Identification of Heavy Metals found in samples collected from the IDM Solvent Recovery and Hazardous Waste incinerator, San Lorenzo, Santa Fe Province, Argentina 1998.

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Sample Description

In May and September 1998, three samples were collected from the IDM incinerator, located in San Lorenzo, in the province of Santa Fe. Wastewater from the plant is discharged into the Arroyo San Lorenzo, which itself discharges into the Parana River. One sample of this effluent was collected in May 1998 (LA8035), along with one sample of sediment, collected from the bank of the Arroyo San Lorenzo, close to the waste stream (LA8036). In addition, one sample of sludge was collected from a treatment tank (LA8079), in September 1998. Due to the recent flooding, no samples of surface sediment from the Arroyo San Lorenzo were collected.

Sampling Methodology

All three samples were collected and stored in glass Duran bottles previously rinsed with nitric acid to remove all heavy metal residues. Effluent sample LA8035 was collected in a 1-litre bottle, rinsed three times with the sample before the final collection, and filled completely, thus ensuring no air bubbles were present. Solid samples LA8036 and LA8079 were collected with polyethylene trowels previously washed with detergent, deionised water and nitric acid. Following collection, samples were transported to the Greenpeace Research Laboratory, kept cold during transit, and refrigerated immediately on arrival. Heavy metals were determined quantitatively using Inductively Coupled Plasma-Atomic Emission Spectroscopy (ICP-AES).

Analytical Methodology

Effluent sample LA8035 was preserved in 5% v/v nitric acid on arrival. After 24 hours, 50 ml was transferred to a 120 ml Teflon microwave vessel microwave vessel fitted with a screw cap and pressure relief valve. The vessel was 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 (630 W).

Solid samples LA8036 and LA8079 were dried in an oven for 48 hours, until dry weight readings became constant. They were then crushed using a pestle and mortar until homogenous and sieved through a 2 mm mesh. 0.5 g of sample was then weighed into a 120 ml Teflon microwave vessel. 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 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 (630 W).

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 blank sample, and two internally prepared quality controls (8mg/l, 0.8 mg/l for mercury) were also prepared, matrix matched to the samples.

ICP-AES Analysis

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: manganese (Mn), chromium (Cr), zinc (Zn), copper (Cu), lead (Pb), nickel (Ni), cobalt (Co) and cadmium (Cd). A multi-element instrument calibration standard was prepared at a concentration of 10 mg/l (matrix matched to the samples). The calibration was validated using a quality control standard, prepared from different reagent stocks, at 8 mg/l. Samples exceeding the calibration range were diluted appropriately, 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). Samples exceeding this range were diluted and re-analysed. The quality control standard was again prepared from a different reagent stock at 80% of the calibration range (i.e. 80 ug/l).

Reporting limits of 0.01 mg/l were used for all metals with the exception of lead (0.03 mg/l) and mercury (0.002 mg/l). These were calculated using statistical methods supplied by the UK Water Research Centre (1989).

Results

Are expressed in ppm (parts per million) i.e. mg/l for LA8035, mg/kg (dry weight) for LA8036 and LA8079.

Sample number	**Mn (ppm)**	**Cr (ppm)**	**Zn (ppm)**	**Cu (ppm)**	**Pb (ppm)**	**Ni (ppm)**	**Co (ppm)**	**Cd (ppm)**	**Hg (ppm)**
LA8035	0.04	<0.01	<0.01	<0.01	<0.03	<0.01	<0.01	<0.01	<0.002
LA8036	814.4	31.2	83.2	26	30.2	17.8	12.4	n/d	1.0
LA8079	1485.6	286.9	4865.4	1074.0	5270.2	298.1	17.3	98.1	3.6

Results of heavy metals analysis, IDM Incinerator, Argentina 1998

Discussion

Effluent sample LA8035 and sediment sample LA8036 did not contain significantly high levels of heavy metals. Only manganese was present at detectable concentrations in LA8035, with manganese, chromium, zinc, copper, lead, nickel, cobalt and mercury, found in sample

LA8036, all present at concentrations typical of uncontaminated sediment (Salomons and Forstner 1984).

However of concern are the levels of heavy metals found in sludge sample LA8079. With the exception of cobalt, all the metals determined were present at significant concentrations, with lead and cadmium present at anomalously high levels.

Over 5g/kg (0.5%) of lead was determined in this sample, along with 98 mg/kg of cadmium. Zinc, copper and manganese were also present at concentrations greater than 1 g/kg. Mercury was present at a concentration of 3.6 mg/kg, which when compared with usual environmental levels of less than 0.5 mg/kg, becomes significant (Bryan and Langston 1992, Solomons and Forstner 1984). Equally, when compared with usual environmental levels of 50 mg/kg (Byran and Langston 1992, Salomons and Forstner 1984), chromium and nickel levels are also significant.

Analysis of this waste further establishes the fact that heavy metals can never be destroyed by incineration, they will only ever be redistributed in the waste, and therefore of great concern is the environmental fate of this sludge. Due to the high levels of heavy metals present, further incineration and / or landfill are not environmentally acceptable options. Many of these metals are toxic at very low concentrations; they are also persistent in the environment and have the potential to bioaccumulate through the food chain. Their toxicological properties can be summarised as follows:

Lead

Concentrations of lead found in uncontaminated sediments range from <10 mg/kg to 50 mg/kg (Salomons and Forstner 1984, Bryan and Langston 1992, Licheng and Kezhun 1992, Goncalves *et al.* 1992). Soil levels can range from <10 mg/kg to 500 mg/kg, with levels over 1000 mg/kg being classed as contaminated, and thus unfit for agricultural or recreational use, by the UK Department of the Environment (1980) (Alloway 1990). Sewage sludges can contain up to 3600 mg/kg of lead, however concentrations greater than 1200 mg/kg cannot be applied to agricultural soil as fertiliser (Alloway 1990). Therefore compared to these environmental levels, 5.27g/kg of lead found in sample LA8079, is indicative of severe contamination. The most likely source being the incineration of lead-based paints. With other, less probable sources, including the incineration of other chemical wastes, lead acid batteries, gasoline additives, and domestic sewage.

Unlike some heavy metals, lead is not required by animals (including humans) or plants for normal growth and development. It has no known nutritional or biochemical function and if present in sufficient quantities will inhibit animal and plant growth, development and health (Nriagu 1988).

It is not considered to be one of the most environmentally mobile metals, often heavily bound to suspended particulate and sediment material (Berg *et al.* 1995, Hapke 1991), however there is appreciable evidence to show that sediment-bound lead is available to deposit-feeding species (Bryan and Langston 1992). With high bioconcentration factors (BCFs) being

determined in studies using oysters (6,600 for *Crassostrea virginica*), freshwater algae (92,000 for *Senenastrum capricornutum*) and rainbow trout (726 for *Salmon gairdneri*) (Eisler 1988). It is toxic to all aquatic biota, and organisms higher up the food chain may experience lead poisoning as a result of eating lead-contaminated food.

In terms of human health the effects of lead are the same irrespective of whether it is inhaled or ingested (ATSDR 1997). Lead can cause irreversible central nervous system damage and decreased intelligence at extremely low doses (Needleman *et al.* 1990, ATSDR 1997). At higher levels of exposure anaemia may result, along with severe kidney damage (ATSDR 1997). Children are especially susceptible to lead poisoning because they absorb and retain more lead in proportion to their weight than adults (ATSDR 1997).

Cadmium

Background levels of cadmium in the environment are extremely low. With water concentrations of ng/l being usual (parts per trillion), and sediment levels less than 0.2 mg/kg, most commonly quoted (Bryan and Langston 1992, Salomons and Forstner 1984, Licheng and Kezhun 1992, Goncalves *et al.* 1990). Soil levels range from <1 to 3 mg/kg, with levels over 3mg/kg classed as contaminated, and thus unfit for agricultural or recreational use, by the UK Department Environment (1980) (Alloway 1990). Sewage sludges can contain up to 3410 mg/kg of cadmium, however those containing more than 30 mg/kg cannot be applied to agricultural land (Alloway 1990). Sample LA8079 contained 98 mg/kg of cadmium, a level indicative of severe contamination.

The most often mentioned sources of cadmium entering the aquatic environment are industrial effluents and sewage (Butler and Timperley 1995). Small amounts of cadmium enter the environment from the natural weathering of minerals, but most is released through human activities (Elinder 1985), it most frequently used in the production of nickel-cadmium batteries (35%) and for metal plating (30%). It is also used for pigments (15%), for plastics and synthetics (10%), and for alloys and other miscellaneous uses (10%) (ATSDR 1997), and the incineration of any of these products could result in high levels of cadmium being present in the residual sludge.

Cadmium has no biological function, and is highly toxic to both animals and plants. The low concentrations of cadmium usually encountered in the environment do not cause acute toxicity, however elevations above background concentrations can have deleterious effects on plant and animal health (Bryan and Langston 1992, Alloway 1990).

Toxic effects on exposure biota include observed correlations between increased levels of cadmium found in limpets and a reduced ability to utilise glucose (Shore *et al.* 1975, Bryan and Langston 1992). Reductions in reproduction rates and thus population numbers in copepods and isopods (Giudici and Guarino 1989) have also been observed. The toxicity of low sediment-cadmium concentrations was also suggested by observations showing that in San Francisco Bay, the condition of certain species of clam declined as cadmium concentrations rose from 0.1 to 0.4 mg/kg (Luoma *et al.* 1990).

Regarding potential human exposure, food, water and cigarette smoke will be the largest sources of cadmium for members of the general population. Eating food or drinking water with very high cadmium levels can severely irritate the stomach, leading to vomiting and diarrhoea (ATSDR 1997). Eating lower levels of cadmium over a long period of time can lead to a build up in the kidneys. This cadmium build-up causes kidney damage, and also leads to the weakening of bone (Nriagu 1988). Studies concerned with the effects of eating and drinking high levels of cadmium are not strong enough to show that such exposure can lead to an increased rate of cancer. However the U.S. Department of Health and Human Services and the U.S. Environment Protection Agency have both determined that cadmium and cadmium compounds may reasonably be anticipated to be carcinogens (ATSDR 1997).

Zinc

Background levels of zinc in sediment are usually quoted as being less than 100 mg/kg (Goncalves *et al.* 1990, Bryan and Langston. 1992, Licheng and Kezhun 1992, ATSDR 1997). Soil levels can range from <10 to 250 mg/kg, with levels greater than 500 mg/kg being indicative of contamination (Department of the Environment, UK 1980 in Alloway 1990). Nearly 5g/kg were detected in sample LA8079, indicative of severe contamination.

Environmental releases of zinc from anthropogenic sources far exceed the releases from natural sources (ATSDR 1997). Such anthropogenic releases include those resulting from electroplating, smelting and ore processing, as well as acid mine drainage, effluents from chemical processes (textiles, pigment and paint, fertiliser production), and discharges of untreated domestic sewage.

Although zinc is not regarded as being especially toxic, it is sometimes released into the environment in appreciable quantities, and can thus have deleterious effects on certain species at specific concentrations. For example, effects on fertilisation and embryonic development have been observed in species of fish and harpacticoid copepods (Ojaveer *et al.* 1980, Verriopoulos and Hardouvelis 1988).

In terms of human health, most of the studies relating to the effects of zinc concentrate on exposure via inhalation (which can cause a specific short-term disease called metal fume fever). Less is known about the long-term effects of ingesting too much zinc, through food, water or dietary supplements. It is an essential trace element, but ingestion of higher than recommended levels can have adverse effects on health. The recommended Dietary Allowances for zinc are 15 mg/day for men and 12 mg/day for women. If doses 10 –15 times higher than these recommendations are taken by mouth, even for a short time, stomach cramps, nausea and vomiting may occur (ATSDR 1997). Ingesting high levels for several months may cause anaemia, damage to the pancreas, and decreased levels of high-density lipoprotein (HDL) cholesterol (ATSDR 1997).

Copper

Background levels of copper in sediment range from less than 10 mg/kg to 75 mg/kg (Salomons and Forstner 1984, Byran and Langston 1992, Licheng and Kezhun 1992,

Goncalves *et al.* 1990). Soil levels range from <10 to 100 mg/kg, with levels above 200 mg/kg classified as contaminated, and thus unfit for agricultural and recreational use, by the UK Department of the Environment (1980) (Alloway 1990). Levels in sewage sludges can be as high as 8000 mg/kg, however such these levels cannot be applied to agricultural land as fertiliser (Alloway 1990). Over 1g/kg was detected in sample LA8079.

Anthropogenic sources of copper arise mainly from mining, smelting and metal plating operations; chemical discharges, agricultural runoff and domestic sewage effluents are also significant sources (ATSDR 1997).

Like zinc, copper is necessary for good health. However very large single or daily intakes of copper, or prolonged exposure to lower levels can have adverse effects on human health. Consumption of food or drinking water containing elevated levels of copper can result in vomiting, diarrhoea, stomach cramps and nausea (ATSDR 1997).

Mercury

Mercury concentrations associated with clean sediments are extremely low, with levels ranging from 0.03 mg/kg to 0.5 mg/kg (Bryan and Langston 1992, Salomons and Forstner 1984, Licheng and Kezhun 1992). Soil levels range from 0.2 to 1 mg/kg, with levels over 3 mg/kg being indicative of contamination (Department of the Environment, UK 1980 in Alloway 1990)).

Mercury is a non-essential trace metal, having no biochemical or nutritional function. Biological mechanisms for its removal are poor, and mercury is the only metal known to biomagnify i.e. progressively accumulate through the food chain (WHO 1989, ICME 1995). It is extremely toxic to both animals and plants at low concentrations. Therefore any elevation above baseline levels could have a deleterious effect on any exposed biota (ATSDR 1997).

Since the poisoning incident that devastated the Japanese town of Minamata, the implementation of widespread regulations on mercury disposal has greatly reduced the threat of similar incidents. However the retention of mercury by sediments may delay the elimination of contamination for many years. Thus for example concentrations as high as 100 mg/kg were still present in sediments at certain sites in Minamata Bay, ten years after discharges ceased (Bryan and Langston 1992, Tsubaki and Irukayama 1977). The importance of this is the fact that mercury accumulation from sediments may be a dominant pathway for uptake in aquatic organisms and accounts for relatively high concentrations in deposit-feeders both in freshwater and estuarine systems (Bryan and Langston 1992, Kiorboe *et al.* 1983). Also it known that inorganic mercury can be methylated by microorganisms within the sediment, and it is widely accepted that organic forms of mercury are even more toxic than the inorganic forms (ATSDR 1997).

The most common form of mercury is methylmercury (MeHg), and although there is evidence which links levels of total mercury in the environment with those in higher predators such as fish, concern centers on MeHg accumulation. MeHg exhibits high lipid solubility. It is able to cross cell membranes easily, and therefore quickly enters the aquatic food chain. It also has a

long biological half-life, and due to increased longevity of top predators in association with these other properties, it provides one of the rare examples of metal biomagnification in food chains. For example, MeHg concentrations in carnivorous fish at the top of freshwater and salt-water food chains (e.g., pike, tuna and swordfish) are biomagnified in the order of 10,000-1000,000 times the concentrations found in ambient waters (Callahan *et al.* 1979, EPA 1980, 1984, ATSDR 1997).

The significance of this is that biomagnification of MeHg in aquatic food chains is considered the most important source of non-occupational human exposure to the element (EPA 1984, ATSDR 1997), and as mercury is highly toxic and persistent, anomalous environmental levels warrant concern. Mercury has no beneficial effects in humans, and there is no known homeostasis for it (i.e. no maintained equilibrium between mercury entering the body and leaving). Any long-term exposure may therefore be expected to progressively cause severe disruptions in the normal functioning of any accumulating organ (Nriagu 1988). Accumulating organs include the kidneys, liver and central nervous system, and exposure to high levels of metallic, inorganic or organic mercury can permanently damage these organs (ATSDR 1997).

Chromium

Background concentrations of chromium in sediment range from less than 50 to 100 mg/kg (Salomons and Forstner 1984). With soil levels typically ranging from <10 to 100 mg/kg. Levels over 200 mg/kg are indicative of contamination (Department of the Environment UK, 1980 in Alloway 1990).

Elevations above the background range are nearly always due to anthropogenic discharges, with two industrial sectors responsible for the majority of releases: metallurgical and chemical (e.g. tanning agents, pigments, catalysts).

Information on the effects of elevated environmental levels on aquatic biota, fish, deposit feeding and wading birds is limited; and how sediment-bound chromium reaches animal and plant tissues is uncertain, although it is recognised that the speciation of chromium determines its bioavailability. Chromium (VI) will be accumulated more readily than chromium (III), as it has been shown to cross biological membranes more readily (Bryan and Langston 1992). Whereas chromium (III) is a trace nutrient at low concentrations, chromium (VI) is non-essential and toxic. Its compounds have been classified as carcinogenic by the International Agency of Research on Cancer (ATSDR 1997).

For freshwater fish and shellfish, the uptake of metals through their food may be quite substantial (Heath 1987, Dallinger *et al.* 1987), and metals taken up by plants can be an important route to make metals in the sediments bioavailable to herbivore species (Berg *et al.* 1995).

An average daily intake of 50-200 ug/day of chromium (III) is recommended for adults (ATSDR 1997), chromium (III) being an essential nutrient, required for normal energy metabolism. However the consumption of contaminated fish, other foodstuffs and drinking

water could increase the daily intake levels far beyond those recommended. Ingesting small amounts of chromium (both III and VI forms) has not been reported to cause harm, however ingesting higher than recommended levels over long periods of time can result in adverse health effects including gastro-intestinal irritation, stomach ulcers, kidney and liver damage (ATSDR 1997).

Dermal exposure to both chromium (III) and chromium (VI) can result in severe redness and swelling of the skin (ATSDR 1997). Whereas breathing in high levels of chromium (far less likely in this case) can cause irritation of the nasal and respiratory membranes. These effects have primarily occurred in factory workers who make or use chromium (VI) for several months to many years. Long term occupational exposure to chromium (VI) is believed to be primarily responsible for the increased lung cancer rates (ATSDR 1997).

Nickel

Background concentrations of nickel are usually quoted as being less than 50 mg/kg (Bryan and Langston, 1992; Solomans and Forstner, 1984). Soil levels range from <10 to 20 mg/kg, with levels above 50 mg/kg being indicative of contamination (Department of the Environment UK, 1980 in Alloway 1990).

The most obvious anthropogenic source of nickel is scrap metal waste, notably alloyed metals including stainless steel. However it is also used in electroplating, ceramics, pigments, and as catalysts. Nickel is also used in alkaline (nickel-cadmium) batteries.

Nickel is considered an essential trace element at very low concentrations. It does bioaccumulate in aquatic systems, and as such elevations above normal concentrations can result in deleterious aquatic effects (ATSDR, 1997).

The most common adverse health effect of nickel in humans is an allergic reaction. People can become sensitive to nickel when jewelry or other things containing nickel are in direct contact with the skin. Once a person is sensitized to nickel, further contact with the metal will produce a reaction. The most common reaction is a skin rash at the site of contact. In some sensitized people dermatitis may develop at a site away from the site of contact.

The most serious effects of nickel, such as cancer of the lung and nasal sinus, have occurred in people who have breathed nickel dust while working in nickel refineries or in nickel processing plants. Other lung effects include chronic bronchitis and reduced lung function. The levels of nickel in the workplace were much higher than background levels. The Department of Health and Human Services has determined that nickel and certain nickel compounds may reasonably be anticipated to be carcinogens. The International Agency for Research on Cancer (IARC) has determined that some nickel compounds are carcinogenic to humans and that metallic nickel may possibly be carcinogenic to humans. The EPA has determined that nickel refinery dust and nickel sub-sulfide are human carcinogens.

Manganese

Like zinc, copper, nickel and chromium, manganese is an essential trace element. However exposure to high levels can cause serious illness.

The main use of manganese is in the production of steel (carbon, stainless, high-temperature), along with the manufacture of cast iron and superalloys. The most important compounds of manganese are manganese dioxide, manganese chloride, manganese sulphate and potassium permanganate. Manganese dioxide is commonly used in the production of dry-cell batteries, matches, fireworks, porcelain and glass- bonding materials, and as the starting material for production of other manganese compounds. Manganese chloride often used as a precursor for other manganese compounds, as well as being used as a catalyst in the chlorination of organic compounds. Manganese sulphate is used in glazes and varnishes, in ceramics, fertilisers, as a fungicide, and as a nutritional supplement; and potassium permanganate is used as an oxidizing agent, a disinfectant, an anti-algal agent, a cleaning agent as a preservative for fresh flowers and fruits. Also of importance is the organo-manganese compound MMT, which is used as an antiknock additive in unleaded gasoline.

Incineration of any of these products can yield high levels of manganese in any residual sludge.

Most of the toxicological information on manganese comes from studies of workers chronically exposed to high levels of manganese in the air. Many have suffered both mental and emotional disturbances (ATSDR 1997), 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.

It is not certain whether eating or drinking elevated levels of manganese can cause manganism or not. In one report, humans exposed to contaminated drinking water, developed symptoms similar to those seen in manganese miners or steel workers, but it is not certain if the effects were caused by the manganese alone. Another report found that people who drank water with above average levels of manganese seemed to have a slightly higher frequency of symptoms such as weakness, stiff muscles, and trembling of the hands. However, these symptoms are not specific for manganese, and might have been caused by other factors (ATSDR 1997).

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

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