Impacts of Metals on Aquatic Ecosystems and Human Health

by Frances Solomon

Metal extraction is an important industry for our modern way of life. However, all phases in the life of a mine can discharge metals to estuaries, rivers, streams, and lakes (Ripley and Redmann, 1978). Metals dissolve in water and are easily absorbed by fish and other aquatic organisms. Small concentrations (levels) can be toxic because metals undergo bioconcentration, which means that their concentration in an organism is higher than in water. Metal toxicity produces adverse biological effects on an organism's survival, activity, growth, metabolism, or reproduction. Metals can be lethal or harm the organism without killing it directly. Adverse effects on an organism's activity, growth, metabolism, and reproduction are examples of sublethal effects (Wright and Welbourn, 2002).

In order for a metal to be toxic, it needs to enter the body of the exposed organism and interact with the surface or interior of cells. There are several pathways by which this happens. In addition to diffusion into the bloodstream via the gills and skin, fish can be exposed by drinking water or eating sediments that are contaminated with the metal, or eating other animals or plants that have been exposed to the metal. Humans are exposed to metals via analogous pathways: diffusion into the bloodstream via the lungs and skin, drinking contaminated water, and eating contaminated food (Wright and Welbourn, 2002).

Table 1 presents water quality standards for drinking water and protection of aquatic life (Wright and Welbourn, 2002). Unless otherwise indicated, these are federal Canadian standards. The stricter the standard, the more toxic the metal. Some metals that are especially toxic to aquatic organisms and humans are mercury, cadmium, chromium, and lead. By giving an overview of the toxicity of these metals, this article endeavours to demonstrate the importance of environmentally sensitive mining practice.

Mercury Toxicity

Mercury is an interesting metal because its organic form, methylmercury, is the most toxic. This compound is formed in aquatic ecosystems when naturally occurring bacteria methylate inorganic mercury. The reaction takes place at the water-sediment interface (Wright and Welbourn, 2002) and is facilitated by low pH and high dissolved organic carbon.

Methylmercury dissolves well in water, crosses biological membranes, and persists in fatty tissues of organisms. In addition to bioconcentration, methylmercury undergoes biomagnification; each level of the food chain has higher tissue concentrations than its prey. Mercury levels at the top of the food chain are thousands or millions of times higher than in water or sediments (Wright and Welbourn, 2002).

In a paper published by Wren et al (1983) the biomagnification of mercury in a food chain in a remote, pristine lake in Ontario is illustrated. Although the available analytical methods did not detect mercury in the lake water, bioconcentration resulted in mercury detection in the lake organisms. Biomagnification resulted in higher mercury levels in clams than in the sediment where they dwell, higher levels in smelt than in clams, higher levels in bass and pike than in the smelt that they eat, and highest levels in otters and waterfowl that are the top predators in this food chain. Mercury levels in bass and pike exceeded the 0.5 parts per million (ppm) Canadian federal advisory level for fish consumption.

Human Health Impacts of Mercury

The public became aware of mercury toxicity in the 1950s when fishermen and their families in the Minamata Bay area of Kyushu Island, Japan were stricken with a mysterious neurological illness that was named Minamata Disease. The symptoms were impaired hand-eye coordination, memory and speech loss, blurred vision, blindness, muscle weakening and spasms, and in some cases death. High concentrations of mercury were found in fish and shellfish in the bay; the median concentration in fish was 11,000 ppm (1.1%). An upstream chemical manufacturing factory had used mercuric oxide as a catalyst since 1932 and discharged it to Minamata Bay, where bacteria transformed it to

METAL	DRINKING WATER STANDARD (PPB)	AQUATIC LIFE PROTECTION STANDARD (PPB)
aluminum	100	5 if pH <6.5, 100 if pH >6.5
arsenic	25	5 freshwater (FW), 12.5 saltwater (SW)
cadmium	5	0.017 FW, 0.12 SW
chromium	50	Cr+6: 1 FW, 1.5 SW; Cr+3: 8.9 FW, 56 SW
copper	1000 (based on taste)	2-4 depending on water hardness
lead	10	1-7 depending on water hardness
manganese	500 (World Health Organization)	none
mercury	1	0.1
nickel	20 (World Health Organization)	25-150 depending on water hardness
selenium	10	1
silver	None	0.1
zinc	5000 (based on taste)	30 FW

Table 1: Canadian Water Quality Standards for Metals: Drinking Water and Aquatic Life

methylmercury that underwent bioconcentration and biomagnification. People ate the contaminated fish (Bradl, 2005; Landis and Yu, 2003; Wright and Welbourn, 2002).

Mercury is now known to be a neurotoxin that causes structural damage to the brain and inhibits the activity of enzymes that are needed for normal neurotransmission. This impact occurs at lower concentrations than previously thought (10 ppm instead of 50 ppm). Children and fetuses are at highest risk because their brains are still developing. Some babies were born with Minamata Disease although their mothers did not always show symptoms, suggesting that mercury crosses the placenta (Bradl, 2005; Landis and Yu, 2003; Wright and Welbourn, 2002).

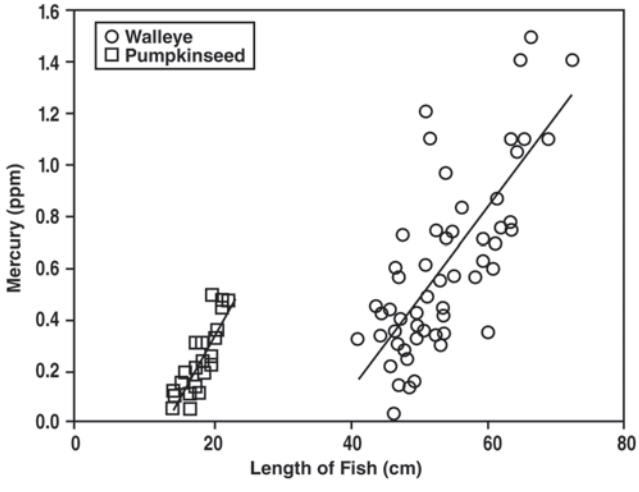
By decreasing the DNA content of cells and adversely affecting cell division, mercury can cause cancer as well as birth defects such as twisted arms and legs. Kidney damage results from long-term exposure to mercury at higher concentrations than those producing neurological damage (Bradl, 2005, Landis and Yu, 2003; Wright and Welbourn, 2002).

Aquatic Ecosystem Impacts of Mercury

The age, size, and species of exposed fish affect the bioconcentration and biomagnification of mercury. Older fish have had more time in which to accumulate mercury in their tissues. Larger fish and predatory species eat further up on the food chain; hence, more biomagnification takes place in their tissues. The graphic illustrates these principles in Lake St. Clair, which has been affected by industrial releases of mercury. Walleye are larger fish than pumpkinseed and feed at a higher level in the food chain. About half of the walleye as well as the larger pumpkinseed had mercury concentrations that exceeded the human health guideline of 0.5 ppm (Ministry of the Environment, Canada, 1992).

Water temperature, pH, and softness, and presence of other metals affect mercury toxicity in fish. Because the body temperature of a fish is the same as the water temperature, the metabolic rate will be higher when the water temperature increases. More mercury will accumulate in fish tissues in summer than in winter (Wright and Welbourn, 2002). When freshwater crayfish were exposed to mercury for 96 hours, the concentration that caused death of 50% of the crayfish was 0.79 parts per billion (ppb) at 20°C, 0.35 ppb at 24°C, and 0.14 ppb at 28°C (Del Ramo et al., 1987).

Mercury is more toxic when the waterbody is acidic because more methylation takes place at lower pH. Mercury is more toxic in softwater (water with low concentrations of calcium ions) than in hardwater because calcium is believed to protect against the uptake of mercury across cell membranes. Selenium and



Environmentally sensitive mining practices are necessary for keeping the balance in ecosystems

cadmium interact antagonistically with mercury; the total toxicity of the metal mixture is less than the sum of the toxicities would predict. These other metals protect aquatic organisms from mercury uptake and toxicity. It is possible that selenium binds with methylmercury, thereby preventing it from inhibiting enzyme activity (Landis and Yu, 2003).

Fish can tolerate ten times as much methylmercury as humans and are more tolerant than their wildlife predators. Storage of methylmercury in the muscle tissue of fish may detoxify it and/or reduce the exposure of brain tissue to it. However, high enough levels of methylmercury cause decreased hatching rate of fish, waterfowl, and marine bird eggs and reduced growth and development of the fish fry and baby birds that have hatched from the eggs. These impacts can have severe repercussions at the population and ecosystem levels because food chains will be impacted and there will be a shift in the species composition of the ecosystem (Wright and Welbourn, 2002).

Cadmium Toxicity

Bioavailability refers to the availability of a metal to enter and affect a biological system. The most bioavailable and therefore most toxic form of cadmium is the divalent ion (Cd+2). Exposure to this form induces the synthesis of a low molecular weight protein called metallothionen, which can then bind with cadmium and decrease its toxicity. This normally takes place in the liver of fish and humans. But if the cadmium concentration is high, the metallothionen detoxification system can become overwhelmed and the excess cadmium will be available to produce toxic effects (Bradl, 2005; Landis and Yu, 2003; Wright and Welbourn, 2002).

Human Health Impacts of Cadmium

At the time that Minamata Disease was detected, the public also became aware of the toxic effects of cadmium. People living in Fuchu, Japan (Jintsu River Basin) complained of joint, bone, and muscle pains and also had symptoms of severe kidney dysfunction. This combination of health problems was called Itai-Itai (Ouch-Ouch) Disease and resulted in the deaths of some residents. A mine located 40 kilometers upstream of Fuchu had discharged untreated effluent, containing cadmium, to rice paddies since the 1920s. Rice plants absorb cadmium easily. Due to bioconcentration, cadmium levels were higher in the rice than in the water in the paddies. There is some evidence for biomagnification of cadmium in the food chain. Therefore, cadmium levels in the bodies of the people who ate the contaminated rice may have been higher than cadmium levels in the rice. Cadmium is toxic to humans when the daily intake is 250 to 300 micrograms. Rice farmers were eating 600 to 1000 micrograms daily – some for up to 30 years (Bradl, 2005; Landis and Yu, 2003; Wright and Welbourn, 2002).

Cadmium is highly persistent in humans, with a biological half-life of 20 to 30 years (Landis and Yu, 2003; Wright and Welbourn, 2002). Exposed humans will never get rid of all of the cadmium in their bodies because it will take 20-30 years to get rid of 50% of the cadmium, 40-60 years to get rid of 75% of the cadmium, and 60-90 years to get rid of 87.5% of the cadmium.

As shown in Itai-Itai Disease, cadmium effects on human health include skeletal deformities and bone loss, kidney damage, and generalized pain. Vitamin D is necessary for deposition of calcium in bones. By blocking Vitamin D synthesis, cadmium prevents the bones from having a normal amount of calcium. The bones become soft or brittle. Chronic cadmium exposure causes kidney damage by inhibiting enzymes responsible for resorption processes. Glucose, protein, and red blood cells are excreted in the urine rather than resorbed into the bloodstream; anaemia is an indicator of kidney damage. The International Agency for Research on Cancer has classvied cadmium as a Category I (human) carcinogen (Bradl, 2005; Landis and Yu, 2003; Wright and Welbourn, 2002).

Aquatic Ecosystem Impacts of Cadmium

Cadmium effects on aquatic organisms are analogous to those in humans, and include skeletal deformities and impaired functioning of kidneys in fish. Cadmium is more toxic in freshwater than in saltwater because cadmium combines with chlorides in saltwater to form a molecule that is less available from solution (Bradl, 2005; Wright and Welbourn, 2002).

The effects of cadmium on aquatic organisms can be directly or indirectly lethal and can impact populations and ecosystems as well as individuals. Skeletal deformities in fish can result in impaired ability of the fish to find food and to avoid predators; hence, this sublethal effect becomes a lethal effect. Cadmium impairs aquatic plant growth. This affects the entire ecosystem because green plants are at the base of all food chains. When aquatic plants that are exposed

to cadmium do not grow normally, there will be less food available for aquatic animals (Bradl, 2005; Landis and Yu, 2003; Wright and Welbourn, 2002).

Cellular damage has been observed in the hepatopancreas of marine crustaceans that experience prolonged exposure to cadmium. The hepatopancreas is a combination of liver and pancreas and therefore has both digestive and metabolic functions. Reduced long-term survival and growth were observed in marine isopods (a group of marine invertebrates) when sublethal cadmium exposure occurred during embryonic and larval development. Some individual animals were more sensitive to the toxic effects of cadmium than others. Differential survival of cadmium-exposed isopods can result in long-term changes in population structure (Bradl, 2005; Landis and Yu, 2003; Wright and Welbourn, 2002).

Metals dissolved in water can kill fish and other aquatic organisms

Chromium Toxicity

Chromium is an essential trace nutrient that is required in small amounts for carbohydrate metabolism, but becomes toxic at higher concentrations. The most bioavailable and therefore most toxic form of chromium is the hexavalent ion Cr+6. Therefore, water quality standards for protection of aquatic life are stricter for hexavalent chromium than for trivalent chromium (Cr+3). Chromium and cadmium interact synergistically; the combined toxicities of these two metals is greater than the sum of their individual toxicities.

Human Health Impacts of Chromium

Four decades after the identification of Minamata Disease from mercury and Itai-Itai Disease from cadmium, the public became aware of the toxic effects of chromium via a film about an exposure incident in the California desert of the U.S. The local utility company used hexavalent chromium as an anti-corrosion agent in cooling towers. Effluent from the cooling towers was discharged to unlined ponds, from where the hexavalent chromium leached into groundwater that supplied drinking water and swimming pool water for the residents of Hinkley, California. Although chromium does not undergo biomagnification, it does undergo bioconcentration. Over 500 people in Hinkley experienced serious health problems including mouth ulcers, nosebleeds, kidney disease, low white blood cell counts – hence depressed immune defense systems, miscarriages, and a variety of cancers. Babies were born with deformed spines (Bradl, 2005; Wright and Welbourn, 2002).

As shown in the film Erin Brockovich and in numerous websites, a clerk at a Los Angeles law firm investigated the health problems and established their association with exposure to hexavalent chromium. Her work resulted in a major lawsuit against the utility company, with financial compensation paid to every resident of Hinkley who had suffered ill health from exposure to hexavalent chromium. An employee of the utility company had been ordered to destroy company records, but instead gave the information to Ms. Brockovich. This case illustrates that one person can make a difference, it is possible to make changes from within a company, and pollution prevention costs less than cleanup or litigation.

Aquatic Ecosystem Impacts of Chromium

Low concentrations of hexavalent chromium cause sublethal toxic effects in aquatic plants and animals. For example, 62 ppb inhibits growth in algae and 16 ppb inhibits growth in chinook salmon (Taub, 2004). Chinook salmon are more sensitive than algae. This is consistent with the overall finding that aquatic animals are more sensitive to metals than are aquatic plants (Wright and Welbourn, 2002). Although reducing the growth of a plant or animal is not directly lethal, the smaller size increases the vulnerability of the organism to predators. What begins as a sublethal effect of a metal may end up as a lethal effect.

As is the case with other metals, chromium toxicity to aquatic organisms increases as water temperature increases and as pH and salinity decrease. Additionally, chromium is more toxic in soft water than in hard water and there are species dvferences in sensitivity. For example, fathead minnows are more sensitive than goldfish. The concentration of chromium that caused death in 50% of the exposed population was 3 ppm in soft water and 72 ppm in hard water for fathead minnows and 18 ppm in soft water and 133 ppm in hard water for goldfish (Puget Sound Water Quality Authority, 1988; Taub, 2004).

Lead Toxicity

The human use of lead goes back at least 5000 years. Because lead is resistant to corrosion and discoloration, its early uses included pipes for the collection, transport, and distribution of water and containers for the storage of food and beverages. Although the use of lead in drinking water pipes has been largely discontinued in developed countries,

Humans are exposed to lead via drinking contaminated water, inhaling contaminated dust, and eating contaminated food

lead-based systems are still found in old buildings in older cities (Wright and Welbourn, 2002). Lead has been used in paint pigments because lead-based paints cling well to wood and lead imparts brightness to the colour (Angier, 2007). This use continues in developing countries. In 2007, U.S. based toy companies and the U.S. Consumer Product Safety Commission recalled toy trains, toy cars, and inexpensive children's jewelry manufactured in China because they contained lead-based paint (Warren, 2007; Lipton and Story, 2007).

Human Health Impacts of Lead

Humans are exposed to lead via inhaling contaminated dust, drinking contaminated water, and eating contaminated food; acidic foods and beverages will solubilise lead from containers. Children are also exposed to lead when they eat leaded paint chips from toys, jewelry, and the walls of old buildings (Bradl, 2005; Landis and Yu, 2003; Wright and Welbourn, 2002).

Lead has adverse behavioural, physiological, and biochemical effects on humans. Fetuses and children under the age of six are most vulnerable. Until the 1970s, a blood lead level of 250 ppb was considered protective of human health. It is now known that this is not a "safe" level for children; 50 – 100 ppb is the current recommended guideline. But whether there is a threshold for effects is unknown (Bradl, 2005; Landis and Yu, 2003; World Health Organization, 1995; Wright and Welbourn, 2002).

Lead accumulates in bones and teeth, where it has a biological half-life of 20 years. As is the case with cadmium, people who are exposed to lead will never completely get rid of it. Although bones and teeth are not harmed, they function as reservoirs for releasing lead into the bloodstream where it then travels to target organs such as the brain. People with a calcium-deficient diet will accumulate more lead in their bones; this results in greater lead toxicity (Bradl, 2005; Landis and Yu, 2003; World Health Organization, 1995; Wright and Welbourn, 2002).

Lead can cross the placenta, resulting in miscarriages, stillbirths, and birth defects such as neurological damage. Neurological impacts of lead include hyperactivity, poor attention span, and low IQ, especially in children. In a Detroit, Michigan study of 5000 children with elevated blood lead levels, a high percentage of the children was found to be in special education classes. Calcium transport across nerve cell membranes is necessary for the normal functioning of the nervous system. Lead mimics calcium, thereby inhibiting neurotransmission (Bradl, 2005; Cunningham and Cunningham, 2004; Landis and Yu, 2003; Wright and Welbourn, 2002).

A major physiological and biochemical impact of lead is inhibition of two key enzymes needed for the synthesis of haemoglobin, the iron-containing protein in red blood cells that binds with and transports oxygen to all cells in the human body. When these enzymes are inhibited and haemoglobin synthesis is disrupted, there is insufficient transport of oxygen and anaemia is the result. Anaemic children fatigue easily and are less able to learn well (Bradl, 2005; Landis and Yu, 2003; Wright and Welbourn, 2002).

Aquatic Ecosystem Impacts of Lead

Lead bioconcentrates in the skin, bones, kidneys, and liver of fish rather than in muscle and does not biomagnify up the food chain. This makes lead less problematic via this route of exposure. However, people who eat the whole fish and wildlife, who, of course, eat the whole fish, can potentially be exposed to high concentrations of lead (Wright and Welbourn, 2002).

When lead concentrations in algae exceed 500 ppb, enzymes needed for photosynthesis are inhibited (Taub, 2004). When less photosynthesis takes place, the algae will produce less food and therefore will not grow as much. Decreased algal growth means less food for animals; this has repercussions for the entire ecosystem.

Fish are more sensitive than algae to lead. When lead concentrations exceed 100 ppb, gill function is affected. Embryos and fry are more sensitive to the toxic effects of lead than are adults. Lead is more toxic at lower pH and in soft water (Taub, 2004; Wright and Welbourn, 2002). As is the case with other metals, the toxicity of lead to fish depends in part on the species. Goldfish are relatively resistant because they can excrete lead via their gills (Landis and Yu, 2003).

Further Information on Metal Toxicity
Further information can be obtained about toxic effects of mercury, cadmium, chromium, lead, and other metals that are mined or emitted as by-products of mining by registering for the online EduMine course.

The University of British Columbia (UBC) Mining Studies Institute will offer a three-day classroom version of the course from April 30 through May 2 of 2008 at UBC Robson Square. Course information and registration are available online.

The term-length course will be offered during the 2008-2009 academic year at the main UBC campus.

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Links and References

- Anaemia
- Aquatic ecosystems
- Bioconcentration
- Biological effects
- Biomagnification
- Calcium-deficient diet
- EduMine course Impacts of Metals on Aquatic Ecosystems and Human Health.
- Erin Brockovich – The Film
- Methylmercury
- Minamata Disease
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