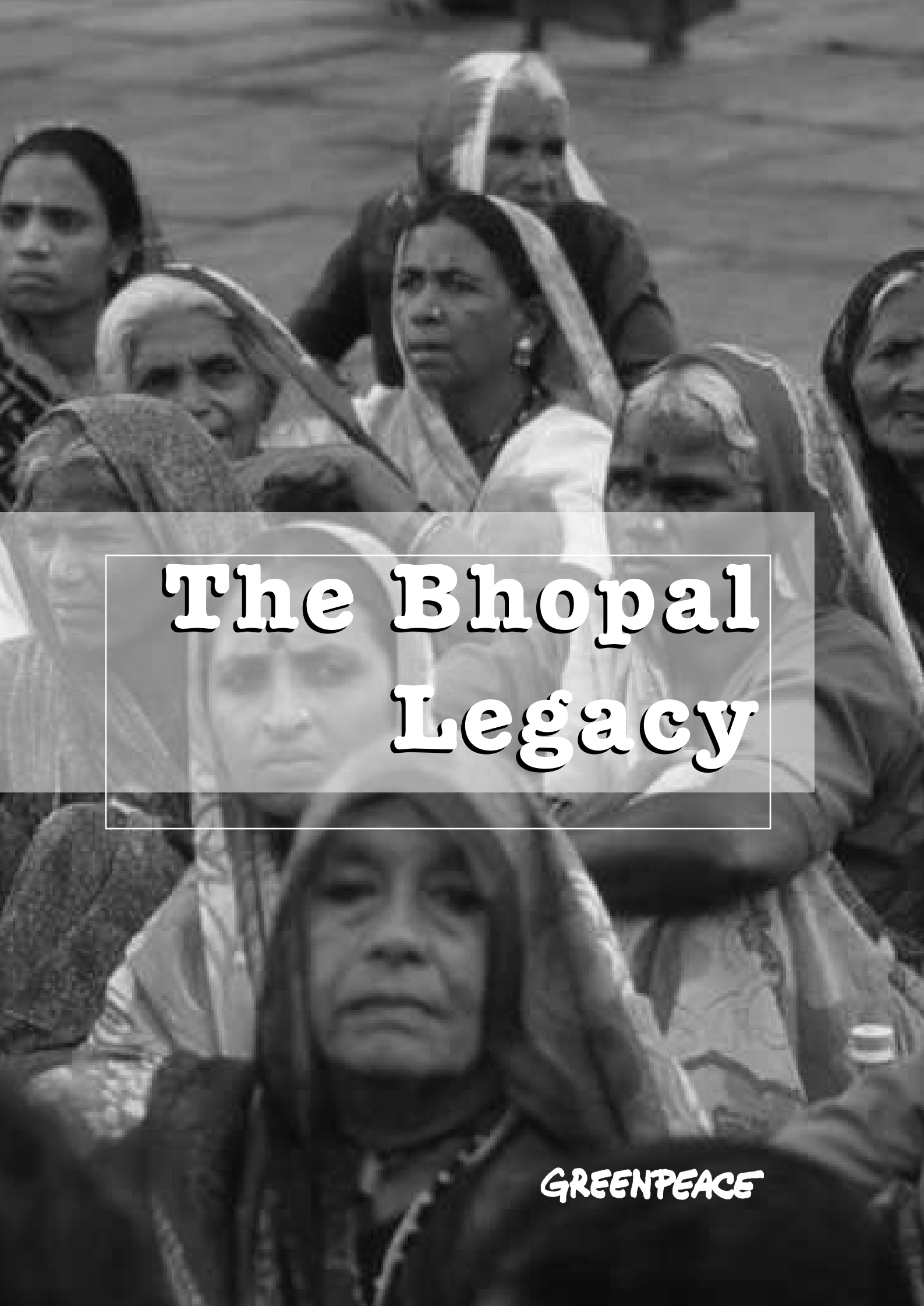

The Bhopal Legacy

GREENPEACE

THE BHOPAL LEGACY

Toxic contaminants at the former Union Carbide factory site, Bhopal, India: 15 years after the Bhopal accident.

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Technical Note 04/99

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Front Cover:
Victims of the Union Carbide disaster attend a weekly meeting.

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Executive summary

The Union Carbide India Ltd. (UCIL) pesticide plant in Bhopal, which used to manufacture (among other products) the pesticide Sevin (carbaryl) gained world-wide recognition as a result of the tragic chemical disaster on the night of 2-3rd December 1984. The accident, involving a massive release of methylisocyanate (MIC) gas, resulted in the death or injury of many thousands of people in the surrounding residential areas. A number of studies, conducted in the aftermath of the accident, understandably focused on the long-term consequences of acute exposure to MIC. Very little attention was paid then to the state of the UCIL site and immediate surroundings with respect to contaminants other than MIC which may have been present not only as a result of the accident, but also the routine operation of the plant. This remains the case today. As legal processes continue to try to establish liability and compensation following the 1984 disaster, responsibility for the contamination which remains on and around the site remains unaddressed. Given the nature of the processes at the plant, and the chemicals handled, it is possible that residents of the communities surrounding the former UCIL site may still be exposed to hazardous chemicals on a daily basis.

In order to gain an insight into the nature and severity of chemical contamination of the former UCIL site and its surroundings, samples of solid wastes, soils and groundwaters from within and surrounding the site were collected by Greenpeace International in May 1999. Samples were returned to the Greenpeace Research Laboratories, based in the University of Exeter, UK, for analysis of heavy metals and organic contaminants. Sludges and soils were collected at locations both within the plant boundary (adjacent to former formulation plant and waste disposal sites) and in an area to the north of the plant at which solar evaporation ponds (SEPs) were formerly located. Groundwater samples were collected from drinking water wells to the north and south of the former UCIL site in order to determine the extent of aquifer contamination with volatile organic compounds.

The results of this survey indicate general contamination of the site and immediate surroundings with chemicals arising either from routine processes during the operation of the plant, spillages and accidents, or continued and ongoing release of chemicals from materials which remain dumped or stored on site. Within this overall contamination,

some locations sampled indicated the presence of "hot-spots" of severe contamination with heavy metals and/or persistent organic pollutants.

1. Samples collected in the vicinity of the former Sevin (carbaryl) formulation plant contained elevated levels of mercury and/or organochlorine compounds. For example: -

> i) Sample IT9012, collected from a drain directly beneath the plant, contained free mercury at over 12% of the overall weight of the sample (between 20000 and 6 million times higher than might be expected as background). Chromium, copper, nickel and lead were also present at elevated levels. The toxic organochlorines hexachloroethane and hexachlorobutadiene (HCBD), common constituents in solid wastes arising from the chlorinated chemicals industry, were also found. HCBD is a potent kidney toxin. Although insufficient information exists to evaluate fully its carcinogenicity, the USEPA list HBCD as a possible human carcinogen.
>
> ii) Sample IT9013, collected from a ditch adjacent to the Sevin plant, contained a complex mixture of organochlorines, including several isomers of hexachlorocyclohexane (HCH also known as benzene hexachloride, BHC), numerous chlorinated benzenes and DDT. The presence of HCH isomers provides further confirmation of the formulation of Sevin/BHC pesticide mixtures, indicated by the presence of containers labelled as such still present on site. Similarly, the presence of chlorinated benzenes suggests their former use or manufacture on site, perhaps predating Sevin production. The reason for the appearance of DDT and metabolites remains unclear as there is no record of it being manufactured or used on site.

2. Samples of solid waste/soil collected from the south-east corner of the plant, the former location of acid waste neutralisation pits, revealed significant, though patchy, contamination. Sample IT9015 from this area also showed mercury levels elevated above background, although much lower than in IT9012. This sample also contained numerous organic contaminants, including 11 identifiable organochlorines with a similar profile (HCH, chlorinated benzenes, DDE) to the sludge sample from the ditch (IT9013). In addition, several other organochlorine compounds were detected which could not be fully identified.

3. Samples of soil collected from the location of the old solar evaporation ponds (SEPs) appeared less contaminated overall, although only a small proportion (as low as 20%) of the organic compounds isolated could be identified to any degree of reliability. This greatly limits any assessment of the nature and extent of contamination in these materials.

4. Volatile organochlorine compounds (VOCs), including chloroform (trichloromethane), carbon tetrachloride (tetrachloromethane) and chlorinated benzenes were detectable in groundwater collected from all three wells close to the northern boundary of the former UCIL plant. Lower, though still significantly elevated, levels were found in samples of groundwater accessed immediately to the south of the boundary and from a well in the

south-east corner of the site itself. No organochlorine contaminants were reported above detection limits in water drawn from wells further north, adjacent to the SEPs, or further south from the plant.

i) Samples IT9030 and IT9032, collected from wells adjacent to the northern plant boundary, contained highly elevated concentrations of carbon tetrachloride (ca 3.4 and 1.7 mg/l respectively) and chloroform (2.59 and 0.1 mg/l respectively). Both these compounds were used as solvents in the Sevin manufacturing process. As the wells sampled lie upstream from the flow of groundwater in this area, the presence of these contaminants probably reflects long-term contamination of the aquifer from routine use or spillages on site. Despite warning signs not to drink the water, these wells remain accessible and in continued use by the local residents.

ii) Chlorobenzenes were also detectable in these samples, IT9030 containing over 2.8 mg/l of 1,2-dichlorobenzene. Trichlorobenzenes, rarely reported in drinking water at levels in excess of 1 ug/l, were present at elevated levels in all three samples north of the boundary, as well as in the wells on and to the south of the boundary. Sample IT9030 again contained the highest concentrations, at approximately 180 ug/l.

iii) Of 10 VOCs found for which WHO guidelines have been established, 8 were present at concentrations above those limits in IT9030. In the case of carbon tetrachloride, concentrations in IT9030 were more than 1700 times above the WHO limit for drinking water.

5. In total, the survey conducted by Greenpeace International has demonstrated substantial and, in some locations, severe contamination of land and drinking water supplies with heavy metals and persistent organic contaminants both within and surrounding the former UCIL pesticide formulation plant. There is an urgent need for a more detailed and extensive survey if the full extent of ongoing contamination from the plant is to be determined.

6. It is also essential that steps are taken to reduce and, as far as possible, eliminate further exposure of communities surrounding the contaminated site to hazardous chemicals. Contaminated wastes and soils must be safely collected and securely contained, until such time as they can be effectively treated. Such treatment must entail the complete removal and isolation of toxic heavy metals from the materials, and complete destruction of all hazardous organic constituents. The treatment process selected for this purpose must operate in a closed loop configuration, such that there are no releases of the chemicals or their hazardous by-products to the environment.

7. For contaminated groundwater, the ultimate goal should be the remediation of the aquifers. This may be achieved, in part, by state of the art filtration technology which traps both volatile and semi-volatile organic contaminants, allowing their isolation, storage and treatment. In the short term, however, the priority, and responsibility of the

Government, must be to provide clean water to the communities and to prevent access to contaminated wells. Urgent action must also be taken to prevent further contamination of aquifers through proper containment of chemicals and contaminated materials both on and surrounding the site.

8. The financial and legal responsibility for the clean-up operation must be borne by the former and/or current owners of the former UCIL site and the Government of India.

Introduction

Between 1977 and 1984, Union Carbide India Limited (UCIL), located within a crowded working class neighbourhood in Bhopal, was licensed by the Madhya Pradesh Government to manufacture phosgene, monomethylamine (MMA), methylisocyanate (MIC) and the pesticide carbaryl, also known as Sevin (Behl *et al.* 1978, UCC 1985, Singh & Ghosh 1987).

Phosgene was manufactured by reacting chlorine, brought to the plant by tanker, and carbon monoxide, produced from petroleum coke and oxygen in an adjacent production facility within the plant (Behl *et al.* 1978, UCC 1985). The MMA, also brought in by tanker, was combined with the phosgene, in the presence of chloroform (used as a solvent throughout the process) to produce methyl carbamoyl chloride (MCC) and hydrogen chloride gas (HCl). HCl was then separated from the MCC so that it could be broken down into MIC and HCl. The MIC was then collected and transferred to stainless steel storage tanks, whilst the HCl, along with residues of MCC, chloroform, and other unwanted by-products (e.g. carbon tetrachloride, MMA, dimethylallophanoyl chloride, ammonium chloride, dimethyl urea, trimethylbiuret and cyanuric acid) were collected and recycled back through the process (Behl *et al.* 1978, UCC 1985).

MIC was manufactured primarily to make the pesticide carbaryl (Sevin) as well as smaller quantities of aldicarb (Temic) and butlyphenyl methylcarbamate, all destined for the Indian market (MacKenzie 1984). Carbaryl was produced by reacting MIC with a slight excess of alpha-naphthol, in the presence of carbon tetrachloride (NEERI 1990), and once made it was sold as the pesticide Sevin. However based on verbal information supplied by ex-workers, and the presence of sacks of hexachlorocyclohexane (lindane) next to the Sevin plant, it is possible that Sevin-lindane formulations were also being manufactured on site.

The reactions described above, once of interest only to the few, are now some of the most widely studied, scrutinised and publicised. The reason for the interest is that on the night of the 2-3 December 1984 one of the world's worst industrial disasters occurred at this Union Carbide plant. Water inadvertently entered the MIC storage tank (number 610), where over 40 metric tonnes of MIC were being stored. The addition of water to the tank caused a runaway chemical reaction, resulting in a rapid rise in pressure and temperature. The heat generated by the reaction, the presence of higher than normal concentrations of chloroform, and the presence of an iron catalyst (resulting from corrosion of the stainless steel tank wall) resulted in a reaction of such momentum, that gases formed could not be

contained by safety systems (UCC 1985). As a result, MIC and other reaction products, in liquid and vapour form, escaped from the plant into the surrounding areas. The effect on the people living in the shanty settlements just over the fence was immediate and devastating (UCC 1985, Gupta *et al.* 1988).

Many died in their beds, others staggered from their homes, blinded and choking, to die in the street. Many more died later after reaching hospitals and emergency aid centres (Gupta *et al.* 1988). The early acute effects were vomiting and burning sensations in the eyes, nose and throat, and most deaths have been attributed to respiratory failure. For some, the toxic gas caused such massive internal secretions that their lungs became clogged with fluids, while for others, spasmodic constriction of the bronchial tubes led to suffocation (ICMR 1985, Gupta *et al.* 1988). Many of those who survived the first day were found to have impaired lung function. However other follow-up studies on survivors have also reported neurological symptoms including headaches, disturbed balance, depression, fatigue and irritability. Abnormalities and damage to the gastrointestinal, musculoskeletal, reproductive and immunological systems were also frequently found (Gupta *et al.* 1988, Rastogi *et al.* 1988, Saxena *et al.* 1988, Bhandari *et al.* 1990, Cullinan *et al.* 1996, Cullinan *et al*, 1997). It is been estimated that at least 3000 people died as a result of this accident, while figures for the number of people injured currently range from 200,000 to 600,000, with an estimated 500,000 typically quoted (Kumar 1994, Kumar 1995, Sriramachari and Chandra 1997).

What followed the night of the 2-3 December 1984 were fierce controversies and legal battles regarding the cause of the accident, liability and compensation; controversies and battles that are still, in part, being fought today, fifteen years later. The conditions surrounding the accident were said to be exceptional, caused by unique and unusual events, However in April 1985, when chlorine gas leaked from the phosgene manufacturing plant into the streets of Bhopal, this conclusion of events was questioned (MacKenzie 1985a).

Hypothesis for the disaster included sabotage, prolonged bulk storage of over 40 tonnes of MIC, non-functioning refrigeration systems, the failure of safety measures (valves, flare towers and alarms) and the malfunctioning of neutralisation facilities (MacKenzie 1984, UCC 1985, MacKenzie 1985b, Singh and Ghosh 1987, Milne 1988, Sriramachari and Chandra 1997). Responsibility for the accident lay somewhere between the Union Carbide Corporation, the Indian operators UCIL, and the Indian Government. However as a result of the controversy and confusion, liability has never been fully accepted by any Party, and compensation has been awarded to only half of the estimated 500,000 victims (Kumar 1995).

The factory was closed down after the accident. The accident also led, as expected, to intensive experimental and epidemiological research into the toxicity of MIC and the tissue damage it could cause. Prior to the Bhopal accident, practically nothing was known, and therefore since 1984 numerous human health investigations and laboratory toxicity studies have been conducted.

However, amongst the controversies regarding blame and accountability, and the research into the toxicity of MIC, the fate of the redundant former UCIL site was largely overlooked. The UCC investigation team examined the site shortly after the accident, reporting its findings in March 1985. Some studies have involved collection of samples from the MIC storage tanks and identification of compounds present (D'Silva *et al.* 1986). However little else is known about the chemicals that still remain on the site. And whilst the importance of on-going studies into MIC toxicity and its effects on those exposed is not in question, the lack of investigation into the human health effects of the material that remains on site still needs to be addressed.

With this in mind, in May 1999, Greenpeace International, along with the Bhopal-based NGOs Bhopal Group for Information and Action and Bhopal Gas Peedit Mahila Udyog Sanghatana, carried out an investigation of the former UCIL site. Samples of soil were collected both from areas once used for waste disposal, and around the former Sevin plant, where a ruptured and leaking storage tank was found. Groundwater samples were also collected from a number of private wells located amongst the shanty settlements of Bhopal. The aim of the sampling program was to identify organic pollutants and heavy metal contaminants present in and around the former UCIL site. Results from this investigation are presented here.

Bhopal Sampling Program

A total of thirty-one samples were taken in and around the former Union Carbide site in Bhopal. Samples collected within the factory complex included five samples of soil/waste and two duplicate samples of groundwater (all groundwater samples were collected in duplicate). Outside the complex, two samples of soil were collected from an area once used as solar evaporation ponds, and twenty-two samples of groundwater were collected from eleven private wells (see Figure 1 for exact sampling locations, and Tables 1a and 1b for full sample descriptions).

General Sampling Procedures

Unless otherwise stated, all soil type samples were taken at a depth of 20-30cm below the surface, and all water samples were taken from hand-pumps. All samples were immediately sealed and cooled upon collection, and remained so until opened for immediate analysis.

Samples from within the factory complex

In order to investigate residual contamination resulting from the activities at the Union Carbide factory, a number of samples were taken from within the main factory complex.

A sediment sample was taken from a covered access hole, over what appeared to be a drainage pipe under the Sevin plant (IT9012). The drain was located approximately 10m from a large pile of brown waste which had leaked from a ruptured tank, and was reported by an ex-worker to be crude Sevin. The drain contained sediment to a depth of

approximately 20cm, and visible traces of metallic mercury could be observed in the drain sediment as well as in the immediate vicinity.

A further sample was taken from a ditch running alongside the former formulation plant, close to the output of a 25cm diameter pipe coming from within the plant (IT9013). A large number of sacks labelled as Sevin and Sevin/BHC mixture remain within this building. Several covered storage areas are located adjacent to the formulation plant, containing a number of highly corroded barrels labelled as 'Sevin residue'. The sample was taken to determine the level of local contamination with Sevin and BHC.

Prior to the installation of the solar evaporation ponds, an area in the south-east of the factory complex was used for dumping a large variety of both liquid and solid waste, including toluene, dichlorobenzylchloride and oxine waste from a pilot plant (based upon verbal knowledge from an ex-worker) Samples were taken from this area in order to determine the environmental contamination as a result of this dumping.

A sample was taken from a 30-40m diameter lime covered pit, where the waste had been dumped (IT9014). The area was covered in a layer of lime to a depth of approximately 10cm. The sample was taken from the soil below the layer of lime. In order to gauge the degree of leaching of chemicals from the dumping area, a further sample was taken from some waste ground adjacent to this site (IT9015). Run off from the old dumping area is reported to flow over this adjacent ground during the monsoon period. The soil in this area appeared to have a high lime content, though it is unclear as to whether this was due to run off from the dumping area, or had been deliberately added.

A capped, though not sealed, bore well located approximately 150m west of the old dumping area was also sampled in order to give an indication of the contamination of the ground water in this area (IT9040/41). The sample was taken at a depth of 4.4m below ground level, just below the surface of the ground water.

Prior to the closure of the site, an incinerator was located close to this dumping ground. The incinerator has now been completely removed, and the area is currently waste ground. A composite sample of soil and ash was taken from this area to determine the legacy of the incinerator in this area (IT9016).

Solar Evaporation Ponds (SEPs)

Located approximately 400m to the north of the factory site, on the opposite side of the railway line, is an area that was previously used as solar evaporation ponds. Waste from the factory was dumped in this area, enabling the water and other volatile material in the waste to evaporate. Approximately 3 years ago the site was partially cleaned up. The residual waste from the area previously used as SEP 1 and 2 was cleared and added to the original site of SEP 3, located in the northwest corner of the SEP site. Based upon verbal communications, this area is lined and capped. According to members of the local community, attempts to grow crops on the cleared area, using local ground water for irrigation, have resulted in failure of the crops.

In order to determine the effectiveness of the previous cleanup of the old SEP site, two samples were taken from this area. A sample was taken from the cleared area within the old SEP site (IT9042). The sample was taken from the lowest lying part of this area, where a pond forms in times of rain. A further sample was taken from the sloping eastern edge of the capped landfill on the site of the old SEP 3, at a point where coloured waste could be seen to be leaching from the capped area (IT9017).

Drinking water and ground water

Twelve water samples were taken. The sample descriptions are given in Table 1B.

A previous official report in 1996 indicated that samples of ground water taken from close to the factory site had high levels of chemical oxygen demand (COD), indicating contamination with oxidisable material, probably organic chemical contamination. No details of the individual chemical contaminants were given. Ground water samples from this area were taken in order to give a clearer picture of the contamination resulting from the activities of Union Carbide.

In the area of the factory site, the ground water is reported to flow in a northeasterly direction (NEERI 1990). In light of this, three sets of samples (IT9030-35) were taken from close to the northeastern wall of the factory complex. These samples were taken to give an indication of the movement of contamination from the site to the adjacent ground water, which is used by the local community for drinking. As noted in the table below, these samples were taken from varying depths. Samples IT9030/31 were taken from a hand-pump that is in regular use despite a sign that was translated by a local as ‘do not use for drinking’

Two further sets of samples were taken slightly further from the main site, to the north of the factory between the main site and the area previously used for the SEPs (IT9022/23 and IT9028/29).

Two more paired samples were taken from an area close to the southern boundary of the factory site (IT 9018/19 and IT9020/21). The wind direction at the time of the MIC gas leak had been in a south-westerly direction (Gupta *et al. 1*988), while the ground water in this area flows in a north-easterly direction (NEERI, 1990). These samples were taken to give an indication of the movement of contamination against the direction of ground water flow, and to determine whether there was any remaining contamination associated with the MIC gas leak. Samples IT9020/21 were taken from a currently unused open well.

In order to determine the quality of the ground water in the general vicinity, two further sets of samples were taken from locations over 1km south of the factory site (IT9036/37 and 9038/39). These samples were also taken to give an indication as to whether any contamination from the factory site had spread more widely, and whether contamination associated with the MIC gas leak remained in the groundwater in this area.

Sample Number	Sample Description
IT9012	Sediment collected from a drain under the former Sevin plant
IT9013	Soil collected from a ditch running alongside the former UCIL formulation plant
IT9014	Soil / lime collected from a lime covered pit, used as a dumpsite prior to the construction of the Solar Evaporation Ponds (SEPs)
IT9015	Soil / lime collected close to the lime covered waste pit
IT9016	Soil / ash collected from the old incinerator area
IT9017	Soil collected from Solar Evaporation Pond (3)
IT9042	Soil collected from an old Solar Evaporation Pond, cleared in 1996

Table 1a Descriptions of solid samples collected around the former Union Carbide (UCIL) site, Bhopal, India 1999

Sample Number	Sample Description	Depth of well (metres)
IT9018	Drinking water collected from a hand-pumped well, J.P. Nagar	Unknown
IT9020	Groundwater collected from an unused open well, J.P. Nagar	10*
IT9022	Drinking water collected from a hand pumped well, Nawab Colony	Unknown
IT9024	Drinking water collected from a hand-pumped well, Shiv Shakti Nagar	37*
IT9026	Drinking water collected from a hand-pumped well, Shiv Shakti, Nagar	24*
IT9028	Drinking water collected from a hand-pumped well, Blue Moon Colony	65-80*
IT9030	Drinking water collected from a hand-pumped wel, Atal Ayub Nagar	9*
IT9032	Drinking water collected from a hand-pumped well, Atal Ayub Nagar	46*
IT9035	Drinking water collected from a hand-pumped well, Atal Ayub Nagar	24*
IT9036	Drinking water collected from a tap, Pirgate, Curfew-Vali-Mata, Mandir	61*
IT9038	Drinking water collected from a hand-pumped well, Railway Colony	Unknown
IT9040	Groundwater collected from a bore-hole within the former Union Carbide (UCIL) site	4.4

Table1b Descriptions of water samples collected from private wells in the vicinity of the former Union Carbide (UCIL) site, Bhopal, India 1999. *Note: * - depth estimated during sampling, or obtained from the local population using the wells.*

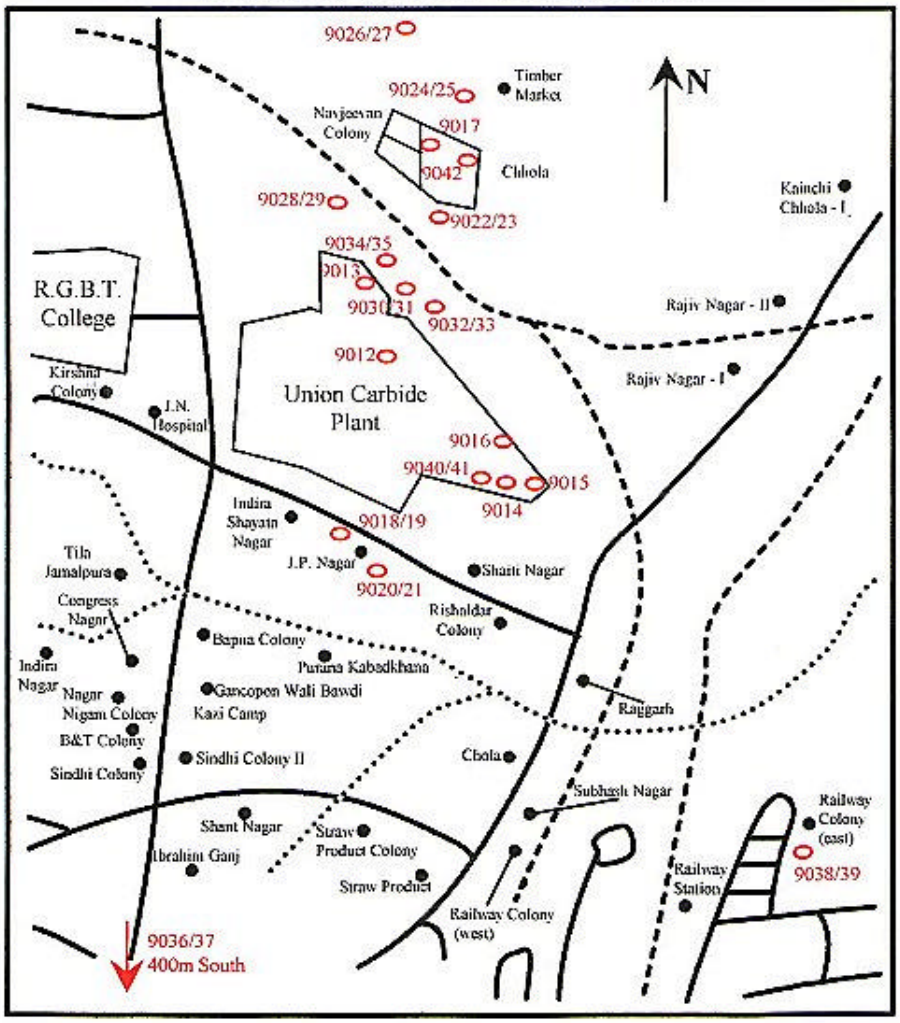
Materials and Methods

See Appendix 1 for details of sample collection, preparation, and analytical methodologies.

Results and discussion

Results of the heavy metal analysis are given in Table 2. Background information on the common sources, environmental behaviour and toxicological properties of these metals can be found in Appendix 4. Possible sources of the metals in these samples are discussed below.

FIG 1 LOCATION OF SAMPLES COLLECTED IN THE VICINITY OF U.C.I.L. PLANT, BHOPAL, INDIA 1999



Approximate Scale
1cm = 136m
1000m (1Km)

KEY
Road
Railway
River

The results of the organoscreen analysis i.e. the groups of organic compounds reliably identified in soil and mixed soil/solid waste samples are presented in Table 3-4. Table 5 of all compounds reliably and tentatively identified is given in Appendix 2. Quantitative results from the volatile organic compounds analysis of the well water samples are given in Table 6. Background information, on the common sources, environmental behaviour and toxicological properties of the organic contaminants found, is given in Appendix 3. Possible sources of these contaminants in these samples are discussed below.

Sample Number	Cd mg/kg	Cr mg/kg	Co mg/kg	Cu mg/kg	Hg mg/kg	Mn mg/kg	Ni mg/kg	Pb mg/kg	Zn mg/kg
IT9012	n/d	480.7	14.9	287.7	128000	1275.4	174.6	174.6	288.6
IT9013	n/d	85.7	21.4	37.8	2.6	920.4	58.2	19.4	71.4
IT9014	n/d	73.0	20.0	42.0	0.8	643.0	54.0	11.0	66.0
IT9015	n/d	52.9	11.8	49.0	8.1	438.2	34.3	15.7	239.2
IT9016	1.0	520.8	8.3	108.3	1.4	526.0	94.8	406.3	426.0
IT9017	n/d	73.0	24.0	40.0	1.1	1136.0	58.0	17.0	59.0
IT9042	n/d	35.5	12.7	17.3	0.4	759.1	29.1	4.6	30.9

Table 2 Results of heavy metal analysis, former Union Carbide (UCIL) site, Bhopal, India 1999

Sample Code	Compounds Isolated	Reliably Identified	Halogenated Compounds	PAHs	Phenolic compounds	Other Aromatics	Aliphatics
IT9012	73	36(49%)	2	12	1	7	12
IT9013	33	21(64%)	14	1	2	0	4
IT9014	45	14(31%)	0	0	2	5	7
IT9015	59	24(41%)	11	1	0	0	12
IT9016	25	12(48%)	1	0	0	1	10
IT9017	15	4(27%)	1	0	0	0	3
IT9042	15	3(20%)	1	0	0	0	2

Table 3 Results of organic screening analysis, former Union Carbide (UCIL) site, Bhopal, India, 1999

Table 2 also shows that IT9012 and IT9016 (collected from the former incinerator site) contained significant levels of chromium, copper, lead, nickel and zinc. Typical background soil concentrations of these metals are quoted as ranging from 50-100 mg/kg, although natural elevations above this range can be found (Alloway 1990). However if the concentrations found in samples IT9013, IT9014, IT9015, IT9017 and IT9042 are taken as being indicative of background levels, elevations of these metals in these two samples are clearly evident.

Former Sevin structure plant

Sample IT9012, a mixture of soil and sludge, was collected from a drain running under the former Sevin plant. Sevin was manufactured at this plant from MIC and alpha-naphthol, in the presence of carbon tetrachloride and an activated carbon catalyst (NEERI 1990). In addition, it is possible that Sevin-lindane formulations were also being produced. During the investigation by Greenpeace, bags of hexachlorocyclohexane (BHC, lindane) were observed next to the plant. It was also observed that, due to

Groups of compounds reliably identified	Number of samples	Sample codes
ORGANOHALOGEN COMPOUNDS		
DDT metabolites	2	IT9013, IT9015
Hexachlorocyclohexanes	2	IT9013, IT9015
Pentachlorocyclohexenes	1	IT9013
Hexachlorobutadiene	1	IT9012
Hexachloroethane	1	IT9012
Dichlorobenzenes	5	IT9013, IT9015, IT9016, IT9017, IT9042
Trichlorobenzenes	2	IT9013, IT9015
Tetrachlorobenzenes	2	IT9013, IT9015
Pentachlorobenzene	2	IT9013, IT9015
Hexachlorobenzene	1	IT9015
Chlorinated toluenes	1	IT9015
Chlorinated pyridines	1	IT9013
POLYCYCLIC AROMATIC HYDROCARBONS		
Naphthalene and its derivatives	3	IT9012, IT9013, IT9015
Phenanthrene and its derivatives	1	IT9012
PHENOLIC COMPOUNDS	3	IT9012, IT9013, IT9014
OTHER AROMATICS		
Alkylated benzene derivatives	2	IT9014, IT9016
Dibenzothiophenes and its derivatives	1	IT9012
1,1'-Biphenyl	1	IT9012
ALIPHATIC HYDROCARBONS	7	IT9012, IT9013, IT9014, IT9015, IT9016, IT9017, IT9042

Table 4 Groups of organic compounds reliably identified in soil and mixed soil/solid waste samples collected in the vicinity of former Union Carbide (UCIL) site, Bhopal, India, 1999

corrosion, a storage tank once used to contain the manufactured Sevin was ruptured and leaking. The contents, along with runoff from other areas of the plant, were being carried by rainwater into this drain.

Table 2 shows the results of the heavy metal analysis carried out on the soil samples collected from the former UCIL site (see Figure 1 and Tables 1a and 1b for exact locations and descriptions). The results show that elevated levels of mercury were found in most samples, with an extraordinarily high 127.9g/kg (12.79%) of mercury found in sample IT9012, a sample of soil and sludge collected from a drain beneath the former Sevin plant. Typical background levels of mercury in soils are quoted as ranging from 0.02-0.6 mg/kg (Alloway 1990, WHO 1989). Depending on which value is used for reference, mercury levels in sample IT9012 therefore exceed background concentrations by approximately 20,000 to 6,000,000 times.

The level of mercury detected in this sample is indicative of gross contamination, with

chromium, copper, lead, nickel and zinc all present at concentrations far higher than those quoted as background levels (Alloway 1990). It is understood, based on verbal information supplied by ex-workers, that the mercury was used around the plant as a sealant, and elemental mercury is readily visible at points throughout the site. However written sources confirming this application have not been found.

High levels of chromium and nickel are thought to result from the fact that most of the processing equipment and storage facilities on site were made of stainless steel or alloys of nickel (e.g. Inconel) (UCC 1985), and some were dosed with potassium dichromate to prevent corrosion. The Union Carbide investigation team also found levels of chromium and nickel salts in the MIC storage tanks, as did later studies conducted by other Union Carbide scientists (UCC 1985, D'Silva *et al.* 1986).

High levels of copper, zinc and lead in sample IT9012 could also result from widespread usage and corrosion. For example, copper is an excellent electrical conductor and therefore would have been used in electrical cables and wires (ATSDR 1997). Lead may have been used in pipe-work for water distribution or in containers used for storing corrosive liquids (e.g. acids). Its alloys may also have been used for welding (ATSDR 1997). Any galvanised steel around the plant would contain zinc, as would any die-casting alloys used (ATSDR 1997).

A total of 73 organic compounds were isolated from this sample; 49% of those were reliably identified. The groups of compounds, including linear alkanes, polycyclic aromatic hydrocarbons (PAHs), and dibenzothiphenes which were found in this sample, could be associated with crude oil or petroleum pollution (Overton 1994). Among those, PAHs are the most toxic and persistent. Once PAHs are released into environment, degradation by micro-organisms is often slow, leading to their accumulation in exposed sediments, soils, aquatic and terrestrial plants, fish and invertebrates. PAHs can have a deleterious effect on human health. People exposed to mixtures of PAHs, through inhalation or skin contact, for long periods of time, have been shown to develop cancer (ATSDR 1997).

1,1'-Biphenyl has been also identified in this sample. In the past biphenyls have been used as a heat transfer fluid (Edwards *et al.* 1991, Budavari *et al*. 1989). According to the UCIL Operating Manual, Dowtherm (a mixture of diphenyl and diphenyl oxide) was used at the plant as a heat transfer fluid in the phosgene and monomethylamine pre-heaters and was also used in the reactivation heater (Behl *et al.* 1978).

Two organochlorine compounds, hexachlorobutadiene and hexachloroethane, were found in this sample. Both may be formed as by-products in a range of industrial processes which involve chlorination (Snedecor 1993, DHHS 1998, US EPA 1986). Hexachloroethane is also formed during incineration of materials containing chlorinated hydrocarbons (ATSDR 1996). Hexachlorobutadiene may enter the environment principally through the disposal of wastes containing hexachlorobutadiene from the chlorinated hydrocarbon industries (ATSDR 1997). If released to soil, both hexachloroethane and hexachlorobutadiene may persist for many months or even years

under aerobic conditions (Howard *et al.* 1991). Persistence is substantially greater under more anaerobic conditions. Both compounds are toxic to humans and may cause damage to animals, birds, fish, and plants (ATSDR 1996 & 1997).

Former UCIL formulation plant

Soil sample IT9013 was collected from a ditch running alongside the former UCIL formulation plant. This sample contained slightly elevated levels of chromium, mercury and nickel. Possible sources of these metals have been described above.

33 organic compounds were isolated from this sample. 64% of these compounds have been reliably identified, the majority (14 compounds out of 21) being organochlorines: four isomers (alpha-, beta-, gamma- and delta-) of hexachlorocyclohexane (HCH) and one isomer of pentachlorocyclohexene; seven chlorinated benzenes (from di- till pentachlorosubstituted); one metabolite of DDT (p,p'-DDD); and tetrachlorinated pyridine.

The presence of some of organochlorine contaminants in the sample undoubtedly results from past manufacture and/or formulation of these or relative compounds by UCIL. Oral information from one of the former workers of UCIL suggested that dichlorobenzenes were manufactured on the site before production of Sevin began. This may also explain the presence of the higher chlorinated benzenes (Bryant 1993).

Hexachlorocyclohexanes, chlorinated benzenes and DDT metabolites are known to persist for a long time in the environment (Howard *et al.* 1991). Therefore these contaminants would be detectable for years after initial introduction into environment. DDT itself was not detected in the sample, but it is known to undergo metabolic conversion and dehydrochlorination to form DDD and DDE metabolites (ATSDR 1997). Both DDD and DDE are more persistent in the environment than DDT; their half-life in soil is more than 15 years even under aerobic biodegradation conditions (Howard *et al.* 1991). In the current study groundwater samples from wells located in and around the former UCIL plant were not analysed for DDT or HCH. The presence of these contaminants was reported by Dikshith and co-workers in 1990 following analysis of water samples collected from wells, hand-pumps and ponds around Bhopal (Dikshith *et al.* 1990). It was reported that three isomers of HCH (alpha-, beta- and gamma-), along with p,p'-DDT, o,p'-DDT, p,p'-DDD and p,p'-DDE have been detected in almost all sixty water samples investigated. Water samples from wells showed the lowest residual content of total HCH (mean concentration 4.654ppm) and total DDT (mean concentration 5.794ppm) followed by water from hand-pumps and ponds. Mean concentration of total HCH and total DDT found in ponds was 9.941ppm and 16.059ppm respectively.

Additionally, a phenolic compound 2,6-bis(1,1-methylethyl)-4-methylphenol, known as butylated hydroxytoluene (BHT), was reliably identified in the sample. The reason for appearance of this compound in the sample is unclear. However, 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). It is known that the UCIL plant

manufactured carbamate insecticides in the past (Lambrech & Charleston 1959) and it is possible that BHT contamination of the soil resulted from past production. 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).

Former UCIL neutralisation pit and incinerator

Two samples (IT9014 and IT9015), mixtures of soil and lime, were collected from an area in the south-east of the factory complex that (according to an ex-plant worker) was used for dumping and neutralisation of a complex mixture of wastes, prior to the introduction of the solar evaporation ponds and the production of MIC. Sample IT9014 was taken from a 30-40m diameter lime covered pit where the waste was reported to have been dumped, while sample IT9015 was taken from adjacent waste ground over which waste from this area is reported, by an ex-worker, to overflow during the monsoon period. It has been reported (Behl *et al.* 1978, NEERI 1990) that acidic wastewater from MIC production, along with sewage and other process wastes, were collected in a neutralisation pit. The location of this pit is not given, and so it is not clear whether the pit they described is the one from which these samples were collected.

Soil sample IT9016 was collected from an area of flattened ground, once the site of the incinerator. The incinerator was used to process waste oil that had been skimmed from the non-acidic process waste (i.e. sewage, floor and equipment washings) in specially designed skimmer pits located within the plant (NEERI 1990). Based on information supplied by ex-workers, it was removed from the site eighteen months after the accident, however it was still used in this interim time to incinerate large quantities of remaining chemicals.

No elevated levels of heavy metals were detected in sample IT9014. However high levels of mercury were found in sample IT9015. As described above, elemental mercury was observed to be distributed widely throughout the site. It is not unsurprising therefore that elevated soil levels of mercury were found at some sampling points.

High levels of chromium, lead and zinc were found in this sample, along with elevated concentrations of copper, nickel, mercury and cadmium. Possible sources of these metals are described above. However based on information supplied by ex-workers, it is possible that bags of potassium dichromate were incinerated here after the accident, therefore explaining the high levels of chromium found in this sample. In addition, as many of these metals can also be present as impurities in crude oil (Kennicutt *et al.* 1996), it is probable that the residues from past incineration still remain in this area.

45 organic compounds were isolated from the sample IT9014, 59 from sample IT9015, and 25 from the sample IT9016. Among those, 31%, 41% and 48% of compounds respectively were reliably identified. The groups of compounds detected in the samples from the former dumpsite differ significantly. Sample IT9014 contained a range of

alkylated benzenes together with linear hydrocarbons (alkanes and alkenes) and two phenolic compounds including BHT. The presence of alkyl benzenes, alkanes and alkenes in the sample could be associated with petroleum contamination (Overton 1994). Again, BHT and its derivative bis(1,1-dimethylethyl)phenol may be present in the soil or wastes as a result of carbamate herbicide degradation, although other potential sources cannot be ruled out.

In contrast, sample IT9015 contained a wide range of organochlorine compounds, including the DDT metabolite p,p'-DDE, the alpha-isomer of hexachlorocyclohexane, eight chlorinated benzenes and one chlorinated toluene. Alkanes, alkenes and naphthalene have also been detected. Furthermore, there were several organohalogen compounds in the sample IT9015 with a relatively high abundance, which could not be reliably or tentatively identified due to unsatisfactory match with existing library.

The difference in the chemical composition of the samples IT9014 and IT9015 could be explained as a result of uneven spread of the contaminants or due to migration processes which have been going for years on this site. What is more interesting is that contaminants which were detected in the sample IT9015 match quite well with those detected in the soil sample IT9013 collected from the ditch near the former UCIL formulation plant. It is possible that the former dumpsite located on the territory of UCIL plant was receiving wastes from its formulating plant and that the area is still polluted with toxic and persistent chemicals as a result.

Soil sample IT9016 contained several linear hydrocarbons and one chlorinated benzene – 1,4-dichlorobenzene. The presence of 1,4-dichlorobenzene in the former UCIL incinerator site could be due to past contamination resulting from activities associated with the incinerator. 1,4-Dichlorobenzene is not usually easily broken down by soil organisms. Plants are thought to take up and retain 1,4-dichlorobenzene, and fish have also been shown to take up and retain this compound (ATSDR 1997).

Former Solar Evaporation Ponds

Once waste from the neutralisation had been treated and mixed, it was pumped to solar evaporation ponds (SEPs) located approximately 400 metres north of the main former UCIL site (Behl *et al.* 1978, NEERI 1990). Soil samples IT9017 and IT9042 were collected from this area.

Sample IT9017 appeared to be the more contaminated by heavy metals of the two samples. Nevertheless, with the exception of mercury (present at 1.1 mg/kg), all concentrations are within background ranges (Alloway 1990).

The organic analysis of these two samples showed very similar contaminant composition. In both samples, 15 compounds have been isolated and only 27% and 20% of those respectively have been reliably identified. The low percentage of reliably identified compounds in these samples is due to the relatively low levels of detected compounds. Only a few linear hydrocarbons and 1,4-dichlorobenzene were identified at high degree

of reliability. These compounds have been determined in almost all samples considered in this study, and their presence in the former SEPs is, therefore, unsurprising. However, these samples were collected after extensive reconstruction and soil redistribution inside the SEPs, which resulted in all surface soil being compressed into one north-east part of the SEPs. Thus, these two samples possibly do not reflect the overall potential contamination of this site; this issue would require further investigation.

Well water analysis for volatile organic compounds (VOC)

Twelve samples of water were collected from the wells in the vicinity of the former Union Carbide site, Bhopal, India. Sample descriptions are presented in Table 1b. Location of the sampling points is presented in Fig.1.

Compound	MDL* ug/l	Concentration, ug/l				
		IT9018	IT9030	IT9032	IT9035	IT9040
Chloroform	10	200	2590	100	160	50
Carbon tetrachloride	10	50	3410	1730	200	<10
Trichloroethene	5	<5	250	<5	<5	<5
Tetrachloroethene	5	20	45	20	15	<5
Hexachloroethane	5	<5	85	<5	15	<5
Chlorobenzene	5	25	56	<5	<5	<5
1,3-Dichlorobenzene	5	15	205	25	10	10
1,4-Dichlorobenzene	5	25	865	10	15	25
1,2-Dichlorobenzene	5	50	2875	20	35	60
1,3,5-Trichlorobenzene	5	<5	<5	<5	15	<5
1,2,4-Trichlorobenzene	5	15	145	25	15	15
1,2,3-Trichlorobenzene	5	10	35	20	15	<5

Table 6 Concentration of volatile organochlorine compounds found in well water samples IT9018, IT9030, IT9032, IT9035 and IT9040 in the vicinity of the former Union Carbide (UCIL) site, Bhopal, India 1999. * MDL – minimum detectable level

These samples were collected both from the area affected by MIC gas during the accident in 1984 and in the vicinity of the former Union Carbide Solar Evaporation Ponds (SEPs). The results of the quantitative analysis for VOC are presented in Table 6, Fig.2 and Fig.3. VOC were detected only in five samples, from wells which were either located on the territory of the plant (sample IT9040) or within a distance of about 50m to the northeast of the former UCIL plant territory (samples IT9030, IT9032 and IT9035). One sample (IT9018) that was collected from the well located to the south of the plant, but closer to its boundary than sample IT9020, also showed the presence of VOC.

Seven samples did not contain detectable levels of (VOC). Three of these samples (IT9022, IT9024 and IT9026) were collected from the wells around SEPs, another three samples were collected from wells located to the south of the plant (IT9020, IT9036 and IT9038) and one sample (IT9028) from the well situated approximately 200m to the north of the plant.

The three wells (samples IT9030, IT9032 and IT9035) in which chlorinated contaminants were detected at the highest concentration are located to the northeast of the former UCIL plant. According to the assessment of pollution from the Solar Evaporation Ponds (NEERI, 1990), the groundwater flows in this direction. Two wells located to the south (sample IT9018) and southeast (sample IT9040) of the plant contain chlorinated compounds at lower concentrations (except chloroform in sample IT9018) than wells located to the northeast of the plant.

Among these five samples the most polluted was sample IT9030, which contained the highest concentration of each of the organochlorine compounds determined except 1,3,5-trichlorobenzene. Sample IT9030 was collected from the hand-pumped well in the immediate vicinity of the plant site, near the former UCIL formulation plant. The depth of this well is not great (approximately 9 meters). There is a sign " Water unfit for consumption" on this well, but water from the well is still used for drinking.

Carbon tetrachloride and chloroform were used in the Union Carbide plant as solvents during the synthesis of Sevin pesticide and synthesis of methyl isocyanate (MIC) respectively (Behl *et al.* 1978, NEERI, 1990). These compounds have been found as contaminants in the glue-like solid by-products that were formed during methyl isocyanate synthesis. While the plant was in operation, solid by-products, termed GLIT, were continuously purged to the hydrochloric acid absorber and then removed to the lime pit (dumpsite) located in the plant territory (Behl *et al.* 1978). Other contaminants of GLIT include methyl isocyanate and its trimer, hydrochloric acid and dimethylallophanoyl chloride, monomethyl amine hydrochloride, ammonium chloride, dimethylallophanoyl chloride, cyanuric acid and dimethyl urea (Behl *et al.* 1978). There is no information on whole chemical composition of GLIT. Nevertheless, it is clearly a highly contaminated waste.

Chlorinated benzenes were not employed in the processes used at the UCIL plant. However, the plant had a storage place for dichlorobenzenes in the northern part of its territory (Behl *et al.* 1978). As was mentioned previously, the source of chlorinated benzenes isomers on the territory of former UCIL plant could be due to past manufacture of dichlorobenzenes on a pilot plant scale before production of Sevin began. Again, information about pilot plant for dichlorobenzene production was received orally from one of the former UCIL workers. Additionally, 1,2,3- and 1,2,4-trichlorobenzene may have been produced from the dehydrohalogenation of the unwanted isomers of the production of the pesticide 1,2,3,4,5,6-hexachlorocyclohexane. As was discussed above, the isomers of various isomers of hexachlorocyclohexane were detected in the soil and soil/solid waste samples IT9013 and IT9015.

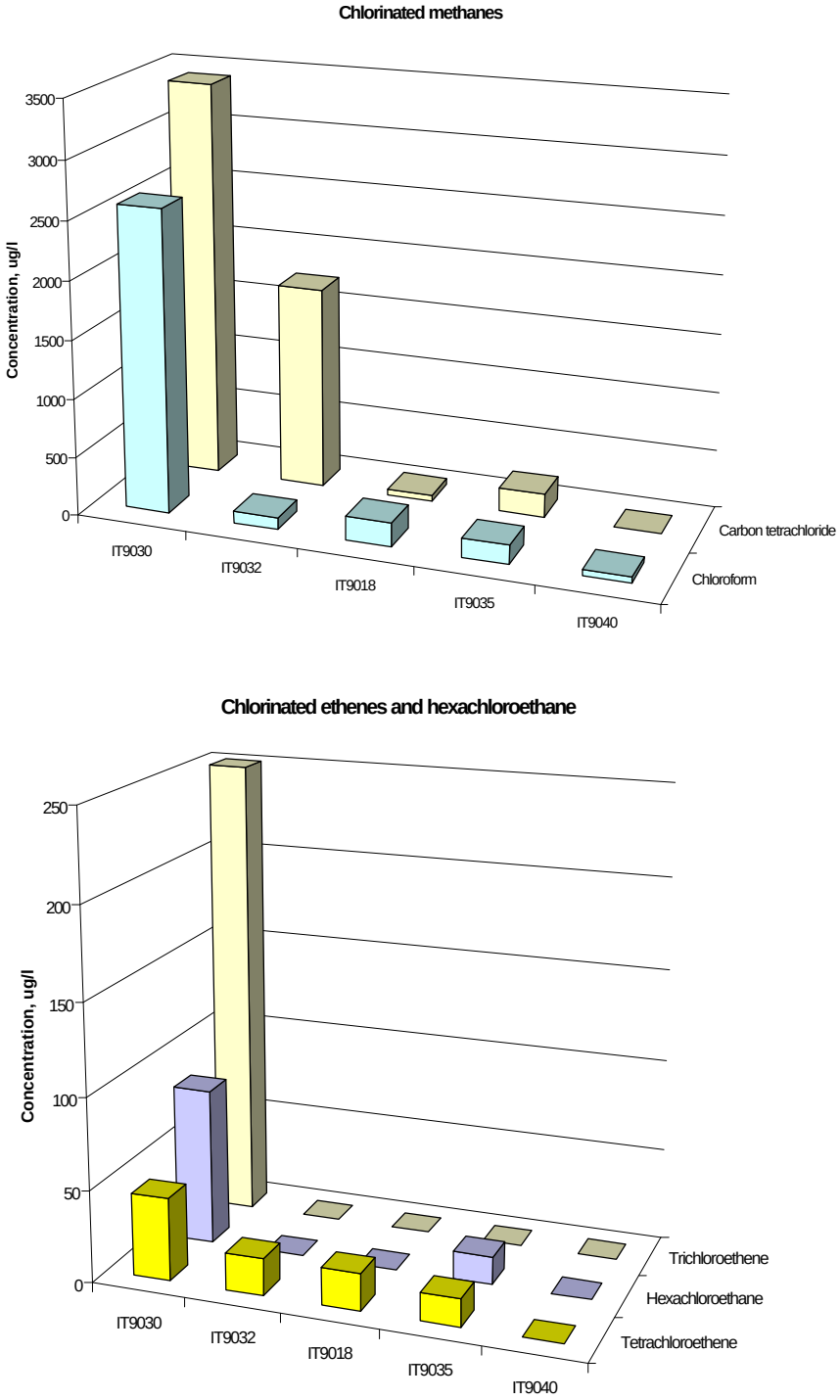


Fig.2 Levels of chlorinated methanes, ethenes and hexachloroethane in well water samples collected in the vicinity of the UCIL plant, Bhopal, India, 1999

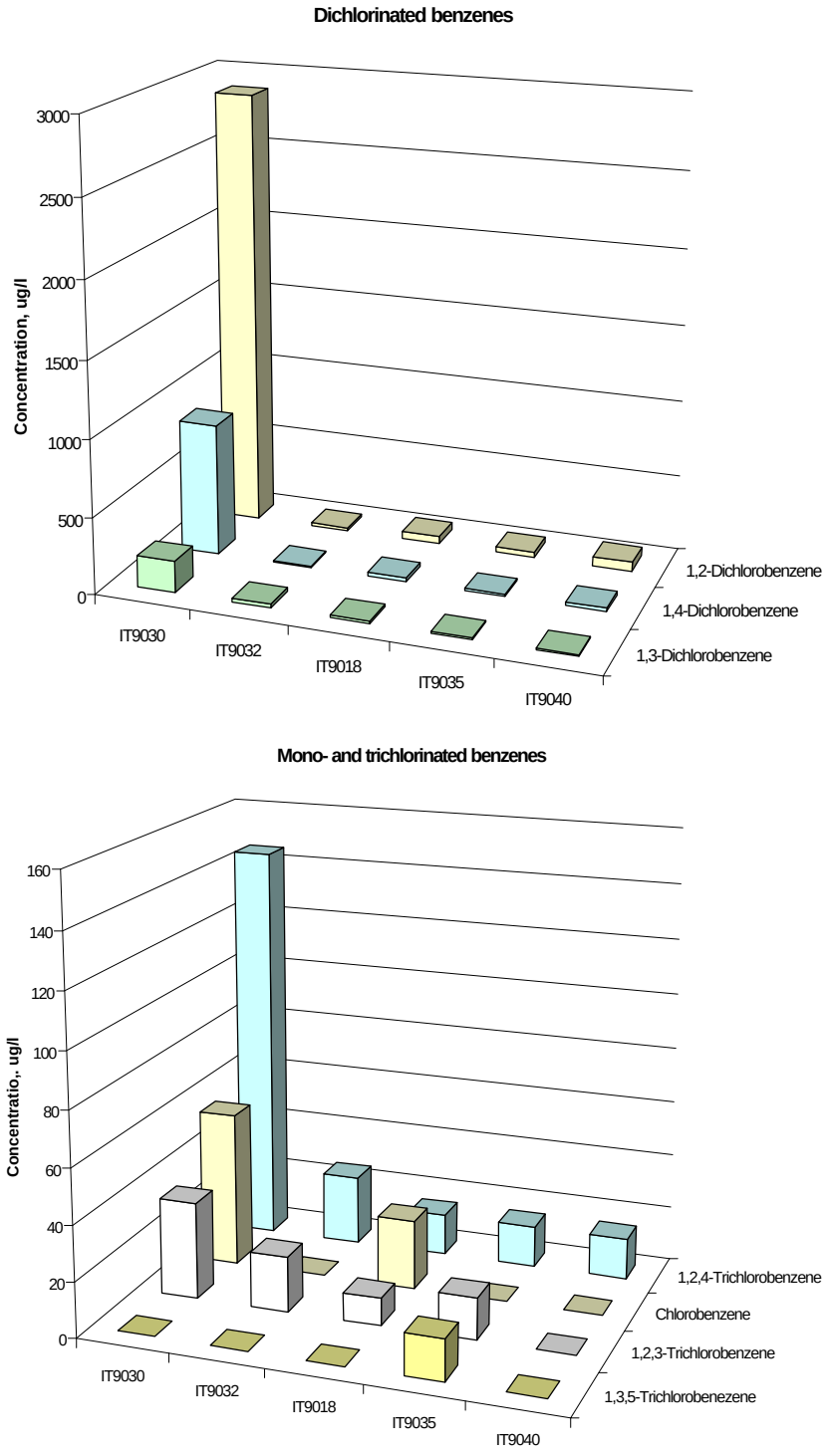


Fig.3. Levels of chlorinated benzenes in well water samples collected in the vicinity of the UCIL plant, Bhopal, India, 1999

Chemical wastes, dump leachates and direct manufacturing effluents have been reported to be the major source of environment pollution by chlorinated benzenes (Howard 1989). Dichlorobenzenes can be moderately to tightly absorbed to particles when released to soil. Nevertheless, they have also been detected in various groundwaters around hazardous waste disposal areas indicating that these contaminants are able to leach (Howard 1989). Trichlorobenzenes have also been found in drinking water but rarely above 1ug/l (WHO 1993).

The occurrence of organochlorine compounds in the groundwater in the vicinity of the plant may be due to a spillage onto the soil or to a leakage from the dump sites which were located at the plant territory before the Solar Evaporating Ponds were constructed (NEERI, 1990). There is no information that chlorinated ethenes and ethanes have been used on the plant, but these compounds (for example hexachloroethane) are known to be an impurity in some chlorinated solvents, or may be formed as a by-product in some chlorination processes (DHHS 1998). Additionally, these compounds could be formed in groundwater contaminated with various other chlorinated solvents under anaerobic conditions (Hashsham *et al.* 1995, Loran & Olsen 1999, Butler & Hayes 1998, Miller *et al.* 1998).

Pollution caused by chlorinated solvents can persist for a long time. For example, carbon tetrachloride is relatively stable in the environment and, if released to land, does not sorb onto soil, but migrates readily to groundwater and can remain in groundwater for months to years (US EPA 1988). Because chlorinated solvents have a density greater than water (CRC 1969), groundwater plumes of these contaminants may form pools of residual solvent below the water table (Rivett *et al.* 1994). Chlorinated solvents may undergo reductive dechlorination under anaerobic conditions, though it has been reported that final transformation into methane and ethane is significantly retarded if several of these compounds are present together (Hughes & Parkin 1996a, Hughes & Parkin 1996b).

Compound	**WHO guidelines, ug/l (WHO 1993)**	**US EPA standards, ug/l (US EPA 1999)**
Chloroform (or total trihalomethanes*)	200	100*
Carbon tetrachloride	2	5
Trichloroethene	70	5
Tetrachloroethene	40	5
Benzene, chloro-	300	100
Benzene, 1,2-dichloro-	1000	600
Benzene, 1,4-dichloro-	300	75
Benzene, 1,2,3-trichloro-	20	-
Benzene, 1,2,4-trichloro-	20	70
Benzene. 1,3,5-trichloro-	20	-

Table 7. Drinking water standards/guidelines for some chloroorganic compounds.

Contamination of the groundwater by chlorinated solvents is a world-wide problem and

occur in most cases in the vicinity of the industrial sites where these compounds are involved in the technological processes. In the investigation of groundwater pollution in the Coventry region (UK), high concentrations of chlorinated solvents have been found inside and outside of the main industrial areas (Lerner *et al.* 1993). Trichloroethene was the most ubiquitous pollutant with a maximum detected concentration of 6000ug/l. Contaminants detected at lower concentrations were 1,1,1-trichloroethane, chloroform, carbon tetrachloride and tetrachloroethene. Chlorinated solvents could dissolve into groundwater from the existing immiscible phase and move with the general flow of groundwater. Modelling studies suggest that individual plumes may extend for several kilometres (Burston *et al.* 1993). The ages of such plumes are unknown, but they may be as old as 55 years.

Comparison of the levels of organochlorine compounds reported in our study with the drinking water standards/guidelines (see Table 7) showed that well water in the study area was not suitable for drinking due to the high level of contamination. In sample IT9030, concentrations of the following compounds exceeded limits set by the World Health Organisation (WHO 1993) and US Environmental Protection Agency (US EPA 1999) for drinking water: carbon tetrachloride (by 1705 and 682 times respectively), chloroform (by 13 and 260 times respectively), trichloroethene (by 3 and 50 times respectively), tetrachloroethene (by 9 times, US EPA only), 1,4-dichlorobenzene (by 3 and 11 times respectively), 1,2-dichlorobenzene (by 3 and 5 times respectively), 1,2,4-trichlorobenzene (by 7 and 2 times respectively), and 1,2,3-trichlorobenzene (by about 2 times, WHO only). Carbon tetrachloride was the only compound in sample IT9032 which exceeded levels set by both regulations – by 865 and 346 times respectively. Two samples IT9018 and IT9035 contained three chloroorganic compounds (carbon tetrachloride, chloroform and tetrachloroethene) each at levels exceeding limits mention above. Only one sample (IT9040) had levels of detected VOC below these limits; water from this well is not used for drinking.

The presence of chlorinated methanes, ethenes, ethanes and benzenes in the well waters near former UCIL plant is undoubtedly due to the long-term industrial contamination of surrounding environment from this plant. Consumption of water, contaminated by chemicals that have been found in this study, for long periods could cause significant health damage. Further information on toxicity, common sources and environmental behaviour of organic compounds found in this study is given in Appendix 3.

Addressing the problem: the need for an internationally verified survey and decontamination programme for the former UCIL plant and surrounding area.

The results of this investigation demonstrate extensive and, in some areas, severe chemical contamination of the environment surrounding the former Union Carbide plant. Analysis of water samples drawn from wells serving the local community has also confirmed the contamination of groundwater reserves with chemicals arising either from previous or ongoing activities and/or incidents. As a result of the ubiquitous presence of

contaminants, the exposure of the communities surrounding the plants to complex mixtures of hazardous chemicals continues on a daily basis. Though less acute than the exposure which took place as a result of the 1984 MIC release, long-term chronic exposure to mixtures of toxic synthetic chemicals and heavy metals is also likely to have serious consequences for the health and survival of the local population.

This open, but largely undocumented, contamination must be urgently and effectively addressed such that the communities of Bhopal are no longer exposed to this legacy of pollution. In order to do this safely and effectively, an effective and fully verified decontamination programme must be undertaken:-

1. Survey and inventory

The study conducted by Greenpeace has highlighted the nature and severity of the problems surrounding the former UCIL production facility. However, a more extensive survey will be necessary in order that the full extent of contamination of soils, sediments and groundwater may be determined and documented.

- The survey should firstly produce an inventory of all accumulations, contained or otherwise, of industrial wastes both within and beyond the plant boundaries.
- Secondly, further samples of topsoil and subsoil, sediments from surface watercourses and groundwater should be analysed in order to gain further information on the geographical extent and complexity of contamination with hazardous organic chemicals and heavy metals.

This survey should be initiated as rapidly as possible, employing internationally accepted techniques and appropriately accredited laboratories. However, the completion of all aspects of the survey should not be seen as a prerequisite for the initiation of programmes to contain and treat those materials (as outlined further below) which are clearly heavily contaminated.

2. Containment of industrial wastes

On the basis of the information obtained in the survey, all accumulations of industrial wastes, particularly those lying beyond the plant boundaries, must be safely and properly contained. Containment must be fully enclosed, above ground and effected in a manner which constitutes safe storage, permits controlled access to the wastes, and which is fully documented, such that any of the wastes may be retrieved for further treatment at any time. The efficacy of containment must also be verified at suitable intervals.

Note that containment is an interim measure only, to prevent continued exposure of humans and wildlife to these contaminated materials and the spread of such contaminants to other environmental media. It must not be viewed as a final solution.

3. Containment of contaminated environmental media

Furthermore, any soils which are found to contain levels of hazardous contaminants significantly above background must be removed (to a depth at which levels of

contaminants are equivalent to background levels) and properly contained as above. This removal and containment process should start with the most heavily contaminated sites first.

4. Complete destruction or recovery of hazardous constituents

For contaminated solid materials covered by 2 and 3 above, the goal must then be the complete destruction or recovery of all hazardous constituents, in a process which ensures that there are no releases to the environment of the hazardous chemicals (or their products of incomplete destruction) during or after treatment.

For persistent organic compounds, the goal must be 100% conversion to non-hazardous final products; for heavy metals, the goal is 100% recovery in a form which can be isolated fully from the matrix and contained separately. The technology used must have the capability to re-introduce any waste streams containing residual quantities of hazardous chemicals (above limits of detection using internationally accepted techniques and suitably accredited laboratories) for further processing, thereby facilitating total contaminant removal.

5. Decontamination of groundwater

For contaminated groundwater aquifers, above-ground containment prior to treatment is not an option. In those cases in which groundwater is determined to contain levels of hazardous substances above background concentrations, the priority must be to ensure that further consumption by humans and livestock is prevented. Pumping equipment should be removed or deactivated and wells capped to prevent access. Alternative water supplies must then be made available as necessary. The responsibility for these public health protection measures must lie with the Government of India.

Wherever possible, efforts should be made to remove contaminants as rapidly and effectively as possible from those aquifers affected. A review of available approaches and technology will be essential in this regard. Filtration through activated charcoal, commonly employed for groundwater clean-up, may effectively remove semi-volatile contaminants, although more volatile compounds (including the halomethanes) may simply escape to the atmosphere. Techniques employing rapid semi-permeable membrane filtration, with extraction into vegetable oil (e.g. Zander *et al.* 1992), may be effective in removing both volatile and semi-volatile contaminants. In any case, all wastes streams generated, which are likely to contain high concentrations of hazardous chemicals, should then be properly contained until such time that they may be decontaminated according to the criteria outlined in 4 above.

6. Avoidance of further contamination

Every effort must be made to ensure that the inventorying, containment and/or treatment of any of the contaminated material does not lead to more widespread contamination of the surrounding environment. Workers employed in handling such materials must be

properly trained, fully aware of the hazards and safety procedures relating to each material and provided with appropriate protective clothing and other necessary safety equipment in order to minimise or eliminate direct exposure.

7. Responsibility for the decontamination programme

The financial, operational and legal responsibility for the decontamination of the site and the surrounding community, in a manner consistent with the criteria set out above, must be borne by the former and/or current owner(s)/operator(s) of the production facility. Legal action against the responsible party must be taken if that party fails to implement a full and effective decontamination programme.

8. Oversight and verification

All components of the decontamination programme, from the survey and design phases through to completion, should be subject to oversight by independent international experts. The effectiveness of any clean-up measures employed must similarly be subject to independent verification using state of the art sampling and analytical techniques.

References

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

ATSDR (1996). Toxicological profile for hexachloroethane (update). Agency for Toxic Substances and Disease Registry, Atlanta, GA. US Department of Health and Human Services

ATSDR (1997). ARSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

Behl, V.K., Iyer, C.R., Choudhary, S.P. and Khanna, S. (1978). Operation Manual. Part I. Methyl isocyanate unit. Reviewed by Ballal, K.D. October, 1978. Union Carbide India Limited. Agricultural Products Division. Bhopal, India. 136pp.

Bhandari, N.R., Syal, A.K., Kambo, I., Nair, A., Beohar, V., Saxena, N.C., Dabke, A.T., Agarwal, S.S. and Saxena, B.N. (1990). Pregnancy outcome in women exposed to toxic gas in Bhopal. Indian J. Med Res. [B] 92: 28-33

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

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

Burston, M.W., Nazari, M.M., Bishop, P.K. & Lerner, D.N. (1993). Pollution if groundwater in the Coventry region (UK) by chlorinated hydrocarbon solvents. Journal of Hydrology 149, 1-4: 137-161

Butler, E.C. & Hayes, K.F (1998). Effects of solution composition and pH on the reductive dechlorination of hexachloroethane by iron sulfide. Environmental Science and Technology 32, 9: 1276-1284

CRC (1969) Handbook of chemistry and physics. A Ready-reference book of chemical and physical data. Weast, R.C. [Ed] Pub. By The Chemical Rubber Co. Library of Congress Card No. 13-11056.

Cullinan, P., Acquilla, S.D. and Dhara, V.R. (1996). Long term morbidity in survivors of the 1984 Bhopal gas leak. National Medical Journal of India 9, 1: 5-10

Cullinan, P., Acquilla, S.D. and Dhara, V.R. (on behalf of the International Medical Commission on Bhopal) (1997). Respiratory morbidity 10 years after the Union Carbide gas leak at Bhopal: a cross sectional survey. British Medical Journal 314: 338-343

D'Silva, T.D.J., Lopes, A., Jones, R.L., Singhawangcha, S. and Chan, J.K. (1986). Studies of methyl isocyanate chemistry in the Bhopal incident. J. Org. Chem. 51: 3781-3788

DHHS (1998). Report on carcinogens. Summary. 8th Edition. US Department of Health and Human Services, 1998, 252pp.

Dikshith, T.S.S., Raizada, R.B., Kumar, S.N., Srivastava, M.K., Kulshrestha, S.K. & Adholia, U.N. (1990) Residues of DDT and HCH in major sources of drinking water in Bhopal, India. Bull. Environ. Contam. Toxicol. 45: 389-393

Edwards I.R., Ferry D.H., Temple W.A. (1991). Fungicides and related compounds. In: Handbook of pesticide toxicology, [Ed] W.J.Hayes, Jr and E.R.Laws, Jr. Academic Press, Inc. Vol.3. Classes of pesticides, pp.1409-1470.

EEC (1979). Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances (OJ L 20, 26.1.1980, p. 43)

Freiria-Gandara, M.J., Lorenzo-Ferreira, R.A., Alvarez-Devesa, A. & Bermejo, F. (1992). Occurrence of halogenated hydrocarbons in the water supply of different cities of Galicia (Spain). Environmental Technology 13: 437-447.

Gupta, B.N., Rastogi, S.K., Chandra, H., Mathur, N., Mahendra, P.N., Pangtey, B.S., Kumar, S., Kumar, P., Seth, R.K., Dwivedi, R.S. and Ray, P.K. (1988). Effect of exposure to toxic gas on the population of Bhopal: part I-epidemiological, clinical, radiological and behavioural studies. Indian Journal of Experimental Biology 26: 149-160

Hashsham, S.A., Scholze, R. & freedman, D.L (1995). Cobalamin-enhanced anaerobic biotransformation of carbon tetrachloride. Environmental Science and Technology 29, 11: 2856-2863

Howard, P. H. (1989). Handbook of environmental fate and exposure for organic chemicals. Volume I: Large production and priority pollutants. Lewis Publishers, Inc., USA

Howard, P.H., Boethling, R.S., Jarvis, W.F., Meylan, W.M. and Michalenko, E.M.[Eds] (1991). Handbook of Environmental Degradation Rates. Lweis Publishers, Inc., USA

Hughes, J.B. & Parkin, G.F. (1996a). Concentration effects on chlorinated aliphatic transformation kinetics. Journal of Environmental Engineering 122, 2: 92-98

Hughes, J.B. & Parkin, G.F. (1996b). Individual biotransformation rates in chlorinated aliphatic mixtures. Journal of Environmental Engineering 122, 2: 99-106

Indian Council of Medical Research (1985). Health effects of exposure to toxic gas at Bhopal: an update on ICMR sponsored researches, New Delhi

Kennicutt, M.C., Green, R.H., Montagna, P. and Roscigno, P.F. (1996). Gulf of Mexico Offshore Operations Monitoring Experiment (GOOMEX), Phase 1: sublethal responses to contaminant exposure- introduction and overview. Can. J. Fish. Aquat. Sci. 53: 2540-2553

Kumar, S. (1994). Independent assessment of Bhopal. The Lancet 343: 283-284

Kumar, S. (1995). Court slams “miserly” attitude to Bhopal victims. New Scientist 148, 2008: 8

Lambrech, J.A. & Charleston, W.Va. (1959). α-Naphthol bicyclic aryl esters of N-substituted carbamic acids. US Patent No.2 903 478. Patented Sept. 8, 1959. US Patent Office

Lerner, D.N., Gosk, E., Bourg, A.C.M., Bishop, P.K., Burston, M.W., Mouvet, C., Egranges, P., & Jakobsen, R. (1993). Postscript - summary of the Coventry groundwater investigation and implications for the future. Journal of Hydrology 149(1-4): 257-272

Loran, M.M. & Olsen, L. (1999). Degradation of 1,1,2,2-tetrachloroethane in a freshwater tidal wetland: field and laboratory evidence. Environmental Science and Technology 33(2): 227-234

MacKenzie, D. (1984). The chemistry behind Bhopal’s tragedy. New Scientist 104, 1434: 3-4

MacKenzie, D. (1985a). Uncharted pipe could explain Bhopal disaster. New Scientist 106, 1451: 4

MacKenzie, D. (1985b). Bhopal: Carbide insists it was sabotage. New Scientist 198: 1482: 15

Miller, P.L., Vasudevan, D., Gschwend, P.M. & Roberts, A.L. (1998). Transformation of hexachloroethane in sulfidic natural water. Environmental Science and Technology 32, 9: 1269-1275

Milne, R. (1988). “Act of sabotage” killed thousands at Bhopal. New Scientist 118, 1613: 28

NEERI (1990). Assessment of pollution damage due to Solar Evaporation Ponds at UCIL, Bhopal. Published by National Environmental Engineering Research Institute, Nagpur-440 020, Madhya Pradesh Pradushan Niwaran Mandal, Bhopal, 1990.

Overton, E.B. (1994). Toxicity of petroleum. In: Basic Environmental Toxicology. Cockerham & Shane [Eds], Chapter 5: 133-156

Rastogi, S.K., Gupta, B.N., Husain, T., Kumar, A., Chandra, S. and Ray, P.K. (1988). Effect of exposure to toxic gas on the population of Bhopal: part II-respiratory impairment. Indian Journal of Experimental Biology 26: 149-160

Rivett, M.O., Feenstra, S., Cherry, J.A. (1994). Transport of a dissolved-phase plume from a residual solvent source in a sand aquifer. Journal of Hydrology 159(1-4): 27-41

Saxena, A.K., Singh, K.P., Nagle, S.L., Gupta, B.N., Ray, P.K., Srivastav, R.K., Tewari, S.P., and Singh, R. (1988). Effect of exposure to toxic gas on the population of Bhopal: part IV-immunological and chromosomal studies. Indian Journal of Experimental Biology 26: 173-176

Singh, M.P. and Ghosh, S. (1987). Bhopal gas tragedy: model simulation of the dispersion scenario. Journal of Hazardous Materials 17: 1-22

Snedecor, G (1993). Hexachloroethane. Chlorocarbons, -hydrocarbons ($CHCl=CCl_2$). In: Kirk-Othmer Encyclopedia of Chemical Technology. [Eds] Jacqueline I. Kroschwits; Mary Howe-Grant. Fourth edition, Vol. 6. John Wiley & Sons, Inc. pp.29-31

Sriramachari, S. and Chandra, H. (1997). The lessons of Bhopal [toxic] MIC gas disaster, scope for expanding global biomonitoring and environmental specimen banking. Chemosphere 34, 9/10: 2237-2250

Suzuki T., Yaguchi K., Nishima T., Suga T. (1996). Degradation of terbutol in soils from turfgrass and drainage basins on golf-courses. Journal Of Agricultural And Food Chemistry 44, 6: 1599-1602

Suzuki T., Yaguchi K., Ohnishi K., Suga T. (1995). Identification of degradation products of terbutol in environmental water from golf-courses. Journal of Agricultural and Food Chemistry 43, 6: 1712-1717.

UCC (1985). Bhopal methyl isocyanate incident investigation team report. Danbury, Connecticut, US. Union Carbide Corporation, March 1995

US EPA (1986). Factsheets for regulated chemicals: Hexachlorobutadiene. Office of Environmental Health Hazard Assessment. US Environmental Protection Agency

US EPA (1988). Reviews of environmental contamination and toxicology. Vol. 106, Environmental Protection Agency, Office of Drinking Water Health Advisories, Springer-Verlag New York Inc. ISSN 0179-5953: 21-35

US EPA (1999). Current drinking water standards. National primary and secondary drinking water regulations. Office of Ground Water and Drinking Water. US Environmental Protection Agency

Wallace, L.A. (1997) Human exposure and body burden for chloroform and other trihalomethanes. Critical Reviews in Environmental Science and Technology, 27(2): 113-194.

WHO (1989). Mercury. Environmental Health Criteria 86. ISBN 92 4 154460 0

WHO (1993) Guidelines for drinking water quality. Vol.1. Recommendations. Second Edition, ISBN 92 4 154460 0

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

Zander, A.K., Chen, J.-S., and Semmens, M.J. (1992). Removal of hexachlorocyclohexane isomers from water by membrane extraction into oil. Water Research 26, 2: 129-137

Appendix 1

Materials and Methods

All samples were collected and stored in clean glass bottles that had been thoroughly washed with detergent, and rinsed with deionised water, analytical grade pentane and nitric acid to remove all organic and heavy metal residues. Soil samples for heavy metal determinations and organic screen analysis were collected in 100ml clear glass bottles. Aqueous samples for volatile organic compounds (VOCs) analysis were collected in 125ml amber glass bottles capped with a ground glass stopper. Bottles were filled completely, ensuring that no air bubbles were present. All samples were stored cold, kept cold during transit, and refrigerated at 4^0C immediately on arrival at the Greenpeace Research Laboratories.

1. Organic Analysis

1.1 Preparation of samples for Volatile Organic Compounds (VOCs) analysis

For volatile organic compound analysis, no sample preparation was required. The original sample was sub-sampled immediately after opening. Two portions of 10ml each were transferred into 20ml headspace vials and sealed with Teflon-lined vial caps. One sub-sample was used for the organic screen analysis to evaluate the whole range of the compounds in the sample. The second sub-sample was used for quantification of the detected compounds with an external standard method. For quantification of VOCs a standard calibration solution was prepared with the following compounds: chloroform, carbon tetrachloride, trichloroethene, tetrachloroethene, hexachloroethane, chlorobenzene, 1,2-dichlorobenzene, 1,3-dichlorobenzene, 1,4-dichlorobenzene, 1,2,3-trichlorobenzene, 1,2,4-trochlorobenzene, and 1,3,5-trichlorobenzene. All standard compounds were obtained from Sigma-Aldrich Co. Ltd./Supelco UK.

1.2 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.

1.3 Chromatographic Analysis

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

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

Instrumentation for the analysis of volatile organic compounds was a Hewlett Packard (HP) 5890 Series II gas chromatograph with HP 19395-A headspace sampler, interfaced with a HP Chem-Station data system and linked to a HP 5970 Mass Selective Detector operated in scan mode. Again, the identification of compounds was carried out using a combination of computer matching (against a HP Wiley 138 library of 138 000 mass spectra) and expert interpretation. Quantification of VOCs was performed in selective ion monitoring (SIM) mode using an external standard method, the target ions and qualifiers are presented in the table below.

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 (using retention times and mass-spectra obtained during calibration) are assumed to give reliable identifications. Tentative identification

refers to qualities between 51% and 90% against HP Wiley 275 library only. Analytes yielding match qualities of 50% or less are assumed to be unidentified.

Standard compound	Target ion	Qualifier ion
Chloroform	83	118
Carbon tetrachlorode	117	119
Trichloroethene	130	95
Tetrachloroethene	166	129
Hexachloroethane	117	201
Chlorobenzene	112	77
1,2-Dichlorobenzene	146	111
1,3-Dichlorobenzene	146	75
1,4-Dichlorobenzene	146	111
1,2,3-Trichlorobenzene	180	145
1,2,4-Trichlorobenzene	180	145
1,3,5-Trichlorobenzene	180	145

2. Heavy Metal Analysis

2.1 Preparation of solid samples for heavy metals analysis

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.

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

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

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

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

Appendix 2

Table 5. List of organic compounds reliably and tentatively identified in soil and soil/solid waste samples associated with the former UCIL, Bhopal, India

Sample Number:IT9042	Sample Number:IT9017	Sample Number:IT9016	Sample Number:IT9015	Sample Number:IT9014	Sample Number:IT9013	Sample Number:IT9012
Number of Compounds isolated:15	Number of compounds isolated:15	Number of compounds isolated: 25	Number of compounds isolated:59	Number of compounds isolated:45	Number of compounds isolated:33	Number of compounds isolated:73
COMPOUNDS RELIABLY IDENTIFIED: 1-Dodecene Benzene, 1,4-dichloro- Cyclotetradecane COMPOUNDS TENTATIVELY IDENTIFIED : 1-Hexadecene 3-Dodecene, (Z)- Benzamide, 3-amino- Cyclohexane, 1,2-dimethyl-, cis- Docosane, 11-butyl- Heneicosane Octadecane Tetratetracontane Tricosane Undecane, 2,4-dimethyl-	COMPOUNDS RELIABLY IDENTIFIED: 1-Hexadecene Benzene, 1,4-dichloro- Docosane Dodecane COMPOUNDS TENTATIVELY IDENTIFIED : 5-Octadecene, (E)- 7-Tetradecene Decane, 2-methyl- Octadecane, 3-ethyl-5-(2-ethylbutyl)- Octane, 4-methyl- Phenol, 2,6-bis(1,1-dimethylethyl)- Triacontane Tridecane, 3-methyl-	COMPOUNDS RELIABLY IDENTIFIED: 1-Hexadecene 1-Tetradecene Benzene 1,4 –dichloro- Benzene, (1-methyldecyl)- Docosane Dodecane Eicosane Heptadecane Octadecane Tetracosane Tetradecane Tridecane COMPOUNDS TENTATIVELY IDENTIFIED : 1-Dodecene Benzene, (1-ethyldecyl)- Benzene, (1-methylundecyl)- Cyclododecane Cyclohexadecane Decane Heptacosane Hexadecane Tridecane, 6-propyl- Tridecanol Undecane	COMPOUNDS RELIABLY IDENTIFIED: 1-Hexadecene 1-Octadecene alpha-HCH Benzene, 1,2 dichloro- Benzene, 1,2,3,4-tetrachloro- Benzene, 1,2,3,5-tetrachloro- Benzene, 1,2,3-trichloro- Benzene, 1,2,4-trichloro-5-methyl- Benzene, 1,3-dichloro- Benzene, 1,4-dichloro- Benzene, hexachloro- Benzene, pentachloro- Docosane Eicosane Heneicosane Hexadecane Hexadecane, 3-methyl- Naphthalene Octadecane p,p'-DDE Pentacosane Tetradecane Tetratriacontane Tricosane COMPOUNDS TENTATIVELY IDENTIFIED : 6-Tridecene Cyclopropane, cis-1-butyl-2-methyl- Cyclotetradecane Decane, 2,3,5,8-Tetramethyl- Decane, 2,4,6-trimethyl- Decane, 2-methyl- Docosane, 11-decyl- Docosane, 7-hexyl- Docosane, 9-butyl- Dodecane Heptacosane Heptadecane Heptadecane, 4-methyl- Hexatriacontane Nonadecane Nonane, 5-butyl- Octadecane, 3-ethyl-5-(2-ethylbutyl)- Undecane, 2-methyl- Undecane, 3,8-dimethyl- Undecane, 5-methyl-	COMPOUNDS RELIABLY IDENTIFIED: 1-Dodecene 1-Tetradecene Benzene, (1-ethyldecyl)- Benzene, (1-ethylundecyl)- Benzene, (1-methylheptadecyl)- Benzene, (1-pentylheptyl)- Benzene, (1-propyldecyl)- Docosane Hentriacontane Nonane, 5-butyl- Pentacosane Phenol, 2,6-bis(1,1-dimethylethyl)-4-methyl- Phenol,bis(1,1-dimethylethyl)- Tetratriacontane COMPOUNDS TENTATIVELY IDENTIFIED : 1-Heptadecanamine 4-Decene Benzene, (1-butylheptyl)- Benzene, (1-butyloctyl)- Benzene, (1-butylpentyl)- Benzene, (1-ethylnonyl)- Benzene, (1-methylundecyl)- Benzene, (1-pentylhexyl)- Benzene, (1-pentyloctyl)- Benzene, (1-propylpentyl)- Docosane Docosane, 11-decyl- Dodecane Eicosane, 10-methyl- Heptacosane Heptadecane Heptane, 2,4-dimethyl- Hexadecane Hexatriacontane Nonane, 4,5-dimethyl- Octacosane Octadecane, 3-ethyl-5-(2-ethylbutyl)- Octane, 2-methyl- Pentadecane, 3-methyl- Tridecane, 3-methyl- Undecane, 2-methyl-	COMPOUNDS RELIABLY IDENTIFIED: 1-Hexadecene 1-Tetradecene alpha-HCH Benzene, 1,2,3,4-tetrachloro- Benzene, 1,2,3,5-tetrachloro- Benzene, 1,2,4-trichloro- Benzene, 1,2-dichloro- Benzene, 1,3-dichloro- Benzene, 1,4-dichloro- Benzene, pentachloro- beta-HCH Cyclohex-1-ene, 1,2,3,4,5-pentachloro- delta-HCH Eicosane gamma-HCH Naphthalene Octadecane p,p'-DDD Phenol, 2,6-bis(1,1-dimethylethyl)-4-methyl- Phenol, bis(1,1-dimethylethyl)- Pyridine, 2,3,4,6-tetrachloro- COMPOUNDS TENTATIVELY IDENTIFIED : 1-Undecene Cyclohexane, 1,1,2,3,4,5,6-heptachloro- Cyclohexene,.gamma.-3,4,5,6-tetrachloro- Docosane Eicosane, 9-octyl-	COMPOUNDS RELIABLY IDENTIFIED: 1,1'-Biphenyl 1,3-Butadiene, 1,1,2,3,4,4-hexachloro- 1-Dodecene 1-Octadecene Dibenzothiophene Dibenzothiophene, 2,8-dimethyl- Dibenzothiophene, 2-ethyl- Dibenzothiophene, 3,4-dimethyl- Dibenzothiophene, 3-methyl- Dibenzothiophene, 4-methyl- Docosane Ethane, hexachloro- Heneicosane Heptadecane Hexadecane Naphthelene, 1-methyl Naphthelene, 2-methyl- Naphthalene Naphthalene, 1,2,3,4-tetrahydro- Naphthalene, 1,4,5-trimethyl- Naphthalene, 1,4,6-trimethyl- Naphthalene, 1,5-dimethyl- Naphthalene, 2,3,6-trimethyl- Naphthalene, 2,6-dimethyl- Naphthalene, 2,7-dimethyl- Nonadecane Octadecane Pentacosane Pentadecane Pentadecane, 2,6,10,14-tetramethyl- Phenanthrene Phenanthrene, 9-methyl- Phenol, 2,4-bis (1,1-dimethylethyl)- Tetracosane Tricosane Tridecane COMPOUNDS TENTATIVELY IDENTIFIED : [8]paracyclophane-2,4-diene 2-Dodecene, (E)- 5-Octadecene, (E)- 9-Undecen-2-one, 6,10-dimethyl- alpha-HCH Azulene, 7-ethyl-1,4-dimethyl- Benzene, (1-chloroethylmethyl)- Benzene, 1,1'-[(methylthio)ethenylidene]bis- Benzene, 1,1'-oxybis- Dodecane, 2,6,10-trimethyl- Dodecane, 3-methyl- Eicosane, 2-methyl- Hexadecane, 2,6,10,14-tetramethyl- Naphthalene, 1-(2-propenyl)- Phenol, 2,4-bis (1,1-dimethylethyl)-4-methyl- Tetradecane Undecane

Appendix 3

Toxicological outlines for key organic compounds

Carbon tetrachloride

Carbon tetrachloride is a manufactured compound that does not occur naturally (US EPA 1988). It is a clear, colourless, non-flammable liquid, which is heavier than water, and it is moderately soluble in water. Carbon tetrachloride itself does not burn but poisonous gases are produced in fire, including phosgene and hydrogen chloride. It uses as solvent for oils, fats, lacquers, varnishes, rubber waxes and resins. Carbon tetrachloride was formerly used as dry cleaning agent and fire extinguisher. Because of its harmful and ozone depleting effects, these uses are now banned and it is only used in some industrial applications. Principally it was used in the production of chlorofluorocarbon (CFC) refrigerants (Budavari et al 1986***;*** WHO 1993) but this use of carbon tetrachloride was stopped in 1996 when CFC-11 and CFC-12 have been banned (UNEP 1997).

Carbon tetrachloride is a substance which can cause cancer in animals and humans (US EPA 1997) and has been classified as Group 2B carcinogen (possibly carcinogenic to humans) by International Agency for Research on Cancer (IARC 1999). Carbon tetrachloride induces hepatic cell prolifiration and DNA synthesis. It also has a mutagenic effect and induces aneuploidy in several *in-vitro* systems (IARC 1999). High exposure to carbon tetrachloride can cause liver, kidney, and central nervous system damage. Liver swells and cells are damaged or destroyed. Kidneys are also damaged, causing a build-up of wastes in the blood. If exposure is low and then stops, the liver and kidneys can repair the damaged cells and function normally again (ATSDR 1997). If exposure is very high, the nervous system, including the brain, is affected. People may feel intoxicated and experience headaches, dizziness, sleepiness, and nausea and vomiting. These effects may subside if exposure is stopped, but in severe cases, coma and even death can occur (ATSDR 1997).

Carbon tetrachloride may enter the environment from industrial effluents, municipal treatment plant discharges, or spills (Menzer *et al.* 1986; US EPA 1988). It has been found in the river waters in the areas influenced by the chlorinated organic solvent plant (Amaral *et al.* 1996). Carbon tetrachloride is relatively stable in the environment. If carbon tetrachloride released to land, it does not sorb onto soil, but migrates readily to ground water and is believed to remain in ground water for several years (US EPA 1988). Under anaerobic conditions carbon tetrachloride can be biotransformed producing hazardous intermediates such as chloroform and methylene chloride (Hashsham *et al.* 1995) and carbon disulphide under sulphate reducing conditions (Delvin & Muller 1999; Hashsham *et al.* 1995).

Carbon tetrachloride has been detected in drinking water (Abernathy 1994). In the EPA Ground Water Supply Survey on drinking water supplies that used groundwater as a source (Cotruvo *et al.* 1986) carbon tetrachloride was among six most frequently

occuring in the samples analysed with the maximum concentration of 16ug/l. In the study carried out in different cities in Galicia, Spain (Freiria-Gandara *et al.* 1992) it was found that concentration of carbon tetrachloride in treated drinking water (if detected) was in the range between 39.5 and 1.5ug/l. In the similar investigation of drinking water in Barcelona, Spain (Amaral *et al.* 1996) the levels of carbon tetrachloride were less than 0.1ug/l.

The maximum level of this compound in drinking water stipulated by the World Health Organization is 2ug/l (WHO 1993). The Environmental Protection Agency (US EPA 1998) has set a limit for carbon tetrachloride of 5ug/l. The EPA recommends that drinking water levels which are "safe" for short-term exposures for a 10kg child consuming 1 litre of water per day: a one-day exposure of 4mg/l; a ten day exposure to 0.2mg/l; up to 7 year exposure to 0.07mg/l (US EPA 1998).

Carbon tetrachloride belongs to the organohalogen compounds whose presence in groundwater are controlled by the European Community Environmental Legislation. Article 3 of EC Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances (EEC 1979) and amended later (EEC 1991) says that Member States shall take necessary steps to prevent the introduction into groundwater of substances in List I and organohalogen compounds are among the groups of the compounds listed there.

The quality objective of 12ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters, and territorial waters) is set for carbon tetrachloride by the EC Council Directive 86/280/EEC (EEC 1986) and amended in 1988 (EEC 1988).

The EC Council Directive 76/769/EEC (EEC 1976) which last was amended in 1996 (EEC 1996) restricts marketing and use of carbon tetrachloride. Carbon tetrachloride may not be used in concentrations equal to or greater than 0.1% by weight in substances and preparation placed on the market for sale to the general public and/or in diffusive applications such as in surface cleaning and cleaning of fabrics.

Carbon tetrachloride is included in Group II of Annex B of controlled substances of the Montreal Protocol (UNEP 1997) as an ozone depleting compound. Article 2D on carbon tetrachloride says that level of consumption and production of this substance, calculated using a complex formula, should not exceed zero from 1 January 1996. However, the developing countries are entitled to delay implementation for ten years in order to meet their basic domestic needs, as specified in the Article 5.

References

Abernathy, C.O. (1994) A retrospective on drinking water. In: Water contamination and health. Wang, R.G.M. [Ed], Marcel Dekker, Inc., New York: 1-14

Amaral, O.C., Otero, R., Grimalt, J.O. & Albaiges, J. (1996) Volatile and semi-volatile organochlorine compounds in tap and riverine waters in the area of influence of a chlorinated organic solvent factory. Water Resources. 30(8): 1876-1884

ATSDR (1997) ARSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease

Registry, U.S. Public Health Service. CRC Publishers
Budavari, S.M., O'Neil, J., Smith A. and Heckleman P.E. [Eds] (1986) The Merck index: an encyclopaedia of chemicals, drugs and biologicals. 11th Edn Merck and Co, Inc., New Jersey, USA
Cotruvo, J., Goldhaber, S. & Vogt, C. (1986) Regulatory significance of organic contamination in the decade of the 1980s. In: Organic carcinogens in drinking water. Ram, N.M., Calabrese, E.J. & Christman, R.F. [Ed]. John Wiley and Sons, New York: 511-530
Delvin, J.F. & Muller, D. (1999) Field and laboratory studies of carbon tetrachloride transformation in sandy aquifer under sulfate reducing conditions. Environmental Science and Technology 33(7): 1021-1027
EEC (1976) Council Directive 76/769/EEC of 27 July 1976 on the approximation of lows, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations. OJ L 262: 201-203
EEC (1979) Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances. OJ L 020: 43-48
EEC (1986) Council Directive 86/280/EEC of 12 June 1986 on limit values and quality objectives for discharges of certain dangerous substances included in List I of the Annex to Directive 76/464/EEC. OJ L 181: 16-27
EEC (1988) Council Directive 88/347/EEC of 16 June 1988 on limit values and quality objectives for discharges of certain dangerous substances included in List I of the Annex to Directive 76/464/EEC. OJ L 158: 35-41
EEC (1991) Council Directive 91/692/EEC of 23 December 1991 standardizing and rationalizing reports on the implementation of certain Directives relating to the environment. OJ L 377: 48-54
EEC (1996) Commission Directive 96/55/EC of 4 September 1996 adapting to technical progress for the 2nd time Annex I to Council Directive 76/769/EEC of 27 July 1976 on the approximation of lows, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations (chlorinated solvents). OJ L 231: 20-21
Freiria-Gandara, M.J., Lorenzo-Ferreira, R.A., Alvarez-Devesa, A. & Bermejo, F. (1992) Occurance of halogenated hydrocarbons in the water supply of different cities of Galicia (Spain). Environmental Technology 13:437-447
Hashsham, S.A., Scholze, R. & freedman, D.L (1995) Cobalamin-enhanced anaerobic biotransformation of carbon tetrachloride. Environmental Science and Technology 29(11): 2856-2863
IARC (1999) Carbon tetrachloride. IARC Monographs Vol.71, p.401
Menzer, R.E. & Nelson, J.O (1986) Water and soil pollutants. In: Casarett and Doull's Toxicology: The Basic Science of Poisons. Third Edition. Klaasen, C.D, Emdur, M.O. & Doll, J. [Ed]. Macmillan Publishing Company. ISBN 0-02-364650-0, pp.825-856
UNEP (1997) The 1987 Montreal Protocol on Substances that Deplete the Ozone Layer, as adjusted and amended by the second Meeting of the Parties (London, 27-29 June, 1990), and by the fourth Meeting of the parties (Copenhagen, 23-25 November 1992), and further adjusted by the seventh Meeting of the Parties (Vienna, 5-7 December 1995), and further adjusted and amended by the ninth Meeting of the Parties (Montreal, 15-17 September 1997)
US EPA (1988) Reviews of environmental contamination and toxicology. Vol. 106, Environmental Protection Agency, Office of Drinking Water Health Advisories, Springer-Verlag New York Inc. ISSN 0179-5953: 21-35
US EPA (1997) Chemicals known to the state to cause cancer or reproductive toxicity. Environmental Protection Agency, Office Of Environmental Health Hazard Assessment Safe Drinking Water And Toxic Enforcement Act Of 1986
US EPA (1998) National Primary Drinking Water Regulations. Technical Factsheet on Carbon tetrachloride. Environmental Protection Agency, Office of Groundwater and Drinking Water
WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

Chloroform

Chloroform is a heavy, colourless, non-flammable liquid. It has a characteristic pleasant, sweet, ethereal odour and a sweetish burning taste. The odour is non-irritant (CEC 1986). It has been extensively used in the past as an anaesthetic (Snyder & Andews 1996). Currently the largest use of chloroform is to make HCFC-22, an ozone-depleting refrigerant (Holbrook 1993). The Montreal Protocol, the international legislation which protects the ozone layer, has set targets for reducing the use of HCFC-22, but it will not be totally phased out until 2030 (UNEP 1997).

Chloroform is the most abundant of the trihalomethanes (THMs) which are generated as by-products during water disinfection using chlorine-containing compounds (Oxenford 1996; ATSDR 1997; Health Canada 1996). Additionally it can be formed in washing machines into which chlorinated bleach has been added (Shepherd & Corsi 1996), in the natural waters where chlorine-containing effluents have been discharged (Mills *et al.* 1998). Exposure to chloroform may occur when breathing contaminated air, drinking contaminated water or through skin contact (Weisel & Chen 1994; Weisel & Jo 1996). Water is possibly now the major source of environmental exposure to chloroform.

Chloroform has been specified by the International Agency for Research on Cancer in the Group 2B as possibly carcinogenic to humans (IARC 1998). Investigation on animals have shown that the main target organs for carcinogenicity from chloroform are liver, kidney, and/or intestine (Dunnick & Melnik 1993; Snyder & Andews 1996; Chiu *et al.* 1996). A guideline value of 200ug/l was calculated to correspond to an excess lifetime cancer risk of 10^{-5} by the World Health Organisation (WHO 1993). The Maximum Contaminant Level (MCL) of 100ug/l in drinking water which is delivered to any user of a public water system is set by EPA for the total THMs (US EPA 1999). There are four contaminants included in this group: chloroform, bromodichloromethane, dibromochloromethane and bromoform (Oxenford 1996).

It is not known whether chloroform causes reproductive effects or birth defects in people, but animal studies have shown that miscarriages occurred in rats and mice that breathed air containing 30–300 ppm chloroform during pregnancy and also in rats that ate chloroform during pregnancy. Offspring of rats and mice that breathed chloroform during pregnancy had birth defects. Abnormal sperm were found in mice that breathed air containing 400 ppm chloroform for a few days (ATSDR 1997).

The levels of chloroform found in treated drinking water depend upon water treatment practice, age of the water, water temperature (Health Canada 1996) and can vary in the range from less than 1ug/l to 200ug/l (Wallace 1997; Health Canada 1996). Levels less than 10ug/l have been found in the US rural ground water (Wallace 1997), mean value of 84ug/l has been reported for the surface waters (if detected) in the same survey.

Chloroform evaporates easily into the air. Most of the chloroform in air breaks down eventually, but it is a slow process. The breakdown products in air include phosgene and hydrogen chloride, which are both toxic (ATSDR 1997). It is poorly absorbed to soil and

can travel through soil to groundwater where it can persist for years. Chloroform dissolves easily in water and some of it may break down to other chemicals (ATSDR 1997).

The presence of chloroform (as organohalogen compound) in groundwater is controlled by European Community Environmental Legislation. Article 3 of EC Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances (EEC 1979) and amended later (EEC 1991) says that Member States shall take necessary steps to prevent the introduction into groundwater of substances in List I and organohalogen compounds are among the groups of the compounds listed there.

The quality objective of 12ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters, and territorial waters) is set for chloroform by the EC Council Directive 86/280/EEC (EEC 1986) and amended in 1988 (EEC 1988).

The EC Council Directive 76/769/EEC (EEC 1976) which last was amended in 1996 (EEC 1996) restricts marketing and use of chloroform. Chloroform may not be used in concentrations equal to or greater than 0.1% by weight in substances and preparation placed on the market for sale to the general public and/or in diffusive applications such as in surface cleaning and cleaning of fabrics.

References

ATSDR (1997) ATSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

CEC (1986) Chloroform. In: Organochlorine solvents: health risk to workers. Publ: Commission of the European Communities, ISBN 0-85186-078-8: 17-53

Chiu, N., Orme-Zavaleta, J., Chiu, A., Chen, C., DeAngelo, A., Brattin, W. & Blancato, J. (1996) Characterization of cancer risk associated with exposure to chloroform. Environmental Carcinogenesis & Ecotoxicology Reviews. Journal of Environmental Science and Health C14(2): 81-104

Dunnick, J.K & Melnik, R.L. (1993) Assessment of the carcinogenic potential of chlorinated water: experimental studies of chlorine, chloramine and trihalomethanes. Journal of the National Cancer Institute 85(10): 818-823

EEC (1976) Council Directive 76/769/EEC of 27 July 1976 on the approximation of laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations. OJ L 262: 201-203

EEC (1979) Council Directive 80/68/EEC of 17 December 1979 on the protection of groundwater against pollution caused by certain dangerous substances. OJ L 020: 43-48

EEC (1986) Council Directive 86/280/EEC of 12 June 1986 on limit values and quality objectives for discharges of certain dangerous substances included in List I of the Annex to Directive 76/464/EEC. OJ L 181: 16-27

EEC (1988) Council Directive 88/347/EEC of 16 June 1988 on limit values and quality objectives for discharges of certain dangerous substances included in List I of the Annex to Directive 76/464/EEC. OJ L 158: 35-41

EEC (1991) Council Directive 91/692/EEC of 23 December 1991 standardizing and rationalizing reports on the implementation of certain Directives relating to the environment. OJ L 377: 48-54

EEC (1996) Commission Directive 96/55/EEC of 4 September 1996 adapting to technical progress for the 2nd time Annex I to Council directive 76/769/EEC of 27 July 1976 on the approximation of lows, regulations and administrative provisions of the Member States relating to restrictions on the

marketing and use of certain dangerous substances and preparations (chlorinated solvents). OJ L 231: 20-21
Health Canada (1996) A one-year survey of halogenated disinfection by-products in the distribution system of treatment plant using three different disinfection processes. Published by authority of the Minister of Health, Canada. ISBN 0-662-25172-5, 59p.
Holbrook, M.T. (1993) Chloroform. IN: Kroschwitz, J.I. & Howe-Grant, (Eds). The Kirk-Othmer Encyclopedia of Chemical Technology, Fourth Edition .Publ. Wiley-Interscience, N.Y. Volume 5, pp 1051-1062
IARC (1998) Overall evaluation of carcinogenicity: an updating of IARC Monographs Volumes 1 to 42. Chloroform. Supplement 7, p.152
Mills, W.B., Lew, C.S. & Loh, J.Y. (1998) Predictions of potential human health and ecological risks from power plant discharges of total residual chlorine and chloroform into rivers. Environmental Science and Technology 32(14): 2162-2171
Oxenford, J.L. (1996) Disinfection by-products: current practices and future directions. In: Disinfection by-products in water treatment: the chemistry of their formation and control. Minear, R.A. & Amy G.A. [Ed] CRC Press, Inc. ISBN 1-56670-136-8: 3-16
Shepherd, J.L. & Corsi, R.L. (1996) Chloroform in indoor air and wastewater: the role of residential washing machines. Journal of Air and Waste Management Association 46: 631-642
Snyder, R. & Andews, L.S. (1996) Toxic effects of solvents and vapours. In: Casarett and Doull's Toxicology: The Basic Science of Poisons. Fifth Edition. Klaasen, C.D, Amdur, M.O. & Doull, J. [Ed]. Mc-Graw-Hill, ISBN 0-07-105476-6, pp.737-772
UNEP (1997) The 1987 Montreal Protocol on Substances that Deplete the Ozone Layer, as adjusted and amended by the second Meeting of the Parties (London, 27-29 June, 1990), and by the fourth Meeting of the parties (Copenhagen, 23-25 November 1992), and further adjusted by the seventh Meeting of the Parties (Vienna, 5-7 December 1995), and further adjusted and amended by the ninth Meeting of the Parties (Montreal, 15-17 September 1997)
Wallace, L.A. (1997) Human exposure and body burden for chloroform and other trihalomethanes. Critical Reviews in Environmental Science and Technology 27(2): 113-194
Weisel, C.P. & Chen, W.J. (1994) Exposure to chlorination by-products from hot water uses. Risk Analysis 14(1): 101-107
Weisel, C.P. & Jo W-K. (1996) Ingestion, inhalation, and dermal exposures to chloroform and trichloroethene from tap water. Environmental Health Perspectives 104(1): 48-51
WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

Trichloroethene

Trichloroethene is a clear, colourless, heavy liquid with a pleasant, sweetish, chloroform-like odour, and a sweet burning taste. It easily evaporates at room temperature. Trichloroethene is non-flammable substance but it can decompose at high temperature in the air producing hydrochloric acid, phosgene and other compounds (CEC 1986). Other names of trichloroethene are trichloroethylene and ethylene trichloride.

Trichloroethene is a chlorinated solvent which was produced since the 1920s in many countries by chlorination of ethylene or acetylene (IARC 1995). Another method of producing trichloroethylene is by direct chlorination of ethylene dichloride to form trichloroethylene and tetrachloroethylene (ATSDR 1997). Oxychlorination of chlorinated wastes from PVC manufacturing (EDC tars) can be used to make chlorinated solvents including trichloroethene, but this method also results in the generation of large quantities of dioxins (EA 1997, ICI 1994). It has been used in vapour degreasing in 1920s, later it was introduced for use in dry cleaning but its use in this industry has declined sharply

since the 1950s. In the 1990s, 80-90% of trichloroethene worldwide was used for degreasing metals (IARC 1995). Use for all applications in Western Europe, Japan and the United States in 1990 was about 225 thousand tonnes. Other uses include processes requiring strong solvent action to dissolve rubbers and resins and in the manufacture of paints, lacquers and adhesives and oils from animal and vegetable matter. The textile industry also uses trichloroethylene as a solvent in waterless dying and finishing operations (ASTDR 1997). It is also used as a chain terminator in the production of polyvinyl chloride, has also been used in fire extinguishing and fire retarding applications (CEC 1986).

Trichloroethene is photochemically reactive compound and it can decompose in the presence of free radicals. Stabilisers such as epoxides (including the carcinogen epichlorohydrin) or combinations of epoxides, esters and amines are added to commercial trichloroethene to prevent it becoming acidic towards equipment and degreased materials (CEC 1986).

Trichloroethylene is not thought to occur naturally in the environment. However, it is present in most underground water sources and many surface waters as a result of the manufacture, use, and disposal of the chemical (Hughes *et al.* 1994, WHO 1993, ATSDR 1997).

Trichloroethene was found at different levels in drinking water supplies: in Galicia (Spain) in range of concentrations between 1 and 11.6ug/l (Freiria-Gandara *et al.* 1992), in drinking water samples from Zagreb, Croatia, contained 0.69 to 35.90 ug/l (ATSDR 1997), and up to 212 ug/l in the drinking water from two villages in Finland (Vartiainen *et al* 1993). It was also detected in the breath of people after inhalation and dermal exposure to tap water contaminated with trichloroethene (Weisel & Jo 1996). The World Health Organisation guideline value for trichloroethene in drinking water is 70ug/l, assuming that this route provides 10% of exposure (WHO 1993). The maximum contaminant level (MCL) for trichloroethylene in drinking water set by US Environmental Protection Agency is 5 ug/l (US EPA 1999). Some surface waters have been found to contain more than 400ug/l of this contaminant (CEC 1986).

Trichloroethylene easily dissolves in water, and it remains there for a long time. Under anaerobic conditions (for example in ground water) trichloroethene may degrade to more toxic compounds including vinyl chloride (Klier *et al.* 1999, Su & Puls 1999, WHO 1993). Trichloroethene itself could be formed as a degradation product of another chlorinated volatile compound – 1,1,2,2-tetrachloroethane (Loran & Olsen 1999).

Trichloroethylene may absorb to particles in water, which will cause it to eventually settle to the bottom sediment (ATSDR 1997). Soil contamination by trichloroethene has been reported with concentration ranging from below 1mg/kg to approximately 1500mg/kg (Ho *et al.* 1999). Trichloroethylene evaporates less easily from the soil than from water and may remain in soil for a long time.

Among the most heavily trichloroethene-exposed people are those working in the degreasing of metals, who are exposed by inhalation. Breathing large amounts of trichloroethylene may cause impaired heart function, coma, and death. Breathing it for long periods may cause nerve, lung, kidney, and liver damage. Inhaling small amounts for short periods of time may cause headaches, lung irritation, dizziness, poor co-ordination, and difficulty concentrating. Drinking large amounts of trichloroethylene may cause nausea, liver and kidney damage, convulsions, impaired heart function, coma, or death. Drinking small amounts of trichloroethylene for long periods may cause liver and kidney damage, nervous system effects, impaired immune system function and impaired foetal development in pregnant women, although the extent of some of these effects is not yet clear. Skin contact with trichloroethylene for short periods may cause skin rashes (ATSDR 1997).

Trichloroethene has been classified by International Agency for Research on Cancer in Group 2A (probably carcinogenic to humans) (IARC 1995). The most important observations are the elevated risk for cancer of the liver and biliary tract and the modestly elevated risk for non-Hodgkin's lymphoma. Trichloroethylene-contaminated groundwater may marginally increase a risk for non-Hodgkin's lymphoma. It has been shown to induce lung and liver tumours in various strains of mice (Fisher & Allen 1993, WHO 1993). It is also a weakly active mutagen in bacteria and yeast (WHO 1993).

Trichloroethene is in the first list of priority substances of the Commission Regulation (EC) No 1179/94 (EC1994) which is a part of the Council Regulation (EEC) No 793/93 on the evaluation and control of the risks of existing substances (EEC 1993).

Discharges of trichloroethene during several industrial processes (including production of tetrachloroethene and trichloroethene) and usage of trichloroethene for degreasing of metals are controlled by the European Community Legislation and special provisions are set relating to trichloroethene in the Council Directive 90/415/EEC (EEC 1990). The quality objective of 10ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters, and territorial waters) is set for trichloroethene in the same Directive.

Article 5(6) of the recent document (EC 1999) concerning limitation of emissions of volatile organic compounds due to the use of organic solvents in certain activities and installations says that substances or preparations which, because of their content of volatile organic compounds classified as carcinogens, mutagens, or toxic to reproduction, shall be replaced as far as possible by less harmful substances or preparations within the shortest possible time.

References

ATSDR (1997) ATSDR’s Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

CEC (1986) Trichloroethylene. In: Organochlorine solvents: health risk to workers. Publ: Commission of the European Communities. ISBN 0-85186-078-8: 93-130

EA (1997) Regulation of dioxin releases from the Runcorn operations of ICI and EVC. Information Report. Environmental Agency Licence GD03177G0003. Pub. By Crown. 36pp.
EC (1994) Commission Regulation (EC) No 1179/94 of 25 May 1994 concerning the first list of priority substances as foreseen under Council Regulation (EEC) No 793/93. OJ L 131: 3-4
EC (1999) Council Directive 1999/13/EC of 11 March 1999 on the limitation of emission of volatile organic compounds due to the use of organic solvents in certain activities and installations. OJ L 85: 1-22
EEC (1990) Council Directive of 27 July 1990 amending Annex II to Directive 86/280/EEC on limit values and quality objectives for discharges of certain dangerous substances included in list I of the Annex to Directive 76/464/EEC. OJ L 219:49-57
EEC (1991) Council Directive 91/692/EEC of 23 December 1991 standardizing and rationalizing reports on the implementation of certain Directives relating to the environment. OJ L 377: 48-54
EEC (1993) Council Regulation (EEC) No 793/93 of 23 March 1993 on the evaluation and control of the risks of existing substances. OJ L 84: 1-75
Fisher, J.W. & Allen, B.C. (1993) Evaluating the risk of liver cancer in humans exposed to trichloroethylene using physiological models. Risk Analysis 13(1): 87-95
Freiria-Gandara, M.J., Lorenzo-Ferreira, R.A., Alvarez-Devesa, A. & Bermejo, F. (1992) Occurance of halogenated hydrocarbons in the water supply of different cities of Galicia (Spain). Environmental Technology 13: 437-447
Ho, S.V., Athmer, C., Sheridan, P.W., Hughes, B.M., Orth, R., McKenzie, D., Brodsky, P.H., Shapiro, A.M., Sivavec, T.M., Salvo, J., Schultz, D., Landis, R., Griffith, R. & Shoemaker, S. (1999) The Lasagna technology for *in situ* soil remediation. 2. Large field test. Environmental Science and Technology 33(7): 1092-1099
Hughes, K., Meek, M.E. & Windle, W. (1994) Trichloroethylene: Evaluation of risks to health from environmental exposure in Canada. Environmental Carcinogenesis and Ecotoxicology Reviews. Journal of Environmental Science and Health 12(2): 527-543
IARC (1995) Trichloroethylene. IARC Monographs Vol.63, p.75
ICI (1994) Report of the Chief Inspector HMIP authorisation AK6039 Improvement Condition part 8, table 8.1, item 2: Formation of dioxines in oxychlorination, significance for human health and monitoring proposals. ICI Chemicals and Polymers report NWJP/BMTD, 27th April, 1994, 16pp.
Loran, M.M. & Olsen, L. (1999) Degradation of 1,1,2,2-tetrachloroethane in a freshwater tidal wetland: field and laboratory evidence. Environmental Science and Technology 33(2): 227-234
Su, C. & Puls, R.W. (1999) Kinetics of trichloroethene reduction by zerovalent iron and tin: pretreatment effect, apparent activation energy, and intermediate products. Environmental Science and Technology 33(1): 163-168
Vartiainen, T., Pukkala, E., Rienoja, T., Strandman, T., Kaksonen, K. (1993) Population exposure to trichloroethene and tetrachloroethene and cancer risk - 2 cases of drinking-water pollution. Chemosphere 27(7): 1171-1181
Weisel, C.P. & Jo, W-K (1996) Ingestin, inhalation, and dermal exposures to chloroform and trichloroethene from tap water. Environmental Health Perspectives 104(1): 48-51
WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

Tetrachloroethene

Tetrachloroethene is a clear liquid, which is heavier than water, with a sweet chloroform-like odour. Tetrachloroethene itself does not burn but it can produce poisonous gases in fire including hydrogen chloride and phosgene (US EPA 1989). Other names for tetrachloroethene include perchloroethylene and tetrachloroethylene.

Tetrachloroethene was first prepared in 1821 by Faraday by thermal decomposition of hexachloroethane (Hickman 1993). Tetrachloroethene is one of the most important chlorinated solvents worldwide and it has been produced commercially since the early

1900s. About 513 thousand tonnes were used in all applications in Western Europe, Japan and the United States in 1990 (IARC 1995). Tetrachloroethene is typically produced as a co-product with either trichloroethene or carbon tetrachloride from from hydrocarbons, partially chlorinated hydrocarbons, and chlorine (Hickman 1993). Oxychlorination of chlorinated wastes from PVC manufacturing (EDC tars) can be used to make chlorinated solvents including tetrachloroethene, but this method also results in the generation of large quantities of dioxins (EA 1997, ICI 1994). Most of the tetrachloroethene produced was used for the dry cleaning garments and smaller amounts were used for degreasing and in the production of chlorofluorocarbons (CFCs) (IARC 1995). However, this latter application will have been reduced since the Montreal Protocol banned CFC-11 and CFC-12 over most of the world (UNEP 1997). Tetrachloroethene was used in the textile industry for processing, finishing and sizing (US EPA 1998). Other uses include: insulating/cooling fluid in electric transformers; in typewriter correction fluids, as veterinary medication against worms, and it was once used as grain fumigant (US EPA 1998).

Tetrachloroethene is well known environmental contaminant. It has been detected in air, lakes, rainwater, seawater, rivers, soil, food and human tissues (ATSDR 1997; Bauer 1990; CEC 1986). It has also been found in drinking water at concentration in the range from 10ug/l to 180 ug/l (Bauer 1990; Freiria-Gandara *et al.* 1992; Vartiainen *et al.* 1993; CEC 1986). Contamination of well water with the concentration of 375ug/l was recorded at a waste disposal site due to tetrachloroethene leaching through soil (CEC 1986). The World Health Organisation guideline value for tetrachloroethene in drinking water is 40ug/l assuming that 10% of exposure comes from this source (WHO 1993). The maximum contaminant level (MCL) for tetrachloroethene in drinking water set by US Environmental Protection Agency is 5 ug/L (US EPA 1999). Tetrachloroethene has also been detected in the effluents from industrial plants and refineries and in sewage treatment plant effluents before and after chlorination (Santillo *et al.* 1997; US EPA 1989; CEC 1986).

The majority of the produced tetrachloroethene (80-85%) is lost in the atmosphere as a result of evaporation during production, storage and use (US EPA 1994; CEC 1986) and only 1% is released to water. Releases of tetrachloroethene to the environment are primarily from alkali and chlorine industries (US EPA 1998). In 1992, more than 12.3 million pounds (5584.2 tonnes) of perchloroethylene were released to the atmosphere, 10 thousand pounds (4.54 tonnes) to surface water, 13 thousand pounds (5.9 tonnes) to underground injection sites, and 9 thousand pounds (4.07 tonnes) to land from U.S. facilities (US EPA 1994). Once released into environment tetrachloroethene can undergo transformation. The degradation of tetrachloroethene through biotic mechanism includes the formation of lesser chlorinated compounds including trichloroethene, cis- and trans-1,2-dichloroethene, and vinyl chloride (Klier et al. 1999). In the air a photochemical degradation occurs with trichloroacethyl chloride as a major degradation product and phosgene a lesser one (CEC 1986).

The major route of human exposure to tetrachloroethene is from inhalation of contaminated urban air, especially near point sources such as dry cleaners, drinking

contaminated water from contaminated aquifers (US EPA 1998), drinking water distributed in pipelines with vinyl liners (Webler & Brown 1993), and inhalation of contaminated occupational atmospheres in metal degreasing and dry cleaning industries (US EPA 1998).

Exposure to very high concentrations of tetrachloroethene can cause dizziness, headaches, sleepiness, confusion, nausea, difficulty in speaking and walking, and unconsciousness (ATSDR 1997). Prolonged and frequently repeated dermal exposure can cause irritation, dryness, and dermatitis due to defatting (US EPA 1994). Tetrachloroethene is classified as Group 2A carcinogen (probably carcinogenic to humans) by the International Agency for Research on Cancer (IARC 1995). This compound induces leukemia in rats and increases risk for oesophageal cancer, non-Hodgkin's lymphoma and cervical cancer (IARC 1995). Tetrachloroethene has been shown to cause liver tumours in mice and kidney tumours in male rats (ASTDR 1997). It has been found that exposure to tetrachloroethene-contaminated drinking water was associated with an increased risk of leukemia and bladder cancer and that the risk was dose related (Aschengrau *et al.* 1993).

Specific provisions are set by the European Community Legislation relating to tetrachloroethene in the Council Directive 90/415/EEC (EEC 1990) which controls discharges of tetrachloroethene during several industrial processes (including production of tetrachloroethene, trichloroethene, carbon tetrachloride and chlorofluorocarbons) and usage of tetrachloroethene for degreasing of metals. The quality objective of 10ug/l for the aquatic environment (including inland surface waters, estuary waters, internal coastal waters other than estuary waters, and territorial waters) is set for tetrachloroethene in the same Directive.

Article 5(6) of the recent document (EC 1999) concerning limitation of emissions of volatile organic compounds due to the use of organic solvents in certain activities and installations says that substances or preparations which, because of their content of volatile organic compounds classified as carcinogens, mutagens, or toxic to reproduction, shall be replaced as far as possible by less harmful substances or preparations within the shortest possible time.

Tetrachloroethene is in the first list of priority substances of the Commission Regulation (EC) No 1179/94 (EC1994) which is a part of the Council Regulation (EEC) No 793/93 on the evaluation and control of the risks of existing substances (EEC 1993).

References

Aschengrau, A., Ozonoff, D., Paulu, C., Coogan, P., Vezina, R., Heeren, T. & Zhang, Y. (1993) Cancer risk and tetrachloroethylene-contaminated drinking water in Massachusetts. Archives of Environmental Health 48(5): 284-292

Bauer, U. (1990) Occurrence of tetrachloroethylene in the FRG. In: Organohalogen compounds. Vol.2: Dioxin'90 – EPRI-Seminar. Hutzinger, O. & Fiedler, H. [Ed] published by Ecoinforma Press, Bayreuth, FRG. pp385-388

CEC (1986) Perchloroethylene. In: Organochlorine solvents: health risk to workers. Publ: Commission of the European Communities, ISBN 0-85186-078-8: 191-224

EA (1997) Regulation of dioxin releases from the Runcorn operations of ICI and EVC. Information Report. Environmental Agency Licence GD03177G0003. Pub. By Crown. 36pp.
EC (1994) Commission Regulation (EC) No 1179/94 of 25 May 1994 concerning the first list of priority substances as foreseen under Council Regulation (EEC) No 793/93. OJ L 131: 3-4
EC (1999) Council Directive 1999/13/EC of 11 March 1999 on the limitation of emission of volatile organic comp[ouinds due to the use of organic solvents in certain activities and installations. OJ L 85: 1-22
EEC (1990) Council Directive of 27 July 1990 amending Annex II to Directive 86/280/EEC on limit values and quality objectives for discharges of certain dangerous substances included in list I of the Annex to Directive 76/464/EEC. OJ L 219: 49-57
EEC (1991) Council Directive 91/692/EEC of 23 December 1991 standardizing and rationalizing reports on the implementation of certain Directives relating to the environment. OJ L 377: 48-54
EEC (1993) Council Regulation (EEC) No 793/93 of 23 March 1993 on the evaluation and control of the risks of existing substances. OJ L 84: 1-75
Freiria-Gandara, M.J., Lorenzo-Ferreira, R.A., Alvarez-Devesa, A. & Bermejo, F. (1992) Occurrence of halogenated hydrocarbons in the water supply of different cities of Galicia (Spain). Environmental Technology, 13: 437-447
Hickman, J.C. (1993) Tetrachloroehylene. Chlorocarbons, -hydrocarbons ($CHCl=CCl_2$). In: Kirk-Othmer Encyclopedia of Chemical Technology. [Eds] Jacqueline I. Kroschwits; Mary Howe-Grant. Fourth edition, Vol. 6. John Wiley & Sons, Inc. pp.50-59
IARC (1995) Tetrachloroethylene. IARC Monographs Vol.63, p.159
ICI (1994) Report of the Chief Inspector HMIP authorisation AK6039 Improvement Condition part 8, table 8.1, item 2: Formation of dioxines in oxychlorination, significance for human health and monitoring proposals. ICI Chemicals and Polymers report NWJP/BMTD, 27th April, 1994, 16pp.
Klier, N.J., West, R.J. & Donberg, P.A. (1999) Aerobic biodegradation of dichloroethylenes in surface and subsurface soils. Chemosphere 38(5): 1175-1188
Santillo, D., Labounskaia, I., Stringer, R.L. & Johnston, P. A. (1997) Report on the analysis of industrial wastewaters from the Frutarom VCM/PVC plant near Haifa, Israel and adjacent shoreline sediments for organic contaminants. Greenpeace Research Laboratories Technical Note 03/97
UNEP (1997) The 1987 Montreal Protocol on Substances that Deplete the Ozone Layer, as adjusted and amended by the second Meeting of the Parties (London, 27-29 June, 1990), and by the fourth Meeting of the parties (Copenhagen, 23-25 November 1992), and further adjusted by the seventh Meeting of the Parties (Vienna, 5-7 December 1995), and further adjusted and amended by the ninth Meeting of the Parties (Montreal, 15-17 September 1997)
US EPA (1989) Tetrachloroethylene. US Environmental Protection Agency factsheets for regulated chemicals. Aquire Database, ERL-Duluth, US Environmental Protection Agency
US EPA (1994) Chemical summary for perchloroethylene. Office of Pollution Prevention and Toxics. US Environmental Agency
US EPA (1998) Technical factsheet on: Tetrachloroethylene. National primary drinking water regulations. Drinking water and health. Office of Ground Water and Drinking Water, US Environmental Protection Agency
US EPA (1999) Current drinking water standards. National primary and secondary drinking water regulations. Office of Ground Water and Drinking Water. US Environmental Protection Agency
Vartiainen, T., Pukkala, E., Rienoja, T., Strandman, T., Kaksonen, K. (1993) Population exposure to trichloroethene and tetrachloroethene and cancer risk - 2 cases of drinking-water pollution. Chemosphere 27(7): 1171-1181
Webler, T. & Brown, H.S. (1993) Exposure to tetrachloroethylene via contaminated drinking pipes in Massachusetts: a predictive model. Archives of Environmental Health 48(5): 293-297
WHO (1993) Guidelines for drinking-water quality. Vol.1: Recommendations. Second Edition, ISBN 92 4 154460 0, 188p.

Hexachloroethane

Hexachloroethane is a white crystalline solid with a camphor-like odour. It is non-flammable compound but it can decompose at high temperature (400-500^{0}C) to give

tetrachloroethylene, carbon tetrachloride and chlorine (Snedecor 1993). Hexachloroethane is formed as a by-product in many industrial chlorination processes designed to produce lower chlorinated hydrocarbons. Commercially it is produced by chlorination or oxychlorination of tetrachloroethene either in the presence of catalysts or under especial conditions (photochemical chlorination) (Snedecor 1993). Chlorination of hexachlorobutadiene has also been used to produce hexachloroethane. Additionally, hexachloroethane can be formed as a pyrolisis product during decomposition of trichloroethene and tetrachloroethene (Yasuhara & Morita 1990; Yasuhara 1993). It is also formed during incineration of materials containing chlorinated hydrocarbons (ATSDR 1996).

Historically hexachloroethane had wide and quite extensive applications. It was used in the past by the military in the production of pyrotechnic devices and screening smoke (DHHS 1998, Snedecor 1993). Hexachloroethane has been used in metal and alloy production, mainly in refining aluminium alloys. It was also used for removing impurities from molten metals, recovering metals from ores or smelting products, and improving the quality of various metals and alloys. Hexachloroethane has been used as a degassing agent for magnesium. Hexachloroethane has also been used as an additive in combustible liquids (ignition suppressant) and fire extinguishing fluids (smoke generated by hexachloroethane is used as a flame retardant). Hexachloroethane has had a variety of applications as a polymer additive. It has flame-proofing qualities, increases sensitivity to radiation cross-linking, and it is used as a vulcanising agent. Added to polymer fibres, hexachloroethane acts as a swelling agent and increases affinity for dyes. Hexachloroethane has also served as a fixer for some types of experimental photography and xerography (DHHS 1998, Snedecor 1993). At present hexachloroethane is not manufactured as an end-use product in the United, but it is formed as a by-product in the production of some chemicals (DHHS 1998, ATSDR 1996).

Hexachloroethane is not produced naturally in the environment. This compound is one of the most common environmental pollutants which was detected in air, surface water, drinking water and soil around chemical dump sites in the USA (DHHS 1998). Potential sources of hexachloroethane release to the environment include: formation during combustion and incineration of chlorinated wastes, release to air due to volatility and inefficient solvent recovery (hexachloroethane is an impurity in some chlorinated solvents), and formation during chlorination of sewage effluent prior to discharge (DHHS 1998). Hexachloroethane is also reported produced in small quantities from chlorination of raw water during drinking water treatment (IARC 1991).

If released to soil, this compound may persist for one year and could potentially contaminate groundwater where it can persist for up to 2 years (Howard *et al.* 1991). Hexachloroethane volatilises slowly from dry soil surfaces.

When hexachloroethane enters water, volatilisation appears to be the dominant removal mechanism (half-life in the surface water is up to 6 month). Moderate to slight adsorption to suspended solids and sediments may occur. Biodegradation is not expected to occur at a rate which would make this an important fate process in natural water systems.

However, under certain conditions (in the presence of sulphides) hexachloroethane can undergo reductive dehalogenation producing lower chlorinated compounds such as tetrachloroethylene, pentachloroethane and trichloroethylene (Miller *et al.* 1998; Butler & Hayes 1998). Bioconcentration of hexachloroethane in aquatic species has been demonstrated. It was shown that absorption of selected waterborne chloroethanes including hexachloroethane occurs in large adult fish and results in distribution kinetics similar to those observed in inhalation exposures (McKim *et al.* 1996).

If released to air, hexachloroethane exists almost entirely in the vapour phase. This compound is not expected to degrade in the troposphere. Half-life of hexachloroethane in the air is about 73 years (Howard *et al.* 1991). As a result of its persistence in the troposphere, long range transport is expected to occur.

The most probable routes of human exposure to hexachloroethane are dermal contact and ingestion of contaminated drinking water and, to a lesser extent inhalation (DHHS 1998). It has been listed among 148 hazardous air pollutants highlighted in the comprehensive evaluation of the potential public health implication of outdoor air toxic compounds concentrations across the United States (Woodruff *et al.* 1998).

Hexachloroethane has been reported to adversely affect the central nervous system and the eye (ATSDR 1996). The neurologic effects are mild, generally reported as an inability to close the eyelid, irritated, inflamed and watering eyes. Hexachloroethane is moderately irritating to the skin, mucous membranes, and liver in humans. Liver and kidney effects have been observed in animals acutely exposed to hexachloroethane by ingestion (ATSDR 1996; Bucher 1996). The US Department of Health and Human Services has determined that hexachloroethane may reasonably be anticipated to be a carcinogen (DHHS 1998). The International Agency for Research on Cancer classified hexachloroethane in Group 2B (possibly carcinogenic to humans).

The use and marketing of hexachloroethane is regulated by the European Community Legislation. EC directive 97/16/EC (EC 1997) added hexachloroethane to Annex I of Council Directive 76/769/EEC (EEC 1976) and banned most its uses in the non-ferrous metal industry. The only exceptions made were for some non-integrated foundries casting aluminium and for certain magnesium alloys.

References

ATSDR (1996) Toxicological profile for hexachloroethane (update). Atlanta, GA. US Department of Health and Human Services. Public Health Service

Bucher, J.R. (1996) NTR Technical Report on renal toxicity studies of selected halogenated ethanes administrated by gavage to F344/N rats. US Department of Health and Human Services. NIH Publication 96-3935, February 1996. 52pp.

Butler, E.C. & Hayes, K.F (1998) Effects of solution composition and pH on the reductive dechlorination of hexachloroethane by iron sulfide. Environmental Science and Technology 32(9): 1276-1284

DHHS (1998) Report on carcinogens. Summary. 8th Edition. US Department of Health and Human Services, 1998, 252pp.

EC (1997) Directive 97/16/EC of the European Parliament and of the Council of 10 April 1997 amending for the 15th time Directive 76/769/EEC on restrictions on the marketing and use of certain dangerous substances and preparations. OJ L 116: 31-32

EEC (1976) Council Directive 76/769/EEC of 27 July 1976 on approximation of the laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations. OJ L 262: 201-203

Howard, P.H., Boethling, R.S., Jarvis, W.F., Meylan, W.M. & Michalenko, E.M. (1991) Handbook of environmental degradation rates. Pub. By Lewis Publishers, Inc. ISBN 0 87371 358 3, 725pp.

IARC (1991) Chlorinated drinking water. IARC Monographs on the evaluation of carcinogenic risks to humans. Vol. 52. ISBN 92 832 1252 5, pp.45-128

McKim, J.M., Nichols, J.W., Lien, G.J., Hoffman, A.D., Galliant, C.A. & Stokes, G.N. (1996) Dermal absorption of three waterborne chloroethanes in rainbow trout (*Oncorhynchus mykiss*) and channel catfish (*Ictalurus punctatus*). Fundamental and Applied Toxicology 31: 218-228

Miller, P.L., Vasudevan, D., Gschwend, P.M. & Roberts, A.L. (1998) Transformation of hexachloroethane in sulfidic natural water. Environmental Science and Technology 32(9): 1269-1275

Snedecor, G (1993) Hexachloroethane. Chlorocarbons, -hydrocarbons ($CHCl=CCl_2$). In: Kirk-Othmer Encyclopedia of Chemical Technology. [Eds] Jacqueline I. Kroschwits; Mary Howe-Grant. Fourth edition, Vol. 6. John Wiley & Sons, Inc. pp.29-31

Woodruff, T.J., Axelrad, D.A., Caldwell, J., Morello-Frosch, R. & Rosenbaum, A. (1998) Public health implication of 1990 air toxics concentrations across the United Staes. Environmental Health Perspectives 106(5): 245-251

Yasuhara, A. & Morita, M. (1990) Formation of chlorinated compounds in pyrolisis of trichloroethylene. Chemosphere 21(4-5): 479-486

Yasuhara, A. (1993) Thermal decomposition of tetrachloroethylene. Chemosphere 26(8): 1507-1512

Chlorinated benzenes

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

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

1 chlorine 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).

1.Mono- and di-chlorobenzenes.

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

One of the earliest uses of chlorobenzene was as an intermediate for the explosive picric acid during the first World War (CEC 1986). It is used as a solvent and as an intermediate in chemical synthesis. In the US in the 1980s, the predominant use was for the production of ortho- and para-chlorobenzenes. Theses are used as intermediates for rubber chemicals, antioxidants, dyes and pigments, pharmaceuticals and agricultural chemicals. The fungicide benomyl, and carbofuran and the parathion group of insecticides are all derived from chlorobenzene. One previously important use was in the manufacture of DDT. Chlorobenzene production has fallen due to the development of other routes to aniline and phenol and the restriction of DDT use. 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 repellant was widespread in the 1970s (CEC 1986). Once absorbed, 1,4-dichlorobenzene is stored in the adipose tissue, and has been detected in human samples (CEC 1986, Ware 1988b). 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).

2.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) and sediment (Labunska *et al.* 1998), though it was not found in human adipose tissue from Canada (Hermanson *et al.* 1997). Little is known about its toxicity other than its ability to damage the liver, kidney and thyroid (Giddings *et al.* 1994a).

More information is available about 1,2,4-trichlorobenzene. According to Giddings et al. (1994a), only 1,2,4-trichlorobenzene has industrial application in Canada. 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).

3.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).

4.Pentachlorobenzene

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

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

5.Hexachlorobenzene

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

HCB emissions from waste incineration, metallurgical industries and burning of chlorinated fuels are highlighted (UNECE 1998)(Annex V).

HCB is toxic to aquatic life, land plants, land animals, and humans. It is listed by the IARC as a Group 2B carcinogen, *i.e.* possible carcinogen to humans and also appears to be a tumour promoter. Hexachlorobenzene may damage the developing foetus, liver, immune system, thyroid and kidneys and CNS. The liver and nervous system are the most sensitive to its effects. Porphyria is a common symptom of HCB toxicity. High or repeated exposure may damage the nervous system, and can cause irritability, difficulty with walking and co-ordination, muscle weakness, tremor and/or a feeling of pins and needles on the skin. Repeated exposure, especially when skin effects occur, can lead to permanent skin changes, such as changes in pigmentation, tight, thickened skin, easy wrinkling, skin scarring, fragile skin, and increased hair growth, especially on the face and forearms (ATSDR 1997, Newhook & Meek 1994). Recent research (van Birgelen 1998) suggests that HCB has dioxin-like toxicity and more epidemiological studies should be undertaken especially concerning infants fed breast milk in countries with HCB exposure levels.

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

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

References

ASTDR (1997). Toxicological profiles. U.S. Department of Health and Human Services, Public Health Service, CRC Press Inc

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

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

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

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

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

EEC (1976) Council Directive 76/464/EEC of 4 May 1976 on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community. OJ L 129: 23-29 as amended. European Community Environmental Legislation Volume 7: Water.

EEC (1986) Council Directive 86/280/EEC of 12 June 1986 on limit values and quality objectives for discharges of certain dangerous substances included in List 1 of the Annex to Directive 76/464/EEC. European Community Environmental Legislation Volume 7: Water.

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

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

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

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

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

Labunska, I., Stringer, R., Santillo, D. & Stephenson, A. (1998) Identification and environmental significance of organic pollutants and heavy metals in samples associated with Rhodia S.A. and Carbocloro Industrias Quimicas S.A., Cubatao and Sao Vicente, Brazil, 1998. Greenpeace Research Laboratories Technical Note 20/98

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

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

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

MINDEC (1995) Ministerial Declaration of the Fourth International Conference on the Protection of the North Sea. 8-9 June 1995, Esjberg, Denmark.
Newhook, R. & Mek, M.E. (1994) Hexachlorobenzene: Evaluation of risks to health from Environmental Exposure in Canada. Environ. Carcino. & Ecotox. Revs. C12(2): 345-360
OSPAR (1998a) OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Sintra Statement, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998.
OSPAR (1998b) OSPAR Convention for the Protection of the Marine Environment of the North East Atlantic. OSPAR Strategy with regard to Hazardous Substances, Reference no. 1998-16, OSPAR 1998/14/1/Annex 34, Ministerial Meeting of the OSPAR Commission, Sintra b22-23 July 1998.
Sala, M., Sunyer, O., Otero, R., Santiago-Silva, M., Ozalla, D., Herrero, C., To-Figueras, J., Kogevinas, M., Anto, J., Camps, C. & Grimalt, J. (1999) Health effects of chronic high exposure to hexachlorobenzene in a general population sample. Arch. Of Environ. Health 54(2): 102-109
Sicko-Goad, L. & Andersen, N.A. (1993a) Effect of lipid composition on the toxicity of trichlorobenzene isomers to diatoms. I. Short-term effects of 1,3,5-trichlorobenzene. Arch. Environ. Contam. Toxicol. 24: 236-242
Sicko-Goad, L. & Andersen, N.A. (1993b) Effect of lipid composition on the toxicity of trichlorobenzene isomers to diatoms. II. Long-term effects of 1,2,3-trichlorobenzene. Arch. Environ. Contam. Toxicol. 24: 243-248
Sicko-Goad, L., Evans, M.S., Lazinsky, D., Hall, J. & Simmons, M.S. (1989d) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. IV. Pentachlorobenzene and comparison with trichlorobenzene isomers. Arch. Environ. Contam. Toxicol. 18: 656-668
Sicko-Goad, L., Hall, J., Lazinsky, D. & Simmons, M.S. (1989b) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. II. 1,3,5-Trichlorobenzene. Arch. Environ. Contam. Toxicol. 18: 638-646
Sicko-Goad, L., Hall, J., Lazinsky, D. & Simmons, M.S. (1989c) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. III. 1,2,3-Trichlorobenzene. Arch. Environ. Contam. Toxicol. 18: 647-655
Sicko-Goad, L., Lazinsky, D., Hall, J. & Simmons, M.S. (1989a) Effect of chlorinated benzenes on diatom fatty acid composition and quantitative morphology. I. 1,2,4-Trichlorobenzene. Arch. Environ. Contam. Toxicol. 18: 629-637
UNECE (1998) Protocol to the 1979 Convention on long-range transboundary air pollution on persistent organic pollutants. Publ UNECE, 61pp.
UNEP (1995) Decision 18/32 of the UNEP Governing Council: Persistent Organic Pollutants. UNEP Governing Council, 25th May 1995.
UNEP (1997) Decisions adopted by the Governing Council at its nineteenth session: 13c. International action to protect human health and the environment through measures which will reduce and/or eliminate emissions and discharges of persistent organic pollutatns, including the development of an international legally binding instrument. UNEP Governing Council, 7th February 1997.
Van Birgelen, A.P.J.M. (1998) Hexachlorobenzene as a possible major contributor to the dioxin activity of human milk. Environ. Health Persp. 106(11): 683-688
Ware, G.W. (Ed.)(1988a) Chlorobenzene. Rev. Environ. Contam.Toxicol. 106: 37-49
Ware, G.W. (Ed.)(1988b) Ortho-, meta- and para- dichlorobenzene. Rev. Environ. Contam.Toxicol. 106: 51-6

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).

References

Andrews, L.S. & Snyder, R. (1986) Toxic effects of solvents and vapors. In Toxicology: the basic science of poisons. Klaasen C.D., Ambur M.O. and Doull J. [Eds], MacMillan Publishing Co., New York: 636-668

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

Chalaux N., Takada H., Bayona J.M. (1995) Molecular markers in Tokyo Bay sediments – sources and distribution. Marine Environmental Research, Vol., 40, No.1, pp.77-92

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

Overton, E.B. (1994). Toxicity of petroleum. In: Basic Environmental Toxicology. Cockerham & Shane [Eds], Chapter 5: 133-156

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

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).

References

ATSDR (1997) ATSDR's toxicological Profiles on CD-ROM. Agency for Toxic Substances and Disease Registry, US Public Health Service, Publ: Lewis Publishers.

Carson, R. (1962) Silent Spring. Publ: Penguin, London, ISBN 0-14-027371-9, 323pp.

De Cruz I., Mougin C. & Grolleau G. (1997). Chlorinated hydrocarbons in eggs of grey heron (Ardea cinerea L.) in France (Lac de Grandlieu). Chemosphere 35 (5): 1003-1009.

de Wit C.A., de March B.G.E. & Muir D.C.G. (1997). An overview of the AMAP assessment of persistent organic pollutants in the Arctic: Biological effects. In: The AMAP International Symposium on Environmental Pollution of the Arctic. Extended Abstracts, volume 1. Tromso, Norway June 1-5, 1997.

DHHS (1998) 8th Report on Carcinogens. 1998 Summary. Publ: US Department of Health and Human Services, 252pp.

Dinham, B (1993) The pesticide hazard: a global health and environmental audit. Publ: the Pesticides Trust/Zed Books, ISBN 1 85649 201 X Hb, 228pp.

FAO/UNEP (1991) Decision guidance documents: aldrin, DDT, dieldrin, dinoseb and dinoseb salts, fluoroacetamide, HCH (mixed isomers). Publ; joint FAO/UNEP Programme for the operation of Prior Informed Consent, Rome/Geneva.

Garcelon, D.K. & Thomas, J.N.(1997) DDE Poisoning in an Adult Bald Eagle. Journal of Wildlife Diseases 32(2):299-303

Haegele, M.A. & Tucker, R.K.(1974) Effects of 15 Common Environmental Pollutants on Eggshell Thickness in Mallards and Coturnix. Bulletin of Enviromental Contamination &Toxicology 11(1):98-102

Longnecker, M.P., Rogan, W.J. & Lucier, G. (1997) The human health effects of DDT (dichlorodiphenyltrichloroethane) and PCBs (polychlorinated biphenyls) and an overview of organochlorines in public health. Annu. Rev. Public Health 18: 211-244

Meister, R.T. [Ed] (1992). Farm Chemicals Handbook '92, Meister Publishing Co., Willoughby, OH.

Newton I., Wyllie I & Asher A. (1993). Long-term trends in organochlorine and mercury residues in some predatory birds in Britain. Environmental Pollution 79: 143-151.

Newton, I. (1995) The contribution of some recent research on birds to ecological understanding. Journal of Animal Ecology 64: 675-696

Newton, I., Bell, A.A. & Wyllie, I. (1982) Mortality of sparrowhawks and kestrels. British birds 75(5): 195-204

Peakall, D.B. & Lincer, J.L.(1996) Do PCBs cause eggshell thinning? Environmental Pollution 91(1):127-129

Provini, A.& Glassi S. (1999) Polychlorinated Biphenyls and Chlorinated Pesticides in Bird Eggs from Calabria (Southern Italy). Ecotoxicology and Enviromental Safety 43:91-97

RSC (1991) The Agrochemicals Handbook, 3rd Edition. Publ: Royal Society of Chemistry Information Services, Cambridge, UK.

Smith, A. G. (1991) Chlorinated hydrocarbon insecticides IN: Handbook of Pesticide Toxicology, Volume 2, Classes of Pesticides, Hayes, W.J.Jr. & Laws, E.R.Jr. (Eds.), Publ: Academic Press, Inc, pp731-915
WHO (1979) DDT and its derivatives. Environmental Health Criteria 9. World Health Organizations, Geneva, 1979, 194pp.
WWF (1998) Resolving the DDT dilemma. Publ: WWF US & WWF Canada, Washington & Toronto, 52pp.

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, and 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).

References

ATSDR (1997) ARSDR's Toxicological profiles on CD ROM. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service. CRC Publishers

Botta, D. Dancelli, E. & Mantica, E. (1996) A case history of contamination by polychloro-1,3-butadiene. Environmental Science and Technology 30(2): 453-462

Choudhary, G. (1995) Human health perspecives on environmental exposure to hexachlorobutadiene: a review. Environ. Carcino. & Ecotox. Revs. C13(2): 179-203

Driscoll, T.R., Hamdam, H.H., Wang, G., Wright, P.F.A. & Stacey, N.H. (1992) Concentrations of individual serum or plasma bile acids in workers exposed to chlorinated aliphatic hydrocarbons. British Journal of Industrial Medicine 49: 700-705

Goodrichmahoney, J.W., I.P. Murarka, L.J. Hocombe and M.E. Horn (1993). Pentachlorophenol-treated wood poles and crossarms - toxicity characteristic leaching procedure (tclp) results. Environment International 19(6): 535-542.

IARC (1999). 1,1,2,3,4,4-Hexachlorobutadiene. IARC Monographs. International Agency for Research on Cancer. Geneva. Vol. 73, p. 277

IRIS (1993) Hexachlorobutadiene. Integrated Risk Information System. US EPA, Washington, DC. May 14, 1993

Johnston, P.A., R.L. Stringer, R. Clayton and R.J. Swindlehurst (1994). Regulation of toxic chemicals in the North Sea: The need for an adequate control strategy. North Sea Monitor June 1994: 9-16.

Jonker, D., Woutersen, R.A. & Feron, V.J. (1996) Toxicity of mixutes of nephrotoxicants with similar or dissimilar mode of action. Food and Chemical Toxicology 34: 1075-1082

Santillo, D. & Labounskaia, I. (1997a) Petkim: Organic Analytical report 1: Analysis of marine sediments and seawater from locations adjacent to the main wastewater discharges of the Petkim chemical complex, Aliaga, Turkey. Greenpeace Research Laboratories Technical note 08/97

Santillo, D. & Labounskaia, I. (1997b) Petkim: Organic analytical report 2: Analysis of wastes generated by integrated chemical production processes, including the manufacture of VCM and chlorinated organic solvents, at the Petkim chemical complex, Aliaga, Turkey. Greenpeace Research Laboratories Technical Note 09/97

US EPA (1986). Factsheets for regulated chemicals: Hexachlorobutadiene. Office of Environmental Health Hazard Assessment.

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.

References

Abd-Allah, A.M.A. (1994) Residue levels of organochlorine pollutants in fish from Abu-Quir Bay and Idku Lake, Alexandria, Egypt. Toxicological and Environmental Chemistry 44: 65-71

ASTDR (1997) ATSDR's toxicological Profiles on CD-ROM. Agency for Toxic Substances and Disease Registry, US Public Health Service, Publ: Lewis Publishers.

Bunton T.E. (1996). Experimental chemical carcinogenesis in fish. Toxicologic Pathology, Vol.24, No.5, Pp.603-618.

EEC (1984) Council Directive 84/491/EEC of 9 October 1984 on limit values and quality objectives for discharges of hexachlorocyclohexane. OJ L 274: 11-17

El-Gendy, K.S., Abdalla, A.A., Aly, H.A., Tantawy, G. & El-Sebae, A.H. (1991) Residue levels of chlorinated hydrocarbon compounds in water and sediment samples from Nile branches in the delta, Egypt. J. Environ. Sci. Health B26(1): 15-36

FAO/UNEP (1998) Rotterdam Convention on the prior informed consent procedure for certain hazardous chemicals and pesticides in international trade. Publ. FAO/UNEP

Hernandez, L.M., Fernandez, M.A. & Gonzales, M.J. (1992) Organochlorine pollutants in water, soils, and earthworms in the Guadalquivir, Span. Bull. Environ. Contam. Toxicol. 49: 192-198

Martijn, A., Bakker, H. & Schreuder, R.H. (1993) Soil persistence of DDT dieldrin, and lindane over long period. Bull. Environ. Contam. Toxicol. 51: 178-184

Nagata K., Huang C.S., Hamilton B.J., Carter D.B., Narahashi T. (1996). Differential-effects of hexachlorocyclohexane on the gaba receptor subunits expressed in human embrionic kidney-cell line. Brain Research, Vol.738, No.1, pp.131-137.

Norstom, R.J. & Muir, D.C.G. (1994) Chlorinated hydrocarbon contaminations in arctic marine mammals. The Science of the Total Environment 154: 107-128

Ramesh, A., Tanabe, S., Murase, H., Subramanian, A.N. & Tatsukawa, R. (1991) Distribution and behaviour of persistant organochlorine insecticides in paddy soil and sediments in the tropical environment: A case study in South India. Environmental Pollution 74: 293-307

Romijn C.A.F.M., Luttik R., Canton J.H. (1994). Presentation of a general algorithm to include effect assessment on secondary poisoning in the derivation of environmental-quality criteria.2. Terrestrial food-chains.Ecotoxicology and environmental safety, 1994, Vol.27, No.2, pp.107-127.

Schulz R., Liess M. (1995).Chronic effects of low insecticide concentrationson fresh-water *Caddisfly Larvae*. Hydrobiologia, Vol.299, No.2, pp.103-113.

Skark, C. & Zullei-Seibert, N. (1995) The occurrence of pesticides in groundwater – results of case-studies. Intern. J. Environ. Anal. Chem. 58: 387-396

Smith A.G. (1991). Chlorinated Hydrocarbon Insecticides. In: Handbook of pesticide toxicology. Volume 2. Classes of pesticides. [Eds] Hayes W.J. and Laws E.R. Academic Press, Inc., pp.731-860.

Tan, G.H. & Vijayaletchumy, K. (1994) Organochlorine pesticide residue levels in peninsular Malaysian rivers. Bull. Environ. Contam. Toxicol. 53: 351-356

Toppari J., Larsen J.C., Christiansen P., Giwercman A., Grandjean P., Guillette L.J., Jegou B., Jensen T.K., Jouannet P., Keiding N., Leffers H., McLachlan J.A., Meyer O., Muller J., Rajpert-De Meyts E., Scheike T., Sharpe R., Sumpter J., Skakkebaek N/E. (1995). Ministry of Environment and Energy. Denmark. Male reproductive health and environmental chemicals with estogenic effects. Miljoproject nr 290 1995. Copenhagen. Danish Environmental Protection Agency, 1995.

UNECE (1998) Protocol to the Convention on Long-Range Transboundary Air Pollution (LRTAP) on Persistent Organic Pollutants. Adopted Aarhus, June 1998, United Nations Economic Council for Europe.

Waliszewski, S.M., Pardio Sedas, V.T., Chantiri P., J.N., Infanzon R., R.M. & Rivera, J. (1996) Organochlorine pesticide residues in human breast milk from tropical areas in Mexico. Bull. Environ. Contam. Toxicol. 57: 22-28

Xu, Y & Zhang, Y. (1994) Hexachlorocyclohexane (HCH) residues in Ya-Er Lake area, China. Intern. J. Environ. Anal. Chem. 57: 53-61

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 its 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).

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 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).

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 a 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 affecting crop yield and soil fertility. Soil concentrations ranging from 70-400 mg/kg are classified as critical, above which toxicity is considered likely (Alloway 1990). It was the observed phytotoxicity of zinc in sewage-sludge amended soils, that led several countries to formulate guidelines for sludge usage (Alloway 1990)

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).

References

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

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

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

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

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

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

Budavari, S.M., O'Neil, J., Smith, A. and Heckleman, P.E. [Eds] (1989). The Merck Index: an encyclopaedia of chemicals, drugs and biologicals. 11TH Edn Merck and Co, Inc., New Jersey, USA
Bureau of Indian Standards (1995). Drinking water specifications. Second Edition, UDC 628.1.033
Dudka, S. and Adriano, D.C. (1997). Environmental impacts of metal ore mining and Processing: a review. J. Environ. Qual. 26: 590-602
Dudka, S., Piotrowska, M., Chlopecka, A. and Witek, T. (1995). Trace metal contamination of soils and crop plants by the mining and smelting industry in Upper Silesia, South Poland. Journal of Geochemical Exploration 52: 237-250
EEC (1975). Council Directive 75/440/EEC of 16 June 1975 concerning the quality required of surface water intended for the abstraction of drinking water in the Member States (OJ L 194, 25.7.1975, p. 26)
EEC (1976). Council Directive 76/464/EEC of 4 May 1976 on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community (OL J 129, 18.5.1976, p. 23)
EEC (1978). Council Directive 78/659/EEC of 18 July 1978 on the quality of fresh waters needing protection or improvement in order to support fish life (OJ L 222, 14.8.1978, p. 1)
EEC (1980). Council Directive 80/778/EEC of 15 July 1980 relating to the quality of water intended for human consumption (OJ L 229, 30.8.1980, p. 11)
Goyer, R.A. (1996). Toxic effects of metals. In Casarett & Doull's Toxicology. The Basic Science of Poisons, Fifth Edition, Klaassen, C.D. [Ed]. McGraw-Hill Health Professions Division, ISBN 0071054766
Jung, M.C. and Thornton, I. (1996). Heavy metal contamination of soils and plants in the vicinity of a lead-zinc mine, Korea. Applied Geochemistry 11: 53-59
Kroschwitz, J.I and Howe-Grant, M. [Eds] (1995). Kirk-Othmer Encyclopaedia of Chemical Technology Volume 7.Fourth Edition. Zinc and zinc alloys. John Wiley and Sons, New York, Chichester, Brisbane, Toronto and Singapore
Mance, G. and Yates, J. (1984). Proposed environmental quality standards for List II substances in water: zinc. Water Research Centre Technical Report TR209
Matthews, H. and Thornton, I. (1982). Seasonal and species variation in the content of cadmium and associated metals in pasture plants at Shipham. Plant Soil 66: 181-193
Nriagu, J.O. (1990). Global metal pollution. Poisoning the biosphere? Environment 32, 7: 7-11;28-33
Nriagu, J.O. and Pacyna, J.M. (1988). Quantitative assessment of World-wide contamination of air, water and soils with trace metals. Nature 333: 134-139
Pain, D.J., Sanchez,A. and Meharg, A.A.(1998). The Donana ecological disaster: contamination of a world heritage estuarine marsh ecosystem with acidified pyrite mine waste. The Science of the Total Environment 222: 45-54
Salomons, W. and Forstner, U. (1984). Metals in the hydrocycle. Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, ISBN 3540127550
UNEP (1993). Preliminary assessment of the state of pollution of the Mediterranean Sea by zinc, copper and their compounds and proposed measures. Mediterranean Action Plan UNEP (OCA)/MED/WG.66/Inf.3, Athens 3-7 May 1993
USPHS (1997). Toxicological profile for zinc on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service
Verriopoulos, G. and Hardouvelis, D. (1988). Effects of sub-lethal concentrations of zinc on survival and fertility in four generations of Tisbe. Marine Pollution Bulletin 19: 162-166
World Health Organisation (1993). Guidelines for drinking-water quality. Volume 1: Recommendations. ISBN 9241544600

Lead (Pb)

1. Occurrence, Production and Use

There is a general agreement that the abundance of lead in the average crustal rock is approximately 16 mg/kg; although black shales, rich in organic matter and sulphide minerals, can have far higher concentrations (Alloway 1990). Lead is chiefly obtained from the sulphide ore galena, by a roasting process; and is currently mined in 47

countries, making it one of the most widespread metals in terms of primary production. In addition to this, secondary smelters, processing lead metal products, are located in 43 countries, reflecting widespread recycling of lead in electric storage batteries (Dudka and Adriano 1997).

Natural sources of atmospheric lead include wind-borne soil particles and volcanic emissions, forest fires, biogenic processes and sea salt sprays. The total amount of lead released to the atmosphere from natural sources is estimated at 12,000 tonnes / year, compared with an estimated anthropogenic load of 332,000 tonnes /year (Nriagu 1990).

Uses of lead and its compounds are extensive. As a metal, it is often used as pipe-work for water distribution, or as containers for storing for corrosive liquids (e.g. sulphuric acid). Its alloys are used in welding, printing and as anti-friction metals; and great quantities, both of the metal and its dioxide, are used in electric storage batteries. Other uses include cable coverings, ammunition, and in the manufacture of lead tetraethyl, used as an anti-knock compound in petrol. Compounds of lead are used as paint pigments, PVC stabilisers, pesticides, varnishes, lubricants, as glazes for pottery and porcelain, and in leaded glass crystal (Budavari et al. 1989, USPHS 1997).

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

SOURCE	Emission (thousand tonnes / year)
Transportation (anti-knock petrol additives)	248
Non ferrous metal production (Pb, Zn, Cu, Ni, Cd)	46.5
Steel and iron manufacturing	15.7
Energy production	12.7
Commercial uses (cement, chemicals, paint, ceramics)	4.5
Mining	2.55
Waste incineration (municipal refuse and sewage sludge)	2.37
TOTAL	332.32

Table 1 World-wide atmospheric emissions of lead from anthropogenic sources

SOURCE	Emission (thousand tonnes / year)
Atmospheric fallout	100
Manufacturing processes (metal, chemicals, paper, petroleum products)	14
Sewage discharges	9.4
Base metal mining and smelting	7.0
Domestic wastewaters	6.8
Electric power plants	0.72
TOTAL	137.92

Table 2 World-wide inputs of lead into aquatic ecosystems

SOURCE	Emission (thousand tonnes / year)
Discarded manufactured products	292
Atmospheric fallout	232
Coal ashes	144
Urban refuse	40
Agricultural and animal wastes	26
Solid wastes from metal fabrication	7.6
Logging and wood wastes	7.4
Municipal sewage and organic waste	7.1
Fertilisers and peat	2.9
TOTAL	759

Table 3 World-wide inputs of lead into soils

2. Environmental Levels, Contamination and Behaviour

lead is present in uncontaminated aquatic and terrestrial ecosystems at relatively low levels (see Table 4).

Environmental Matrix	Concentration	Reference
Seawater (estuarine waters around England and Wales)	24-880 ng/l	Law et al. 1994
Seawater (open ocean)	5 ng/l 1-14 ng/l 20-71 ng/l	UPHS 1997 Bryan and Langston 1992 Law et al. 1994
Freshwater (mean value from 39,490 measurements)	3.9 ug/l	USPHS 1997
Drinking water	<5-30 ug/l	USPHS 1997
Soil	10-30 mg/kg	Alloway 1990
Freshwater / marine sediment	20-30 mg/kg	USPHS 1997

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

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

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

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

Table 5 lead concentrations associated with anthropogenic contamination

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

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

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

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

concentrations are found in aquatic and terrestrial organisms that live near to lead mining, smelting, and refining facilities; storage battery recycling plants; areas affected by high automobile and truck traffic; sewage sludge and spoil disposal areas; sites where dredging has occurred; areas of heavy hunting (spent lead shot); and in urban and industrialised areas (USPHS 1997).

3. Toxicity

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

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

Animals studies have reproduced many of the toxic effects listed above, and animals feeding close to smelting, mining and recycling facilities, have often ingested levels of lead that have resulted in poisoning and death (Henny *et al.* 1991, Blus *et al.* 1991, USPHS 1997, WHO 1989, Collivignarelli *et al.* 1986). In addition, birds feeding on contaminated prey or ingesting lead shot into their gizzards, can be exposed to severe levels of lead. Resulting in high kidney, liver and bone concentrations, reduced growth and development, behavioral abnormalities, and sometimes death (Mateo *et al.* 1997, WHO 1989).

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

report a reduction in growth, fertility and reproduction suppression, and mortality, at ug/l (parts per billion) concentrations (WHO 1989).

4. Legislation

European legislation concerned with water quality and permissible environmental levels, does not generally treat lead as a priority pollutant. However, anthropogenic discharges of lead into the aquatic environment still need to cease, and therefore lead is included on the majority of subsidiary and secondary lists. 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 50 ug/l 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. lead is included in List II, and as such water pollution caused by its presence must be reduced in keeping with National Environmental Quality Standards.

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 Bureau of Indian Standards, which currently set a maximum permissible concentration for lead of 50 ug/l (1995); the USEPA, which limits the concentration of lead to 15 ug/l (USPHS 1997); and the WHO, which currently recommends a limit of 50 ug/l, however there is much current discussion and desire to reduce this to 10 ug/l (WHO 1993).

The Water Research Centre in the UK recommends the following Environmental Quality Standards for lead: protection of freshwater salmonid fish 4-20 ug/l; protection of freshwater coarse fish 50-250 ug/l; protection of other freshwater life and associated non-aquatic organisms 5-60 ug/l; protection of saltwater fish, shellfish, other salt water life and associated non-aquatic organisms 25 ug/l (Brown *et al.* 1984).

Regarding soil contamination, the UK Department of the Environment (ICRCL) regards a level of 0-500 mg/kg as being typical of uncontamination. Anything above this is regarded as being contaminated, and as such, restrictions on recreational and agricultural uses apply (Alloway 1990). In terms of permissible sewage sludge levels, acceptable concentrations for lead range from 300-1200 mg/kg. However resulting soil concentrations should not exceed 50-100 mg/kg (recommended EC, UK, France, Germany) (Alloway 1990).

Finally, lead 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 level within the next 25 years. An objective further reinforced in the Sintra Statement at the 1998 Ministerial Meeting of the OSPAR Commission (OSPAR 1998a). Lead and organic lead compounds were selected for priority action, and as such as included in Annex 2 of the OSPAR Strategy with regard to Hazardous Substances (OSPAR 1998b).

References

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

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

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

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

Brown, V.M, Gardiner, J. and Yates, J. (1984). Proposed environmental quality standards for List II substances in water. Inorganic lead. Water Research Centre Technical Report TR208

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

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

Bureau of Indian Standards (1995). Drinking water specifications. Second Edition, UDC 628.1.033

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

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

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

EEC (1975). Council Directive 75/440/EEC of 16 June 1975 concerning the quality required of surface water intended for the abstraction of drinking water in the Member States (OJ L 194, 25.7.1975, p. 26)

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

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

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

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

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

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

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

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

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

MINDEC (1995) Ministerial Declaration of the Fourth International Conference on the Protection of the

North Sea. 8-9 June 1995, Esjberg, Denmark.
Nriagu, J.O. (1988). A silent epidemic of environmental metal poisoning. Environmental Pollution 50: 139-161
Nriagu, J.O. (1990). Global metal pollution. Poisoning the biosphere? Environment 32, 7: 7-33
Nriagu, J.O. and Pacyna, J.M. (1988). Quantitative assessment of world-wide contamination of air, water and soils with trace metals. Nature 333: 134-139
OSPAR (1998a). OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Sintra Statement, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998.
OSPAR (1998b). OSPAR Convention for the Protection of the Marine Environment of the North East Atlantic. OSPAR Strategy with regard to Hazardous Substances, Reference no. 1998-16, OSPAR 1998/14/1/Annex 34, Ministerial Meeting of the OSPAR Commission, Sintra b22-23 July 1998.
Pain, D.J., Sanchez,A. and Meharg, A.A.(1998). The Donana ecological disaster: contamination of a world heritage estuarine marsh ecosystem with acidified pyrite mine waste. The Science of the Total Environment 222: 45-54
Pirkle, J.L., Kaufman, R.B., Brody, D.J., Hickman, T., Gunter, E.W. and Paschal, D.C. (1998). Exposure of the U.S. population to lead, 1991-1994. Environmental Health Perspectives 106, 11: 745-750
Sadiq, M. (1992). Toxic metal chemistry in marine environments. Marcel Dekker Inc., New York, Basel, Hong Kong. ISBN 0824786475
Sanchez, J., Carmen Vaquero, M. and Legorburu, I. (1994). Metal pollution from old lead-zinc mine works: biota and sediment from Oiartzun Valle. Environmental Technology 15: 1069-1076
Sauve, S., McBride, M.B. and Hendershot, W.H. (1997). Speciation of lead in contaminated soils. Environmental Pollution 98, 2: 149-155
USPHS (1997). Toxicological profile for lead on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service
World Health Organisation (1989). Lead- environmental aspects. Environmental Health Criteria 85. ISBN 9241542853
World Health Organisation (1993). Guidelines for drinking-water quality. Volume 1: Recommendations. ISBN 9241544600

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 non-sensitised individuals would have to ingest or inhale a large amount of nickel to suffer adverse health effects, sensitised individuals react adversely to far lower concentrations (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).

References

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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).

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

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).

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 chlor-alkali 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 affected 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).

References

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

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

Barbosa, A.C. (1997). Mercury in Brazil: present or future risks? Journal of the Brazilian Association for the Advancement of Science 49,1/2: 111-116

Barbosa, A.C., Boischio, A.A., East, G.A., Ferrari, I., Goncalves, A., Silva, P.R.M. and da Cruz, T.M.E. (1995). Mercury contamination in the Brazilian Amazon. Environmental and occupational aspects. Water, Air and Soil Pollution 80: 109-121

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

Bureau of Indian Standards (1995). Drinking water specifications. Second Edition, UDC 628.1.033

D'Itri, F.M. (1991). Mercury contamination: what we have learned since Minamata. Environmental Monitoring and Assessment 19: 165-182

EEC (1975). Council Directive 75/440/EEC of 16 June 1975 concerning the quality required of surface water intended for the abstraction of drinking water in the Member States (OJ L 194, 25.7.75, p. 26)

EEC (1976). Council Directive 76/464/EEC of 4 December 1976 on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community (OJ L 353, 17.12.90, p. 59)

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

Gonzalez, H. (1991). Mercury pollution caused by a chlor-alkali plant. Water, Air and Soil Pollution 56: 83-93

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

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

Harada, M. (1997). Neurotoxicity of methylmercury; Minamata and the Amazon. In Mineral and Metal Neurotoxicology. Yasui, M., Strong, M.J., Ota, K. and Verity, M.A.[Eds]. CRC Press Inc., ISBN 0849376645

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

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

Naimo, T.J. (1995). A review of the effects of heavy metals on freshwater mussels. Ecotoxicology 4: 341-362
Nriagu, J.O. (1990). Global metal pollution. Poisoning the biosphere? Environment 32, 7: 33
Nriagu, J.O. and Pacyna, J.M. (1988). Quantitative assessment of world-wide contamination of air, water and soils with trace metals. Nature 333: 134-139
OSPAR (1998a). OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Sintra Statement, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998.
OSPAR (1998b). OSPAR Convention for the Protection of the Marine Environment of the North East Atlantic. OSPAR Strategy with regard to Hazardous Substances, Reference no. 1998-16, OSPAR 1998/14/1/Annex 34, Ministerial Meeting of the OSPAR Commission, Sintra b22-23 July 1998.
Ratcliffe, H.E., Swanson, G.M. and Fischer, L.J. (1996). Human exposure to mercury: a critical assessment of the evidence of adverse health effects. Journal of Toxicology and Environmental Health 49: 221-270
Reuther, R. (1994). Mercury accumulation in sediment and fish from rivers affected by alluvial gold mining in the Brazilian Madeira River Basin, Amazon. Environmental Monitoring and Assessment 32: 239-258
Salomons, W. and Forstner, U. (1984). Metals in the hydrocycle. Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, ISBN 3540127550
USPHS (1997). Toxicological profile for Mercury on CD-ROM. Agency for Toxic Substances and Disease Registry. U.S. Public Health Service
World Health Organisation (1989). Mercury. Environmental Health Criteria 86. ISBN9241542861
World Health Organisation (1993). Guidelines for drinking water quality. Volume 1: Recommendations. ISBN 9241544600
Zalups, R.K., Lash, L.H. (1994). Advances in understanding the renal transport and toxicity of mercury. Journal of Toxicology and Environmental Health 42: 1-44

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

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

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).

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, than if it is bound to organic matter or present as an insoluble precipitate. Therefore, copper sulphate, or chloride, present in MSW incinerator ash or mine tailings, is far more bioavailable and migratory than the organically bound copper found in sewage sludge (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-linking 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 affected; and in the embryos of oysters and mussels concentrations of 5 ug/l were seen to induce abnormalities. A similar concentration resulted in increased mortalities in populations of the isopod crustacean *Idothea baltica* (UNEP 1993, Bryan 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).

References

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

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

Bureau of Indian Standards (1995). Drinking water specifications. Second Edition, UDC 628.1.033

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

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

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

EEC (1975). Council Directive 75/440/EEC of 16 June 1979 concerning the quality required of surface water intended for the abstraction of drinking water in the Member States (OJ L 194, 25.7.1975, p. 26)

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

EEC (1978). Council Directive 78/659/EEC of 18 July 1978 on the quality of fresh waters needing protection or improvement in order to support fish life (OJ L 222, 14.8.1978, p. 1)

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

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

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

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

Hong, S., Candelone, J.P., Soutif, M. and Boulton, C.F. (1996). A reconstruction of changes in copper production and copper emissions to the atmosphere during the past 7000 years. The Science of the Total Environment 188: 183-193

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

Mance, G., Brown, V.M. and Yates, J. (1984). Proposed environmental quality standards for List II substances in water. Copper. Water Research Centre Technical Report TR210

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

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

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

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

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

Thornton, I. (1995). Metals in the global environment: facts and misconceptions. A publication by the International Council on Metals and the Environment (ICME). ISBN 1895720095

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

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

World Health Organisation (1993). Guidelines for drinking-water quality. Volume 1: Recommendations. ISBN 9241544600

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

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

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).

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 migrate significantly in natural systems. Instead, it is rapidly precipitated and adsorbed onto suspended particles and bottom sediments. However changes in the chemical and physical properties of an aquatic environment, can result in changes to the chromium (III)-chromium (VI) equilibrium (Richard and Bourg 1991).

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

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

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

References

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

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

Bureau of Indian Standards (1995). Drinking water specifications. Second Edition, UDC 628.1.033

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

EEC (1975). Council Directive 75/440/EEC of 16 June 1975 concerning the quality required of surface water intended for the abstraction of drinking water in the Member States (OJ L 194, 25.7.1975, p. 26)

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

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

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

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

IARC (1982). Chromium and certain chromium compounds. In: IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Chemicals, industrial processes and industries associated with cancer in humans. IARC monographs, Vol. 1 to 29

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

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

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

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

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

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

Richard, F.C. and Bourg, A.C.M. (1991). Aqueous geochemistry of chromium: a review. Wat. Res. 25, 7: 807-816

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

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

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

World Health Organisation (1993). Guidelines for drinking-water quality. Volume 1: Recommendations. ISBN 9241544600

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).

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

Cadmium is more mobile in aquatic environments than most other metals. It is also bioaccumulative and persistent in the environment ($t^{1/2}$ of 10-30 years) (USPHS 1997). It is found in surface and groundwater as either the +2 hydrated ion, or as an ionic complex with other inorganic or organic substances. While soluble forms may migrate in water, cadmium in insoluble complexes or adsorbed to sediments is relatively immobile. Similarly, cadmium in soil may exist in soluble form in soil water, or in insoluble complexes with inorganic and organic soil constituents (USPHS 1997, WHO 1992). 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).

References

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

Alloway, B.J. (1996). Soil pollution and land contamination. In Pollution, causes, effects and control, 3rd Edition. Harrison, R.M. [Ed]. The Royal Society of Chemistry, Cambridge, UK. ISBN 0854045341

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

Bureau of Indian Standards (1995). Drinking water specifications. Second Edition, UDC 628.1.033

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

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

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

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

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

EEC (1975). Council Directive 75/440/EEC of 16 June 1975 concerning the quality required of surface water intended for the abstraction of drinking water in the Member States (OJ L 194, 25.7.75, p. 26)

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

EEC (1980). Council Directive 80/778/EEC of 15 July 1980 relating to the quality of water intended for human consumption (OJ L 229, 30.8.80, p. 11)

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

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

Goyer, R.A., Epstein, S., Bhattacharyya, M., Korach, K.S. and Pounds, J. (1994). Environmental risk factors for osteoporosis. Environmental Health Perspectives 102, 4: 390-394

IARC (1982). Cadmium and certain cadmium compounds. In: IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Chemicals, industrial processes and industries associated with cancer in humans. IARC monographs, Vol. 1 to 29

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

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

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

MINDEC (1995) Ministerial Declaration of the Fourth International Conference on the Protection of the

North Sea. 8-9 June 1995, Esjberg, Denmark
Mitchell, D.J., Wild, S.R. and Jones, K.C. (1992). Arrested municipal solid waste incinerator fly ash as a source of heavy metals to the UK environment. Environmental Pollution 76: 79-84
Mueller, P.W., Paschal, D.C., Hammel, R.R., Klincewicz, S.L. and MacNeil, M.L. (1992). Chronic renal effects in three studies of men and women occupationally exposed to cadmium. Arch. Environ. Contam. Toxicol. 23: 125-136
Nriagi, J.O. (1988). A silent epidemic of environmental metal poisoning. Environmental Pollution 50: 139-161
Nriagu, J.O. (1990). Global metal pollution. Poisoning the biosphere? Environment 32, 7: 7-11;28-33
OSPAR (1998a). OSPAR Convention for the Protection of the Marine Environment of the North-East Atlantic. Sintra Statement, Ministerial Meeting of the OSPAR Commission, Sintra 22-23 July 1998.
OSPAR (1998b). OSPAR Convention for the Protection of the Marine Environment of the North East Atlantic. OSPAR Strategy with regard to Hazardous Substances, Reference no. 1998-16, OSPAR 1998/14/1/Annex 34, Ministerial Meeting of the OSPAR Commission, Sintra b22-23 July 1998.
Pain, D.J., Sanchez,A. and Meharg, A.A.(1998). The Donana ecological disaster: contamination of a world heritage estuarine marsh ecosystem with acidified pyrite mine waste. The Science of the Total Environment 222: 45-54
Roels, H., Bernard, A.M., Cardenas, A., Buchet, J.P., Lauwerys, R.R., Hotter, G., Ramis, I., Mutti, A., Franchini, I., Bundshuh, I., Stolte, H., De Broe, M.E., Nuyts, G.D., Taylor, S.A. and Price, R.G. (1993). Markers of early renal changes induced by industrial pollutants. III. Application to workers exposed to cadmium. British Journal of Industrial Medicine 50: 37-48
Sadiq, M. (1992). Toxic metal chemistry in marine environments. Marcel Dekker Inc., New York, Basel, Hong Kong. ISBN 0824786475
Salomons, W. and Forstner, U. (1984). Metals in the hydrocycle. Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, ISBN 3540127550
Thuvander, A. (1989). Cadmium exposure of rainbow trout, Salmo gairdneri Richardson: effects on immune functions. J. Fish Biol. 35: 521-529
USPHS (1997). Toxicological profile for cadmium on CD-ROM. Agency for Toxic Substances and Disease Registry
USPHS (1998). 8th Report on Carcinogens 1998 Summary
World Health Organisation (1992). Cadmium. Environmental Health Criteria 135. ISBN 9241571357
World Health Organisation (1993). Guidelines for drinking water quality. Volume 1: Recommendations. ISBN 9241544600