

Analysis of liquid and solid wastes from aluminium refinery, Japan, October 1996.

Angela Stephenson, Irina Labounskaia, David Santillo, Ruth Stringer & Paul Johnston

Greenpeace Research Laboratories Technical Note 02/97

28th February 1997

Summary

The aluminium refinery which Greenpeace Japan investigated in Japan discharges waste effluents to a collection channel which subsequently flow in to an open creek and, ultimately to the sea. Processes employed at the refinery, in particular the use of hexachloroethane (HCE) as a degassing agent, result in the release of a complex waste stream which was found to contain a wide range of organic and heavy metal contaminants at levels greatly elevated above background. Among the organic compounds discharged, the organochlorines, including HCB, HCBD and HCE itself, are the most significant as a consequence of their high toxicity, persistence and ability to bioaccumulate. Such discharges have led to accumulation of organochlorines and heavy metals, including lead, copper and chromium, to extremely high levels in sediments within the effluent channel and, in turn, to contamination of the waters and sediments of the creek downstream from the plant. Levels of dioxin (I-TEQ) in sediment sampled at the point of discharge from the plant exceeded levels reported for contaminated sediments elsewhere by 100-1000 times, and were within the range reported for very highly contaminated chlorinated wastes from the PVC industry.

The release of such a complex and toxic waste stream from the refinery, and the consequent contamination of the downstream environment, is clearly cause for serious concern. As an immediate step, the commitment to the elimination of HCE as a degassing agent for aluminium production within the OSPAR region should be noted and the industry should seek to phase out the use of this compound with immediate effect.

Introduction

Among the principle waste streams arising from the refining of aluminium are the solid and liquid wastes from the degassing process. This process involves the removal of gaseous impurities, particularly hydrogen, from the molten aluminium immediately prior to pouring the molten metal into casts. This avoids the formation

of bubbles and blisters in the cast aluminium which would otherwise weaken the final product (McKetta 1989).

The aluminium refinery investigated by Greenpeace Japan utilises the organochlorine compound hexachloroethane (HCE) as a degassing agent. The use of HCE as a degassing agent in the aluminium and other non ferrous metal industries has been addressed by the Oslo and Paris Commissions, as an important point source of organochlorines to the marine environment. PARCOM Decision 96/1 outlines timetables for elimination of HCE from such uses (OSPARCOM 1996). The decision states that:-

“All uses of hexachloroethane in the non-ferrous metal industry shall be phased out by 31 December 1997.” (OSPARCOM 1996)

- with the exception that the use of HCE in grain refining for the production of magnesium alloys, and in small, non-integrated aluminium foundries may, be permitted under certain circumstances until 31 December 1998.

This phase out has been agreed by contracting parties in recognition of the status of HCE as an organohalogen, included on the PARCOM black priority list of chemicals for elimination, and of the existence of less hazardous alternatives for degassing which are also “...capable of attaining similar, or even superior, technical efficiency and performance...”.

In practice, very few of the contracting parties to the Conventions continue to use HCE for degassing, having already substituted for less hazardous alternatives. In Switzerland, for example, the use of HCE in the aluminium industry was phased out as early as 1987 (OSPARCOM 1995).

The agreement to phase out this application of HCE relates principally to its toxicity, persistence and potential to accumulate in biological tissues and the recognised need to eliminate discharges of such substances to the marine environment. The use of HCE at the plant therefore represents a significant potential source of persistent organohalogens to the surrounding environment. In addition, there is a possibility that inorganic impurities in the aluminium, or heavy metals employed at other stages of the refining or onward manufacturing processes, may be carried through in to the liquid and solid waste streams.

The principal aims of the current study were, therefore, to determine the nature and levels of organic and heavy metal contaminants in liquid and solid wastes arising from the degassing process at the refinery. The refinery discharges liquid wastes into an open channel, which also receives wastewater from a wire production plant upstream. These combined discharges subsequently flow into a natural creek and, in turn, to the sea. The study, therefore, also involved analysis of heavy metals and organics in sediments from the wastewater channel and creek in order to determine the extent to which processes at the refinery have led to contamination of the downstream environment. This included analysis of a single sample of sediment from the receiving channel for chlorinated dioxins and furans. Results are discussed in terms of the nature and complexity of contamination, levels compared to published data for sediments and solid wastes and the toxicological significance of the

contaminants identified.

Analytical Methodology

Sample collection

A total of 10 samples, 3 liquid and 7 solid, were collected from locations on or adjacent to the refinery site; these are described in detail below:-

MI6161 - wastewater from degassing plant, before neutralisation (pH 11);

MI6162 - wastewater from degassing plant, after neutralisation (pH 6-6.5), at point of discharge to combined effluent channel (downstream from another plant);

MI6163 - wastewater from combined effluent channel at point of discharge to creek;

MI6164a and b - duplicate samples of solid waste from degassing process;

MI6165 - sediment from combined effluent channel, at point of discharge of wastewater from the degassing plant;

MI6166 - sediment from combined effluent channel, upstream from degassing plant, at point of discharge of wastewater from another plant (no water flow above this point);

MI6167 - sediment from creek, at point of confluence with combined effluent channel;

MI6168 - sediment from creek, upstream from confluence with combined effluent channel, at point of discharge of wastewater from an additional plant;

MI6169 - sediment from combined effluent channel, immediately downstream from discharge of wastewater from the degassing plant.

Liquid samples were collected in 1 litre glass Duran bottles, precleaned with Aristar grade hydrochloric acid, to remove metal contaminants, and with pesticide residue analysis grade (PRAG) acetone and pentane, to remove organic contaminants remaining on the surfaces after washing. Bottles were filled completely to avoid evaporative loss of the more volatile components, and samples placed as soon as possible into a precooled, darkened container.

Solid samples were collected in 100ml Duran bottles, similarly decontaminated, using prewashed untreated wooden spoons. These samples were also cooled immediately.

All samples were transferred to a refrigerator for storage until return to the UK for analysis at the Greenpeace Research Laboratories.

a) Organic screen analysis

Organic contaminants were extracted using standard liquid:liquid or liquid:solid solvent extraction techniques and analysed using gas chromatography/mass spectrometry (GC/MS).

Sample preparation

Samples were prepared for analysis using extraction methods based on those described in Swindlehurst *et al.* (1995), modified as described below.

Liquid samples

Following removal of a sub-sample of 100ml for analysis of heavy metals the remaining 900ml of sample was spiked with a small volume of a standard solution of deuterated-naphthalene (to a final concentration of 100 ppm) to act as an internal standard. 25ml of analytical grade pentane were added to the spiked sample and the bottle rolled for 2 hours in order to improve contact between solvent and sample matrix. The solvent fraction was removed using a separating funnel, the remaining sample acidified (to enable extraction of organic acids), 25 ml of fresh solvent added and the sample rolled for a further two hours before separation.

For each sample the two solvent fractions (before and after acidification) were combined and the whole volume cleared of very high molecular weight compounds by one or more passages through an activated Florasil column. Following clean-up, the extract was evaporated down to approximately 2 ml under a stream of analytical grade nitrogen. A small volume of a standard solution of brominated naphthalene was added to allow accurate determination of the final volume of extract.

Solid samples

A sub-sample of approximately 30g was taken for analysis from each sediment sample. This was spiked with deuterated naphthalene as for the liquid samples, and 20 ml of a 3:1 (by volume) mixture of analytical grade pentane:acetone added. Subsamples were sonicated for two hours to improve efficiency of extraction. This first extract was decanted off, the sediment acidified, and the extraction process repeated.

Following the two extraction phases, the two extracts were again combined and prepared for analysis as described for the liquid:liquid extracts above.

Sample analysis

Extracts were analysed using a Hewlett Packard GC/MS system, injecting 1ul on to a 25m BPX-5 fused silica capillary column held in an HP 5890 GC connected in line to an HP 5972 Mass Selective Detector (MSD). Analytes were identified through a combination of computer matching of mass spectra, against a library of 136 000 compounds, and expert interpretation.

Data are reported for each sample as the number of individual compounds isolated as discreet peaks and a list of those identified. Compounds identified to a match quality of 90% or greater are assumed to be reliable identifications. Those with match qualities between 50 and 89% are assumed to be tentatively identified only. All compounds yielding lower match qualities than 50% are assumed to be unidentified.

b) Dioxin analysis

A single sediment sample, MI6165 (sediment from combined effluent channel at point of discharge of wastewater from degassing process) was submitted to an independent, accredited analytical laboratory for determination of concentrations of dioxins (polychlorinated dibenzo-p-dioxins and furans). Further details on analytical methodology can be provided on request.

c) Heavy metal analysis

Solid samples

Samples of solid waste, sediment and soil were oven-dried for 48 hours until constant weight. They were then crushed using a pestle and mortar until homogenous. 0.5g of each sample was weighed into a 120ml Teflon microwave vessel fitted with a screw cap and pressure relief valve. To this 10ml of deionised water was added, followed by 7.5ml of concentrated hydrochloric acid and 2.5ml of concentrated nitric acid. The vessels were then sealed, placed on a rotating table in a microwave oven (model MDS-2000, CEM Corp.), and allowed to digest for one hour at full power (630W).

After cooling to ambient temperature, the digests were filtered into volumetric flasks, diluted with deionised water, made up to a volume of 50ml and mixed. Certified reference materials and blanks were prepared with every batch of 20 samples. Reference materials used were PACS-1 and BEST-1 (for mercury), trace elements in marine sediment certified by the National Research Council, Canada; and BCR-143, trace elements in a sewage sludge amended soil, certified by the Commission of the European Communities. Reference materials were spiked with a 1 mg/l standard, and acid matrix matched to the samples. Recoveries were calculated for all metals:

$$\% \text{ Recovery} = (X1 - X2) / Y * 100$$

Where X1 = the mean value of the certified reference material

X2 = the mean value of the certified reference material and spike

Y is the spike concentration for X2

Liquid samples

All samples were preserved in 10% v/v concentrated nitric acid on arrival. 50ml was transferred to a 120ml Teflon microwave vessel and digested using the same procedure and programming conditions as mentioned above. After cooling to ambient temperature, samples were filtered into volumetric flasks, diluted with deionised water, made up to a volume of 50ml and mixed. A quality control standard (8 mg/l) and blank standard were prepared with every 20 samples.

Following preparation the samples were analysed by Inductively Coupled Plasma Atomic Emission Spectroscopy (ICP-AES), using a Varian Liberty-100 Sequential Spectrometer. The following metals were quantified directly: Chromium (Cr), copper (Cu), lead (Pb), cobalt (Co), cadmium (Cd), etc.. Instrument calibration standards were matrix matched to the samples (15% v/v hydrochloric acid, 5% v/v nitric acid for solid samples; 10% v/v nitric acid for aqueous samples), and the calibration itself was validated using quality control standards set at 20% and 80 % of the calibration range (0.01 to 10 mg/l). Samples exceeding this range were diluted, in duplicate,

appropriately. The spectrometer was re-calibrated after every 20 samples to compensate for fluctuations in sensitivity. All other instrument quality control procedures were adhered to.

Mercury (Hg) was determined using Cold Vapour Generation ICP-AES. Hg(II) was reduced to Hg (0) i.e. a vapour, following reduction by sodium borohydride (0.5% w/v) and hydrochloric acid (10 Molar), the vapour was carried, in a stream of argon, into the spectrometer. The calibration employed ranged from 2 ug/l to 100 ug/l, the spectrometer was re-calibrated after every 10 samples, and each new calibration was validated using quality control standards as mentioned above.

Results and Discussion

Results from the organic screen and heavy metal analyses are discussed separately below.

a) organic screens

Analysis revealed significant and complex organic contamination of solid and liquid waste streams from the degassing plant, and evidence that the discharge of such liquid wastes has led to contamination of sediments with organochlorines both in the combined effluent channel and in the creek to which the channel discharges.

Wastewaters MI6161-6163

Wastewaters MI6161 and 6162 yielded a similar number and range of organic chemicals, with a clear predominance of organochlorines. For example, of the 11 compounds identified reliably in MI6161, before neutralisation, all were persistent organochlorine chemicals (Table 1). Both MI6161 and 6162 yielded hexachlorobenzene (HCB), pentachlorobenzene, pentachlorofluorobenzene, pentachloro-(trichloroethenyl)benzene, octachloropentafulvalene, a chlorinated cyclopentadiene and higher chlorinated naphthalenes. Additional compounds identified in MI6161 include nonachlorobiphenyl (a higher chlorinated PCB), hexachlorobutadiene (HCBBD) and hexachloroethane (HCE) itself. Note that these compounds may well have been present in MI6162, but not identified to reasonable degree of reliability. Indeed, HCE was tentatively identified in MI6163, taken immediately downstream from MI6162. Overall, therefore, neutralisation appears to have had limited impact on the extent of organic contamination of the wastewater. Analysis of the wastewater sample MI6163 revealed that many of these extremely hazardous chemicals were being discharged to the open creek as part of a complex effluent stream.

The compounds identified have very severe toxicological implications if released to the environment. For example, HCB is highly toxic, persistent and bioaccumulative. Howard et al. (1991) estimates half lives for HCB in soils and aerobic aquatic systems of between 2.7 and 5.7 years; in anaerobic sediments this could be as long as 10-23 years.

HCB is acutely toxic in high doses (Merck 1989, USPHS 1990a), but also exerts a wide range of toxic effects in a range of organisms exposed chronically to lower

doses. Much of our knowledge about effects on humans originates from the study of

Sample code	isolated	No. of compounds			percentage unidentified
		identified >90%	organohalogens	tentatively identified	
MI6161	22	11	11	2	41%
MI6162	26	8	7	9	35%
MI6163	19	8	7	2	47%
MI6164a	64	8	0	33	36%
MI6164b	61	14	1	14	54%
MI6165	69	22	2	31	23%
MI6166	58	7	0	28	40%
MI6167	8	1	1	1	75%
MI6168	10	0	0	1	90%
MI6169	29	6	1	8	52%

Table 1: Numbers of organic compounds identified and proportion remaining unidentified in each of the samples, including numbers identified as organohalogens to greater than 90% reliability.

a population in Turkey who, between 1955 and 1959, consumed bread made with grain contaminated with HCB (USPHS 1990a). Cumulative doses were relatively high, particularly in children, who received a large proportion of their dose pre-natally in the womb, during the most sensitive period of their development. Very high levels of infant mortality were associated with this exposure. Sublethal effects in children and adults were extremely wide-ranging, including toxicity to the liver, kidneys, skin and thyroid, interference with bone development, complex changes to blood biochemistry, and damage to the immune and nervous systems.

Although no cancer statistics appear to be available for this population, the high incidence of enlarged thyroid glands may well have been associated with an increase in thyroid cancer (USPHS 1990). HCB is a known initiator and promoter of cancer in a number of laboratory organisms, targeting the liver and thyroid in particular (Newhook and Meek 1994), and has also been implicated in suppression of the immune system (Thomas 1990). There is some evidence for a link between environmental exposure to HCB and an increased incidence of breast cancer (Dewailly *et al.* 1993a).

Direct effects on the reproductive system in mammals have also been described, particularly in female monkeys, where low chronic doses of HCB have led to degeneration of ovarian epithelial and germ cells (Jarrell *et al.* 1993) and alteration of levels of steroid sex hormones circulating in the blood. These effects may occur at levels of exposure which do not cause any other visible symptoms of toxicity. On the basis of such observed effects on hormone levels, Colborn *et al.* (1993) list HCB as a hormone disrupting chemical.

Discharge of HCB to the aquatic environment has a number of implications. Although many studies report levels of HCB in aquatic organisms, relatively few address the potential or actual toxicity of this compound in marine systems. Part of the problem relates to the fact that HCB is generally one of many organochlorine and other chemical contaminants isolated from the tissues of marine organisms, whose concentrations may co-vary (i.e. follow similar patterns of contamination levels). Identifying one agent as a causal factor for an observed effect is, therefore, largely impossible (Myers *et al.* 1994). In a study of genetic toxicity to marine organisms of sediments from the Western Mediterranean, in which hexachlorobenzene was present at high concentrations, the greatest toxicity appeared to be related to other contaminants which were present at much lower concentrations (Fernandez *et al.* 1992).

Nevertheless, direct toxicity of HCB to marine organisms has been demonstrated; Anderson *et al.* (1981), in a study of bacterial infection in the clam *Mercenaria mercenaria*, reported complete suppression of antibacterial activity in the haemolymph resulting from exposure to relatively low doses of HCB. The effect was greatly enhanced if the clams were exposed to low levels over a prolonged period as a result of accumulation of HCB in the body tissues. This has obvious implications for disease resistance in marine organisms and suggests that HCB may act on the immune systems of a very wide range of organisms.

On the basis of its toxicity, persistence in the environment and tendency to bioaccumulate in tissues, HCB is included on the UK "Red List" of priority substances for control (Agg and Zabel 1990), and is listed as a moderate to high priority for reduction under the Canadian Environmental Protection Act (Newhook and Meek 1994).

Very little toxicological information is available for the structurally-related chemicals pentachlorobenzene, pentachlorofluorobenzene or pentachloro(trichloroethenyl)-benzene. Nevertheless, these are also likely to be highly persistent and toxic compounds with some tendency to bioaccumulate in fatty tissues.

HCBD is a fairly common contaminant produced as a by-product in a number of industrial processes involving organic chlorine chemistry, including the production of PVC and the secondary oxychlorination of EDC wastes in the manufacture of solvents (Johnston *et al.* 1994). It is also reported as a contaminant in technical formulations of pentachlorophenol, used widely as a wood preservative (Goodrichmahoney *et al.* 1993). It is included on the UK Red List of priority pollutants for control (Agg and Zabel 1990), on account of its acute and chronic toxicity and persistence in the environment.

HCBD is a potent kidney toxin in laboratory animals (Werner *et al.* 1995), often showing greater toxicity in males than females for equivalent doses (Birner *et al.* 1995). It is a known animal carcinogen and a suspected carcinogen in humans (listed by the USEPA). 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 (USPHS 1992a). 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.

PCBs are stable chlorinated organic chemicals which are extremely resistant to chemical and microbial degradation. Their high persistence, toxicity and tendency to accumulate in biological tissues has led to restrictions and/or bans on their manufacture and use and careful control of disposal in many countries. The presence of a PCB in this effluent sample, identified with a high degree of reliability, is surprising. It is possible that such a compound could be generated through chemical conversion of other organochlorine compounds at the high temperatures in the refinery process, although it must also be considered that its presence results from residual presence of PCBs in plant machinery on site.

A number of sub-lethal toxic effects have been linked to exposure of animals and human populations to high levels of PCBs and related chemicals, including altered thyroid activity and related changes in brain biochemistry and function (Seegal and Shain 1992, Brouwer *et al.* 1995), suppression of the immune system (Dewailly *et al.* 1993b) and impaired bone development (Hodges *et al.* 1994). Prenatal exposure to PCBs has been linked to behavioural changes and mild learning difficulties in children (Jacobson and Jacobson 1993). Many of these effects may result from interference with the endocrine (hormone) system, which plays a central role in development and regulation of a wide range of body functions. As hormones are effective at very low concentrations in the body, xenobiotic chemicals which can interfere with such systems may also exert effects at concentrations orders of magnitude lower than those causing acute toxic effects (Allsopp *et al.* 1995). In addition, as many aspects of the endocrine system, in particular the sex steroid hormones, are practically identical in a wide range of organisms (from fish to mammals), effects observed in laboratory animals may well be translated to related effects in wildlife and humans (e.g. Sumpter and Jobling 1995).

Exposure of the developing foetus *in utero* to elevated levels of PCBs, and to other anthropogenic chemicals, as a result of transfer from maternal blood, may be of greatest concern, since the foetus is generally more susceptible than later life stages. This is because growing and dividing cells are less resistant to injury, and because the mechanisms for protection of the body against toxic chemicals in adults are not fully developed in the foetus.

Hexachloroethane itself is a highly toxic and irritant chemical which is readily absorbed through the skin. Given that HCE is used in bulk in the degassing process, during which it is exposed to molten aluminium and gaseous impurities at extremely high temperatures, it seems likely that this would represent the principal, if not only, source of organohalogen contaminants in the wastewater. This implies that the formation and release of organohalogenes from this process could be eliminated through substitution of HCE for an alternative, less hazardous, degassing agent. Without such substitution, the plant will undoubtedly continue to act as a point source discharge of toxic and persistent organochlorines to the creek and, in turn, the marine environment.

It should be noted that knowledge of the toxicological properties of the vast majority of industrial chemicals is extremely limited. The discharge from the refinery, as in

the case for many other industrial waste streams, contains a range of compounds which have never been properly assessed in terms of their toxicity and environmental fate. Even for those compounds which have been most studied, the degree of understanding of their modes of action remains, for the most part, very poor. Added to this is the fact that most of the toxicological data which are available relate to single compounds. Few industrial effluents contain one or two individual compounds in isolation, a situation made clear by the current analyses. Inability to identify a significant proportion of the chemicals isolated (Table 1) adds further to the complexity of the problem. The resulting combined effluent may have toxicological properties which are wholly unpredictable, and which may be greater or lesser than the sum of the properties of the individual components (Johnston *et al.* 1996). Conversely, even when a complex discharge can be linked directly to a particular environmental response, one is still left with the task of determining which components of the effluent, acting alone or in concert, are responsible for the impacts observed.

A number of recent studies have reported strong synergistic (ie. more than additive) interactions between chemicals, in some cases enhancing the toxicity of individual components several hundred fold (e.g. van Birgelen *et al.* 1996). The mechanisms behind such interactions are unclear, although it is thought that certain chemicals may act as facilitators, increasing the permeability of cell membranes to other toxicants and thereby enhancing the toxic response (Witte *et al.* 1996).

Solid wastes MI6164a and b

The two samples of solid waste arising from the degassing process were found to be largely inorganic in nature (primarily aluminium salts, see below). Nevertheless, a wide range of organic contaminants were detected in both samples, with hexachloroethane among the most abundant compounds present. The majority of the other compounds identified were long-chain (aliphatic) hydrocarbons, possibly present as contaminants in the gas phase in the molten aluminium.

We currently have no information regarding the fate of the solid wastes from the degassing plant although it is likely that they would ultimately be disposed to landfill. Any waste management strategy would need to take account of the presence of a complex mixture of organic contaminants, and of the HCE content in particular.

Sediments MI6165-6169

Discharge of the contaminated wastewater stream from the degassing plant has led to contamination of the sediment in the combined effluent channel with persistent organic pollutants, including organochlorines and polycyclic aromatic hydrocarbons (PAHs). Only two organochlorine compounds were reliably identified in the sediment, penta-chloro(trichloroethenyl)benzene and a chlorinated cyclopentadiene derivative, although these appeared to be present at very high levels. The organic screen analysis is a non-quantitative analytical technique, so it is not possible to determine concentrations of these contaminants in the sediment. Nevertheless, their presence could well indicate the presence of other organochlorines, including dioxins and furans. On the basis of the presence of these compounds, this sediment sample was submitted for quantitative

dioxin analysis; these results are presented and discussed below.

PAHs, including fluoranthene, pyrene, phenanthrene, benzo[ghi]fluoranthene and chrysene, were the other predominant contaminants. As no PAHs were detected in the sediment sampled from the channel upstream from the degassing plant (MI6166), it seems likely that these also stem from processes carried out at the plant. These compounds could have originated as contaminants in the unrefined aluminium or in the gas stream arising from the incomplete combustion of fuels used to fire the refining operation. Whatever their source, their presence is of some significance because of their toxicity. Some PAHs, including chrysene and some benzofluoranthene isomers

are confirmed carcinogens in animals. Other reported effects in exposed organisms include damage to skin, blood and immune systems and interference with development in fetal and juvenile organisms (USPHS 1990b). Although exposure of organisms to these contaminants in situ is unlikely, these toxicological properties would need to be taken into account when planning onward treatment or disposal of these sediments.

Analysis of the other sediment samples from the combined effluent channel and the creek appears to confirm that the refinery, specifically the degassing plant, is responsible for emissions of organochlorine compounds to the environment. Neither MI6166 (upstream of the plant in the effluent channel) nor MI6168 (upstream of the discharge in the creek) contained any identifiable organochlorines. In contrast, HCE was identified reliably in sediments MI6169 (a short way downstream from the degassing plant discharge) and MI6167 (in the creek itself at the point of confluence with the combined effluent channel). This is clear evidence that the plant is acting as an important point source of chlorinated organic chemicals to the aquatic environment and that HCE, and possibly other organochlorines which could not be reliably identified, are accumulating in sediments beyond the plant boundaries.

b) dioxin analysis

As discussed above, sediment MI6165 was selected for quantitative dioxin analysis by an accredited laboratory. Concentrations of principal dioxin and furan congeners are presented in Table 2, along with their toxicity equivalence factors and their relative contributions to the toxicity of the sediment. In concentration terms, the octachlorinated forms were predominant, 43 ug/kg dry weight for octachlorodibenzo-p-dioxin (OCDD) and >260 ug/kg dry weight for octachlorodibenzofuran (OCDF). Note that, in the latter case, a minimum value is quoted. This uncertainty results from interference (peak overlap) in the OCDF region of the chromatogram which could not be removed by further clean-up or simple dilution of the sample extract. The cause of the interference is currently unknown. It is considered likely, therefore, that the value presented is an underestimate of the true concentration of OCDF in the sediment.

Also particularly abundant were 1234678-HpCDD (46 ug/kg) and 1234678-HpCDF (74 ug/kg) although, owing to their higher toxicity equivalence factors, the pentachlorinated forms 12378-PCDD and 23478-PCDF dominated the overall dioxin-related toxicity of the sediment.

Congener-specific concentrations in this sediment sample are very much greater than values reported for natural sediments, even in areas heavily impacted by industrial and urban activities. Note that, in Table 2, concentrations are reported as ug/kg (ppb), rather than the more usual units of ng/kg (ppt). For example, concentrations reported

PCDD/PCDFs	conc. (ug/kg)	I-TEF	I-TEQ (ug/kg)
2378-TCDD	0.31	1	0.31
12378-PCDD	4.20	0.5	2.10
123478-HxCDD	5.90	0.1	0.59
123678-HxCDD	9.10	0.1	0.91
123789-HxCDD	7.10	0.1	0.71
1234678-HpCDD	46.0	0.01	0.46
OCDD	43.0	0.001	0.043
2378-TCDF	3.60	0.1	0.36
12378-PCDF	9.50	0.05	0.475
23478-PCDF	6.80	0.5	3.40
123478-HxCDF	21.0	0.1	2.10
123678-HxCDF	11.3	0.1	1.13
123789-HxCDF	3.20	0.1	0.32
234678-HxCDF	6.20	0.1	0.62
1234678-HpCDF	74.0	0.01	0.74
1234789-HpCDF	13.3	0.01	0.133
OCDF	260 *	0.001	0.26 *
Total ITEQ			14.66 *

Table 2: Congener profile for polychlorinated dibenzo-p-dioxins and furans in sample MI6165 (sediment from combined effluent channel, at point of discharge of effluent from degassing plant). Note that all concentrations are in ug/kg dry weight (ppb).

* Interference on chromatogram may have led to underestimation of OCDF concentration; value presented is therefore a minimum concentration.

here are approximately 100 to 1000 times greater than those reported for the most highly contaminated sediments in the Lower Passaic River (New York, USA), an area receiving chlorinated wastes from a large number of heavy industries including pulp and paper production, metal refining, wood treatment and large-scale chemical manufacture (Wenning *et al.* 1993). Similarly, levels in the Saguenay Fjord and St Lawrence Estuary are three or more orders of magnitude lower than concentrations in sample MI6165 (Brochu *et al.* 1995). Friege and Klos (1990) reported OCDF concentrations of between 1.5 and 2 ug/kg immediately downstream from a large chemical plant producing vinyl chloride and other organochlorines on the River Lippe, a tributary of the Rhine (Germany). This can be compared to the minimum value of 260 ug/kg OCDF in the sample from the refinery. Values from the current study more closely resemble those reported by Muto *et al.* (1991) for contaminated soils and sludges from various chemical manufacturing and waste disposal sites in Japan (not detected-74.7 ug/kg for individual congeners, although the highest values in their data set corresponded to 2378-TCDF).

In terms of toxicity equivalence, levels of dioxins and furans in MI6165 are also

considerably in excess of levels expected in contaminated sediments. Levels reported for sediments in the UK range from 2-267 ng/kg dry weight I-TEQ, the highest levels being recorded in the Mersey and the Humber, both very heavily industrialised areas (Evers *et al.* 1993, Rose *et al.* 1994). Concentrations in other coastal and estuarine areas of the North Sea range from 5-48 ng/kg I-TEQ. The heavily contaminated sediments of Ketelmeer lake in the Netherlands contain approximately 70 ng/kg I-TEQ (Beurskens *et al.* 1993). Against this, the 14.66 ug/kg I-TEQ (14,660 ng/kg) in sediment MI6165 is extremely high.

Indeed, the levels in this sediment sample compare better with highly contaminated solid waste streams associated with some sectors of the chlorinated chemical manufacturing industries. For example, ICI (1994) reported I-TEQs of 3.1-7.6 ug/kg in heavy ends from the manufacture of vinyl chloride. A range of PVC sludge wastes collected from PVC manufacturing sites in the US yielded values in the range 3.9-6370 ug/kg I-TEQ (Stringer *et al.* 1995). Mixed light and heavy ends from the manufacture of PVC sampled from a site in the UK contained 16.5 ug/kg I-TEQ (unpublished data, 1996). The levels reported in this study are, therefore, cause for serious concern, particularly in relation to the onward handling of such wastes and the possibilities for contamination of the wider environment. It was not possible to carry out similar analyses on sediments from the creek downstream of the plant, but it is extremely likely, given the nature of the wastes being discharged, that elevated levels of dioxins and furans would also be detectable in these sediments.

c) heavy metals

Wastewaters MI6161-6163

As expected, aluminium levels in the degassing effluents (MI6161-6162) were high, 2.4 and 2.3 mg/l respectively. By the time the effluent reaches the creek, the concentration has fallen to 0.7 mg/l. Aluminium occurs ubiquitously in natural waters, with levels in surface waters increased directly or indirectly by human activity through surface run-off, tributary inflow, groundwater seepage, industrial and municipal effluents, and wet and dry deposition. Background concentrations of aluminium in surface waters range from 0.06 to 0.3 mg/l (USPHS 1992b), and even though this range is exceeded in this sample, the concentration detected is unlikely to have deleterious effects on the aquatic environment.

Regarding the other metals, copper and lead were present at detectable levels in both the degassing effluents and the creek. Similar concentrations were found in both the wastewater samples. There was a slight reduction in the levels of copper and lead following neutralisation. By the time the effluent reached the creek, levels had fallen: copper 210 ug/l and lead 150 ug/l. These creek concentrations do exceed natural background concentrations, but again, they are unlikely to have a deleterious effect on the aquatic environment.

Solid wastes MI6164a and b

X-ray diffraction analysis of the solid waste from the degassing process revealed it to contain between 65-68% aluminium. Other elements present at significant levels included oxygen, iron, silicon, chlorine, titanium, chromium and potassium. It is not known what treatment and/or disposal operations are in use at the plant, and thus the fate of this solid waste requires further investigation.

Sediments MI6165-6169

All the sediment samples contained elevated levels of aluminium, ranging from 10 g/kg upstream from the refinery discharge, to 223 g/kg close to the neutralisation tank. Analysis of sediment provides an excellent picture of the extent of pollution within a defined area. Water, due to fluctuations in emission and flow follows a less well defined pattern. Heavy metals will bind predominantly to suspended material and finally accumulate in the sediment, providing a reliable history of pollution.

Sediments will always contain some level of heavy metal ions, levels very much dependant on the geology of the area as well as anthropogenic inputs. Elevated levels of heavy metals in these sediments however are clearly due to anthropogenic inputs. Comparison with available published data, shows that as well as aluminium being present in abundance, chromium, copper and lead are all present at levels hugely exceeding background concentrations. Background concentrations of these metals range from 40 to 100 mg/kg (Salomons and Forstner 1984). The most contaminated sample is MI6165, collected from the outfall leaving the neutralisation tank, with g/kg levels of the above stated metals present. Similar levels are found in the creek sediment, downstream from the refinery.

The levels of heavy metals determined in the sediment samples indicate quite clearly that contamination from the refinery is migrating over the site and into the creek. Analysis of sediment samples, collected upstream and downstream from the plant, shows that the anthropogenic input of heavy metals into the creek is considerable. For example chromium levels (one of the elements found as an impurity in the solid waste, see sample MI6164) are over fifty times higher downstream than upstream from the refinery. Aluminium levels are five times higher, with lead concentrations more than doubling.

Chromium concentrations in UK estuarine sediments, collected from a number of industrially polluted sites (including the Mersey, Tyne, Humber and Severn), range from 30 to 200 mg/kg. The highest levels were found in the Loughor Estuary in South Wales, a site of tin plate production. Levels over ten times greater than this are detected in the sediment collected from the creek. There is limited information on the effect of such levels on aquatic biota, plants, fish, deposit feeding and wading birds (USPHS 1992c). It is recognised that the speciation of chromium determines its bioavailability. The speciation will dictate the bioaccumulation of sediment bound chromium by marine and fresh water organisms (Bryan and Langston, 1991). Chromium (VI) will be accumulated more rapidly than chromium (III), as it has been shown to readily cross biological membranes (and for this reason is considered more toxic). The results from these samples give us the total concentration of chromium present, the speciation of the chromium was not identified. It is likely that the dominant species present in the creek sediments is chromium (III), as this is naturally

more abundant. However as levels are so high it would be naive to presume uptake from the sediment to aquatic animals and plants would not occur. Also due to the catalytic properties of other metals, present in high concentrations (e.g. aluminium and manganese), the oxidation of chromium (III) to chromium (VI) is quite possible.

Conclusions

Operations at the aluminium refinery, and particularly the use of hexachloroethane (HCE) in the degassing process, result in the discharge of heavily contaminated wastewater to a combined effluent channel and, ultimately, to an open creek and the sea. This discharge has led to contamination of sediments with organochlorines and heavy metals, many of which are toxic, persistent in the environment and have the ability to bioaccumulate.. The organochlorines are of particular significance because of their relatively high abundance. Analysis of a single sediment sample from the effluent channel for dioxins and furans revealed high levels of contamination (14.66 ug/kg or ppb I-TEQ), within the range previously reported for sludge and tar wastes from the chlorinated chemical industry. Clearly the plant represents a significant point source of persistent organic pollutants to the wider environment. There is, therefore, an urgent need to review operations at the refinery with a view to replacing the HCE degassing process with existing, less hazardous but equally effective processes.

References

- Agg, A.R., and T.F. Zabel (1990). Red-list substances: selection and monitoring. *J. IWEM* 4: 44-50.
- Allsopp, M., P. Costner and P. Johnston (1995). *Body of Evidence: The effects of organochlorines on human health*. Publ. Greenpeace International, May 1995: 90 pp.
- Anderson, R.S., C.S. Giam, L. Ray and M.R. Tripp (1981). Effects of environmental pollutants on immunological competency of the clam *Mercenaria mercenaria*: Impaired bacterial clearance. *Aquatic Toxicology* 1: 187-195.
- Beurskens, J.E.M., Mol, G.A.J., Barreveld, H.L., van Munster, B., and Winkels, H.J., (1993). Geochronology of priority pollutants in a sedimentation area of the Rhine River. *Environmental Toxicology and Chemistry* 12: 1549-1566.
- van Birgelen, A.P.J.M., K.M. Fase, J. van der Kolk, H. Poiger, A. Brouwer, W. Seinen and M. van den Berg (1996). Synergistic effect of 2,2',4,4',5,5'-hexachlorobiphenyl and 2,3,7,8-tetrachlorodibenzo-p-dioxin on hepatic porphyrin levels in the rat. *Environmental Health Perspectives* 104(5): 550-557.
- Birner, G., M. Werner M.M. Ott and W. Dekant (1995). Sex-differences in hexachlorobutadiene biotransformation and nephrotoxicity. *Toxicology and Applied Pharmacology* 132(2): 203-212.
- Brochu, C., Moore, S., and Pelletier, E., (1995). Polychlorinated dibenzo-p-dioxins and dibenzofurans in sediments and biota of the Saguenay Fjord and the St Lawrence Estuary. *Marine Pollution Bulletin* 30(8): 515-523.
- Brouwer, A., .G. Ahlborg, M van den Berg, L.S. Birnbaum, E. Rund-Boersma, B. Bosveld, M.S. Denison, L.E. Gray, L. Hagmar, E. Holene, M. Huisman, S.W. Jacobson, J.L. Jacobson, C. Koopman-Esseboom, J.G. Koppe, B.M. Kulig, D.C. Morse, G. Muckle, R.E. Peterson, P.J.J. Sauer Jr, R.F. Seegal, A.E. Smits-van Prooije, B.C.L. Touwen, N. Weisglas-Kupertis and A. Winneke (1995). Functional aspects of developmental toxicity of polyhalogenated aromatic hydrocarbons in experimental animals and human infants. Outcome of a meeting held from May 9-11 1994 in Wageningen, Netherlands, under the auspices of the EERO Foundation Training and Assesment Programme. *Eur. J. Pharmacol. Environ. Toxicol. & Pharmacol. Section* 293: 1-40.
- Bryan, G.W. and Langston, W.J. (1991). Bioavailability, accumulation and effects of heavy metals in sediments with special reference to UK estuaries: A Review. *Environmental Pollution* 76: 89-113.
- Colborn, T., F.S. vom Saal, and A.M. Soto (1993). Developmental effects of endocrine-disrupting chemicals in wildlife and humans. *Environmental Health Perspectives* 101(5): 378-384.
- Dewailly, E., S. Dodin, R. Verreault, P. Ayotte, L. Sauve and J. Morin (1993a).

High organochlorine body burden in breast cancer women with oestrogen receptors. In Dioxin '93, 13th International Symposium on Chlorinated Dioxins and Related Compounds, Vienna, September 1993: Organohalogen Compounds 13: 403-406.

Dewailly, E., S. Bruneau, C. Laliberte, M. Belles-Iles, J.P. Weber and R. Roy (1993b). Breast milk contamination by PCBs and PCDD/Fs in Arctic Quebec. Preliminary results on the immune status of Inuit infants. In: Dioxin '93. 13th International Symposium on Chlorinated Dioxins and Related Compounds, Vienna, September 1993. Organohalogen Compounds 13: 403-406.

Evers, E.H.G., Klamer, H.J.C., Laane, R.W.P.M., and Govers, H.A.J., (1993). Polychlorinated dibenzo-p-dioxin and dibenzofuran residues in estuarine and coastal North Sea sediments: sources and distribution. Environmental Toxicology and Chemistry 12: 1583-1598.

Fernandez, P., M. Grifoll, A.M. Solanas, J.M. Bayona and J. Albaiges (1992). Bioassay-directed chemical analysis of genotoxic components in coastal sediments. Environmental Science and Technology 26(4): 817-829.

Friege, H., and Klos, H., (1990). Historical development of sediment contamination in an industrialised area. I. PCDDs and PCDFs in river sediments. Organohalogen Compounds 1: 521-528.

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.

Hodges, S.J., S. Meghji, B. Henderson, C. Hopper and M. Harris (1994). The effects of environmental pollutants on bone cell function. Bone Miner. 25: S45.

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

ICI (1994). Report to the Chief Inspector HMIP, Authorisation AK6039, Improvement Condition Part 8, Table 8.1, Item 2: Formation of dioxins in oxychlorination, significance for human health and monitoring proposals. ICI Chemicals and Polymers Report NWJP/BMTD, 27th April 1994: 16pp

Jacobson, J.L., and S.W. Jacobson (1993). A 4-year follow up study of children born to consumers of Lake Michigan fish. J Great Lakes Res. 19(4): 776-783.

Jarrell, J.F., A. McMahon, D. Villeneuve, C. Franklin, A. Singh, V.E. Valli and S. Bartlett (1993). Hexachlorobenzene toxicity in the monkey primordial germ cell without induced porphyria, Reproductive Toxicology 7: 41-47.

- 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.
- Johnston, P.A., R.L. Stringer and D. Santillo (1996). Effluent complexity and ecotoxicology: Regulating the variable within varied systems. *Toxicology and Environment News* 3(4): 115-120.
- McKetta, J., [Ed.](1989). *Encyclopaedia of Chemical Processing and Design*, Volume 3. Marcel Dekker Inc., New York and Basel. 4th Edn.
- Merck (1989). *The Merck Index: An Encyclopedia of Chemicals, Drugs and Biologicals*. Budavari, S., O'Neil, M.J., Smith, A., and Heckelman, P.E., [Eds]. 11th Edn. Merck and Co., Inc., Rahway, N.J., USA
- Muto, H., Saito, K., Shinada, M., and Takizawa, Y., (1991). Concentrations of polychlorinated dibenzo-p-dioxins and dibenzofurans from chemical manufactureres and waste disposal facilities. *Environmental Research* 54: 170-182.
- Myers, M.S., C.M. Stehr, O.P. Olsen, L.L. Johnson, B.B. McCain, S.-L. Chan and U. Varanasi (1994). Relationships between toxicopathic hepatic lesions and exposure to chemical contaminants in English Sole (*Pleuronectes vetulus*), starry flounder (*Platichthys stellatus*) and white croaker (*Genyonemus lineatus*) from selected marine sites on the Pacific Coast, USA. *Environmental Health Perspectives* 102(2): 201-215.
- Newhook, R., and M.E. Meek (1994). Hexachlorobenzene: evaluation of risks to health from environmental exposure in Canada. *Environ. Carcino. & Ecotox. Revs.*, C12(2): 345-360.
- OSPARCOM (1995). Overview of Implementation Reports on PARCOM Decision 93/1 on the Phasing-Out of the Use of Hexachloroethane (HCE) in the Non-Ferrous Metal Industry. Presented by the Secretariat to the Programmes and Measures Committee of the Oslo and Paris Conventions for the Prevention of Marine Pollution, Oviedo 20-24 February 1995.
- OSPARCOM (1996). PARCOM Decision 96/1 on the Phasing-Out of the Use of Hexachloroethane in the Non-Ferrous Metal Industry.
- Rose, C.L, McKay, W.A., and Ambidge, P.F., (1994). PCDD and PCDF levels in river systems in England and Wales, UK. *Chemosphere* 29(6): 1279-1292.
- Sadiq, M. (1992). *Toxic Metal Chemistry in Marine Environments*. Marcel Dekker, Inc. New York, Basel, Hong Kong.
- Salomons, W. and Forstner, U. (1984). *Metals in the hydrocycle*. Springer-Verlag, Berlin, Heidelberg, New York, Tokyo.

- Seegal, R.F., and W. Shain (1992). Neurotoxicity of polychlorinated biphenyls. The role of ortho-substituted congeners in altering neurochemical function. In: *The Vulnerable Brain and Environmental Risks Vol. 2: Toxins in Food*. Isaacson, R.L., and Jensen, K.F., [Eds]. Plenum Press, New York: 169-195.
- Stringer, R.L., Costner, P., and Johnston, P.A., (1995). PVC manufacture as a source of PCDD/Fs. *Organohalogen Compounds* 24: 119-123.
- Sumpter, J.P., and S. Jobling (1995). Vitellogenesis as a biomarker for oestrogenic contamination of the aquatic environment. *Environmental Health Perspectives* 103 (Suppl. 7): 173-178.
- Swindlehurst, R.J., P.A. Johnston, S. Troendle, R.L. Stringer, A.D. Stephenson and I.M. Stone (1995). Regulation of toxic chemicals in the Mediterranean Sea: the need for an adequate strategy. *The Science of the Total Environment* 171: 243-264.
- Thomas, P.T., (1990). Approaches used to assess chemically induced impairment of host resistance and immune function. *Toxic Substances Journal* 10: 241-278.
- USPHS (1990a). Toxicological Profile for Hexachlorobenzene. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry. December 1990.
- USPHS (1990b). Toxicological Profile for Polycyclic Aromatic Hydrocarbons. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry. December 1990.
- USPHS (1992a). Toxicological profile for hexachlorobutadiene. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, July 1992.
- USPHS (1992b). Toxicological Profile for Aluminium. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, July 1992.
- USPHS (1992c). Toxicological profile for Chromium. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, 1992.
- Wenning, R.J., Harris, M.A., Finley, B., Paustenbach, D.J., and Bedbury, H., (1993). Application of pattern recognition techniques to evaluate polychlorinated dibenzo-p-dioxin and dibenzofuran distributions in surficial sediments from the Lower Passaic River and Newark Bay. *Ecotoxicology and Environmental Safety* 25: 103-125.
- Werner, M., G. Birner and W. Dekant (1995). The role of cytochrome p4503a1/2 in the sex-specific sulfoxidation of the hexachlorobutadiene metabolite, n-acetyl-s-(pentachlorobutadienyl)-l-cysteine in rats. *Drug Metabolism and Disposition* 23(8): 861-868.
- Witte, I., H. Jacobi and U. Juhlstrauss (1996). Suitability of different cytotoxicity assays for screening combination effects of environmental chemicals in human fibroblasts. *Toxicology Letters*, 87(1): 39-45.