Appendix 1 Analytical methodology

1. Organic analysis of wastewater, sediment and soil samples

1.1 Preparation of samples for organic screen analysis

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

Solid Samples

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

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

Aqueous Samples

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

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

1.2 Chromatographic Analysis

Organic compounds were identified qualitatively using Gas Chromatography Mass Spectrometry (GC-MS). Instrumentation was a Hewlett Packard (HP) 5890 Series II gas chromatograph, interfaced with a HP Chem-Station data system and linked to a HP 5972 Mass Selective Detector operated in scan mode. The identification of compounds was carried out by computer matching against a HP Wiley 275 library of 275,000 mass spectra combined with expert interpretation.

Results are reported as a list of those compounds reliably and tentatively identified. Match qualities of 90% or greater against HP Wiley 275 library or identification confirmed against standard compounds are assumed to give reliable identifications. Tentative identification refers to qualities between 51% and 90% against HP Wiley 275 library only. Analytes yielding match qualities of 50% or less are assumed to be unidentified.

2. Heavy Metal Analysis

2.1 Preparation of samples for heavy metals analysis

Solid samples

Samples were air dried until weighing readings became constant (approx. 5 days). They were then crushed using a pestle and mortar until homogenous and sieved through a 2-mm mesh. 0.5 g of sample was weighed into a glass 100 ml boiling tube and to this 10 ml of deionised water was added, followed by 7.5 ml of concentrated hydrochloric acid and 2.5 ml of concentrated nitric acid. Boiling tubes were then 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 five hours.

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

Aqueous samples

On arrival, 100ml of sample was transferred to a clean glass bottle and acidified with nitric acid (10% v/v). 50 ml was then transferred to a 100ml boiling tube, placed onto the Gerhardt Kjeldatherm digestion block, and refluxed at 130 °C for five 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.

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

Following preparation, samples were analysed by ICP-AES, using a Varian Liberty-100 Sequential Spectrometer. The following metals were quantified directly: manganese, chromium, zinc, copper, lead, nickel, cobalt and cadmium. Two multi-element instrument calibration standard were prepared at a concentration of 10 mg/l. One in an acid matrix of 5% v/v hydrochloric acid and 5% v/v nitric acid (for solid samples), the other in an acid matrix of 10% v/v nitric acid (for aqueous samples). Calibrations were validated using quality control standards (8 mg/l), prepared internally from different reagent stocks. Samples exceeding the calibration range were diluted accordingly, in duplicate, and re-analysed.

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

Appendix 2

List of compounds reliably identified and groups of compounds tentatively identified in the samples (Table 5)

Sample Number: 119006	Sample Number: 119007	Sample Number: 119008	Sample Number: 119009	Sample Number: 119010	Sample Number: 119011
Number of compounds isolated: 18	**Number of compounds isolated: 29**	**Number of compounds isolated: 20**	**Number of compounds isolated: 62**	**Number of compounds isolated: 111**	**Number of compounds isolated: 16**
Sample Type: Soil	**Sample Type: Soil**	**Sample Type: Soil**	**Sample Type: Industrial waste water**	**Sample Type: Sediment**	**Sample Type: Sediment**
COMPOUNDS RELIABLY IDENTIFIED: 1-Hexadecene 1-Octadecene Benzene, 1,4-dichloro- Docosane o,p'-DDD or Chloditan o,p'-DDE p,p'-DDD P,P'-DDE Tetracosane **GROUPS OF COMPOUNDS TENTATIVELY IDENTIFIED:** Linear alkanes and alkenes	**COMPOUNDS RELIABLY IDENTIFIED:** 1-Tetradecene Benzene, (1-ethyldecyl)- Benzene, (1-ethylnonyl)- Benzene, (1-ethylundecyl)- Benzene, (1-methyldecyl)- Benzene, (1-methyldodecyl)- Benzene, (1-methylundecyl)- Benzene, (1-pentylheptyl)- Benzene, (1-propylnonyl)- o,p'-DDD or Chloditan p,p'-DDD p,p'-DDE **GROUPS OF COMPOUNDS TENTATIVELY IDENTIFIED:** Alkylated benzene Cyclic aliphatic hydrocarbons DDT derviatives Linear alkanes and alkenes	**COMPOUNDS RELIABLY IDENTIFIED:** Benzophenone, 4,4'-dichloro- Cyclohexadecane o,p'-DDD or Chloditan o,p'-DDE o,p'-DDMU o,p'-DDT p,p'-DDD P,P'-DDE p,p'-DDMU p,p'-DDT **GROUPS OF COMPOUNDS TENTATIVELY IDENTIFIED:** Linear alkanes and alkenes	**COMPOUNDS RELIABLY IDENTIFIED:** 1,3-Butadiene, 1,1,2,3,4,4-hexachloro- alpha-HCH Azulene, 2,4,6-trimethyl- Benzene, 1,2,3,4-tetrachloro- Benzene, 1,2,4-trichloro- Benzene, 1,2-dichloro- Benzene, 1,3-dichloro- Benzene, 1,4-dichloro- Cyclopentene, trichloro- Docosane Docosane, 11-decyl- Eicosane gamma-HCH Heptacosane Hexadecane Naphthalene Naphthalene, 1,3-dimethyl- Naphthalene, 1-methyl- Naphthalene, 2-methyl- o,p'-DDD or Chloditan o,p'-DDE o,p'-DDT p,p'-DDD p,p'-DDE p,p'-DDMU p,p'-DDT Pentacosane **GROUPS OF COMPOUNDS TENTATIVELY IDENTIFIED:** Alkanes and alkenes Biphenyl Chlorinataed ethane derivative Chlorinated toluenes Flourene Mirex Naphthalene Organonitrogen compounds	**COMPOUNDS RELIABLY IDENTIFIED:** 1,3-Butadiene, 1,1,2,3,4,4-hexachloro- alpha-HCH Azulene, 2,4,6-trimethyl- Benzaldehyde Benzene , 1,2,4,5-tetrachloro- Benzene 2,4,-dichloro-1-(2-chloroethenyl)- Benzene, 1,1'-sulfonylbis[4-chloro- Benzene, 1,1'-thiobis[4-chloro- Benzene, 1,2,3,4-tetrachloro- Benzene, 1,2,4-trichloro- Benzene, 1,2-dichloro- Benzene, 1,3,5-trichloro- Benzene, 1,3-dichloro- Benzene, 1,3-dichloro-2-ethenyl- Benzene, 1,4-dichloro- Benzene, 1,4-dichloro-2-(2-chloroethenyl)- Benzene, 1,4-dichloro-2-ethenyl- Benzene, 1,4-dichloro-2-ethyl- Benzene, pentachloro- Benzophenone, 2,4,4'-trichloro- Benzophenone, 2,4'-dichloro- Benzophenone, 3,4' -dichloro- Benzophenone, 3,4,4-trichloror- Benzophenone, 4,4'-dichloro- Benzothiazole, 2-(methylthio)- beta-HCH Cyclohex-1-ene, .gamma.-2,3,4,5,6-pentachloro- DDM DEHP delta-HCH Eicosane Endosulfan I Endosulfan II Endosulfane ether gamma-HCH Heptadecane Hexadecane Naphtalene, 2-methyl- Naphthalene Naphthalene, 1,4,6-trimethyl- Naphthalene, 1,5-dimethyl- Naphthalene, 1-methyl- Naphthalene, 2,7-dimethyl- Nonadecane o,p'-DDD or Chloditan o,p'-DDE o,p'-DDMU o,p'-DDT p,p'-DDD p,p'-DDE p,p'-DDMU p,p'-DDT PCB 3 Pentadecane, 2,6,10,14-tetramethyl- Sulfur, mol. (S8) Tridecane **GROUPS OF COMPOUNDS TENTATIVELY IDENTIFIED:** Biphenyl Chlorinated methanoindene Chlorinated benzeneacetic acid Chlorinated pyrrole Chlorinated quinoxaline Chlorinated 11H-dibenzo[b,e][1,4]dioxepin Cyclic aliphatic hydrocarbons Linear alkanes and alkenes Mirex PCBs Phenol Terphenyl	**COMPOUNDS RELIABLY IDENTIFIED:** 3-Hexadecene, (Z)- 5-Octadecene, (E)- Dodecane Phenol, 2,4-bis (1,1-dimethylethyl)- Phenol, 2,6-bis(1,1-dimethylethyl)-4-methyl- **GROUPS OF COMPOUNDS TENTATIVELY IDENTIFIED:** Cyclic aliphatic hydrocarbons Linear alkanes and alkenes

Appendix 3

Toxicological outlines for key organic compounds

DDT and metabolites

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

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

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

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

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

DDT is moderately to slightly toxic to studied mammalian species via the oral route (RSC 1991; Meister 1992; ASTDR 1997). The primary target of DDT is the nervous system and

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

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

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

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

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

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

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

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

DDT is controlled under numerous international legal instruments - notably the PIC Convention, the LRTAP POPs protocol, the Barcelona Convention, the Helsinki Convention, the IJC and the draft UNEP POPs Convention. It is also, of course, included under wider groupings of organochlorine pesticides or organohalogens under the various waste trade Conventions and the OSPAR Convention. Agricultural use of DDT is almost totally banned, but its use is frequently retained for public health purposes. According to FAO/UNEP (1991) DDT is banned in Chile, Cuba, the EC, Liechtenstein, Mexico, Panama, Republic of Korea, Singapore, Sri Lanka, Sweden, Togo and the USSR and has been withdrawn from sale in Canada and Poland. It is severely restricted in Argentina, Belize, China, Colombia, Dominica, Ecuador, Japan, Kenya, Mauritius, the USA, Venezuela, and Yugoslavia. In many of these countries, use is only permitted for control of critical disease vectors and would be carried out only at the behest of the government health department. In addition, DDT is banned (except for drug use) in the countries which are party to the 1992 Helsinki Convention. Unfortunately, DDT is still diverted illegally from government health programmes to agricultural use on a regular basis. This is known or suspected to have happened in Bangladesh, Belize, Ecuador, India, Kenya, Madagascar, Mexico and Tanzania (WWF 1998).

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Hexachlorocyclohexane

Mixture of hexachlorocyclohexanes is produced by the photochemical reaction between chlorine and benzene (Safe 1993). Technical grade hexachlorocyclohexane (HCH) comprised of different isomeric forms. The approximate isomer content is alpha-HCH (60-70%), beta-HCH (7-10%), gamma-HCH (14-15%), delta-HCH (7%), and epsilon-HCH (1-2%). Lindane is the gamma isomer of hexachlorocyclohexane and it is commercially produced by purification of the technical HCH (Safe 1993). This compound has been produced worldwide for use as an insecticide to control grasshoppers, cotton and rice pests, wireworms, and other soil pests. Lindane has been used for protection of seeds, for treatment of poultry and livestock, and for control of household insects. It is also still used as a scabicide and pediculocide, usually as lotions, creams, and shampoos.

Alpha-, beta-, and gamma-HCH are the most important isomers in terms of environmental impact. The relatively high stability and lipophilicity of HCH and its global use pattern has resulted in significant environmental contamination by this chlorinated hydrocarbon. Once introduced into environment HCH may persist for many years (Martijn & Schreuder 1993). The beta-isomer is more persistent than others (ATSDR 1997).

Human intake of HCH compounds is largely through food consumption (Toppari *et al.* 1995). Alpha-, beta- and gamma-HCH have been recorded in human breast-milk with the beta-isomer being the most ubiquitous (Waliszewski *et al.* 1996; Safe 1993). The generally less widespread nature of the alpha- and gamma-isomers in comparison to beta-HCH is due to the more rapid clearance of these isomers from the body. Like many persistent organochlorines, HCH levels in the body have been found to increase with age (ASTDR 1997).

Hexachlorocyclohexane isomers have been detected in air, surface and ground water, soil and sediments (El-Gendy *et al.* 1991; Safe 1993; Xu 1994; Tan & Vijayaletchumy 1994; Skark & Zullei-Seibert 1995; Ramesh *et al.* 1991), plants (Xu 1994), birds, fish and mammals (Smith 1991; Xu 1994; Abd-Allah 1994; Norstrom & Muir 1994). In humans lindane mostly concentrates in adipose tissue (Safe 1993). It has been reported that lindane and other organochlorine compounds can be transferred through the pathway soil→earthworm→bird/mammal (Hernandez, *et al.* 1992; Romijn *et al.* 1994) thereby causing secondary poisoning.

Lindane, the gamma-isomer of hexachlorocyclohexane, is toxic to animals, humans, and aquatic species. Acute animal poisoning by lindane causes increased respiratory rate, restlessness accompanied by increased frequency of urination, intermittent muscular

spasms of the whole body, salivation, grinding of teeth and consequent bleeding from the mouth, backward movement with loss of balance and somersaulting, retraction of the head, convulsions, gasping and biting, and collapse and death usually within a day (Smith 1991).

Chronic health effects can occur at some time after exposure to lindane and can last for months or years. Lindane has been shown to cause liver, lung, endocrine gland and certain other types of cancer in animals (Smith 1991). Repeated overexposure may damage the liver. Chronic toxic effects may also include shortened lifespan, reproductive problems, lower fertility, and changes in appearance or behaviour. The differential actions of hexachlorocyclohexane isomers may produce variable effects on different regions of the nervous systems and in different species of animals (Nagata *et al.* 1996).

Hexachlorocyclohexane may be introduced to the environment from industrial discharges, insecticide applications or spills, and may can cause significant damage. Acute toxic effects may include the death of animals, birds, or fish, and death or low growth rate in plants (Bunton 1996, Smith 1991). The insecticide load in surface waters does not ordinarily reach concentrations acutely toxic to aquatic fauna. However, lindane has high chronic toxicity to aquatic life. The effects of the low insecticide concentrations often appear only after relatively long exposure times. Chronic exposure to insecticides, such as lindane, (Schulz *et al.* 1995) can be hazardous to freshwater macroinvertebrates even at unexpectedly low concentrations. The low-concentration effects may depend on both species and substance and therefore cannot be predicted from toxicity data at higher concentrations.

Hexachlorocyclohexane, as a toxic, persistent and bioaccumulative chemical, is a subject to the European Community legislation. The limit values and quality objectives for discharges of hexachlorocyclohexane are set by the Council Directive 84/491/EEC (EEC 1984) as amended. The uses of hexachlorocyclohexane (including lindane) were severely restricted under the Persistent Organic Pollutants (POPs) Protocol, which was adopted in 1998 and has 36 contracting parties encompassing not only Europe but also Canada and the United States of America (UNECE 1998). The POPs Protocol is part of the 1979 Convention on Long-Range Transboudary Air Pollution (LRTAP), which is under the auspices of the United Nations Economic Council for Europe. Lindane is also included in the Annex III of the 1998 Rotterdam Convention on the Prior Informed Consent procedure (PIC procedure) among 27 other chemicals (FAO/UNEP 1998). Under the PIC procedure countries should not export any chemical to any other country without first receiving explicit permission. In order to avoid unfair trade barriers arising through the implementation of the Convention, any country that has denied import of any chemical must also stop producing it domestically and may not import it from any country that is not a Party to the Convention.

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Endosulfan

Endosulfan is a cyclodiene insecticide. The main intermediates that are used in the manufacture of endosulfan are hexachlorocyclopentadiene, 1,4-dihydroxy-2-butene and thionyl chloride (Safe 1993, Weil & Sandeer 1997). Technical grade endosulfan is a brownish solid which consists of about four parts of α-endosulfan and one part of β-endosulfan. These two stereoisomers are also known as endosulfan 1 and endosulfan 2 (Metcalf 1995). The two endosulfan isomers have different physical properties and α-endosulfan is the more toxic (Smith 1991).

Endosulfan was first produced by Farbwerke Hoechst AG in the 1950s and was also manufactured in the USA by FMC Corporation. In 1984, worldwide production was estimated at 10,000 metric tonnes annually. It is a broad-spectrum insecticide used on food, non-food and forage crops as well as for wood preservation and in home gardening

products. It is particularly effective against the Colorado potato beetle and is not regarded as highly toxic to bees (ATSDR 1997).

High levels of contamination by endosulfan is limited to areas where it is manufactured, formulated, applied or disposed of. However, long distance transport can occur and endosulfan has been recorded at measurable concentrations in Arctic snow.

The two major metabolites of endosulfan are endosulfan sulphate and endosulfan diol. Endosulfan sulphate, the product of biological degradation and the major metabolite in soil, is formed where bacterial populations are high. It is as toxic as the parent compound and apparently more potent than the β-isomer of endosulfan. It has been extensively researched because of links to fish kills (Smith 1991). The non-toxic diol is probably the final product of endosulfan metabolism in mosquitofish, in which it has been found in greater abundance than α-endosulfan and endosulfan sulphate residues (Nowak & Sunderam 1991). In experiments in mice, rapid excretion of endosulfan followed its conversion to water-soluble compounds and only 20% of the metabolites were found to be lipophilic (Smith 1991).

Pseudomonad microbes have been reported to isomerize and biodegrade endosulfan to endosulfan alcohol and endosulfan ether. Technical-grade endosulfan may also contain up to 2% endosulfan alcohol and 1% endosulfan ether (ATSDR 1997).

Endosulfan residues found in food samples were recorded by a number of U.S. states in 1988 and 1989. In eight cases contamination was considered significant (Minyard & Roberts 1991). In Portugal, levels exceeding the EEC MRL of 1 mg/kg have been found in broccoli (Magalhães *et al.* 1989). When administered in large doses endosulfan appears in milk, largely as the sulphate (Smith 1991). This metabolite has been reported in cows' milk samples from various locations in Switzerland (Rappe *et al.* 1987).

Oral LD_{50} values of 43 mg/kg for male rats and 18 mg/kg for female rats have been determined in laboratory tests. Observed sub-lethal effects included liver enlargement, seizures, reduced growth and survival, raised blood sugar, more aggressive behaviour, depressed immune responses and interstitial changes in the kidney of male rats (Smith 1991). Oral doses of or exceeding 5mg/kg/day during gestation increased both the mortality of rat dams and the incidence of resorption and skeletal abnormality in their foetuses. Doses of 10mg/kg/day caused degeneration and weight loss of testes (Smith 1991). Endosulfan has also been found to be oestrogenic (Zou & Fingerman 1997).

Endosulfan's persistence and high toxicity to fish are of special concern and numerous studies have investigated the risks and adverse effects of run-off water on non-target organisms. Technical grade endosulfan 96h LD_{50} values of 2.0µg/l at 30°C and 4.6µg/l at 35°C have been determined for mosquitofish (Nowak & Sunderam, 1991). The reported LC_{50} for rainbow trout is 1.4µg/l. The No Observed Effect Concentration (NOEC), based on growth and reproduction, is 49µg/l for *Daphnia cephalata* (Barry *et al.* 1996). At concentrations greater than 50ng/l, pathological changes in mitochondria and peroxisomes have been observed and noted as one mechanism of endosulfan intoxification, consistent with its acting as a mixed-type inducer of MFO systems in fish hepatocytes (Arnold *et al.* 1996). *Channa punctatus* suffered ovarian steroidogenesis inhibition at sublethal concentrations (Inbaraj & Haider 1988). The liver, kidney and brain supported highest

levels of accumulated endosulfan in catfish, the distribution probably being influenced by lipid content (Nowak 1991).

Endosulfan may be lethal to humans and animals by inhalation, oral or dermal exposure. Acute effects observed in laboratory animals or humans are typical of those following exposure to other cyclodiene compounds. The main target is the central nervous system but the liver, kidney, gastrointestinal, haematopoietic and dermal systems and developing foetus have also been affected in exposed experimental animals (Nowak 1991).

Reported toxic effects in exposed livestock include ataxia, progressing to complete inability to stand, in lambs grazing a sprayed field, blindness in sheep (with full recovery after a month) (Smith 1991) and death and acute illness in cattle mistakenly sprayed with endosulfan for lice control. Neuromuscular symptoms associated with organochlorine toxicosis were observed in the cattle and included abnormal posturing, spastic gait, apprehension, hypersensitivity, belligerence, loss of coordination and non-stop chewing movements. These signs of intense nervous system stimulation may be caused by impairment of glutamine synthesis, resulting in elevated ammonia concentrations in the brain and enhanced neurotransmitter release at cholinergic synapses (Nowak 1991).

For the general population, the main source of exposure to endosulfan is via ingestion of food which contains endosulfan as a result of direct pesticide application or bio-concentration. A tragic case of extensive food contamination occurred in Sudan in 1991. Maize, which had been intentionally treated with endosulfan for use as poisonous bait for birds, was instead used in baking flour, causing the deaths of thirty-one people (Dinham 1993).

There are some records of accidental and intentional poisoning. A 70 year old woman died about three hours after taking only ‘drops’ of an endosulfan formulation. An unsuccesful suicide resulted in an acute cardiac and convulsion stage, a sub-acute pulmonary and convulsive stage and finally a slow recovery. After one month’s occupational exposure, a worker reported ‘’flu-like’ symptoms and headache, followed tbe next day by collapse, convulsions and temporary unconsciousness and amnesia. Subsequent effects were consistent with epilepsy (Smith 1991).

The US EPA recommends that, to prevent human exposure to harmful concentrations in drinking water, fish and seafood, levels of endosulfan in lakes, rivers, and streams should not exceed 74µg/l (74 ppb). The Food and Drug Administration (FDA) permits a maximum of 24 ppm endosulfan on dried tea and EPA limits are set from 0.1-2.0 ppm endosulfan on other raw agricultural products. The Occupational Safety and Health Administration (OSHA) has set the permissible exposure limit (PEL) at 0.1 mg/m^3 workroom air for workers on 8-hour shifts over a 40-hour working week (ATSDR 1997).

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Chlorinated Benzenes and Toluenes

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 monchlorobenzene,
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 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, 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, fungicideand 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 1,2,3,4,5,6-hexachlorocyclohexane. 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-tatrachlorobenzene being the most toxic. Not enough information was available to classify tetrachlorobenzenes as to carcinogenicity.

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

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

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

Pentachlorobenzene

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

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

Hexachlorobenzene

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

HCB is toxic to aquatic life, land plants, land animals, and humans. It is listed by the IARC as a Group 2B carcinogen, i.e. possible carcinogen to humans and also appears to be a tumour promoter. Hexachlorobenzene may damage the developing foetus, liver, immune system, thyroid and kidneys and CNS. The liver and nervous system are the most sensitive to its effects. Porphyria is a common symptom of HCB toxicity. High or repeated exposure may damage the nervous system, and can cause irritability, difficulty with walking and co-ordination, muscle weakness, tremor and/or a feeling of pins and needles on the skin. Repeated exposure, especially when skin effects occur, can lead to permanent skin changes, such as changes in pigmentation, tight, thickened skin, easy wrinkling, skin scarring, fragile skin, and increased hair growth, especially on the face and forearms (ATSDR 1997, Newhook & Meek 1994, van Birgelen 1998). Recent research (van Birgelen 1988) suggests that HCB has dioxin-like toxicity and that, based on a preliminary toxic equivalence factor (TEF) of 0.0001, HCB could contribute significantly to the dioxin-type toxicity of human milk based on PCB/PCDD/PCDF toxicity equivalents. In many countries, this could mean an increase of 10%-60%, but in countries with high HCB

exposure levels, the effects could be even greater. In Spain and the Czech Republic, inclusion of HCB in total breastmilk TEQ estimates could lead to totals 6 times higher than based only on PCBs and PCDFs. Slovakia and India also have very high HCB levels; other countries (eg Austria) high levels in previous decades. It has been suggested that more epidemiological studies should be undertaken, especially in the most highly contaminated countries.

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

Once introduced into environment, HCB strongly absorb to soil materials and almost no desorption take place (Bahnick & Doucette 1988). It is bioaccumulative and biomagnifies. It can be measured in ambient air, drinking water, soil, food and breast milk (Newhook 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 a convention currently being drawn up (UNEP 1995, 1997). Furthermore, HCB is included on Annex I of the Draft UNECE POPs Protocol under the Convention on Long-Range Transboundary Air Pollution (LRTAP)(UNECE 1998).
Within the EC, discharges of HCB are controlled as stipulated by 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 IJ has called for all uses to be eliminated. See the Chapter on international legal instruments for further details.

Chlorinated Toluenes

The chlorinated toluenes (chloromethylbenzenes) occur in five chlorination stages of which four have several isomers. Some are produced by reacting liquid toluene with gaseous chlorine and the monochlorotoluenes are produced without substantial by-production of the dichlorotoluenes. Both batch and continuous methods of production are used. Since only a few of the isomeric chlorotoluenes can be produced economically by direct chlorination, others are produced by indirect syntheses.

They are principally manufactured in the United States, Germany and Japan with much of the production being consumed in captive applications. In the USA chlorinated toluenes were first manufactured by Hooker Electrochemical Co. and Heyden Chemical Corp. Heyden ceased manufacturing in the 1970s and Hooker were bought by Occidental, which is now the only producer in the USA (Lin & Krishnamurti (1993). World production is estimated at several tens of kilotons for polychlorinated toluenes, while non captive end use sales account for between 0.1 and 1kt for o- and p- chlorotoluenes (Lin & Krishnamurti 1993).

Chlorination of both the aromatic ring and the side chain can take place. The commercially important ring substituted compounds are as follows:

2-Chlorotoluene (o-chlorotoluene)
4-Chlorotoluene (p-chlorotoluene)
2,4-Dichlorotoluene
2,6-Dichlorotoluene
3,4-Dichlorotoluene
2,3,6-Trichlorotoluene

Monochlorotoluenes

2-Chlorotoluene is usually thr predominat isomer generated by chlorination of toluene (about 70% under normal condition (Lin & Krishnamurti 1993. It is a raw material in the production of 2-chlorobenzyl chloride, 2-chlorobenzaldehyde and 2-chlorobenzoic acid. These are used as precursors in the production of dyes, pharmaceuticals, optical brighteners and fungicides. It is also used in the production of dichlorotoluenes and other chlorotoluenes for specialty applications.

3-chlorotoluene has similar solvent properties to 2-chlorotoluene, but little further information is available. It occurs at only a rate of about 1% on the chlorination of toluene and must be synthesised indirectly. During purification of technical mixes, it partitions into the p-chloro-toluene fraction (Lin & Krishnamurti 1993).

4-Chlorotoluene is used mainly to produce 4-chlorobenzotrichloride which as a precursor to 4-chlorobenzotrifluoride is important in the manufacture of dinitroaniline and diphenylether herbicides such as Trifluralin. Other side chain chlorinated derivatives are insecticides and dyes.

Monochlorotoluenes can be used to synthesis benzyl, benzal and benzotrichloride (see

below); chlorinated benzaldehydes and chlorinated cresols.

Dichlorotoluenes

Chlorination of o-chlorotoluene yields all four possible dichlorotoluenes, with 2,5-dichlorotoluene making up some 60% of the reaction mix (Lin & Krishnamurti 1993). 2,4-Dichlorotoluene is used via its chlorinated side chain derivatives in the production of fungicides, dyes and pharmaceuticals, preservatives and peroxides.

The remainder of the commercially important ring substituted chlorotoluenes are used in small amounts to generate precursors for a variety of dyes and herbicides. Derivatives which are also chlorinated on the ring have little industrial significance.

Tri-, tetra- and penta-chlorinated toluenes.

These have relatively few industrial applications. 2,3,6-trichlorotoluene is used as a herbicide manufacturing intermediate (Lin & Krishnamurti 1993).

alpha-chlorinated toluenes

The important side chain chlorinated compounds, each of which has a majority end use, are:

Benzyl chloride ([chloromethyl]benzene)
Benzal chloride ([dichloromethyl]benzene)
Benzotrichloride ([trichloromethyl]benzene)

The IARC has classified benzyl chloride, benzal chloride, benzotrichloride and benzoyl chloride (C_6H_6COCl) as group 2A carcinogens on the basis of combined exposures.

Benzyl chloride

In 1989, the developed world had capacity to manufacture over 140,000 tonnes per annum of benzyl chloride. Monsanto had the largest capacity, with plants in US and Belgium. Other manufacturers were Bayer (West Germany), Tessenderlo Chemical (Belgium) and Akzo (USA). Velsicol ceased production in 1986 after poor market forecasts and other facilities were not utilising full capacity. Only a total 92,700 tonnes were manufactured in 1989; in the US only 54% of a possible 49,000 tonnes were produced (Lin & Bieron 1993). Benzyl chloride is largely utilised in the manufacture of benzyl butyl phthalate, a plasticiser for vinyl resins accounting for over two thirds of production. Other uses include the manufacture of benzyl alcohol, and quaternary ammonium compounds. Triphenylmethane dyes are also manufactured from this compound as are a number of flavour and perfume products, dyes, synthetic tannins, pharmaceuticals and resins (Lin & Bieron 1993, Saxena & Abdel-Rahman 1989). It has been identified as a constituent of oil refinery effluents (Saxena & Abdel-Rahman 1989).

Carcinogenicity of the chlorinated toluenes is discussed above. It is mutagenic and may damage the liver and heart and depress the central nervous system; developmental and foetal toxicity have also been reported. It is extremely irritating to the eyes, skin and respiratory system, causing pulmonary oedema (Lin & Bieron 1993). Because of its lacrymatory properties, it has been used as a chemical warfare agent (Saxena & Abdel-Rahman 1989).

Some 2,400 tonnes of benzyl chloride were amongst the waste dumped by Hooker Chemical in the Love Canal in New York State (Saxena & Abdel-Rahman 1989). Hooker Chemical bough the Canal (built by William T. Love in the 1890s as part of an unsuccessful hydroelectric project) in the 1940s. Between then and 1953, they filled it with an estimated 20,000 tonnes of drummed chemical wastes and in 1953 Occidental (who had bought Hooker in 1968) sold the site to the local school board for $1. A school and houses were built but residents suffered health problems including miscarriages and birth defects. In 1978 pregnant women and children under two years of age were encouraged to leave the area and in 1980 the government offered to purchase the homes of another 710 families. After a cleanup costing tens of millions of dollars, the USEPA declared the site clean in 1990 and homes there were again offered for sale. However, environmental groups still believe that health hazards remain (Miller 1992).

Benzyl chloride is an irritant, dysregulator of liver bichemistry (Saxena & Abdel-Rahman 1989) and is mutagenic. After oral dosing, highest concentrations are found in the digestive tract, followed by the liver. Most was excreted in urine within three days (Saxena & Abdel-Rahman 1989).

Benzal chloride

Benzal chloride can be made by chlorination of toluene and is used mostly for the manufacture of benzaldehyde. It has a strong pungent odour, and is highly irritating, with lacrymatory properties (Lin & Bieron 1993). As discussed above, it is probably carcinogenic to humans in concert with other chlorinated aromatics.

Benzotrichloride

Further chlorination of benzal chloride produces benzotrichloride. It is important in the manufacture of a variety of compounds but is quantitatively mostly converted to benzoyl chloride. In 1988, of an estimated 68kt production capacity in the western world, approximately 31500 tonnes was realised. Bayer (FRG) has capacity of 14,000 tpa; Occidental, NY (USA)(20,000 tonnes) and Velsicol (USA)(11,00 tonnes)(Lin & Bieron 1993).

As with the other alpha-chlorinated toluenes, benzotrichloride is extremely irritating and lacrymatory (Lin & Bieron 1993). Its carcinogenic potential is discussed above.

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Hexachloro-1,3-butadiene

Hexachloro-1,3-butadiene (HCBD) is a colourless liquid with a turpentine-like odour. This compound is not found naturally in the environment. HCBD either commercially manufactured or is known to be a by-product of the manufacture of chlorinated hydrocarbons such as tetrachloroethene, trichloroethene, abd carbon tetrachloride (ATSDR 1997; US EPA, 1986, Johnston *et al.* 1994; Botta *et al.* 1996). It is always present in relatively small quantities (up to 5% and more of hexachlorobutadiene in the chlorolysis process of 1,2-dichloroethane for the production of carbon tetrachloride and tetrachloroethene), but, because of the huge production of volatile chlorinated solvents, the amounts of hexachlorobutadiene from the different processes are relevant (Botta *et al.* 1996). It is also reported as a contaminant in technical formulations of pentachlorophenol, used widely as a wood preservative (Goodrichmahoney *et al.* 1993). Hexachlorobutadiene was first prepared in 1877 by the chlorination of hexyl oxide (ATSDR 1997).

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

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

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

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

The International Agency for Research on Cancer concluded that there was limited evidence of HCBD carcinogenicity in rats and classified HCBD as compound not

classifiable as to human carcinogenicity (Group 3) (IARC 1999). The US Environmental Protection Agency considers HCBD to be a possible human carcinogen and has classified it as a Group C carcinogen (IRIS 1993).

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Alkylbenzenes

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

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

The acute toxicity of inhaled alkylbenzenes is best described as central nervous system (CNS) depression (Andrews & Snyder, 1986). Acute toxicity does not vary very much within the group. In animal models, relatively similar concentrations of inhaled alkylbenzene vapours were found to be lethal. Impaired reaction times and impaired speech are the two most commonly noted CNS effects (Klaassen *et al.* 1996). All alkylbenzenes mention above are irritating to the eyes and mucous membranes, can cause irritation and burning of the skin, and all are narcotics at high concentrations. Benzene itself is a known carcinogen. Chronic exposure can lead to bone marrow depression, which in a few cases, can progress to leukemia (Budavari *et al.* 1989).

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Phthalate esters

Phthalate esters are more often referred to as the phthalates. They are used in every major product category (Kemi 1994). 90% of all plasticizers is used in the production of soft PVC (Cadogan *et al.* 1993) but they are also used in inks and dyes (Jobling *et al.* 1995), which may be of particular relevance to the industrial estates under study. Bis(2-ethylhexyl) ester of 1,2-benzenedicarboxylic acid, also known as bis(2-ethylhexyl) phthalate (DEHP), was one of the phthalates produced in the greatest quantities ten to fifteen years ago (Menzert & Nelson 1986).

Phthalates are relatively persistent and are among 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 can cause liver cancer in laboratory animals and has been classified as possibly carcinogenic to humans by the IARC and as a probable human carcinogen by the US Environmental Protection Agency. The US Department of Health and Human Services (DHHS) has determined that DEHP may reasonably be anticipated to be a carcinogen (DHHS 1998). In its scientific opinion expressed in November 1998, the European Commission's Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE 1998) noted that the most sensitive effect of DEHP may be damage to the development of the testes, based on tests involving exposure of rats to relatively low concentrations both in the womb and for the first three weeks after birth. The Committee also judged that such testicular toxicity may have greater relevance for humans than carcinogenic effects.

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

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

Because of its recognised hazardous properties, DEHP is included on the OSPAR List of Chemicals for Priority Action (Annex 2 to the OSPAR Strategy with Regard to Hazardous Substances, OSPAR 1998).

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Chlorinated biphenyls

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

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

PCBs were always sold as technical mixes rather than individual chemicals. de Voogt and Brinkman (1989) list some 46 trade names used for PCBs and PCB containing products. The Aroclor range manufactured by Monsanto was probably the most widely used. The most important PCB applications in tonnage terms were transformer oils and capacitors (de Voogt & Brinkman 1989). In transformer oils, the PCBs were mixed with chlorobenzenes (mainly trichlorobenzenes and tetrachlorobenzenes) as solvents (Swami *et al.* 1992, de Voogt and Brinkman 1989). PCBs may also be generated as a by-product particularly in the chlorine chemical industy. Such industries include the PVC industry, where waste EDC tars are contaminated, together with aqueous effluents, by dichlorobiphenyls.

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

Levels of PCBs in biological material may be several orders of magnitude higher than ambient. PCBs are bioconcentrated to a factor of 6000 for fish and 47000 for invertebrates (Jones *et al.* 1988). Train (1979) reports bioconcentration factors of between 2500 and 100,000.

PCBs can be absorbed through the skin as well as through ingestion and inhalation. For the general population today, food is the primary source, though dermal exposure may be

dominant amongst those directly handling PCBs or PCB-contaminated materials (Lees *et al.* 1987).

Safe (1993) lists the following symptoms of PCB toxicity:

enzyme induction
decreased vitamin A levels
lymphoid involution
thymic and splenic atrophy
immunosuppression
chloracne
alopecia
oedema
hyperkeratosis
blepharitis
hyperplasia of the epithelial lining of the extrahepatic bile duct
the gall bladder and urinary tract
hepatomegaly and liver damage including necrosis
haemorrhage
altered porphyrin metabolism
tumour promotion
altered levels of steroid and thyroid hormones
reproductive toxicity including menstrual irregularities, reduced conception, early abortion, excessive menstrual and postconceptual haemorrhage, anovulation, testicular atrophy, decreased spermatogenesis, teratogenesis and developmental toxicity.

In addition, low levels of PCBs caused behavioural impairment in monkeys (Rice 1999). Aroclor 1254 compromised the immune response of earthworms (Roch & Cooper 1991). Aroclors 1221, 1254 and 1268 all reduced in vitro fertilisation rates in mice. PCB 1254 was the most potent mix (Kholkute *et al.* 1994).

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

Invertbrates display a differential response to individual PCB congeners. In the aquatic snail, *Lymnaea stagnalis*, 2,2'-dichlorobiphenyl was substantially more toxic than the other congener under test, 4,4-dichlorobiphenyl (Wilbrink *et al.* 1987), being rapidly fatal to over 60% of the test animals (Wilbrink *et al.*, 1990) and inhibiting production of egg masses more rapidly.

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

system, the corticosteroid system and several other endocrine pathways. In addition, effects on the thyroid system on wild populations of fish-eating birds and captive seals have been correlated with PCB exposure (Brouwer *et al.* 1999).

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

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

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

The control of PCBs is addressed under most of the international legal instruments relating to organochlorines, *inter alia*, the Barcelona, Helsinki, Basel, Bamako, Rotterdam OSPAR and LRTAP Conventions and the International Joint Commission on the Great Lakes. In addition, PCBs are targeted for global production ban under the UNEP POPS Convention currently being drafted. Within the EC, applications for the PCBs were first restricted by Directive 76/769/EEC, which deals with the marketing and use of dangerous substances and preparations (EC 1976). It, and its amendment (EC 1991), restricted the applications of PCBs and their replacements, the PCTs.

EC regulations on disposal of PCBs, as set out in a 1996 Directive, dictate that the phase-out PCBs should be completed by 2010. Further, national enabling legislation should have been emplaced by March 1998. Several countries have missed this deadline and in mid 1999, the EC initiated action through the European Court of Justice against Germany, Greece, Spain, Portugal and UK for failing to implement the directive (ENDS 1999).
Within the European Community, PCBs are regarded as "blacklist substances" (Gardiner & Mance 1984) although no regulatory directive has yet been proposed. PCBs are also included in the proposed UK "red list" (Jones *et al*. 1988). The US Toxics Substances Control Act (TOSCA) designates wastes containing greater than 50ppm PCBs are designated as hazardous (Rogan 1995).

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Butylated hydroxytoluene (BHT)

2,6-bis(1,1-dimethylethyl)-4-methylphenol, also known as butylated hydroxytoluene (BHT), is frequently employed as an antioxidant in food products, rubbers, soaps and in the production of plastics and other petrochemical products (Jobling *et al.* 1995). It is also manufactured as an antiskinning agent in paints, varnishes and other surface finishes, and as an antioxidant. The use of BHT as an antioxidant in food has been associated with certain allergic reactions (Dean 1986). There is some evidence that BHT can act as a promoter of liver cancer, in combination with carcinogenic substances, through induction of abnormal liver metabolism (Williams *et al.* 1986). Additionally, BHT is one of the main degradation products of the herbicide Terbutol (2,6-di-tert-butyl-4-methylphenyl N-methylcarbamate) (Suzuki *et al.* 1995 & 1996).

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Appendix 4

Toxicological outlines for heavy metals

Zinc (Zn)

1. Natural Occurrence

Zinc is a relatively common metal, being 23rd in order of chemical abundance. It is found in the Earth's crust at an average concentration of 80 mg/kg, although some clay sediments and shales may contain higher concentrations (Alloway 1990, Salomons and Forstner 1984). It is not found naturally in it's pure form (as a lustrous, blue-white metal) but as a mineral (most commonly sphalerite, zinc sulphide), often associated with the ores of other metals (e.g. copper, lead and cadmium) (Kroschwitz and Howe-Grant 1995).

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

2. Production, Use and Anthropogenic Sources

Once the zinc ore has been mined, broken and crushed, the Zinc minerals, on treatment with water and chemicals, can be concentrated efficiently. Surplus chemicals, washings and waste rock from these processes form the "tailings", and are separated and discharged. The concentrated ore is then heated in a furnace in the presence of air to produce Zinc oxide. This is then combined with coke or coal, and retorted to approximately 1,100°C to produce metallic Zinc. Alternatively, the roasted Zinc oxide can be leached with sulphuric acid, and electrolysed to produce Zinc of >99.9% purity, an increasingly popular practice (USPHS 1997, Kroschwitz and Howe-Grant 1995). Zinc can also be recovered from secondary sources i.e. "old" scrap, such as die castings and engraver's plates, and "new" scrap, such as drosses, skimmings, flue dust, and clippings (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 (USPHS 1997, Annema and Ros 1994, UNEP 1993, Budavari *et al.* 1989).

Estimates of anthropogenic emissions of Zinc are given in Tables 1-3 (Nriagu 1990, Nriagu and Pacyna 1988).

Source	Emission (thousand tonnes / year)
Non-ferrous metal production (Zn, Cu, Pb, Cd, Ni)	72.0
Steel and iron manufacturing	33.4
Energy production (coal and oil combustion)	16.8
Waste incineration (municipal refuse and sewage sludge)	5.90
Commercial uses (e.g. phosphate fertilisers, cement, paper, chemicals)	3.25
Mining	0.46
TOTAL	131.81

Table 1 World-wide atmospheric emissions of zinc from anthropogenic sources.

Source	Emission (thousand tonnes / year)
Manufacturing processes (metal, chemicals, paper, petroleum products)	85
Domestic wastewaters	48
Atmospheric fallout	40
Base metal mining and smelting	29
Electric power plants	18
Sewage discharges	17
TOTAL	237

Table 2 World-wide inputs of zinc into aquatic ecosystems

Source	Emission (thousand tonnes / year)
Discarded manufactured products	465
Agricultural, animal wastes, food wastes	316
Coal ashes	298
Atmospheric fallout	92
Urban refuse	60
Logging and wood wastes	39
Municipal sewage and organic waste	39
Solid wastes from metal fabrication	11
Fertilisers and peat	2.5
TOTAL	1322.5

Table 3 World-wide inputs of zinc to soils.

3. 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 4). However, as anthropogenic emissions of zinc far exceed those from natural sources, elevations above these natural, background concentrations are often found (see Table 5).

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 (USPHS 1997). In soils, it often remains strongly sorbed, and in the aquatic environment it will predominantly bind to suspended material before finally accumulating in the sediment (USPHS 1997, Bryan and 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

(USPHS 1997). Zinc in a soluble form (e.g. sulphate or chloride, present in incinerator ash, or mine tailings) is far more likely to migrate through the environment than if it is bound to organic matter or present as an insoluble precipitate (e.g. as in sewage sludge) (USPHS 1997).

Environmental Matrix	Concentration	Reference
Seawater	<1 ug/l (open ocean) 0.3-70 ug/l (coastal and estuarine)	Bryan and Langston 1992. UNEP 1993
Freshwater	<50 ug/l	USPHS 1997
Drinking water	0.02-1.2 mg/l	USPHS 1997
Soil	10-300 mg/kg (50 mg/kg average)	Alloway 1990
Freshwater sediment	<100 mg/kg	USPHS 1997, Salomons and Forstner 1984
Marine Sediment	<100 mg/kg	Bryan and Langston 1992, UNEP 1993

Table 4 Background concentrations of zinc found in water, sediments and soil.

Site Description	Concentration	Reference
Restronguet Creek sediment, branch of the Fal estuary, UK. Receiving acidic drainage from past and present mining activities.	3000 mg/kg	Bryan and Langston 1992
Seawater, Restronguet Creek Seawater, polluted harbours in the Mediterranean	20-20460 ug/l 450 ug/l	Bryan and Langston 1992 UNEP 1993
Mediterranean coastal sediments (Venice Lagoon, River Ebro Delta, Izmir Bay, Kastela Bay, Gulf of Elefsis, Abu Kir Bay). Sites receiving large quantities of industrial and urban wastes	200-6200 mg/kg	UNEP 1993
Concentrations in surface soils in the vicinity of mines and smelters. Poland, UK, USA, Russia, Korea	11-10500 mg/kg 2040-50000 mg/kg 400-4245 mg/kg 329-25800 mg/kg	Dudka et al. 1995 Matthews and Thornton 1982 Dudka and Adriano 1997 Jung and Thornton 1996
River water, from sites receiving urban and industrial waste, USA	0.01-2.4 mg/l	USPHS 1997
Donana National Park, Spain. Contaminated river sediments close to mining sites	879-12200 mg/kg	Pain et al. 1998

Table 5 Zinc concentrations associated with sites of anthropogenic contamination.

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 (USPHS 1997).

4. Toxicity and Essentiality

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

Due to the essentiality of zinc, dietary allowances of 15 mg/day for men, and 12 mg/day for women are recommended (USPHS 1997). Seafood provides a major source, and

several species such as oysters, mussels, shrimps and crabs have bioconcentration factors ranging from several hundreds to several thousands (UNEP 1993). 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 month, resulted in anaemia and injury to the pancreas and kidney; and rats that ate very large amounts of zinc became infertile (USPHS 1997). Humans taking supplements at higher than recommended doses (400-500 mg/day) suffered severe gastro-enteritis (Abernathy and Poirier 1997); and humans who drank water from galvanised pipes, over a prolonged period, suffered irritability, muscular stiffness and pain, loss of appetite and nausea (UNEP 1993).

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 (USPHS 1997, 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 ug/l, carbon fixation rates in natural phytoplankton populations were depressed. Others observed that the growth of cultured diatoms was inhibited at 20 ug/l (Bryan and Langston 1992). Effects on fertilisation and embryonic development in Baltic spring-spawning herring at low salinity were detected at only 5 ug/l (UNEP 1993); and the fertility of successive generations of harpacticoid copepod *Tisbe holothuria* was reduced by continuous exposure to only 10 ug/l (Verriopoulos and Hardouvelis 1988).

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

Plant studies have shown that although an essential element for higher plants, in elevated concentrations zinc is considered phytotoxic, directly effecting 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)

5. Legislation

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

European Council Directive 75/440/EEC concerning the quality required of surface water intended for the abstraction of drinking water in the Member States. Water containing more than 5 mg/l must be subjected to intensive physical and chemical treatment prior to use, with some degree of physical treatment still required for zinc levels of 0.5 mg/l.

European Council Directive 76/464/EEC on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. Zinc is included in List II, and as such water pollution caused by its presence must be reduced in keeping with National Environmental Quality Standards.

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

European Council Directive 80/778/EEC relating to the quality of water intended for human consumption. Guide levels of 100 ug/l (for outlets of pumping and / or treatment works) and 5000 ug/l (after water has been standing for 12 hours in the piping / made available to the consumer) are set.

The Water Research Centre in the UK recommends the following Environmental Quality Standards for zinc: protection of freshwater salmonid fish 8-125 ug/l; protection of freshwater coarse fish 75-500 ug/l; protection of other freshwater life and associated non-aquatic organisms 100 ug/l; protection of saltwater fish, shell fish and associated non-aquatic organisms 40 ug/l (Mance and Yates 1984).

The USEPA recommends a maximum permissible concentration for drinking water of 5 mg/l (USPHS 1997), as do the WHO (1993) and the Bureau of Indian Standards (1995). For the protection of fresh and saltwater life, environmental quality standards of 570 and 170 ug/l respectively, are set. Furthermore, any release of more than 1,000 pounds (or in some cases 5,000 pounds) of zinc or its compounds into the environment (i.e., water, soil, or air) must be reported to EPA (USPHS 1997).

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

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Nickel (Ni)

1. Natural Occurrence

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

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

2. Production, Use and Anthropogenic Sources

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

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

Estimates of anthropogenic emissions of nickel are given in Tables 1-3 (Nriagu 1990, Nriagu and Pacyna 1988):

Source	**Emission (thousand tonnes / year)**
Energy production (coal and oil combustion)	42.0
Steel and iron manufacture	4.47
Non-ferrous metal production (Ni, Cu, Pb)	3.99
Mining	0.80
Waste incineration (municipal refuse and sewage sludge)	0.35
TOTAL	51.61

Table 1 World-wide atmospheric emissions of nickel from anthropogenic sources.

Source	Emission (thousand tonnes / year)
Domestic wastewaters	62
Base metal mining and smelting	13
Electrical power plants	11
Sewage discharges	11
Atmospheric fallout	10
Manufacturing processes (metal, chemicals, paper, petroleum products)	7.4
TOTAL	114.4

Table 2 World-wide inputs of nickel into aquatic ecosystems.

Source	Emission (thousand tonnes / year)
Coal ashes	168
Agricultural and animal wastes	45
Atmospheric fallout	24
Discarded manufactured products	19
Municipal sewage and organic waste	15
Logging and wood wastes	13
Urban refuse	6.1
Fertilisers and peat	2.2
Solid wastes from metal fabrication	1.7
TOTAL	294

Table 3 World-wide inputs of nickel to soils.

3. Environmental Levels, Contamination and Behaviour

Nickel is found in varying concentrations in nearly all uncontaminated aquatic and terrestrial ecosystems (see Table 4). However, as anthropogenic emissions of nickel far exceed those from natural sources, elevations above these natural, background concentrations, are often found (see Table 5).

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

Table 4 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 (USPHS 1997, Mance and 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 (USPHS 1997).

Site Description	Concentration	Reference
Drinking water, near a large, open-pit mine, USA	200 ug/l	USPHS 1997
Seawater, coastal and estuarine sites of industrial and domestic discharges, UK	0.23-4.9 ug/l	Law et al. 1994
Sediment, Elsburgspruit-Natalspruit Rivers, South Africa (mining discharges and sewage)	54.5-890 mg/kg	Steenkamp et al. 1995
Soil, Sudbury Basin / Coniston, Canada (nickel mining and smelting)	100-3000 mg/kg 160-12300 mg/kg	Freedman and 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 5 nickel concentrations associated with anthropogenic contamination and waste.

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 (USPHS 1997, Alloway 1990).

4. 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 (USPHS 1997, Alloway 1990). However, at the other extreme, there is sufficient evidence for the carcinogenicity of nickel and certain nickel compounds e.g. oxide, subsulphide, carbonate, acetate, carbonyl and hydroxide. The US Department of Health and Human Services, in its 8th Report on Carcinogens, therefore lists nickel and these compounds as Reasonably Anticipated to be Human Carcinogens (USPHS 1998). Whereas metallic nickel and its alloys are listed as possible human carcinogens (Group 2B), by the International Agency for Research on Cancer (1998).

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

Allergic contact dermatitis is the most prevalent adverse effect of nickel in the general population (2-5% may be nickel sensitive). Here, people become sensitive to nickel when

jewellery or other nickel- containing objects are in direct contact with the skin. Once a person is sensitised to nickel, any further contact will produce a reaction. A rash at the site of contact is visible, and in some cases eczema may develop. Therefore, although none-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 (USPHS 1997).

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

5. Legislation

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

European Council Directive 76/464/EEC on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. Nickel is included in List II, and as such, water pollution caused by its presence must be reduced in keeping with National Environmental Quality Standards.

European Community Council Directive 80/778/EEC relating to the quality of water intended for human consumption. A Maximum Permissible Limit of 50 ug/l is set.

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

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

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Mercury (Hg)

1. Natural Occurrence

Mercury is a very rare metal, found in the earth's crust at concentrations frequently below 0.03 mg/kg (Alloway 1990). Cinnabar (mercury sulphide) is the only commercially important ore, and is found along lines of previous volcanic activity. The most famous and extensive deposits of cinnabar are in Spain (containing 6-7% mercury), with other deposits (<1% mercury) found in the CIS, Algeria, Mexico and Italy (USPHS 1997, Greenwood and Earnshaw 1984). Volcanic activity and biogenic processes are the largest natural sources of atmospheric mercury, followed by emissions from wind-borne soil particles, sea salt sprays and forest fires. It is estimated that the total amount of mercury released to the atmosphere from natural sources is 2500 tonnes / year, compared with an estimated anthropogenic load of 3550 tonnes (Nriagu 1990).

2. Production, Use and Anthropogenic Sources

Mercury ores are processed easily and inexpensively to produce metallic mercury. Due to the low boiling point of elemental mercury, refining can be achieved by heating the ore and condensing the vapour, a method that is 95% efficient, and yields mercury that is 99.9% pure (USPHS 1997).

Uses of mercury are extensive, due to its unique properties of fluidity, its high surface tension, and its ability to alloy with other metals. It is primarily used in the electrical industry in alkaline batteries, electric lamps, and wiring and switching devices, such as thermostats and cathode tubes. It is also used in the chemical industry as a catalyst used to form polymers (e.g. vinyl chloride and polyurethane), and as the cathode in the chlor-alkali electrolytic separation of brine to produce chlorine and sodium hydroxide (caustic soda). Mercuric oxide and mercuric sulphide are used as pigments in paints; and gold mining operations utilise mercury to extract gold from ores through amalgamation. Metallic mercury is used in dental restorations, and in medical equipment, such as thermometers and manometers. Until 30 years ago, mercury compounds were used extensively as pharmaceuticals and agrochemicals, e.g. as components of antiseptics, diuretics, skin lightening creams, laxatives, anti-syphilitic drugs, fungicides, bactericides, wood and felt preservatives. However, due to the high toxicity of mercury, most of these applications are banned in most parts of the World (USPHS 1997).

Estimates of anthropogenic emissions of mercury are given in table 1-3 (Nriagu 1990, Nriagu and Pacyna 1988).

Source	**Emission (thousand tonnes / year)**
Energy production (coal combustion)	2.26
Waste incineration (municipal refuse and sewage sludge)	1.16
Non ferrous metal production (Pb, Cu, Ni)	0.13
TOTAL	3.55

Table 1 World-wide atmospheric emissions of mercury from anthropogenic sources.

Source	**Emission (thousand tonnes / year)**
Manufacturing processes (metal, chemicals, petroleum products)	2.1
Atmospheric fallout	2.0
Electric power plants	1.8
Domestic wastewaters	0.30
Sewage discharges	0.16
Base metal mining and smelting	0.10
TOTAL	6.46

Table 2 World-wide inputs of mercury into aquatic ecosystems.

Source	Emission (thousand tonnes / year)
Coal ashes	2.6
Atmospheric fallout	2.5
Logging and wood wastes	1.1
Agricultural and animal wastes	0.85
Discarded manufactured products	0.68
Municipal sewage and organic waste	0.44
Urban refuse	0.13
Solid wastes from metal fabrication	0.04
Fertilisers and peat	0.01
TOTAL	8.35

Table 3 World-wide inputs of mercury to soils.

3. Environmental Levels, Contamination and Behaviour

Mercury is found at very low concentrations in many aquatic and terrestrial ecosystems (see Table 4). However, as anthropogenic emissions of mercury exceed those from natural sources, elevations above these natural, background, concentrations, can be found (see Table 5).

Due to the fact that mercury is the only metal that can exist as both a liquid and a vapour at ambient temperatures, its environmental behaviour differs from that of most other toxic elements (USPHS 1997, WHO 1989). mercury can exist in three valence states, Hg (0), Hg (I) and Hg (II). In the atmosphere, elemental mercury is by far the most common form, and, as a vapour, it is responsible for the long-range, global cycling of mercury. In addition, to a far lesser degree, mercury may be associated with particulates, which are removed by dry or wet deposition. Atmospheric inputs may be more significant in areas where other sources, such as contaminated rivers, are less important or non-existent (USPHS 1997, WHO 1992).

In the aquatic environment, mercury is most commonly found in the mercuric (II) state, and its fate, once released, is dominated by rapid adsorption to soluble and particulate organic material; followed by flocculation, precipitation and final accumulation in the bottom sediment. Because of the strength with which mercury is bound to sediment, exchange back to the water column is generally slight, although it can be accelerated in saline waters, and in the presence of high concentrations of sulphide (anoxic conditions) (USPHS 1997, Bryan and Langston 1992). Dredging or re-suspension of bed materials may cause short-term release of mercury, although levels of dissolved metal quickly return to pre-disturbance values. mercury accumulation from sediments may therefore be a dominant pathway for uptake in aquatic organisms and accounts for relatively high concentrations in deposit feeders, in both freshwater and marine systems (Bryan and Langston 1992).

Inorganic mercury can be methylated by micro-organisms, indigenous to soils, fresh water and marine sediments. The most common form of organic mercury is methylmercury (MeHg), which is soluble, mobile, and quick to enter the aquatic food chain. The selective retention of MeHg at each step in the food chain, relative to inorganic mercury, is related to its high lipid solubility, its long biological half-life, and the increased longevity of top predators (Bryan and Langston 1992). As a result, MeHg provides one of the rare examples of metal biomagnification in food chains (USPHS 1997, WHO 1989). For

example, concentrations in carnivorous fish at the top of freshwater and salt water food chains (e.g., pike, tuna, and swordfish) are biomagnified 10,000-100,000 times the concentrations found in ambient waters (USPHS 197). The significance of this bioaccumulation is that it is generally the most important source of human, non-occupational mercury exposure (USPHS 1997, WHO 1989).

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

Table 4 Background concentrations of mercury found in water, sediments, soil and fish.

Environmental Matrix	Concentration	Reference
Marine sediment (Mersey estuary, UK)	6 mg/kg	Bryan and Langston 1992
Freshwater sediment (affected by gold mining, Brazil)	0.14-9.82 mg/kg	Reuther 1994
Soil (varying distances from chlor-alkali plant)	0.1-10 mg/kg	Gonzalez 1991, Alloway 1990
Fish (Madeira River, gold mining activities, Brazil)	<0.3-11.15 mg/kg	Barbosa et al. 1995

Table 5 mercury concentrations associated with anthropogenic contamination and waste.

4. Toxicity

Mercury is an extremely toxic, non-essential trace metal, having no biochemical or nutritional function. Biological mechanisms for its removal are poor, and, as mentioned above, mercury is the only metal known to biomagnify i.e. progressively accumulate though the food chain (WHO 1989).

Acute inhalation of high levels of mercury vapour may cause nausea, vomiting, diarrhoea, increases in blood pressure or heart rate, skin rashes, eye irritation, corrosive bronchitis and pneumonitis. And, if not fatal, may be associated with central nervous system (CNS) effects such as tremor or increased excitability (USPHS 1997, Goyer 1996). With chronic exposure, the major effects are on the CNS (tremor, spasms, loss of memory, increased excitability, severe depression personality changes, even delirium and hallucination), although renal damage, associated with chronically exposed workers, has also been shown (Ratcliffe *et al.* 1996, Goyer 1996). These effects have also been reported in animal studies (USPHS 1997)

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

Exposure to MeHg has resulted in permanent damage to the CNS, kidneys, and the developing foetus. The levels of MeHg that result in these effects are not usually encountered by the general population, however they were encountered by the population

of Minamata, in Japan, who were exposed to high levels of MeHg from eating contaminated fish and seafood collected from the Bay (USPHS 1997). Symptoms such as brain damage, numbness of extremities, and paralysis, along with the loss of hearing, speech and sight were reported (D'Itri 1991). However even today, the full range of neurological symptoms caused by the ingestion of MeHg in fish and shellfish has not been fully characterised, and the total number of Minamata Disease sufferers has not been determined (D'Itri 1991). Furthermore, whilst only the Japanese cases have been confirmed as Minamata Disease, other populations in Canada (from chlo-ralkali discharges) and Brazil (from gold mining) are potentially at risk. The problem of methylation of past and present inorganic mercury discharges continues, and the long retention time of mercury by sediments, delays the elimination of contamination for many years (Harada 1997, Barbosa 1997, Akagi *et al.* 1995, Bryan and Langston 1992, D'Itri 1991).

Studies on the aquatic toxicity of mercury are numerous, and again show that MeHg is more toxic than any of the inorganic forms. Invertebrate studies have reported significant reductions in the growth rate of the mussel *Mytilis edulis* at concentrations of 0.3 ug/l, with growth almost ceasing at 1.6 ug/l, and acute lethal effects observed at 25 ug/l (WHO 1989). In addition, changes in filtering activity, oxygen consumption, blood osmotic pressure, ciliary and valve activity have also been reported (Naimo 1995) In the American oyster *Crassostrea virginica* embryonic abnormalities were evident at concentrations of 5-10 ug/l. With survival rates of exposed clams and barnacles, copepods, shrimps and crustaceans all greatly effected by increased levels of mercury (WHO 1997, Bryan and Langston 1992).

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

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

5. Legislation

European legislation on water quality and permissible environmental levels treat mercury as a priority pollutant i.e. legislation is concerned with the elimination of pollution caused by mercury and not just the reduction. For example:

Council Directive 75/440/EEC concerning the quality required of surface water intended for the abstraction of drinking water in the Member States. Water containing more than 0.5 ug/l of mercury must be subjected to intensive physical and chemical treatment prior to use.

Council Directive 76/464/EEC on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. Mercury is included in List I, and as such water pollution caused by its presence should be eliminated.

Council Directive 80/778/EEC relating to the quality of water intended for human consumption. A maximum permissible concentration of 1 ug/l is set.

Other drinking water standards include those set by the Bureau of Indian Standards (1 ug/l) (1995), the USEPA (2 ug/l) (USPHS 1997) and the WHO (1 ug/l) (1993).

In terms of soil contamination, the UK Department of the Environment (ICRCL) classifies a level of 0-1 mg/kg as being typical of uncontamination. Anything above this is classified as contaminated, and as such, restrictions on recreational and agricultural uses apply (Alloway 1990). In terms of permissible sewage sludge levels, acceptable concentrations for mercury range from 2-25 mg/kg. However resulting soil concentrations should not exceed 1-2 mg/kg (EC, UK, France, Germany) (Alloway 1990).

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

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Copper (Cu)

1. Natural Occurrence

Copper was almost certainly one of the first three metals discovered (the others being gold and silver), and although opinion on the earliest use varies, 5000 BC is not an unreasonable estimate (Hong et al. 1996, Greenwood and Earnshaw 1984). Abundance in the Earth's crust is reported as ranging from 24-55 mg/kg (Alloway 1990), although higher levels are associated with some shales and clays (Thornton 1995). copper can occur in the elemental state, however it is found more commonly as a sulphide (copper pyrite), oxide (cuprite) or carbonate (malachite). The largest deposits of copper are found in the USA, Chile, Canada, the Commonwealth of Independent States, Zambia and Peru (Dudka and Adriano 1997, Alloway 1990).

Volcanic activity is the major source of copper released to the atmosphere, followed by emissions from wind-borne soil particles, forest fires, sea salt spray and biogenic processes. It is estimated that the total amount of copper released to the atmosphere from

natural sources is 28,000 tonnes / year, compared with an estimated anthropogenic load of 35,000 tonnes / year (Nriagu 1990).

2. Production, Use and Anthropogenic Sources

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

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

Estimates of anthropogenic emissions of copper are given in Tables 1-3 (Nriagu 1990, Nriagu and Pacyna 1988).

Source	**Emission (thousand tonnes / year)**
Non-ferrous metal production (Cu, Pb, Zn, Cd, Ni)	23.2
Energy production (coal and oil combustion)	8.04
Steel and iron manufacturing	2.01
Waste incineration (municipal refuse and sewage sludge)	1.58
Mining	0.42
TOTAL	35.25

Table 1 World-wide atmospheric emissions of copper from anthropogenic sources.

Source	**Emission (thousand tonnes / year)**
Manufacturing processes (metal, chemicals, paper, petroleum products)	34
Domestic wastewaters	28
Base metal mining and smelting	14
Electric power plants	13
Sewage discharges	12
Atmospheric fallout	11
TOTAL	112

Table 2 World-wide inputs of copper into aquatic ecosystems.

3. 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 4). However, as anthropogenic emissions of copper exceed those from natural sources, elevations above these natural, background concentrations are often found (see Table 5).

Source	Emission (thousand tonnes / year)
Discarded manufactured products	592
Coal ashes	214
Agricultural and animal wastes	67
Logging and wood wastes	28
Urban refuse	26
Atmospheric fallout	25
Municipal sewage and organic waste	13
Solid wastes from metal fabrication	*4.3*
Fertilisers and peat	*1.4*
TOTAL	*970.7*

Table 3 World-wide inputs of copper to soils.

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

Table 4 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 (USPHS 1997, Mance et al. 1984).

Site Description	Concentration	Reference
Seawater, Restronguet Creek, UK (receives acidic drainage from past and present mining activities)	>2000 ug/l	Bryan and Langston 1992
Sediment, Restronguet Creek	3000 mg/kg	Bryan and 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 5 copper concentrations associated with sites of anthropogenic contamination and waste.

In soils, copper has a high affinity for sorption by organic and inorganic ligands (e.g. humic and fulvic acids, hydroxides of iron, aluminium and manganese). However it can also exist as soluble ions and complexes. copper in a soluble form is far more bioavailable

and far more likely to migrate through the environment, that if it is bound to organic matter or present as an insoluble precipitate. Therefore, copper sulphate, or chloride, present in MSW incinerator ash or mine tailings, is far more bioavailable and migratory than the organically bound copper found in sewage sludge (USPHS 1997, Alloway 1990, Mance et al. 1984).

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

4. 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-lining of collagen, elastin and hair keratin (USPHS 1997). Human deficiency is characterised by anaemia, resulting from defective haemoglobin synthesis (Goyer 1996). However at the other extreme, vomiting, hypotension, jaundice, coma and even death, can result from acute poisoning (USPHS 1997).

Therefore, even though copper is essential for good health, a very large single dose, or long term elevated exposure can be harmful. Inhalation of dust and vapours can irritate the nose, mouth and eyes, and cause headaches, dizziness, nausea and diarrhoea. Oral exposure to high levels can cause vomiting, diarrhoea, stomach cramps and nausea (USPHS 1997). Copper homeostasis plays an important role in the prevention of copper toxicity, in humans, terrestrial animals, and aquatic organisms. copper is readily absorbed from the stomach and small intestine; and after requirements are met, there are several mechanisms that prevent copper overload e.g. bile excretion, increased storage in the liver or bone marrow (USPHS 1997). However, failure of this homeostatic mechanism can occur in humans and animals following exposure to high levels of copper. This rare disease, known as Wilson’s disease, is characterised by the excessive retention of copper in the liver and impaired copper excretion in the bile. Resulting in liver and kidney damage and haemolytic anaemia (USPHS 1997).

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

Aquatic toxicity to copper is well studied, and there is experimental evidence that a considerable number of species are sensitive to dissolved concentrations as low as 1-10 ug/l (Bryan and Langston 1992). For example, studies have shown that at levels of 2 ug/l, the survival rate of young bay scallops was significantly effected; and in the embryos of oysters and mussels concentrations of 5 ug/l were seen to induce abnormalities. A similar concentration resulted in increased mortalities in populations of the isopod crustacean Idothea baltica (UNEP 1993, Bryan and Langston 1992, Giudici et al. 1989). Other studies have reported reductions in the survival, growth and fertility of amphipods and copepods (Conradi and DePledge 1998, UNEP 1993), and embryonic sensitivity in fish exposed to levels of 25 ug/l (UNEP 1993, Mance et al. 1984) Furthermore, a study of species diversity in benthic communities from Norwegian fjords, led to the conclusion that the most sensitive animals were missing from sites where sediment-copper levels exceeded

200 mg/kg. In the UK, such concentrations are exceeded in a number of estuaries, including the Fal and the Tamar. Here, many species of bivalves, including some mussels, clams and cockles are absent, and at best distribution is severely limited. The toxicity of the surface sediment containing over 2000 mg/kg of copper, towards juvenile bivalves appears to be the reason (Bryan and Langston 1992).

5. Legislation

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

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

European Council Directive 76/464/EEC on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. copper is included in List II, and as such, water pollution caused by its presence must be reduced in keeping with National Environmental Quality Standards.
European Council Directive 78/659/EEC on the quality of fresh waters needing protection or improvement in order to support fish life. An Environmental Quality Standard of 40 ug/l is set.

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

Other drinking water standards include those set by the Bureau of Indian Standards (1995) (50ug/l), the USEPA (1300 ug/l) (USPHS 1997) and the WHO (1000 ug/l)(1993)

The Water Research Centre in the UK recommends the following Environmental Quality Standards for copper: protection of freshwater fish, other freshwater life and associated non-aquatic organisms 1-28 ug/l; protection of saltwater fish, shellfish, other saltwater life and associated non-aquatic organisms 5 ug/l (Mance et al. 1984).

Regarding soil contamination, the UK Department of the Environment (ICRCL) classifies a level of 1-100 mg/kg as being typical of uncontamination. Anything above this is classified as contaminated, and as such, restrictions on recreational and agricultural uses apply (Alloway 1990). In terms of permissible sewage sludge levels, acceptable concentrations for copper range from 500-3000 mg/kg. However soil concentrations should not exceed 50-100 mg/kg (EC, France, Germany) (Alloway 1990).

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Chromium (Cr)

1. Natural Occurrence

Chromium is the 21st most abundant element in the Earth's crust, with an average concentration of 100 mg/kg. However in some igneous rocks, clays and shales, higher concentrations can be found (Alloway 1990). The only ore of chromium of any

commercial importance is chromite ($FeCr_2O_4$) which is produced principally in South Africa, Albania, Turkey, India and Zimbabwe. Other less plentiful sources are the ores crocoite ($PbCrO_4$) and chrome ochre (Cr_2O_3) (Mukherjee 1998, USPHS 1997, Alloway 1990, Greenwood and Earnshaw 1984). The gem stones, emerald and ruby, owe their colours to traces of chromium (Alloway 1990).

Emissions from wind-borne soil particles are the largest natural sources of atmospheric chromium, followed by emissions from volcanic activity, biogenic sources, forest fires and sea salt sprays. It is estimated that the total amount of chromium released to the atmosphere from natural sources is 43,000 tonnes / year, compared with an estimated anthropogenic load of 30,400 tonnes / year (Nriagu 1990).

2. Production, Use and Anthropogenic Sources

Chromium is produced in two forms. Firstly as ferrochrome, formed by the reduction of chromite with coke in an electric arc furnace (a low-carbon ferrochrome can be made using silicon, instead of coke, as the reductant). This iron-chromium alloy is used directly as an additive to produce chromium-steels, which are "stainless" and hard. Alternatively, following aerial oxidation of chromite, leaching, precipitation and reduction, chromium metal can be obtained (USPHS 1997, Greenwood and Earnshaw 1984).

Of the 10 million tonnes of 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. The refractory properties of chromium (resistance to high temperatures) are exploited in production of refractory bricks for lining furnaces and kilns, accounting for approximately 15% of the chromate ore used. 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 (USPHS 1997, Alloway 1990, Greenwood and Earnshaw 1984).

Estimates of anthropogenic emissions of chromium are given in Tables 1-3 (Nriagu 1990, Nriagu and Pacyna 1988):

Source	**Emission (thousand tonnes / year)**
Steel and iron manufacturing	15.6
Energy production (coal and oil combustion)	12.7
Cement production	1.3
Waste incineration (municipal refuse and sewage sludge)	0.8
TOTAL	30.4

Table 1 World-wide atmospheric emissions of chromium from anthropogenic sources.

3. Environmental Levels, Contamination and Behaviour

Chromium is found in varying concentrations in nearly all uncontaminated aquatic and terrestrial ecosystems (see Table 4). However, in areas associated with anthropogenic

emissions, ecosystem levels can far exceed natural, background concentrations (see Table 5).

Source	Emission (thousand tonnes / year)
Manufacturing processes (metal, chemicals, paper, petroleum products)	51
Domestic wastewaters	46
Sewage discharges	19
Base metal mining and smelting	12
Atmospheric fallout	9.1
Electric power plants	5.7
TOTAL	142.8

Table 2 World-wide inputs of chromium into aquatic ecosystems.

Source	Emission (thousand tonnes / year)
Discarded manufactured products	458
Coal ashes	298
Agricultural and animal wastes	82
Atmospheric fallout	22
Urban refuse	20
Logging and wood wastes	10
Municipal sewage and organic wastes	6.5
Solid wastes from metal fabrication	1.5
Fertilisers and peat	0.32
TOTAL	898.32

Table 3 World-wide inputs of chromium into soils.

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

Table 4 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 and Langston 1992
Marine sediment, Sawyer's Bay, New Zealand (tannery waste)	3700 mg/kg	Bryan and Langston 1992
Soil, chromium smelting, Japan	30-4560 mg/kg	Dudka and 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 5 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 intra-media transport, however chromium (VI) will eventually be converted to chromium (III), by reducing species such

as organic substances, hydrogen sulphide, sulphur, iron sulphide, ammonium and nitrite (USPHS 1997, Kimbrough *et al.* 1999). This trivalent form is generally not expected to significantly migrate in natural systems. Instead, it is rapidly precipitated and adsorbed onto suspended particles and bottom sediments. However changes in the chemical and physical properties of an aquatic environment, can result in changes to the chromium (III)-chromium (VI) equilibrium (Richard and Bourg 1991).

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

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

The importance of this lies in the fact that whilst chromium (III) is an essential trace elements 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 and Sheuhammer 1993, UNEP 1991, Richard and Bourg 1991).

4. 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 (USPHS 1997, Goyer 1996). The minimum human daily requirement of chromium (III) for optimal health is not known, but a daily ingestion of 50-200 ug/day has been estimated to be safe and adequate. However, although an essential food nutrient, very large doses may be harmful (USPHS 1997).

Chromium (VI) is non-essential and toxic. Compounds are corrosive, and allergic skin reactions readily occur following exposure, independent of dose. Short-term exposure to high levels can result in ulceration of exposed skin, perforations of respiratory surfaces and irritation of the gastrointestinal tract. Damage to the kidney and liver have also been reported (USPHS 1997). In addition, the International Agency for Research on Cancer (IARC) classifies chromium (VI) compounds as known carcinogens (1998). Long-term occupational exposure to airborne levels of chromium higher than those in the natural

environment has been associated with lung cancer. Individuals at most risk include those in chromate-production industries and chromium pigment manufacture and use; and similar risks may exist amongst chromium-alloy workers, stainless steel welders, and chrome-platers (Kimbrough 1999, USPHS 1998).

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

5. Legislation

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

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

European Council Directive 76/464/EEC on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. Chromium is included in List II, and as such water pollution caused by its presence must be reduced in keeping with National Environmental Quality Standards.

European Community Council Directive 80/778/EEC relating to the quality of water intended for human consumption. A Maximum Permissible Concentration of 50 ug/l is set.

Other drinking water legislation includes that set by the by the Bureau of Indian Standards (1995), the WHO (1993), and the USEPA (USPHS 1997). All of these set a guideline / recommended limit of 50 ug/l

Regarding soil contamination, the UK Department of the Environment (ICRCL) classifies a level of 0-100 mg/kg as being typical of uncontamination. Anything above this is classified as contaminated, and as such, restrictions on recreational and agricultural uses apply (Alloway 1990). In terms of permissible sewage sludge levels, acceptable concentrations for chromium range from 200-1200. However resulting soil concentrations should not exceed 150 mg/kg (EC, France, Germany) (Alloway 1990).

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Cadmium (Cd)

1. Natural Occurrence

Cadmium is a relatively rare metal, being 67th in order of chemical abundance. It is found in the Earth's crust at an average concentration of 0.1 mg/kg (WHO 1992), although some sedimentary rocks, black shales and marine phosphates can accumulate higher levels (WHO 1992, Alloway 1990). It is usually found in association with the sulphide ores of zinc, copper and lead, and is obtained as a by-product during the processing of these ores. Volcanic activity is the major natural source of cadmium released to the atmosphere, followed by emissions from wind-borne soil particles, forest fires, sea salt spray and biogenic processes. It is estimated that the total amount of cadmium released to the atmosphere from natural sources is 1400 tonnes / year, compared with an estimated anthropogenic load of 7600 tonnes / year (Nriagu 1990).

2. Production, Use and Anthropogenic Sources

Cadmium is a by-product of zinc and lead mining and smelting, and is currently used primarily for the production of nickel-cadmium batteries (37%) and for metal plating

(25%). It is also used in pigments for glasses and plastics (22%), as a stabiliser in polyvinyl chloride (12%), and as a component of various alloys (4%) (USPHS 1997, WHO 1992). Estimates of anthropogenic emissions of cadmium are given in Tables 1-3 (WHO 1992, Nriagu 1990).

Source	**Emission (thousand tonnes / year)**
Non-ferrous metal production (Zn, Cd, Cu, Pb, Ni)	5.43
Energy production (coal and oil combustion)	0.79
Waste incineration (municipal refuse and sewage sludge)	0.75
Manufacturing processes (steel, iron, phosphate fertilisers, cement)	0.60
TOTAL	7.57

Table 1 World-wide atmospheric emissions of cadmium from anthropogenic sources.

Source	**Emission (thousand tonnes / year)**
Manufacturing (metal, batteries, pigments, plastics)	2.4
Atmospheric fallout	2.2
Base metal mining and smelting	2.0
Domestic wastewaters	1.7
Sewage discharges	0.69
Electric power plants	0.12
TOTAL	9.11

Table 2 World-wide inputs of cadmium into aquatic ecosystems.

Source	**Emission (thousand tonnes / year)**
Coal ashes	7.2
Atmospheric fallout	5.3
Urban refuse	4.2
Agriculture and animal wastes	2.2
Discarded manufactured products	1.2
Logging and wood wastes	1.1
Fertilisers and peat	0.2
Municipal sewage / organic waste	0.18
Solid waste from metal fabrication	0.04
TOTAL	21.62

Table 3 World-wide inputs of cadmium to soils.

3. Environmental Levels, Contamination and Behaviour

Cadmium is a rare metal, found naturally as very low concentrations (see Table 4). However, as anthropogenic emissions far exceed those from natural sources, elevations above these natural, background levels, are often found (see Table 5).

Cadmium is more mobile in aquatic environments than most other metals. It is also bioaccumulative and persistent in the environment ($t^{1/2}$ of 10-30 years) (USPHS 1997). It is found in surface and groundwater as either the +2 hydrated ion, or as an ionic complex with other inorganic or organic substances. While soluble forms may migrate in water, cadmium in insoluble complexes or adsorbed to sediments is relatively immobile.

Environmental matrix	*Concentration*	Reference
Freshwater, groundwater, drinking water	<1 ug/l	USPHS 1997, WHO 1992
Seawater (open ocean)	0.02-0.12 ug/l	Sadiq 1992, Bryan and Langston 1992
Seawater (coastal)	0.01-0.17 ug/l	Bryan and Langston 1992
Marine sediment	<1 mg/kg	Sadiq 1992, Salomons and Forstner 1984
Estuarine sediment	0.2 mg/kg	Bryan and Langston 1992
River sediment	1 mg/kg	Salomons and Forstner 1984
Soil	0.01-2.0 mg/kg	USPHS 1997, Alloway 1990

Table 4 Background concentrations of cadmium found in water, sediment and soil.

Similarly, cadmium in soil may exist in soluble form in soil water, or in insoluble complexes with inorganic and organic soil constituents (USPHS 1997, WHO 1992). In soils, the agricultural use of phosphate fertilisers or cadmium-containing sewage sludge, can dramatically increase cadmium concentrations. Furthermore, cadmium is readily available for uptake in grain, rice and vegetables, and there is a clear association between the cadmium concentration in soil and the plants grown on that soil (Elinder and Jarup 1996, Cabrera *et al.* 1994, WHO 1992).

Site description	Concentration	Reference
Seawater, Restronguet Creek, UK (receives acid mine drainage from past and present mining activities)	50 ug/l	Bryan and Langston 1992
Sediment, Donana National Park, Spain (sites contaminated by mining waste)	2.4-38.6 mg/kg	Pain et al. 1998
Soils and sediments, Taiwan, close to a plastic stabiliser factory	0.22-1,486 mg/kg(soil) 134-4,700 mg/kg (sed.)	Chen 1991
Soil, Zn-Pb smelting, Upper Silesia, Poland	0.3-102 mg/kg	Dudka et al. 1995a
Soil, Cu-Ni mining site, Sudbury, Ontario	0.1-10 mg/kg	Dudka et al. 1995b
Garden soil, Shipham, UK (site of past Zn-Pb mining)	360 mg/kg (max.)	Alloway 1996
MSW fly ash (UK incinerators)	21-646 mg/kg	Mitchell et al. 1992

Table 5 cadmium concentrations associated with sites of anthropogenic contamination and waste.

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

4. Toxicity

Cadmium has no biochemical or nutritional function, and it is highly toxic to both plants and animals (USPHS 1997, WHO 1992, Alloway 1990). In humans and animals, there is

strong evidence that the kidney is the main target organ of cadmium toxicity, following extended exposure (USPHS 1997, Elinder and Jarup 1996, Goyer 1996, Roels *et al.* 1993, Iwata *et al.* 1993, WHO 1992, Mueller *et al.* 1992). Renal damage includes tubular proteinuria (the excretion of low molecular weight proteins) and a decrease in the glomerular filtration rate. The latter results in a depressed re-sorption of enzymes, amino acids, glucose, calcium, copper, and inorganic phosphate. Furthermore, studies have shown that even when cadmium exposure ceases, proteinuria does not decrease, and renal tubular dysfunction and reduced glomerular filtration increase in severity (USPHS 1997, Jarup *et al.* 1997, Elinder and Jarup 1996, Goyer 1996, Iwata *et al.* 1993, WHO 1992, Nriagu 1988).

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

Case studies indicate that calcium deficiency, osteoporosis, or osteomalacia (softening of the bones) can develop in some workers after long-term occupational exposure to high levels of cadmium. A progressive disturbance in the renal metabolism of vitamin D and an increased urinary excretion of calcium is often seen, suggesting that bone changes may be secondary to disruption in kidney vitamin D and calcium metabolism (USPHS 1997, Goyer *et al.* 1994, WHO 1992). In the Jinzu River Basin, a cadmium-contaminated area in Japan, a cadmium induced skeletal disorder known as Itai-Itai disease disabled many children born to women of middle age and poor nutrition (Alloway 1996).

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

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

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

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

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

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

5. Legislation

European Directives and Decisions on water quality and permissible discharges treat cadmium as a priority pollutant. Therefore legislation is concerned with the elimination of pollution caused by cadmium, and not just the reduction. Examples include:

Council Directive 75/440/EEC concerning the quality required of surface water intended for the abstraction of drinking water in the Member States. Water containing more than 1 ug/l of cadmium must be subjected to intensive physical and chemical treatment prior to use.

Council Directive 76/464/EEC on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. Cadmium is included in List I, and as such water pollution caused by its presence should be eliminated.

Council Directive 80/778/EEC relating to the quality of water intended for human consumption. A maximum permissible concentration of 1 ug/l is set.

Other drinking legislation includes that devised by the Bureau of Indian Standards (1995) and the USEPA (USPHS 1997), which both set a maximum permissible concentration for cadmium of 10 ug/l, although the USEPA does have plans to reduce this limit to 5 ug/l. The WHO currently recommends a guideline level of 5 ug/l (WHO 1993).

Regarding soil contamination, the UK Department of the Environment (ICRCL) classifies a level of 0-1 mg/kg as being typical of uncontamination. Anything above this is classifies as contaminated, and as such, restrictions on recreational and agricultural uses apply (Alloway 1990). In terms of permissible sewage sludge levels, acceptable concentrations for cadmium range from 8-30 mg/kg. However resulting soil concentrations must not exceed 3 mg/kg (EC, UK, France, Germany) (Alloway 1990).

Finally, cadmium is included in the list of priority hazardous substances agreed by the Third North Sea Conference (MINDEC 1990), Annex 1A to the Hague Declaration, and confirmed at the Fourth Conference in Esjberg, Denmark, in 1995 (MINDEC 1995). Here it was agreed that environmental concentrations of hazardous substances should be

reduced to near background levels within the next 25 years. An objective further reinforced in the Sintra Statement at the 1998 Ministerial Meeting of the OSPAR Commission (OSPAR 1998a). Cadmium has been selected for priority action, and as such as included in Annex 2 of the OSPAR Strategy with regard to Hazardous Substances (OSPAR 1998b).

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Manganese (Mn)

Manganese is an essential trace metal, although human and animal exposure to high levels can cause serious illness. Workers chronically exposed to high levels of manganese in the air have suffered both mental and emotional disturbances, along with increased slowness and clumsiness of body movements. This combination of symptoms is a disease called manganism. The symptoms can be reduced by medical treatment, but due to the high levels of manganese accumulated in the brain, any brain injury is often permanent (ATSDR 1997). It is not certain whether eating or drinking elevated levels of manganese can cause manganism or not. In one report, humans exposed to contaminated drinking water, developed symptoms similar to those seen in manganese miners or steel workers, but it is not certain if the effects were caused by the manganese alone. Another report found that people who drank water with above average levels of manganese seemed to have a slightly higher frequency of symptoms such as weakness, stiff muscles, and trembling of the hands. However, these symptoms are not specific for manganese, and might have been caused by other factors (ATSDR 1997).

Studies in animals have shown that very high levels of manganese in food or water can cause changes in the brain, suggesting that high levels might cause brain injury. In addition, animal studies have indicated that manganese may also be a reproductive toxicant, especially to males, injuring the testes and causing impotence.

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