture of isomers COMPOUNDS GROUPS TENTATIVELY IDENTIFIED Phenanthrene derivatives Oxygenated alkyl benzenes Aliphatic hydrocarbons

APPENDIX 3
TOXICOLOGICAL OUTLINES FOR ORGANIC COMPOUNDS

A3.1 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 2000, 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 DBP (also called DnBP) can both damage the male and female reproductive systems (Chan & Meek 1994, ATSDR 2000). Both can damage sperm production (ATSDR 2000, Wine *et al.* 1997), impair reproductive success (Chan & Meek 1994, Ema *et al.* 1995, ATSDR 2000, Wine *et al.* 1997) and cause teratogenicity (malformation of the offspring)(Chan & Meek 1994, Ema *et al.* 1993; Ema *et al.* 1995, ATSDR 2000). The liver and kidneys can also be affected by DnBP (Chan & Meek 1994; ATSDR 2000) and DEHP (ATSDR 2000).

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).

References

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

ATSDR (2000) Toxicological Profiles. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service (CD-ROM)

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

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

Cole, R.A., Hardwick, R.C. & Fyfield, T.P. (1984) Plastics as a cause of plant damage and death: a review of the role of di butyl phthalate. Scientific Horticulture 35: 1-10

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

CSTEE (1998) Phthalate migration from soft PVC toys and child-care articles. Opinion expressed at the CSTEE third plenary meeting , Brussels, 24 April 1998. Publ: EU Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE), Brussels, 32pp

CSTEE (1999) Opinion on TNO, LGC and U.S. CPSC reports on phthalate migration test validation-Opinion adopted a the 11th CSTEE plenary meeting on the 28th of September 1999. Publ: EU Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE), Brussels, 32pp

DHHS (2000) 9th report on carcinogens. Publ: US Department of Health and Human Services, 252pp + appendices.

Dirven, H.A.A.M., van den Broek, P.H.H., Arends, A.M.M., Nordkamp, H.H., de Lepper, A.J.G.M., Henderson, P.Th. & Jongeneelen, F.J. (1993) Metabolites of the plasticizer di(2-ethylhexyl)phthalate in urine samples of workers in polyvinylchloride processing industries. International Archives of Occupational and Environmental Health 64(8): 549-554

EC (1999) Commission Decision of 7 December 1999 adopting measures prohibiting the placing on the market of toys and childcare articles intended to be placed in the mouth by children under three years of age made of soft PVC containing one or more of the substances di-iso-nonyl phthalate (DINP), di(2-ethylhexyl) phthalate (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

EC (2000) Commission Decision of 6th December 2000 prolonging for the fourth time the validity of decision 1999815/EC concerning measures prohibiting the the placing on the market of toys and childcare articles intended to be placed in the mouth by children under three years of age made of soft PVC containing certain phthalates (2000/769/EC). OJ L 306, 7.12.2000: 37

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

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

Jaakola, J.J.K., Oie, L., Nafstad, P., Botten, G., Samuelsen, S.O. & Magnus, P. (1999) Interior surface materials in the home and the development of bronchial obstruction in young children in Oslo, Norway. American Journal of Public Health 89(2): 188-192

Janssen, P., van Veen, M., van Appeldorn, M. & Speijers, G. (1998) Phthalates in teething-rings/animal figures for infants (CSR Advisory Report no 5293). Dutch National Institute for Public Health and the Environment (RIVM), Centre for Substances and Risk Assessment, 11pp

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

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

MacKenzie, D. (1997) Alarm sounds over toxic teething rings. New Scientist 154, 14 June 1997, p10

MAFF (1987) Ministry of Agriculture Fisheries and Food, UK. Survey of Plasticiser levels in food contact materials and in foods. Food Surveillance Paper No: 21, Publ HMSO, London, 107pp

MAFF (1990) Ministry of Agriculture Fisheries and Food, UK. Plasticisers: Continuing Surveillance. Food Surveillance Paper No: 30. Publ HMSO London, 53pp

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

Oie, L., Hersoug, L.-G. & Madsen, J.O. (1997) Residential exposure to plasticizers and its possible role in the pathogenesis of asthma. Environmental Health Perspectives 105(9): 972-978

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

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

Stringer, R., Labunska, I., Santillo, D., Johnston, P., Siddorn, J. & Stephenson, A. (2000) Concentrations of Phthalate Esters and Identification of Other Additives in PVC Children's Toys. Environmental Science and Pollution Research 7(1): 27-36

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

A3.2 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 2000, Newhook & Meek 1994, van Birgelen 1998). Recent research (van Birgelen 1988) suggests that HCB has dioxin-like toxicity and that, based on a preliminary toxic equivalence factor (TEF) of 0.0001, HCB could contribute significantly to the dioxin-type toxicity of human milk based on PCB/PCDD/PCDF toxicity equivalents. In many countries, this could mean an increase of 10% - 60%, but in countries with high HCB exposure levels, the effects could be even greater. In Spain and the Czech Republic, inclusion of HCB in total breastmilk TEQ estimates could lead to totals 6 times higher than based only on PCBs and PCDFs. Slovakia and India also have very high HCB levels; other countries (eg Austria) high levels in previous decades. It has been suggested that more epidemiological studies should be undertaken, especially in the most highly contaminated countries.

With the exception of occupational settings, almost all human exposure occurs via food. The greatest body of information on HCB toxicity to humans derives from an incident in Turkey between 1955 and 1959, when HCB-treated grain was made into bread. More than 600 people experienced porphyria cutanea tarda. Children of exposed women had skin lesions and 95% of them died at less than one year old. In the long term (20-30 years), some people continued to have abnormal porphyrin biochemistry and neurological, orthopaedic and dermatological symptoms persisted. Hexachlorobenzene is also thought to have caused porphyria cutanea tarda in populations exposed industrially and through food (Newhook & Meek 1994). High concentrations of HCB were found in the air around a chlor-alkali and organochlorine manufacturing plant at Flix in Spain and in blood of workers and local residents (Sala *et al.* 1999, Grimalt *et al.* 1994). One study found a significant elevation in incidence of cancer of the thyroid, soft tissues and at unspecified

sites in the men of the community (Grimalt *et al.* 1994) and the authors of one study stated that HCB exposure was associated with specific health effects in the most highly exposed subjects (Sala *et al.* 1999).

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

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

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

HCB is also included in the list of priority hazardous substances agreed by the Third and Fourth North Sea Conferences (MINDEC 1990 & 1995), where continuous reduction of all hazardous substances was agreed with the ultimate aim of reducing environmental concentrations of hazardous substances to near background levels (synthetic substances to zero) within the next 25 years. The 1998 Ministerial Meeting of the OSPAR Commission (OSPAR 1998a) further reinforced these objectives. HCB is included on the OSPAR 1998 List of Candidate Substances, Annex 3 of the OSPAR Strategy with regard to Hazardous Substances (OSPAR 1998b). In addition, HCB is regulated under the 1995 Barcelona Convention, the Rotterdam (PIC) Convention and the International Joint Comission on the Great Lakes (IJC) has called for all uses to be eliminated.

References

ASTDR (2000) ATSDR's toxicological profiles on CD-ROM. U.S. Department of Health and Human Services, Public Health Service, CRC Press Inc

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

UNEP (1997) Decisions adopted by the Governing Council at its nineteenth session: 13c. International action to protect human health and the environment through measures which will reduce and/or eliminate

emissions and discharges of persistent organic pollutatns, including the development of an international legally binding instrument. UNEP Governing Council, 7th February 1997.
US EPA (1989). EPA Factsheets for Regulated Chemicals. Hexachlorobenzene. Washington, DC.
Van Birgelen, A.P.J.M. (1998) Hexachlorobenzene as a possible major contributor to the dioxin activity of human milk. Environ. Health Persp. 106(11): 683-688
Ware, G.W. (Ed.)(1988) Chlorobenzene. Rev. Environ. Contam.Toxicol. 106: 37-49
Ware, G.W. (Ed.)(1988) Ortho-, meta- and para- dichlorobenzene. Rev. Environ. Contam.Toxicol. 106: 51-56

APPENDIX 4
TOXICOLOGICAL OUTLINES FOR HEAVY METALS

A4.1 CHROMIUM

Production and Use

Of the total chromium produced annually, about 60-70% is used in alloys, including stainless steel, which contains varying amounts of iron, chromium (10-26%) and nickel, depending on the properties required in the final product. About 15% is also used in the general chemical industry, where chromium compounds are commonly used as tanning agents, textile pigments and preservatives, anti-fouling paints, catalysts, corrosion inhibitors, drilling muds, high temperature batteries, fungicides, wood preservatives, and in metal finishing and electroplating (ATSDR 2000, Alloway 1990, Greenwood & Earnshaw 1984).

Environmental Levels, Contamination and Behaviour

Chromium is found in varying concentrations in nearly all uncontaminated aquatic and terrestrial ecosystems (see Table A4.1.1). However, in areas associated with anthropogenic emissions, ecosystem levels can far exceed natural, background concentrations (see Table A4.1.2).

Environmental Matrix	Concentration	Reference
Seawater (open ocean)	0.057-0.234 μg/l	Bryan & Langston 1992
Freshwater	1.30 μg/l	ATSDR 2000
Drinking water	0.4-8.0 μg/l	ATSDR 2000
Marine sediment	30-200 mg/kg	Bryan & Langston 1992,
Freshwater sediment / suspended particulates	1-500 mg/kg	ATSDR 2000
Soil	<1-100 mg/kg 4-80 mg/kg	Alloway 1990 Dudka & Adriano 1997

Table A4.1.1. Background concentrations of chromium found in water, sediment and soil

Environmental Matrix	Concentration	Reference
Marine sediment, Loughor Estuary (tin plate production) in South Wales	800 mg/kg	Bryan & Langston 1992
Marine sediment, Sawyer's Bay, New Zealand (tannery waste)	3700 mg/kg	Bryan & Langston 1992
Soil, chromium smelting, Japan	30-4560 mg/kg	Dudka & Adriano 1997
Soil, of sewage sludge amended farms, UK	138-2020 mg/kg	Alloway 1990
MSW incinerator ash, UK	44-1328 mg/kg	Mitchell *et al.* 1992

Table A4.1.2. Chromium concentrations associated with anthropogenic contamination and waste

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 transport between different media, 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 2000, Kimbrough *et al.* 1999). This trivalent form does not generally 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 & 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. For example, the landfilling of chromium (III) tannery waste with other acidic industrial wastes, or domestic sewage, which on decomposition can yield acidic conditions, can result in the oxidation of chromium (III) to chromium (VI) (Mukherjee 1998, Outridge & Sheuhammer 1993, UNEP 1991, Richard & 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 2000, Goyer 1996). The minimum human daily requirement of chromium (III) for optimal health is not known, but a daily ingestion of 50-200 μg/day has been estimated to be safe and adequate. However, although an essential food nutrient, very large doses may be harmful (ATSDR 2000).

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 2000). In addition, long-term occupational exposure to airborne levels of chromium higher than those in the natural environment has been associated with lung cancer, and the US. Department of Health and Human Services has classified chromium (VI) compounds as 'known to be human carcinogens' (USPHS 2002). Individuals at most risk include those in chromate-production industries and chromium pigment manufacture

and use; and similar risks may exist amongst chromium-alloy workers, stainless steel welders, and chrome-platers. (Kimbrough 1999, USPHS 2002).

The aquatic toxicology of chromium is also dependent 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 μg/l of dissolved chromium (VI) (Bryan & Langston 1992).

Legislation

Applicable standards set by the Ministry of Science, Technology and Environment of Thailand for chromium include 'Effluent standards for industrial and industrial estate sources' (No.3, BE 2539). These standards set limits for chromium in effluents of 0.25 mg/l chromium (VI) and 0.75 mg/l chromium (III) (Homchean 1998).

References

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

ATSDR (2000) Toxicological profile for chromium on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

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

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

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

Greenwood, N.N. & Earnshaw, A. (1984) Chemistry of the Elements. Pergamon Press Ltd. ISBN 0080220576

Homchean, K. (1998) Industrial Estate Authority of Thailand strategy for environmental compliance, Proceedings of the fifth international conference on environmental compliance and enforcement, Monterey, California, pp101-112

Kimbrough, D.E, Cohen, Y., Winer, A.M., Creelman, L. & Mabuni, C. (1999) A critical assessment of chromium in the Environment. Critical Reviews in Environmental Science and Technology 29(1): 1-46

Mitchell, D.J., Wild, S.R. & Jones, K.C. (1992) Arrested municipal solid waste incinerator fly ash as a source of heavy metals to the UK environment. Environmental Pollution 76: 79-84

Mukherjee, A.B. (1998) Chromium in the environment of Finland. The Science of the Total Environment 217: 9-19

Outridge, P.M. & Schuehammer, A.M. (1993) Bioaccumulation and toxicology of chromium: implications for wildlife. Reviews of Environmental Contamination and Toxicology 130: 31-77

Richard, F.C. & Bourg, A.C.M. (1991) Aqueous geochemistry of chromium: a review. Water Research 25(7): 807-816

UNEP (1991) Tanneries and the environment. A technical guide to reducing the environmental impact of tannery operations. Technical Report Series No. 4. United Nations Environment Programme Industry and Environment Office

USPHS (2002). 10th Report on Carcinogens. U.S. Department of Health and Human Services, US Public Health Service, National Toxicology Program

A4.2 COPPER

Production and Use

Copper is can produced from ore, or recovered from secondary sources (i.e. scrap). The latter process is far less energy intensive, and therefore is playing an increasingly important role in terms of global copper production (UNEP 1993).

Copper is a highly malleable and ductile metal, as well as being an excellent conductor of heat and electricity. Its principal use is as an electrical conductor (copper cables and wires), however it is also widely employed in coinage alloys, in traditional alloys such as bronze (copper and tin), brass (copper and zinc) and Monel (copper and nickel), in corrosive-resistant and decorative plating, in munitions and in dental alloys. Its compounds are used as chemical catalysts, wood preservatives, algicides, fungicides, anti-fouling paints, disinfectants, nutritional supplements in fertilisers and feeds, in petroleum refining and as printing inks and dyes (ATSDR 2000, UNEP 1993).

Environmental Levels, Contamination and Behaviour

Copper is a relatively abundant "trace" metal, found at varying concentrations in nearly all uncontaminated aquatic and terrestrial ecosystems (see Table A4.2.1). However, as anthropogenic emissions of copper exceed those from natural sources, elevations above these natural, background concentrations are often found (see Table A4.2.2).

Environmental Matrix	Concentration	Reference
Seawater (English Channel, Irish Sea, North Sea)	0.35-4.0 μg/l (coastal) 0.14-0.9 μg/l (open ocean)	Law *et al*. 1994
Seawater (background)	0.1 μg/l	Sadiq 1992, Bryan & Langston 1992
Freshwater, UK	<20 μg/l	Mance *et al*. 1984
Soil	20 –30mg/kg	Alloway 1990
Marine sediment	10-30 mg/kg	UNEP 1993, Bryan & Langston 1992
Freshwater sediment	45-50 mg/kg	Salomons & Forstner 1984

Table A4.2.1. Background concentrations of copper found in water, sediment and soil

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 2000, 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.

Site Description	Concentration	Reference
Seawater, Restronguet Creek, UK (receives acidic drainage from past and present mining activities)	>2000 μg/l	Bryan & Langston 1992
Sediment, Restronguet Creek	3000 mg/kg	Bryan & Langston 1992
Sediment, Izmir Bay (receives large quantities of industrial and domestic wastes)	33-866 mg/kg	UNEP 1993
Soil (nickel-copper mining and smelting, Sudbury, Ontario)	11-1890 mg/kg	Dudka *et al*. 1995
Soil treated with copper fungicidal sprays	110-1500 mg/kg	Alloway 1990
MSW incinerator ash (UK)	296-1307 mg/kg	Mitchell *et al*. 1992

Table A4.2.2 copper concentrations associated with sites of anthropogenic contamination and waste

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 sulfate, 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 2000, 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 2000).

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 2000). Human deficiency is characterised by anaemia, resulting from defective haemoglobin synthesis (Goyer 1996). At the other extreme, however, vomiting, hypotension, jaundice, coma and even death, can result from acute poisoning (ATSDR 2000).

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 2000). 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 2000). 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 and can result in liver and kidney damage and haemolytic anaemia (ATSDR 2000).

In addition to these effects, developmental and reproductive damage following exposure to high levels of copper has been seen in animals. No such effects have been reported in humans (ATSDR 2000).

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 μg/l (Bryan & Langston 1992). For example, studies have shown that at levels of 2 μg/l, the survival rate of young bay scallops was significantly affected; and in the embryos of oysters and mussels concentrations of 5 μg/l were seen to induce abnormalities. A similar concentration resulted in increased mortality 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 & DePledge 1998, UNEP 1993), and embryonic sensitivity in fish exposed to levels of 25 μg/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).

Legislation

Applicable standards set by the Ministry of Science, Technology and Environment of Thailand for copper include 'Effluent standards for industrial and industrial estate sources' (No.3, BE 2539). These standards set limits for copper in effluents of 2.0 mg/l (Homchean 1998).

References

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

ATSDR (2000) Toxicological profile for copper on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

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

Conradi, M. & DePledge, M.H. (1998) Population responses of the marine amphipod *Corophium volutator* (Pallas, 1766) to copper. Aquatic Toxicology 44: 31-45

Dudka, S., Ponce-Hernandez, R. & Hutchinson, T.C. (1995) Current level of total element concentrations in the surface layer of Sudbury's soils. The Science of the Total Environment 162: 161-171

Giudici, M., Milgiore, L. & Guarino, S.M. (1989) Effects of chronic exposure to cadmium or copper on *Idothea baltica* (Crustacea: Isopoda). Marine Pollution Bulletin 20(2): 69-73

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

Homchean, K. (1998) Industrial Estate Authority of Thailand strategy for environmental compliance, Proceedings of the fifth international conference on environmental compliance and enforcement, Monterey, California, pp101-112

Law, R.J., Waldock, M.J., Allchin, C.R., Laslett, R.E. & Bailey, K.J. (1994) Contaminants in seawater around England and Wales: results from monitoring surveys, 1990-1992. Marine Pollution Bulletin 28(11): 668-675

Mance, G., Brown, V.M. & Yates, J. (1984) Proposed environmental quality standards for List II substances in water. Copper. Water Research Centre Technical Report TR210
Mitchell, D.J., Wild, S.R. & Jones, K.C. (1992) Arrested municipal solid waste incinerator fly ash as a source of heavy metals to the UK environment. Environmental Pollution 76: 79-84
Sadiq, M. (1992) Toxic metal chemistry in marine environments. Marcel Dekker Inc., New York, Basel, Hong Kong. ISBN 0824786475
Salomons, W. & Forstner, U. (1984) Metals in the hydrocycle. Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, ISBN 3540127550
UNEP (1993) Preliminary assessment of the state of pollution of the Mediterranean Sea by zinc, copper and their compounds and proposed measures. Mediterranean Action Plan UNEP (OCA)/MED/WG.66/Inf.3, Athens 3-7 May 1993

A4.3 LEAD

Production and Use

Lead is primarily obtained from the sulphide ore galena, but is also obtained from the secondary smelting of secondary sources including recycled lead from electric storage batteries (Dudka and Adriano 1997).

Uses of lead and its compounds are extensive. As a metal, it has historically been used as pipe-work for water distribution, or as containers for storing for corrosive liquids (e.g. sulphuric acid). Its alloys are used in welding, printing and as anti-friction metals; and great quantities, both of the metal and its dioxide, are used in electric storage batteries. Other uses include cable coverings, ammunition, and in the manufacture of lead tetraethyl, used as an anti-knock compound in petrol. Compounds of lead are used as paint pigments, PVC stabilisers, pesticides, varnishes, lubricants, as glazes for pottery and porcelain, and in leaded glass crystal (Budavari *et al.* 1989, ATSDR 2000).

Environmental Levels, Contamination and Behaviour

Lead is present in nearly all uncontaminated aquatic and terrestrial ecosystems, though typically at very low levels (see Table A4.3.1).

Environmental Matrix	Concentration	Reference
Seawater (estuarine waters around England and Wales)	24-880 ng/l	Law *et al*. 1994
Seawater (open ocean)	5-71 ng/l	ATSDR 2000, Bryan & Langston 1992, Law *et al*. 1994
Freshwater (mean value from 39,490 measurements)	3.9 µg/l	ATSDR 2000
Drinking water	<5-30 µg/l	ATSDR 2000
Soil	10-30 mg/kg	Alloway 1990
Freshwater / marine sediment	20-30 mg/kg	ATSDR 2000

Table A4.3.1. Background concentrations of lead found in water, sediments and soil

As anthropogenic emissions far exceed those from natural sources, elevations above these natural, background concentrations are often found (Table A4.3.2).

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. In such cases, 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 2000, Alloway 1990). As with all metals, however, speciation is critical when assessing bioavailability and the potential threat to the environment.

Site Description	***Concentration***	Reference
River water, Donana National Park, Spain (close to mining site)	<5-2500 μg/l	Pain *et al.* 1998
Drinking water, USA (contaminated from lead pipes / lead solder).	500 μg/l	ATSDR 2000
Soil, Socorro, New Mexico (USA), close to an abandoned lead smelter	25-10,000 mg/kg	Brandvoid *et al.* 1996
Paddy soil, Taiwan, close to plastic stabiliser manufacturing plant	6.3-12,740 mg/kg	Chen 1991
Soil close to lead smelting sites, Montreal, Canada	40-14,860 mg/kg	Sauve *et al.* 1997
Gannel estuary sediments, UK. Received waste from old lead mines	2700 mg/kg	Bryan & Langston 1992
Oiartzun river sediments, Spain, close to lead-zinc mining sites	68-5540 mg/kg	Sanchez *et al.* 1994

Table A4.3.2. Lead concentrations associated with anthropogenic contamination

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 2000).

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 2000, Sadiq 1992, Alloway 1990). As can the downward movement of lead from soil to groundwater by leaching (ATSDR 2000).

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

areas of heavy hunting (spent lead shot); and in urban and industrialised areas (ATSDR 2000).

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 μg/dl in children, and 10-100 μg/dl in adults have been associated with a wide range of adverse effects. Including 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 2000, Bernard *et al.* 1995, Goyer 1993, Nriagu 1988).

It is currently thought that there may be no level of blood-lead that does not produce a toxic effect, particularly in the developing central nervous system (ATSDR 2000, 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 2000, WHO 1989, Collivignarelli *et al.* 1986). In addition, birds feeding on contaminated prey or ingesting lead shot into their gizzards, can be exposed to severe levels of lead. This can result in high kidney, liver and bone concentrations, reduced growth and development, behavioral abnormalities, and sometimes death (Mateo *et al.* 1997, WHO 1989).

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

Legislation

Applicable standards set by the Ministry of Science, Technology and Environment of Thailand for lead include ‘Effluent standards for industrial and industrial estate sources’ (No.3, BE 2539). These standards set limits for lead in effluents of 0.2 mg/l (Homchean 1998).

References

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

ATSDR (2000) Toxicological profile for lead on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

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

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

Brandvold, L.A., Popp, B.R. & Swartz, S.J. (1996) Lead content of plants and soils from three abandoned smelter areas in and near Socorro, New Mexico. Environmental Geochemistry and Health 18: 1-4

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

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

Chen, Z.S. (1991) Cadmium and lead contamination of soils near plastic stabilising materials producing plants in Northern Taiwan, Water, Air and Soil Pollution 57-58: 745-754

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

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

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

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

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

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

Law, R.J., Waldock, M.J., Allchin, C.R., Laslett, R.E. & Bailey, K.J. (1994) Contaminants in seawater around England and Wales: results from monitoring surveys, 1990-1992. Marine Pollution Bulletin 28(11): 668-675

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

Pain, D.J., Sanchez,A. & Meharg, A.A.(1998) The Donana ecological disaster: contamination of a world heritage estuarine marsh ecosystem with acidified pyrite mine waste. The Science of the Total Environment 222: 45-54

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

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

Sanchez, J., Carmen Vaquero, M. & Legorburu, I. (1994) Metal pollution from old lead-zinc mine works: biota and sediment from Oiartzun Valle. Environmental Technology 15: 1069-1076

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

World Health Organisation (1989) Lead; environmental aspects. Environmental Health Criteria 85. ISBN 9241542853

A4.4 NICKEL

Production, Use and Anthropogenic Sources

Primary nickel is recovered from mined ore; secondary nickel can also be recovered from scrap metal (Greenwood and Earnshaw 1984).

Nickel 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 2000, 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 A4.4.1). However, as anthropogenic emissions of nickel far exceed those from natural sources, elevations above these natural, background concentrations, are often found (see Table A4.4.2).

Environmental Matrix	Concentration	Reference
Seawater	0.1-0.5 μg/l	ATSDR 2000, Law *et al*. 1994
Freshwater	<10-20 μg /l	ATSDR 2000, Mance & Yates 1984
Freshwater sediment	45-65 mg/kg	Salomons & Forstner 1984
Soil	50 mg/kg (range 5-500 mg/kg)	ATSDR 2000, Alloway 1990

Table A4.4.1. 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 2000, Mance & Yates 1984).

Nickel is significantly bioaccumulated in some, but not all, aquatic organisms. Typical bioconcentration factors for significant bioaccumulators include marine phytoplankton <20-2000, seaweeds 550-2000 and algae 2000-40,000 (ATSDR 2000).

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 2000, Alloway 1990).

Site Description	Concentration	Reference
Drinking water, near a large, open-pit mine, USA	200 µg/l	ATSDR 2000
Seawater, coastal and estuarine sites of industrial and domestic discharges, UK	0.23-4.9 µg/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)	100-3000 mg/kg 160-12300 mg/kg	Freedman & Hutchinson 1980 Hazlett *et al.* 1983
Soil, Upper Silesia, Poland (mining and smelting)	5-2150 mg/kg	Dudka *et al*. 1995
MSW incinerator ash, UK	45-2204 mg/kg	Mitchell *et al.* 1992

Table A4.4.2. Nickel concentrations associated with anthropogenic contamination and waste

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 2000, 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 10th Report on Carcinogens, therefore lists nickel compounds as known to be human carcinogens (USPHS 2002).

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 2000, Goyer 1996). Other serious consequences of long term exposure to atmospheric nickel may include chronic bronchitis and reduced lung function (ATSDR 2000). 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 2000).

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

Applicable standards set by the Ministry of Science, Technology and Environment of Thailand for nickel include 'Effluent standards for industrial and industrial estate sources' (No.3, BE 2539). These standards set limits for nickel in effluents of 1.0 mg/l (Homchean 1998).

References

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

ATSDR (2000). Toxicological profile for nickel on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

Chashschin, V.P., Artunia, G.P. and Norseth, T. (1994). Congenital defects, abortion and other health effects in nickel refinery workers. The Science of the Total Environment 148: 287-291

Dudka, S., Piotrowska, M., Chlopecka, A. and Witek, T. (1995). Trace metal contamination of soils and crop plants by mining and smelting industry in Upper Silesia, South Poland. J. Geochem. Explor. 52: 237-250

EEC (1976). Council Directive 76/464/EEC of 4 May 1976 on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community (OJ L 129, 18.5.1976, p. 23)

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

Florence, T.M., Stauber, J.L. and Ahsanullah, M. (1994). Toxicity of nickel ores to marine organisms. The Science of the Total Environment 148: 139-155

Freedman, B. and Hutchinson, T.C. (1980). Pollutant inputs from the atmosphere and accumulations in soils and vegetation near a nickel-copper smelter at Sudbury, Ontario, Canada. Canadian Journal of Botany 58: 108-132

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

Greenwood, N.N. and Earnshaw, A. (1984). Chemistry of the Environment. Pergamon Press Ltd. ISBN 0080220576

Hazlett, P.W., Rutherford, G.K. and van Loon, G.W. (1993). Metal contaminants in surface soils and vegetation as a result of nickel/copper smelting at Coniston, Ontario, Canada. Reclam. Reveg. Res. 2: 123-137

Homchean, K. (1998) Industrial Estate Authority of Thailand strategy for environmental compliance, Proceedings of the fifth international conference on environmental compliance and enforcement, Monterey, California, pp101-112

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

Law, R.J., Waldock, M.J., Allchin, C.R., Laslett, R.E. and Bailey, K.J. (1994). Contaminants in seawater around England and Wales: results from monitoring surveys, 1990-1992. Marine Pollution Bulletin 28, 11: 668-675

Mance, G. and Yates, J. (1984). Proposed environmental quality standards for List II substances in water. Nickel. Water Research Centre Technical Report TR211

Mitchell, D.J., Wild, S.R. and Jones, K.C. (1992). Arrested municipal solid waste incinerator fly ash as a source of heavy metals to the UK environment. Environmental Pollution 76: 79-84

Nriagu, J.O. (1980). Global cycle of nickel. In Nickel in the Environment, John Wiley and Sons, New York: 1-26

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

Nriagu, J.O. and Pacyna, J.M. (1988). Quantitative assessment of World-wide contamination of air, water and soils with trace metals. Nature 333: 134-139

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

Steenkamp, V.E., du Preez, H.H., Schoonbee, H.J. and Shafir, A. (1995). Accumulation of nickel in three freshwater crab populations. S. Afr. J. Wildl. Res. 25, 3: 67-76

USPHS (2002). 10th Report on Carcinogens U.S. Department of Health and Human Services

A4.5 ZINC

Production and Use

Zinc is primarily produced from ores through smelting or electrolyses, but can also be recovered from secondary sources i.e. "old" scrap (ATSDR 2000, Kroschwitz and Howe-Grant 1995).

Zinc is one of the most extensively utilised "trace" metals (Nriagu 1990). It is most commonly employed as a protective coating for other metals e.g. galvanised steel, or as a component of bronze, brass and die-casting alloys. In addition, zinc salts are widely employed as wood preservatives, herbicides, catalysts, analytical reagents, vulcanisation accelerators for rubber, and stabilisers in PVC. They can also be found in ceramics, textiles, fertilisers, paints, pigments, batteries and dental, medical, and household products (ATSDR 2000, Annema & Ros 1994, UNEP 1993, Budavari *et al.* 1989).

Environmental Levels, Contamination and Behaviour

Zinc is a relatively abundant "trace" metal, found at varying concentrations in nearly all uncontaminated aquatic and terrestrial ecosystems (see Table A4.5.1). However, as anthropogenic emissions of zinc far exceed those from natural sources, elevations above these natural, background concentrations are often found (see Table A4.5.2).

Environmental Matrix	Concentration	Reference
Seawater	<1 μg/l (open ocean) 0.3-70 μg/l (coastal and estuarine)	Bryan & Langston 1992. UNEP 1993
Freshwater	<50 μg/l	ATSDR 2000
Drinking water	0.02-1.2 mg/l	ATSDR 2000
Soil	50 mg/kg average (10-300 mg/kg)	Alloway 1990
Freshwater sediment	<100 mg/kg	ATSDR 2000, Salomons & Forstner 1984
Marine Sediment	<100 mg/kg	Bryan & Langston 1992, UNEP 1993

Table A4.5.2 Background concentrations of zinc found in water, sediments and soil

Site Description	Concentration	Reference
Restronguet Creek sediment, UK Receiving acidic drainage from past and present mining activities.	3000 mg/kg	Bryan & Langston 1992
Seawater, Restronguet Creek	20-20 460 μg/l	Bryan & Langston 1992
Mediterranean coastal sediments. Sites receiving large quantities of industrial and urban wastes	200-6 200 mg/kg	UNEP 1993
Concentrations in surface soils in the vicinity of mines and smelters.	11-50 000 mg/kg	Dudka et al. 1995, Matthews & Thornton 1982, Dudka & Adriano 1997
River water, from sites receiving urban and industrial waste, USA	0.01-2.4 mg/l	ATSDR 2000
Donana National Park, Spain. Contaminated river sediments close to mining sites	879-12 200 mg/kg	Pain et al. 1998

Table A4.5.2 Zinc concentrations associated with sites of anthropogenic contamination

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 2000). 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 2000, Bryan & Langston 1992, Alloway 1990). However re-solubilisation back into an aqueous, more bioavailable phase is possible under certain physical-chemical conditions, e.g. 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 2000). 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 2000).

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 2000).

Toxicity and Essentiality

Zinc is a nutritionally essential metal, having enzymatic, structural and regulatory roles in many biological systems (Goyer 1996, Aggett & Comerford 1995). Deficiency in humans can result in severe health consequences including growth retardation, anorexia, dermatitis, depression and neuropsychiatric symptoms (Aggett & Comerford 1995). At the other extreme, excessive dietary exposure, in both humans and animals, can cause gastrointestinal distress and diarrhoea, pancreatic damage and anaemia (ATSDR 2000, 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 2000). 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 2000). Humans taking supplements at higher than recommended doses (400-500 mg/day) suffered severe gastro-enteritis (Abernathy & Poirier 1997); and humans who drank water from galvanised pipes, over a prolonged period, suffered irritability, muscular stiffness and pain, loss of appetite and nausea (UNEP 1993).

With regard to industrial exposure, metal fume fever resulting from the inhalation of zinc oxide fumes presents the most significant effect. Attacks usually begin after 4-8 hours of exposure, and last between 24-48 hours. Symptoms include chills and fever, profuse sweating and weakness (ATSDR 2000, Goyer 1996).

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 μg/l, carbon fixation rates in natural phytoplankton populations were depressed. Others observed that the growth of cultured diatoms was inhibited at 20 μg/l (Bryan & Langston 1992). Effects on fertilisation and embryonic development in Baltic spring-spawning herring at low salinity were detected at only 5 μg/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 & 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 μg/l (UNEP 1993). Shell growth in the mussel *Mytillus edulis* was effected at a concentration of 200 μg/l. With oxygen uptake, feeding and filtration rates were reduced at concentrations ranging between 750-2000 μg/l. Harmful effects on mollusc larva were seen to occur at levels as low as 40 μg/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).

Legislation

Applicable standards set by the Ministry of Science, Technology and Environment of Thailand for zinc include 'Effluent standards for industrial and industrial estate sources' (No.3, BE 2539). These standards set limits for zinc in effluents of 5.0 mg/l (Homchean 1998).

References

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

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

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

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

ATSDR (2000) Toxicological profile for zinc on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service

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

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

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

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

Dudka, S., Piotrowska, M., Chlopecka, A. & Witek, T. (1995) Trace metal contamination of soils and crop plants by the mining and smelting industry in Upper Silesia, South Poland. Journal of Geochemical Exploration 52: 237-250

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

Homchean, K. (1998) Industrial Estate Authority of Thailand strategy for environmental compliance, Proceedings of the fifth international conference on environmental compliance and enforcement, Monterey, California, pp101-112

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

Matthews, H. & Thornton, I. (1982) Seasonal and species variation in the content of cadmium and associated metals in pasture plants at Shipham. Plant Soil 66: 181-193

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

Pain, D.J., Sanchez,A. & Meharg, A.A.(1998) The Donana ecological disaster: contamination of a world heritage estuarine marsh ecosystem with acidified pyrite mine waste. The Science of the Total Environment 222: 45-54

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

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

Verriopoulos, G. & Hardouvelis, D. (1988) Effects of sub-lethal concentrations of zinc on survival and fertility in four generations of Tisbe. Marine Pollution Bulletin 19: 162-166