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Organochlorine and heavy metal pollutants associated with the Gerdau Iron smelter in Sapucaia do Sul, Brazil 2000

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GREENPEACE

TABLE OF CONTENTS

EXECUTIVE SUMMARY

Gerdau is a steel-making company whose headquarters are in Porto Alegre, Rio Grande do Sul State. The company has over a dozen sites in Brazil and in addition to its Brazilian operations, it has factories in Uruguay, Argentina, Chile, the USA and Canada. Its products range from nails to speciality steels, including products for the construction industry (such as bars for reinforcing concrete) and the agricultural industry (e.g. fencing wire).

The Gerdau Riograndense facility is situated in Sapucaia do Sul, Rio Grande do Sul State. Here scrap iron and steel are smelted for the manufacturing of new products. The most commonly employed technology for resmelting steel is the electric arc furnace, which the Gerdau facility is known to employ. This process consumes enormous quantities of electricity and emits large quantities of atmospheric emissions, dusts and slags, often contaminated with a wide range of toxic heavy metals and persistent organic chemicals.

In August 2000, Greenpeace visited the Gerdau facility and collected several samples in and around the site. The analysis of these samples for organic contaminants and heavy metals demonstrated the following:

- Waste slags are contaminated with high concentrations of a number of toxic and potentially toxic heavy metals including chromium, lead, manganese and zinc.

- Slag contaminating the ground outside the factory is also contaminated with of the toxic and persistent polychlorinated biphenyls (PCBs). The profile of these compounds in the slag is consistent with that of the technical PCB mixture Aroclor 1254.

- Dust collected from a house close to the Gerdau facility are contaminated with high levels of a similar range of toxic metals, as well as PCBs, with a very similar profile to that found in the slag.

- The sample of effluent from the Gerdau facility did not show organic contaminants or high levels of metals. However, the quality of the effluent is known to be highly variable, so the results may not represent the worst releases from this site

The release of toxic heavy metals and persistent organic chemicals from the Gerdau facility has been clearly demonstrated. These releases will have detrimental impacts not only on the health of workers at the facility, but also on humans, animals and the general environment, both in the immediate vicinity and further afield.

The Gerdau facility is known to utilise electric arc furnaces in the smelting process. This process is well known to produce large quantities of dust with high concentrations of heavy

metals such as zinc, lead, cadmium and chromium. It is very likely that these dusts are contributing to the release of toxic heavy metals to the environment. In addition, integrated steel works have been shown to be significant sources of the highly toxic and persistent organochlorines such as the polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs). The formation of these compounds is generally a result of chloro-organic contaminants in the scrap, including PCB contaminants and plastics (especially PVC).

1 INTRODUCTION

Gerdau is a steel-making company whose headquarters are in Porto Alegre, Rio Grande do Sul State. The company has over a dozen sites in Brazil, and in addition to its Brazilian operations, it has factories in Uruguay, Argentina, Chile, the USA and Canada. Its products range from nails to speciality steels, including products for the construction industry (such as bars for reinforcing concrete) and the agricultural industry (e.g. fencing wire) (Gerdau 2000).

The iron and steel industry employs a wide variety of technologies, depending upon the feedstock and product. Commonly, several units will be grouped together at an integrated steelworks, though this is by no means always the case. The pollution emitted from plants is also variable, depending upon the materials used and produced as well as the technology employed and the efficacy of any pollution control measures.

The Gerdau Riograndense facility is situated in Sapucaia do Sul, Rio Grande do Sul State. Here scrap iron and steel are smelted for the manufacturing of new products. The most commonly employed technology for resmelting steel is the electric arc furnace and this is known to be in use at this site. The process consumes enormous quantities of electricity and emits large quantities of atmospheric emissions, dusts and slags. The most toxic pollutants emitted are likely to be heavy metals, and highly toxic and persistent organochlorines such as the polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs) (EC 1999). Henceforth, these compounds are referred to simply as dioxins and furans.

2 SAMPLING PROGRAM

In August 2000, Greenpeace visited the Gerdau facility and collected seven samples in and around the site. The samples included two river sediments, one effluent and one water runoff sample, two samples of slag and a sample of dust from a house located very close to the Gerdau facility.

2.1 General Sampling Procedures

All samples were collected and stored in pre-cleaned glass bottles that had been rinsed thoroughly with nitric acid and analytical grade pentane in order to remove all heavy metal and organic chemical residues. Solid samples were collected in 100ml bottles, and the water samples were collected in 1-litre bottles. All samples were immediately sealed and cooled upon collection and returned to the Greenpeace Research Laboratories for analysis. Detailed description of sample preparation and analytical procedures for qualitative organic screen and quantitative heavy metal determination are presented in Appendix 1.

2.2 Sample Descriptions

A sample of effluent was collected from a pipe discharging into the Rio dos Sinos from the Gerdau facility in the vicinity of the treatment lagoon. Significant variation in the quality of the effluent had previously been observed at this site. At times, the effluent was coloured orange, indicating a high iron content, which would probably also be accompanied by an increased loading of other metals. However, on the occasion on which these samples were collected, the effluent was almost colourless. A sample of sediment was collected from the location of this point discharge, as well as from approximately 300 metres upstream of the Gerdau effluent discharge point.

Two samples of slag were collected from locations within and just outside the Gerdau site. The slag was not contained in any way, enabling dispersion to take place into the wider environment. Also collected were samples of water from a runoff channel and of dust from a house located downwind of the factory, approximately 10 metres from the factory wall. A summary of the sample descriptions is presented in Table 1.

Sample Number	Sample Description	Sample Location
AM0072	effluent	from pipe discharging into Rio dos Sinos
AM0073	sediment	adjacent to discharge pipe
AM0074	water	from runoff channel running through plant
AM0075	solid waste	from ground near plant
AM0076	sediment	from river upstream of plant
AM0077	solid waste	from southern area of plant; stored in open air
AM0078	house dust	from house immediately downwind from plant

Table 1. Description of samples collected from Gerdau Riograndense in August 2000.

3 RESULTS AND DISCUSSION

The results of the organic screen analysis and heavy metals analysis of the are presented in Table 2, including a breakdown of the groups of organic compounds reliably identified in the samples.

For more information on the common sources, environmental behaviour and toxicological outlines for key pollutants detected during this study see Appendices 2 and 3.

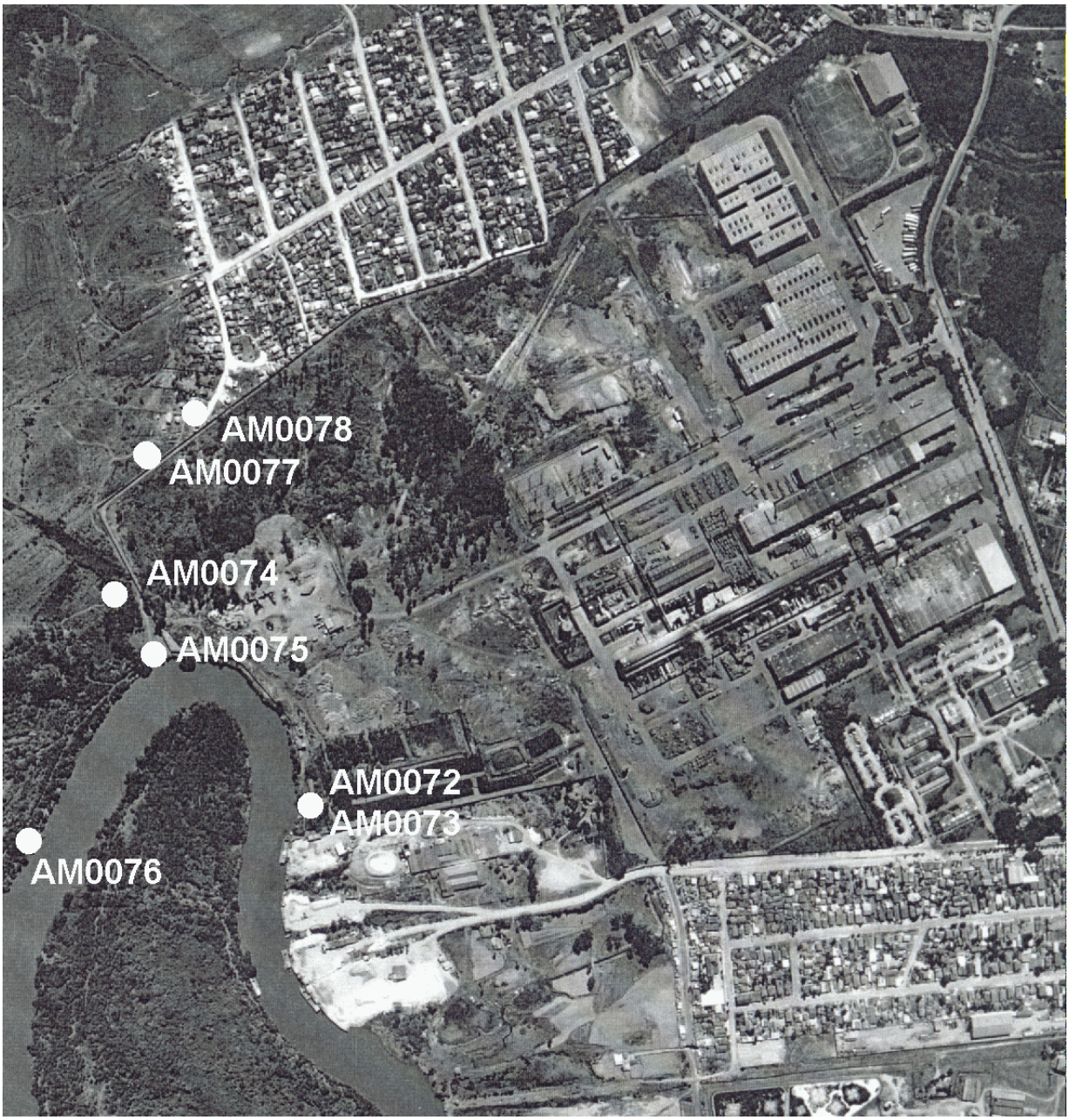

Figure 1. Map of sampling locations around the Gerdau Riograndense facility. Note that sample AM0077 was collected from within the factory site; the location marked on the map is that at the perimeter as close as possible to the sampling location.

Sample Number	AM0074	AM0075	AM0077	AM0078	AM0076	AM0073	AM0072
Sample Type	water	soil/slag	slag	house dust	sediment	soil/sediment	effluent
Sample Information	runoff from site	near factory gates	stored inside plant	house downwind of plant	upstream from plant	adjacent to discharge pipe	discharge to Rio dos Sinos
METALS	ug/l	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	ug/l
Cadmium	<20	3	<2	8	<2	<2	<20
Chromium	<20	2410	1349	289	287	74	<20
Cobalt	<20	20	21	16	22	13	<20
Copper	63	490	289	326	87	21	53
Lead	<30	173	118	624	63	23	<30
Manganese	120	30690	15484	2739	278	131	<10
Mercury	<0.50	0.53	<0.05	0.66	0.12	0.16	<0.50
Nickel	<20	201	119	71	38	22	<20
Zinc	13	2209	1166	4363	123	138	<10
ORGANIC COMPOUNDS							
No. of organic compounds isolated	None	128	74	219	23	13	None
No. of these reliably identified	None	116(91%)	18(24%)	162(75%)	5(22%)	8(62%)	None
ORGANOHALOGEN COMPOUNDS							
Benzene, 1,4-dichloro-		*		*	*	*	
Dichlorobiphenyl isomers		*(2)		*(2)			
Trichlorobiphenyl isomers		1, *(7)		*(8)			
Tetrachlorobiphenyl isomers		1, *(11)		*(13)			
Pentachlorobiphenyl isomers		3, *(9)		*(19)			
Hexachlorobiphenyl isomers (other than 138&153)		*(15)		*(15)			
Heptachlorobiphenyl isomers (other than 180)		*(3)		*(6)			
PCB-138		1		*			
PCB-153		1		*			
PCB-180		*		*			
PAHs							
Naphthalene and/or its derivatives				2			
Anthracene and/or its derivatives				2			
Phenanthrene and/or its derivatives				2			
Pyrene and/or its derivatives				3			
9H-Fluorene and/or its derivatives				1			
Fluoranthene				1			
Benz[a]anthracene and/or its derivatives				1			
Chrysene and/or its derivatives				1			
PHENOLIC COMPOUNDS							
Phenol, 2,4-bis(1,1-dimethylethyl)-				1			
OTHER AROMATIC COMPOUNDS							
Alkylated benzenes		1		1			
Benzaldehyde			1	1	1	1	
Benzenemethanol			1				
Benzo[b]naphtho[2,3]furan				1			
HYDROCARBONS							
Linear		4	14	16	3	6	
Cyclic			1				
MISCELLANEOUS							
Terpenoids				8			

*Table 2. Results of organic chemicals and heavy metals identified in samples collected in and around Gerdau Riograndense, in August 2000. For the groups of organic compounds reliably identified; # signifies the number of compounds identified using general GC/MS screening method; * (#) signifies compounds identified only at trace levels using a selective ion monitoring (SIM) method, with the number of compounds given in parentheses for groups with more than one compound. Metal concentrations are given in mg/kg dry weight for solid samples and ug/l for liquid samples.*

3.1 In and around the factory

3.1.1 Waste Slag

Both samples of slag contained high levels of a number of toxic metals, including chromium, copper, lead, manganese and zinc. Of the two samples, the sample of slag from just outside gates at the rear of the factory near the river, (AM0075) contained all these metals at higher concentrations, as well as containing significant concentrations of cadmium and mercury. For comparison, typical background concentrations of these metals in soil are given in Table 3.

Metal	Background Concentrations in soil (mg/kg dry weight)	Reference
Cadmium (Cd)	0.01-2.0	USPHS 1997, Alloway 1990
Chromium (Cr)	<1-100	Alloway 1990
Copper (Cu)	20 –30	Alloway 1990
Lead (Pb)	10-30	Alloway 1990
Manganese (Mn)	80-7000	Alloway 1990
Mercury (Hg)	0.02-0.625	Alloway 1990, WHO 1989
Zinc (Zn)	10-300 (50 average)	Alloway 1990

Table 3. Typical background concentrations of certain metals found in soil.

The presence of galvanised steel in the scrap is likely to be a major source of zinc in the slag samples as well as in dusts produced at the facility. This type of steel has been coated with zinc to reduce corrosion. The relative amount of galvanised steel being produced has been increasing in recent years, resulting in increased levels of zinc in scrap feed stocks (Dudek *et al*. 1990).

The use of scrap metals containing various alloys of iron and other metals such as stainless steel, or coated steels such as ferrochrome-steel, will be contributing to the levels of non-ferrous metals in the slag.

The behaviour of trace metals once released to the environment, particularly the mobility and toxicity, is highly dependant on the oxidation state and structure of the metal species. The overall impacts caused by the release of these metals to the environment depends on both the quantities released and the form that the metals are in.

Many heavy metals exert a broad range of toxic effects to humans, terrestrial and aquatic life and plants. A number of these metals also have the potential to bioaccumulate, including cadmium, chromium, lead and mercury and zinc (USPHS 1997, Kimbrough et *al.* 1999,

MINDEC 1995). In addition, certain forms of cadmium and chromium have carcinogenic properties (USPHS 1998).

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 (Roels *et al.* 1993). A number of studies indicate a relationship between occupational (respiratory) exposure to cadmium and lung and prostatic cancer (IARC 1998, Goyer 1996).

While certain forms of chromium are essential trace nutrients, chromium (VI) is non-essential and toxic and chromium (VI) compounds have been classified as known carcinogens (IARC 1998). Long-term occupational exposure to airborne levels of chromium higher than those in the natural environment has been associated with lung cancer (USPHS 1998).

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 (USPHS 1997).

Lead has no known nutritional, biochemical or physiological function, and is toxic to most living things. When lead is released into the environment it has a long residence time compared with most pollutants (Goyer 1996). Animals feeding close to smelting facilities have often ingested levels of lead that have resulted in poisoning and death (Blus *et al.* 1991, USPHS 1997). More information on the human health impacts of releases of lead to the environment is further discussed in Section 3.1.3.

Manganese is an essential trace metal, although human and animal exposure to high levels can cause serious illness. Workers chronically exposed to high levels of manganese in the air have suffered both mental and emotional disturbances, along with increased slowness and clumsiness of body movements (ATSDR 1997). Manganese exposure in workers at steel smelters has been linked with early signs of neurological impairment (Wennberg 1991).

Mercury is an extremely toxic, non-essential trace metal, having no biochemical or nutritional function, and biological mechanisms for its removal are poor (WHO 1989).

Zinc is an essential element, however, if plants and animals are exposed to high concentrations of bioavailable zinc, significant bioaccumulation can results, with possible toxic effects (USPHS 1997). Excessive dietary exposure, in both humans and animals, can cause gastrointestinal distress and diarrhoea, pancreatic damage and anaemia (USPHS 1997, Goyer 1996).

More information on the environmental behaviour and toxicology of these metals is given is Appendix 2.

In addition to heavy metals, a number of organic compounds were identified in the sample of slag collected near to the factory (AM0075), including 56 isomers of polychlorinated biphenyls (PCBs). No PCB isomers were identified in the second sample of slag (AM0077).

PCBs are a group of synthetic organic chemicals that contain 209 individual compounds (known as congeners) with varying harmful effects. There are no known natural sources of PCBs. These chemicals usually enter the environment as mixtures of congeners. PCBs are highly persistent chemicals, and levels of PCBs in biological material may be several orders of magnitude higher than ambient concentrations due to the bioconcentration of these fat-soluble compounds.

PCBs have been used in a wide variety of applications, the most important in tonnage terms were in transformer and capacitor oils. PCBs have always been sold as technical mixes rather than individual chemicals. The Aroclor range manufactured by the American company Monsanto was probably the most widely used (de Voogt & Brinkman 1989, ATSDR 1997). The technical mixture Aroclor 1254 has been widely used in transformer, lubricant and capacitor oils (Kimbrough 1989).

Previous studies have demonstrated the presence of PCBs in contaminated scrap metal used for resmelting (Harrad 1994). This is the most likely source of the PCB isomers in the sample of slag at the Gerdau facility.

The profile of isomers identified in the slag sample AM0075 most closely matches with the profile for the technical mix Aroclor 1254. Additional dichlorinated isomers were identified in the slag sample AM0075. While the composition of the Aroclor mixtures can vary from batch to batch, Aroclor 1254 does not generally contain dichlorinated isomers (Kimbrough 1989). The dichlorinated isomers in the slag sample may be due to the presence of lower chlorinated Aroclor technical mixes along with Aroclor 1254, or from dechlorination of higher chlorinated isomers in Aroclor 1254.

PCBs can be absorbed through the skin as well as through ingestion and inhalation. A wide range of effects due to PCB toxicity have been reported, most noticeably reproductive toxicity, teratogenesis and developmental toxicity, immunosuppression and endocrine disruption (Safe 1984). In one study, Aroclor 1254 was shown to be the most potent Aroclor mixture at reducing in-vitro fertilisation rates in mice (Kholkute *et al*. 1994).

More information on the common sources, environmental behaviour and toxicology of PCBs is given is Appendix 3.

Both samples of slag also contained a number of hydrocarbons, and sample AM0075 contained an alkyl benzene. These compounds are components of crude oils or petroleum products and as such are very widespread environmental pollutants (Mackay 1988). It is, therefore, not possible to identify the source or sources of these compounds.

In addition to the slags that are produced during steel production, the use of electric arc furnaces also produces large quantities of dust. The amount of electric arc furnace dust produced is generally 1-2% of the amount of steel obtained. In addition to iron oxide, these dusts have been shown to contain zinc, lead, cadmium and chromium at concentrations that make the material a hazardous waste, making them unsuitable for landfilling. The concentrations of toxic metals in these dusts have been shown to vary considerably over time at a number of steel plants (Cruells *et al.*1992, Perrault *et al.* 1992).

Reported levels of metals in electric arc furnace dusts include high levels of chromium (3.0-3.9 g/kg), copper (3.2-5.0 g/kg), lead (31-57 g/kg), manganese (17-40 g/kg), and zinc (160-244 g/kg). Elevated levels of cadmium (0.4-0.7g/kg) and mercury (0.001 g/kg) have also been reported in electric arc furnace dusts (Cruells *et al.*1992, Andres *et al.* 1995). It should be noted that these figures are given in g/kg while the reported data for this study are in mg/kg, a factor of 1000 different.

Studies have shown that levels of zinc and lead in workplace dusts at coated scrap melting facilities are often close to, or above, limits set by the regional authorities (Lindblad 1992). Dust exposure of foundry workers has been related to fibrosis, bronchial obstruction, and possible lung cancer (Perrault et *al.* 1992, and references therein).

Recovery of up to 99% of these toxic metals from the dusts is possible through a number of different methods (Cruells *et al.* 1992). Stabilisation of these dusts in solid form using materials such as cement has been shown to greatly reduce the leaching rate of these metals from the dusts. However, this process does not eliminate leaching completely (Andres *et al*. 1995).

In addition to the organic compounds discussed above, integrated steel works have been shown to be significant sources of dioxins (ENDS 1995).

Where electric arc furnaces are employed, scrap is often pre-heated, utilising the hot gases from the electric arc furnace. This can cause the volatilisation of volatile compounds including hydrocarbons and alkyl benzenes. At temperatures above 300°C the formation of new organic compounds can occur. These can include polyaromatic hydrocarbons (PAHs) as well as dioxins and furans (Lindblad 1992). The chlorine required for the formation dioxins and furans generally comes from chloro-organic contaminants in the scrap, including chlorinated paraffin cutting oils, PCB contaminants, and plastics (especially PVC). Studies have shown that PVC coated scrap used in electric arc furnaces processes produce the highest amounts of dioxins and furans (Lindblad 1992). Some steel plants have restrictions on the maximum temperature in the scrap pre-heating unit in order to minimise dioxin formation (Lindblad 1992).

In addition to heavy metals and organic pollutants, steel mills can also be major sources of very fine particulate pollution (PM_{10}). The increased levels in these air pollutants from steel mills has been demonstrated to increase the levels of pneumonia, pleurisy, bronchitis and asthma in the vicinity of the mill, particularly amongst children (Pope 1989).

3.1.2 Runoff water

The sample of water (AM0074) collected from a runoff channel running through the plant contained elevated concentrations of copper and manganese; 63 ug/l and 120 ug/l. Typical background concentrations of these metals in freshwater are 20 ug/l and 12 ug/l for copper and manganese respectively (Mance et *al*. 1984, Bowen 1966). The elevation of these metals may be due to leaching of metals from contaminated wastes store within the plant. The elevation of these metals due to regional variations cannot, however, be ruled out.

No organic compounds were identified in this sample of water.

3.1.3 House dusts

The sample of dust collected from a house very close to the factory walls contained elevated levels of a similar range of toxic metals to those identified in the two slag samples, including cadmium, chromium, copper, lead, manganese and zinc.

Significant concentrations of a number of metals have been reported in airborne dusts from steel smelters, including iron, lead, manganese, zinc, and copper. Elevated levels of chromium and cobalt have also been found, though to a lesser degree. Especially high concentrations of lead have been found in dusts at a foundry where untreated scrap metal is used. These metals have been found predominantly in the small particle fraction of the dusts. Higher concentrations of lower boiling metals are generally found in dust samples, which is consistent with their presence being due to smelting activities at the facilities (Perrault *et al.* 1992).

Childhood exposure to lead has been demonstrated to result in adverse health effects, and lead-contaminated household dust is often a primary exposure source, usually via ingestion (Ewers *et al.* 1994, Sterling et *al.* 1999, Lanphear *et al.* 1998). For young children, hand-to-mouth behaviour with ingestion of lead-contaminated dust has been clearly identified as a significant route of exposure for young children (Thornton *et al.* 1990). Lead in house dust samples, at levels below that found in the house dust sample collected for this study (AM0078), have been shown to result in elevation of the amount of lead in the blood of children to levels that could cause health impacts (Lanphear *et al.* 1998). Current recommendations suggested 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).

The toxic effects of lead are the same, irrespective of whether it is ingested or inhaled. Elevated levels of lead within the body have been associated with a wide range of adverse health effects. Of particular concern is the effect of relatively low exposure on cognitive and behavioural development in children (Pirkle *et al.* 1998, USPHS 1997).

Information on the environmental behaviour and toxicology of the additional metals of concern has been previously discussed in Section3.1.1, and more is given is Appendix 2.

A total of 67 isomers of polychlorinated biphenyls (PCBs) were also identified in the sample of house dust (AM0078. The profile of PCB isomers identified in the house dust sample matches very closely the profile found in the sample of slag (AM0075). It seems highly likely therefore, that the presence of these PCB isomers in the house dust is a direct result of activities at the Gerdau facility.

In addition, a range of hydrocarbons, alkyl benzenes and polycyclic aromatic hydrocarbons (PAHs) were identified in this sample. As previously mentioned, hydrocarbons and alkyl benzenes are components of crude oils or petroleum products, as are many PAHs. As such they are very widespread environmental pollutants, and it is, therefore, not possible to identify all the sources of these compounds (Mackay 1988).

PAHs are also commonly found as product of incomplete combustion of organic substances (Jones 1991, Overton 1994). The use of scrap fee stocks in steel smelting has been shown to result in the production of PAHs which can be found in the dusts produced (Lindblad 1992). This source may be contributing to the PAHs found in this sample of house dust.

A relationship has been found between the levels of PAH metabolites (including those of pyrene) found in the blood and urine of iron foundry workers with exposure levels of PAHs in the workplace. The levels of a number of these markers are relatively independent of other factors such as smoking and age (Santella *et al.* 1993).

3.2 River sediments and effluents

Analysis of the sample of effluent from the Gerdau facility (AM0072) for a range of heavy metals showed only the presence of copper at concentrations above detection limits. The level of copper in this sample is only slightly elevated above typical background concentrations of copper in freshwater, 20 ug/l (Mance *et al.* 1984). The concentration of copper may be due to incomplete treatment of contaminated wastewaters at the facility, although regional variations in natural concentrations of copper in freshwater cannot be ruled out.

At times, the effluent is coloured orange, indicating a high iron content, which would probably also be accompanied by an increased loading of other metals. This would be in keeping with use of technologies such as electric arc furnaces, which generally operate on a batch basis

However, on the occasion on which this sample was collected, the effluent was almost colourless, so the results may not represent the worst releases from this site.

The sample of sediment collected at the point of discharge of this effluent (AM0073) did not contain any metals analysed for at elevated levels. A sample of sediment (AM0076) collected from the river approximately 300 km upstream of the discharge point contained a number of metals at higher concentrations than those found at the effluent discharge point. These concentrations were not significantly elevated, and may due to variations in the nature of the sediments between these two locations.

4 CONCLUSIONS

The production of steel from scrap at the Gerdau facility is resulting in the production of large quantities of slag which are contaminated with high concentrations of a number of toxic and potentially toxic heavy metals including chromium, lead, manganese and zinc. Slag stored close to, but outside the factory, is also contaminated with a large number of toxic and persistent polychlorinated biphenyls (PCBs). The profile of these compounds in the slag is consistent with that of the technical PCB mixture Aroclor 1254.

The slag wastes are not contained in any way to prevent the spreading of these contaminated wastes to the environment. A noticeable consequence of this has been demonstrated by the presence of high levels of a similar range of metals in dust collected from a house close to the Gerdau facility. This dust was similarly contaminated with large number of PCBs, with a very similar profile to that found in the slag.

The Gerdau facility is kown to utilise electric arc furnaces in the smelting process. This process is well known to produce large quantities of dust with high concentrations of heavy metals such as zinc, lead, cadmium and chromium. It is very likely that these dusts are contributing to the release of toxic heavy metals to the environment.

In addition, integrated steel works have been shown to be significant sources of the highly toxic and persistent organochlorines such as the polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs). The formation of these compounds is generally a result of chloro-organic contaminants in the scrap, including PCB contaminants, and plastics (especially PVC).

While the analysis of effluent from the Gerdau facility did not show organic contaminants nor high levels of metals, the quality of the effluent is known to be highly variable, so the results may not represent the worst releases from this site

The release of toxic heavy metals and persistent organic chemicals from the Gerdau facility will been clearly demonstrated. These releases will have detrimental impacts not only on the

health of workers at the facility, but also on humans and the general environment, both in the immediate vicinity and further afield.

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APPENDIX 1 ANALYTICAL METHODOLOGY

A1.1. Organic screen analysis

A1.1.1. Preparation of samples

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

A1.1.1.1. Solid Samples

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

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

A1.1.1.2. Aqueous Samples

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

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

A1.1.2. Chromatographic Analysis

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

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

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

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

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.

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

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

A1.2. Heavy Metal Analysis

A1.2.1. Preparation of samples for heavy metal analysis

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

A1.2.1.1. Solid Samples

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

concentrated nitric acid. The samples were digested at room temperature overnight prior to being placed onto a Gerhardt Kjeldatherm digestion block (40 space) connected to a Gerhardt Turbosog scrubber unit (filled with 10% w/v sodium hydroxide). The samples were then refluxed at 130°C for four hours.

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

A1.2.1.2. Aqueous samples

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

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

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

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

APPENDIX 2 TOXICOLOGICAL OUTLINES FOR KEY HEAVY METALS

A2.1. Cadmium

A2.1.1. Environmental Contamination and Behaviour

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

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

A2.1.2. Toxicity

Cadmium has no biochemical or nutritional function, and it is highly toxic to both plants and animals (USPHS 1997, WHO 1992, Alloway 1990). In humans and animals, there is strong evidence that the kidney is the main target organ of cadmium toxicity, following extended exposure (USPHS 1997, Elinder & 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 & Jarup 1996, Goyer 1996, Iwata *et al.* 1993, WHO 1992, Nriagu 1988).

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

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

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

In addition to these toxic effects, it has also been suggested that cadmium may play a role in the development of hypertension (high blood pressure) and heart disease (USPHS 1997, Goyer 1996, Elinder & 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 & Langston 1992). Deleterious effects have also been reported in limpets, where correlations between increased levels of cadmium and reduced ability to utilise glucose were found. Reductions in reproduction rates and population numbers in copepods and isopods have been shown at concentrations as low as 5 ug/l, and exposure to similar levels has resulted in changes in the immune function in some fish, and depressed growth in juvenile fish and invertebrates (Bryan & 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 & Langston 1992).

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A2.2. Chromium

A2.2.1. Environmental Contamination and Behaviour

Although many different oxidation states of chromium exist in the environment, only the trivalent (III) and hexavalent (VI) forms are considered to be of biological importance. In aquatic environments, chromium (VI) will be present predominantly in a soluble form. These soluble forms may be stable enough to undergo intra-media transport, however chromium (VI) will eventually be converted to chromium (III), by reducing species such as organic substances, hydrogen sulphide, sulphur, iron sulphide, ammonium and nitrite (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 & Bourg 1991).

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

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

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

A2.2.2. 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 & Langston 1992).

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A2.3. Copper

A2.3.1. Environmental Contamination and Behaviour

Copper may exist in natural waters either in the dissolved form as the cupric (+2) ion or complexed with inorganic anions or organic ligands (e.g. carbonates, chlorides, humic and fulvic acids). It may also be present as an insoluble precipitate (e.g. a hydroxide, phosphate, or sulphide) or adsorbed onto particulate matter. Alternatively, it can be adsorbed to bottom sediments or exist as settled particulates. The relative concentrations of each of these forms is dependant upon a number of chemical parameters, including pH, salinity, alkalinity, and the

presence of organic ligands, inorganic anions and other metal ions. However, studies have frequently shown that the free +2 ion concentration is low, compared to the levels of copper associated with suspended and bottom sediments (USPHS 1997, Mance *et al*. 1984).

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

A2.3.2. 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. This can result 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 & Langston 1992). For example, studies have shown that at levels of 2 ug/l, the survival rate of young bay scallops was significantly affected; and in the embryos of oysters and mussels concentrations of 5 ug/l were seen to induce abnormalities. A similar concentration resulted in increased mortalities in populations of the isopod crustacean *Idothea baltica* (UNEP 1993, Bryan & Langston 1992, Giudici *et al*. 1989). Other studies have reported reductions in the survival, growth and fertility of amphipods and copepods (Conradi & DePledge 1998, UNEP 1993), and embryonic sensitivity in fish exposed to levels of 25 ug/l (UNEP 1993, Mance *et al*. 1984). Furthermore, a study of species diversity in benthic communities from Norwegian fjords, led to the conclusion that the most sensitive animals were missing from sites where sediment-copper levels exceeded 200 mg/kg. In the UK, such concentrations are exceeded in a number of estuaries, including the Fal and the Tamar. Here, many species of bivalves, including some mussels, clams and cockles are absent, and at best distribution is severely limited. The toxicity of the surface sediment containing over 2000 mg/kg of copper, towards juvenile bivalves appears to be the reason (Bryan & Langston 1992).

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A2.4. Lead

A2.4.1. Environmental Contamination and Behaviour

When lead is released into the environment it has a long residence time compared with most pollutants. As a result, it tends to accumulate in soils and sediments. There, 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.

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.

A2.4.2. Toxicity

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

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

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A2.5. Manganese

Manganese is an essential trace metal, although human and animal exposure to high levels can cause serious illness. Workers chronically exposed to high levels of manganese in the air have suffered both mental and emotional disturbances, along with increased slowness and clumsiness of body movements. This combination of symptoms is a disease called manganism. The symptoms can be reduced by medical treatment, but due to the high levels of manganese accumulated in the brain, any brain injury is often permanent (ATSDR 1997). It is not certain whether eating or drinking elevated levels of manganese can cause manganism or not. Low-level manganese exposure in workers at two steel smelters has been linked with early signs of neurological impairment (Wennberg 1991). In one report, humans exposed to contaminated drinking water, developed symptoms similar to those seen in manganese miners or steel workers, but it is not certain if the effects were caused by the manganese alone. Another report found that people who drank water with above average levels of manganese seemed to have a slightly higher frequency of symptoms such as weakness, stiff muscles, and trembling of the hands. However, these symptoms are not specific for manganese, and might have been caused by other factors (ATSDR 1997).

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

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A2.6. Mercury

A2.6.1. Environmental Contamination and Behaviour

Due to the fact that mercury is the only metal that can exist as both a liquid and a vapour at ambient temperatures, its environmental behaviour differs from that of most other toxic elements (USPHS 1997, WHO 1989). Mercury can exist in three valence states, Hg (0), Hg (I) and Hg (II). In the atmosphere, elemental mercury is by far the most common form, and as a

vapour it is responsible for the long-range, global cycling of mercury. In addition, to a far lesser degree, mercury may be associated with particulates, which are removed by dry or wet deposition. Atmospheric inputs may be more significant in areas where other sources, such as contaminated rivers, are less important or non-existent (USPHS 1997, WHO 1993).

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

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

A2.6.2. Toxicity

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

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

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

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

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

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

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

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A2.7. Zinc

A2.7.1. Environmental Contamination and Behaviour

Zinc occurs in the environment primarily in the +2 oxidation state, either as the free (hydrated) zinc ion, or as dissolved and insoluble complexes and compounds (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 & Langston 1992, Alloway 1990). However, re-solubilisation back into an aqueous, more bioavailable phase is possible under certain physical-chemical conditions, e.g. in the presence of soluble anions, the absence of organic matter, clay minerals and hydrous oxides of iron and manganese, low pH and increased salinity (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).

A2.7.2. Toxicity and Essentiality

Zinc is a nutritionally essential metal, having enzymatic, structural and regulatory roles in many biological systems (Goyer 1996, Aggett & Comerford 1995). Deficiency in humans can result in severe health consequences including growth retardation, anorexia, dermatitis, depression and neuropsychiatric symptoms (Aggett & Comerford 1995). At the other extreme,

excessive dietary exposure, in both humans and animals, can cause gastrointestinal distress and diarrhoea, pancreatic damage and anaemia (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). 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 & Poirier 1997); and humans who drank water from galvanised pipes, over a prolonged period, suffered irritability, muscular stiffness and pain, loss of appetite and nausea (UNEP 1993).

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

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

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

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APPENDIX 3 TOXICOLOGICAL OUTLINES FOR KEY ORGANIC COMPOUNDS

A3.1. Polychlorinated biphenyls (PCBs)

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

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

PCBs have always been sold as technical mixes rather than individual chemicals. de Voogt & Brinkman (1989) list some 46 trade names used for PCBs and PCB-containing products. Of these, the Aroclor range manufactured by the American company Monsanto was probably the most widely used. The most important PCB applications in tonnage terms were transformer oils and capacitors (de Voogt & Brinkman 1989). In transformer oils, the PCBs were mixed with chlorobenzenes (mainly trichlorobenzenes and tetrachlorobenzenes) as solvents (Swami *et al*. 1992, de Voogt & Brinkman 1989). PCBs are also synthesised as by-products in processes ranging from incinerators (USEPA 1998, Ballschmiter *et al.* 1989, Alcock *et al.* 1998) to metallurgical processing (Knutzen & Oehme 1989, Alcock *et al.* 1998, Thiesen *et al.* 1993) to dye manufacturing (USEPA 1998).

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

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

In a review of PCB toxicity, Safe (1984) lists the following symptoms of PCB toxicity: enzyme induction, decreased vitamin A levels, lymphoid involution, thymic and splenic atrophy, immunosuppression, chloracne, alopecia, oedema, hyperkeratosis, blepharitis,

hyperplasia of the epithelial lining of the extrahepatic bile duct, the gall bladder and urinary tract, hepatomegaly and liver damage including necrosis, haemorrhage, hepatotoxicity (altered porphyrin metabolism), tumour promotion, altered levels of steroid and thyroid hormones, reproductive toxicity including menstrual irregularities, reduced conception, early abortion, excessive menstrual and postconceptual haemorrhage, anovulation, testicular atrophy, decreased spermatogenesis, teratogenesis and developmental toxicity. In addition, low levels of PCBs have caused behavioural impairment in monkeys (Rice 1999).

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

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

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

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

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

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

Cummins (1988) has suggested that unless further escape of PCBs is prevented then the eventual extinction of marine mammals is a very real possibility.

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

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

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

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

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

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A3.2. Polycyclic aromatic hydrocarbons (PAHs)

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

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

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

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

PAHs are found to cause harm to human health. Individuals exposed by breathing or skin contact for long period of time to mixtures of PAHs and other compounds can develop cancer (ATSDR 1997). Many of the carcinogenic polycyclic aromatic hydrocarbons are derived from

an angular benz[a]anthracene skeleton. Anthracene itself is not carcinogenic, but benz[a]anthracene appears to have weak carcinogenicity. Addition of another benzene ring in select positions result in agents with powerful carcinogenicity such as dibenz[a,h]anthracene or benzo[a]pyrene. In addition, substitution of methyl groups on specific carbons of the ring also enhances carcinogenity. Thus, 7,12-dimethylbenz[a]anthracene (DMBA) is one of the most powerful synthetic, polycyclic aromatic hydrocarbon carcinogenes known (Williams 1986). Studies in laboratory animals have demonstrated the ability of benz[a]anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[a]pyrene, chrysene, dibenz[a,h,]anthracene, and indeno[1,2,3-c,d]pyrene to induce skin tumors (i.e., they are complete carcinogens) following intermediate dermal exposure. Anthracene, fluoranthene, fluorene, phenanthrene, and pyrene do not act as complete carcinogens (ATSDR 1997).

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

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

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