A Literature Review of Effects of Cadmium on Fish

November 2010

For

The Nature Conservancy
Protecting nature. Preserving life.™
nature.org

By

CSP²

Stuart M. Levit, MS, JD
Center for Science in Public Participation
Bozeman, Montana

Introduction

The purpose of this paper is to review relevant literature regarding fisheries and the threat posed by potential cadmium contamination that could result from mining near Bristol Bay, Alaska. Mining sites may pose a unique risk because metal concentrations in water at mines tend to be several orders of magnitude higher than those for uncontaminated sites (EPA 2000).

Proposed mining of copper-sulfide ore bodies near Bristol Bay, Alaska, will unearth rock and soil with elevated cadmium concentrations (*see e.g.*, Pebble Limited Partnership's Pre-Permitting Environmental / Socio-Economic Data Report Series).[1]

Cadmium in the Environment

- Once rocks with elevated cadmium concentrations are exposed at the earth's surface, cadmium will leach from the solids and dissolve in water, especially under acidic pH conditions.
- Dissolved cadmium and its compounds are toxic to humans, wildlife, and especially aquatic biota, at low concentrations.
- Like all metals, cadmium can move from one environmental compartment to another (e.g., from groundwater to soil), but it does not degrade by breaking down to a less toxic form.

See CSP2 Fact Sheet, http://www.csp2.org/reports/Fact_Sheets--Trace_Elements_in_Mining_Waste.pdf

Past studies and recent data from the region indicate that mining of copper-sulfide ore in Bristol Bay has the potential to create acid mine drainage, which will mobilize metals, including cadmium.[2] Acid mine drainage forms when iron sulfide minerals at a mine are exposed to air and water. This acidic drainage can dissolve metals and metalloids, including cadmium, causing them to leach from the mined rock (whether in waste rock, tailings, mine workings, pit walls, ore, etc.) and into the environment. Once started, acid mine drainage is difficult to stop or

[1] Pre-Permitting Environmental / Socio-Economic Data Report Series Report Series Trace Elements (Sediments & Soils) Report E-2 Summary of Mine Area Sediment Inorganic Analytical Results, 2004 - 2007 (Identified by the report as "preliminary," but no final report has yet been identified. Data should be considered preliminary). The data includes soils and sediment cadmium concentrations in materials that are proposed for mining by that company. http://www.pebblepartnership.com/documents/report-e-trace-elements-soils-and-sediments (accessed 19 July 2010).

[2] Current exploration and other data evidence the presence of sulfide, which is the mineral that forms sulfuric acid upon contact with water and oxygen. In the West zone; "50% of Cominco holes in Pebble West bottomed in sulfide mineralization with grades of 0.6% CuEQ and 96% bottomed in mineralization with grades higher than 0.30% CuEQ" (Rebagliati et al. 2009). There is also pyrite (FeS2) beneath the overburden material of the East zone (Rebagliati and Payne 2005). These data, notably with pyrite, indicate that iron sulfides are present and therefore that acid mine drainage is possible. Further characterization is necessary to identify the actual potential for acid mine drainage.
See also, Pre-Permitting Environmental / Socio-Economic Data Report Series Report Series E. Trace Elements (Sediments & Soils) Report E-2 Summary of Mine Area Sediment Inorganic Analytical Results, 2004 - 2007 (Identified by the report as "preliminary", but no final report has yet been identified. Data should be considered "preliminary").

reverse. Cadmium can contaminate water by leaching from waste rock piles, tailings materials, and exposed/mined-surfaces (especially in the open pit and underground workings).

Cadmium Background

Cadmium is a metal with no known beneficial properties that support life - there is no evidence that it is either biologically necessary or beneficial (Eisler 1985; Eisler 2000; Nordberg et al. 2007). At low concentrations it is toxic to all life, including plants, fish, birds, mammals (including humans), and microorganisms (Eisler 1985; Eisler 2000; Nordberg et al. 2007, ATSDR 2008). It causes cancer, birth defects and genetic mutations (Eisler 2000, Nordberg et al. 2007). In one study that used comparative acute toxicity testing of 63 heavy metals, *cadmium was the most toxic metal* (Borgmann et al. 2005).

In its elemental form Cadmium does not break down but it can change form into different species and compounds. Some species can bind strongly to soil or sediment particles, depending primarily on the acidity of the surrounding water. In sulfide ore bodies, cadmium is usually associated with sphalerite (a zinc sulfide mineral) (USGS 1996). Other sources of cadmium are coal and other fossil fuels, and shale. It can also be released to the atmosphere during volcanic eruptions. It can enter the atmosphere during the burning of coal and household wastes, and can enter water around a sulfide mine as a byproduct of acid mine drainage (*See e.g*. Edmonds and Peplow 2000).

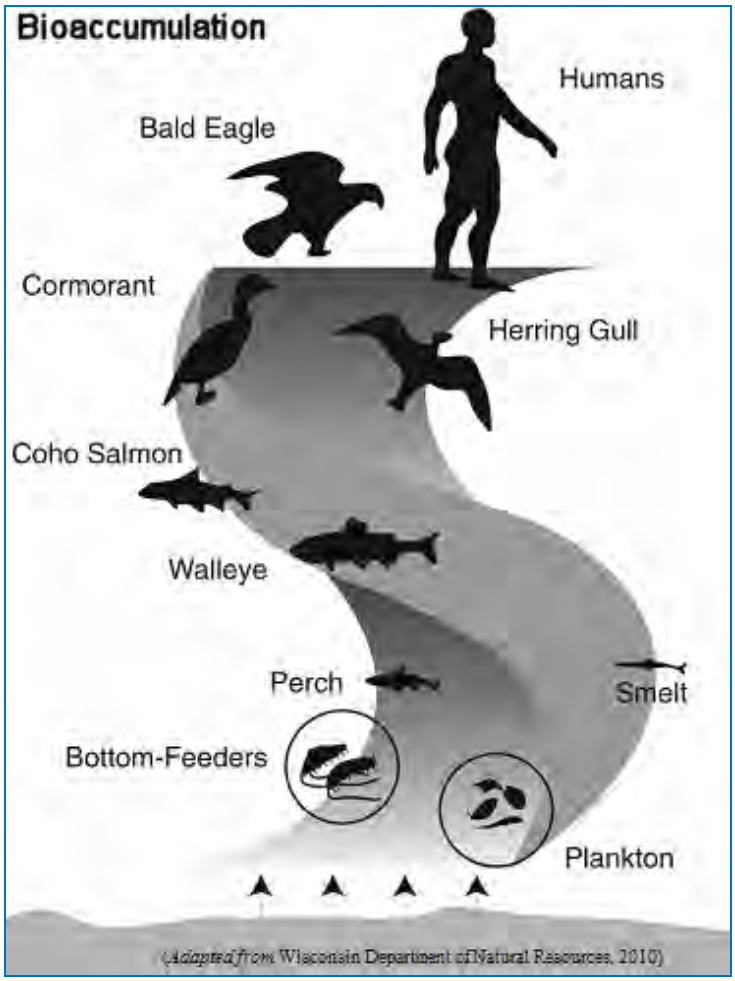
In unpolluted waters, natural cadmium concentrations are generally less than 1 µg/L or 1 part per billion (ppb) (Nordberg et al. 2007). Most cadmium is released from human activities such as mining and smelting of sulfide ores, fuel combustion, and application of phosphate fertilizers or sewage sludge (USPHS 1993). For example, one estimate indicated over 300,000 kg of cadmium was released to the environment from human activities in 1988 (USPHS 1993). Because it is a nondegradable, cumulative pollutant, continued releases are of global concern (ATSDR 2008; Hutton 1983; Tjell 1983).

Cadmium Behavior and Toxicity

Cadmium and other metals released from mining sites can contaminate drinking and other water sources (*See e.g.* Peplow and Edmonds 2004; Younger et al. 2002). Metal contamination in aquatic systems is a particular concern because metals are both persistent and toxic (Clark 1992).

When, where, and how an animal eats can play a role in cadmium behavior:

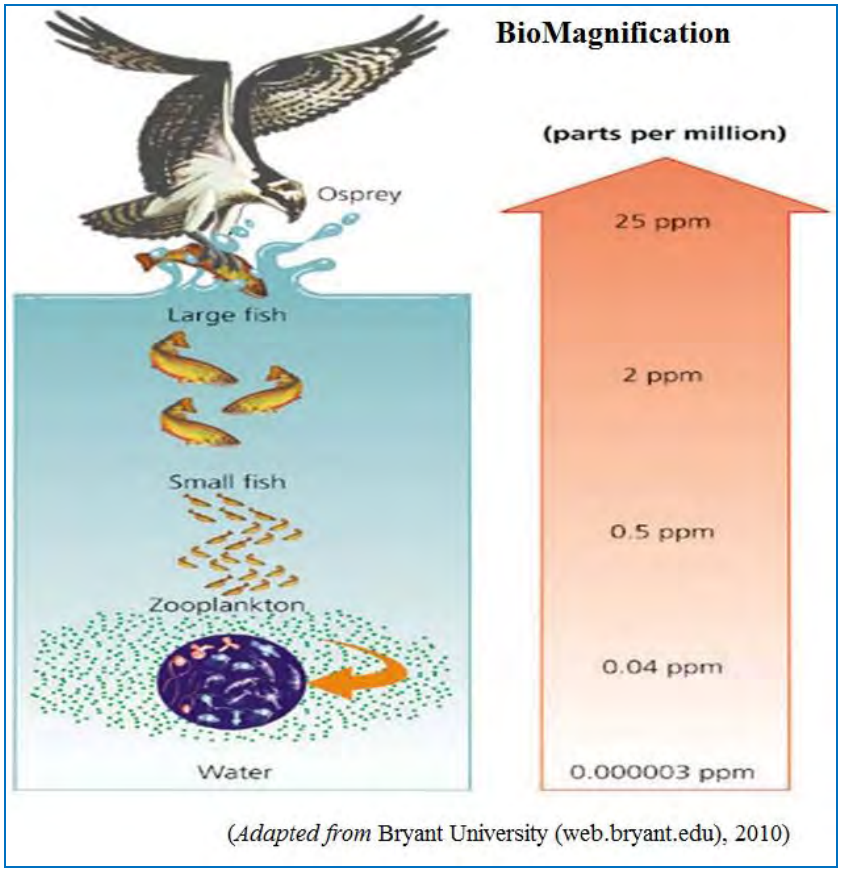
- Animals that accumulate cadmium in their bodies ("body burden") can be eaten by others, and so on, such that cadmium will both accumulate and biomagnify in the food chain (see below) (EPA 2000).



- Fish can accumulate cadmium from the water and eating foods contaminated with cadmium (contaminated food chain). It is important to note that bioaccumulation-magnification occur when a substance cannot be easily metabolized or excreted. Cadmium exhibits this persistence (ATSDR Medical FactSheet).
    - ***Bioaccumulation*** occurs when an animal accumulates cadmium from the environment into its body.
    - ***Biomagnification*** of cadmium occurs when the concentration of cadmium increases from one link in the food chain to another link in the food chain. A bigger fish eats multiple small fish and accumulates the cadmium from those fish in its body tissue. As that fish ages, it keeps accumulating the cadmium from all of the contaminated smaller fish it eats. The cadmium further accumulates - or *magnifies* - as a bigger fish eats that fish, and so on.
    - Biomagnification can happen when eagles, bears, or any animals eat prey species that have bioaccumulated cadmium.
    - For example:
        - Spawning salmon, incubating salmon embryos, fry rearing up to 3 years, and migrating salmon can all boiaccumulate and biomagnify cadmium. They may therefore be harmed as individuals from their exposure and

accumulation of cadmium. They may also cause cadmium to biomagnify in humans who eat them.

- Mussels accumulate cadmium (*see e.g.* Das et al. 2004; Sunila 1986). If a humpback whitefish eats contaminated mussels, it will biomagnify cadmium. If a human eats the humpback whitefish, the human will biomagnify cadmium in his or her body. This is particularly important for subsistence diets based on fish in Native Alaskan communities.



(*Adapted from* Bryant University (web.bryant.edu), 2010)

- The most likely source of cadmium toxicity in humans is contaminated foods (ATSDR 2008). People who regularly consume shellfish and fish organ meats (liver and kidney) may have increased cadmium exposure (ATSDR 2008). As described below, mining copper-sulfide ores near Bristol Bay poses a risk of cadmium releases to the environment, which in turn could lead to fish and subsequent human contamination.[3]
- Cadmium exposures can be acute or chronic.

[3] The ATSDR warns: Elevated cadmium levels in water sources in the vicinity of cadmium emitting industries (historical and current) have been reported. Aquatic organisms will accumulate cadmium, possibly entering the food supply. People who fish in local waters as a means of food should be cautious and abide by any advisories. ATSDR reports: "Recreational and subsistence fishers that consume appreciably higher amounts of locally caught fish from contaminated waterbodies may be exposed to higher levels of cadmium associated with dietary intake (EPA 1993a). Cadmium contamination has triggered the issuance of several human health advisories. As of December 1997, cadmium was identified as the causative pollutant in five fish and shellfish consumption advisories in New York and another in New Jersey."

- Cadmium would be acutely toxic when an animal dies from exposure to a high concentration over a short period of time.
- Cadmium would be chronically toxic when the animal is exposed to cadmium over a long period of time at a lower concentration. While chronic exposure can lead to mortality, sublethal effects such as reduced growth and reproductive success are more common. Cadmium may also accumulate in aquatic biota chronically exposed to sublethal concentrations.

Cadmium Bioaccumulation

- Aquatic and terrestrial organisms bioaccumulate cadmium.
- Cadmium concentrates in freshwater and marine animals to concentrations hundreds to thousands of times higher than in the water.
- Reported bioconcentration factors (BCFs) range from <200 to 18,000 for invertebrates and from 3 to 4,190 for fresh water aquatic organisms.
  - Bioconcentration in fish depends on the pH and the humus content of the water.
- Cadmium bioaccumulates in all levels of the food chain. Cadmium accumulation has been reported in grasses and food crops, and in earthworms, poultry, cattle, horses, and wildlife

(ATSDR Draft Toxicological Profile for Cadmium, 2008)

Even if contaminants are not expected to be bioavailable (such as refractory metal sulfide solids), processes in the environment or in the body of those exposed to cadmium compounds may alter or render the metals bioavailable (*See* Schaider et al. 2007).

Cadmium biomagnifies in the food chain and can accumulate in humans. For example, cadmium can enter freshwaters due to mining activities (*see e.g*. Edmonds and Peplow 2000) where it is taken up by algae, insects, and passed up the food chain to fish, which can accumulate it in their gills, liver and kidneys (*See e.g*. Eisler 1985; Nordberg et al. 2007; ATSDR 2008; Edmonds and Peplow 2000). Humans accumulate cadmium primarily in the kidneys and liver, and its impacts are greatest to the lungs (from inhalation) and kidneys (from ingestion) (ATSDR 2008).

A comparison of metals body burdens in grayling and lake trout from an arctic lake suggested that dietary, physiological, or microhabitat differences play a role in heavy metal accumulation in Arctic lakes (Allen-Gil et al. 1997). The dietary component may be explained by grayling exclusively consuming insects, which are lower on the food chain and likely had lower metal burdens (Allen-Gil et al. 1997). By comparison, lake trout tend to consume a mixture of snails, insects, and small fish, representing a diet higher on the food chain - which can increase the probability of them eating prey species with greater body burdens of cadmium (Allen-Gil et al. 1997). In other words, lake trout would likely have higher dietary exposure to cadmium than grayling because lake trout eat more species from higher in the food chain - and higher food

chain species are more likely to have higher cadmium levels. Lake trout may have also taken-up more metals than grayling because grayling spend most of their time near the water surface, whereas lake trout tend to be dispersed through the water column or reside in the lower layers throughout most of the year (Allen-Gil et al. 1997).

Cadmium has been shown to increase in agricultural soils and cause contamination and impaired kidney function in people eating foods from those soils (Olsson et al. 2005). Similarly, consumption of a cadmium-contaminated rice and fish diet in north central Sri Lanka has been shown to cause chronic renal failure (Bandara et al. 2008). Cadmium has been demonstrated to persist in the human body for up to 38 years, underscoring the importance of decreasing human exposure to cadmium (ATSDR 2008; Nordberg 2006).

Most studies indicate that cadmium is no more toxic in a mixture than when tested alone. However, results from toxicity studies on cadmium and copper mixtures were contradictory; some indicated that cadmium and copper mixtures can result in greater toxicity than cadmium alone or than cadmium and zinc mixtures (Mebane 2006).

Fish species often respond differently to the same environmental impact. For example, Bull trout - but not rainbow trout - were more sensitive when exposed to a mixture of cadmium and zinc than when exposed to cadmium only (Hansen et al. 2002). The cause of these differences in toxicity on the different species is unclear (*See* Hansen et al. 2002). But the results and the unknowns underscore that many fundamental impacts of cadmium are yet unknown and that how cadmium will impact fish in a mine-impacted environment is not clear.

Increasing water temperature (from 8^0 to 12^0 C) increased the rate of cadmium and zinc toxicity in Bull trout and rainbow trout (Hansen et al. 2002). Researchers have observed similar findings for other fish, including Atlantic salmon (*Salmo salar*) (Hansen et al. 2002).

Cadmium Toxicity

- Fish, plants, and animals uptake cadmium from the environment - through food, water, and breathing (including the gills in fish).
- Animals given cadmium in food or water develop high blood pressure, iron-poor blood, liver disease, and nerve/brain damage.
- Cadmium affects birth weight and skeleton development in animals.
- Cadmium is toxic to aquatic organisms at low concentrations.
    - Clean Water Act Fresh Water Criteria – Criterion Continuous Concentration: 0.25 µg/L (the highest concentration to which an aquatic community can be exposed *briefly* without resulting in an unacceptable effect).
    - Criterion Maximum Concentration: 2.0 µg/L (an estimate of the highest concentration to which an aquatic community can be exposed *indefinitely* without resulting in an unacceptable effect).

(CSP2 Fact Sheet, http://www.csp2.org/reports/Fact_Sheets--Trace_Elements_in_Mining_Waste.pdf

Cadmium Accumulation

Cadmium accumulates in the kidney, liver, and gills of freshwater fish (Chowdhury et al. 2004; Dallinger et al. 1996; Thomas et al. 1985; Norey et al. 1990a; Kraal et al. 1995). Cadmium accumulation in these organs appears to be related to the presence of cadmium-binding molecules called metallothioneins (Dallinger et al. 1996; Carpene and VaSik, 1989; Hogstrand et al. 1991).

There is evidence that a high accumulation of cadmium in fish (Arctic char) might be the result of increased metal absorption in the gills from the water due to low alkalinity (Dallinger et al. 1996; Wograth and Psenner 1995; Isock et al. 1995). Alkalinity plays a role in what and where cadmium and other metals accumulate, such that liver metallothionein was dominated by copper and zinc in spite of high cadmium levels in the kidney (Dallinger et al. 1996). The role of metallothionein in fish accumulation is well demonstrated (See e.g. Dallinger et al. 1996 and Yudkovski et al. 2008). As a result, in the Bristol Bay waters, which tend to have low alkalinity (see Northern Dynasty Mines 2005), fish may uptake environmental cadmium at a higher rate than in comparable higher alkalinity waters.

In Arctic Char, cadmium tended to accumulate in the kidney, while copper and zinc tend to accumulate in the liver (Dallinger et al. 1996). However, as cadmium levels increase in these organs, zinc levels tend to decrease (Dallinger et al. 1996). Studies of rainbow trout showed that an accumulation of cadmium in the kidney was accompanied by an increased urinary excretion of zinc (Dallinger et al. 1996; Giles, 1988).

As fish age, cadmium and cadmium (and zinc)-metallothionein concentrations in the liver increase (Dallinger et al. 1996). Cadmium in the kidney has also been shown to increase with age (Dallinger et al. 1996; Kock et al. 1995). These findings are consistent with findings that both cadmium and metallothionein concentrations in fish tissues increase under conditions of chronic exposure to cadmium (Dallinger et al. 1996; Brown et al. 1990; Bonwick et al. 1991; Chatterjee and Maiti, 1991) regardless of species-specific differences (Norey et al. 1990a).

As a related matter, it has been shown that while some fish, such as Arctic char, may eliminate cadmium (Dallinger et al 1996; Kock et al. 1996b), long term net accumulation still occurs in Arctic char that are exposed to a continuous supply of cadmium in water (Dallinger et al. 1996). The distinctions between and impacts of water versus food/ingestion cadmium sources have not been clearly identified (*See. e.g.* Saiki, et al. 2000).

Where cadmium accumulates in fish may be as important as sources and exposure pathways. Fish exposed to cadmium in their diet versus via water took up high cadmium burdens in the gut versus gills, respectively, in addition to other target tissues such as kidney and liver (Chowdhury et al. 2004; Harrison and Klaverkamp 1989; Szebedinszky et al. 2001). In the gills, cadmium burdens can result from both dietary and waterborne cadmium exposure (Chowdhury et al. 2004; Szebedinszky et al. 2001; Handy, 1996). Gill burdens alone therefore are not diagnostic of the exposure route (Chowdhury et al. 2004). Even within the gut cadmium burdens vary, although causation is not clear (and acclimation to cadmium may also impact burden and excretion

(Chowdhury et al. 2004). Among the non-gut, internal tissues, cadmium accumulation from both waterborne and dietary sources is greatest in the kidney and liver -- which are considered to be most significant for cadmium metabolism and detoxification (Chowdhury et al. 2004; Harrison and Klaverkamp 1989; Farag et al. 1994; Kraal et al. 1995; Szebedinszky et al. 2001). It appears that muscle and the brain are protected tissues, accumulating only negligible levels of total and new cadmium from both dietary and waterborne exposures (Chowdhury et al. 2004; Szebedinszky et al. 2001; Chowdhury et al. 2003).

Hardness and pH

Ambient water quality criteria for cadmium (and chromium III, copper, lead, nickel, silver, and zinc) for the protection of aquatic life are based on water hardness. Fish incubated in higher hardness water were about two times more resistant to cadmium toxicity than the fish incubated in extremely soft water (Mebane et al. 2009). Fish are more sensitive to metals such as cadmium and zinc that inhibit ion-regulation because of the energy demands required to maintain a constant condition/homeostasis in lower hardness water (Mebane et al. 2009). To accurately test the impacts of cadmium and other metals on fish, it is important that the role of hardness and acclimation be evaluated (*See* Mebane et al. 2009).

Cadmium's acute toxicity decreases with increasing water hardness (Niyogi et al. 2008; Calamari et al. 1980; Davies et al. 1993; Brinkman and Hansen 2007). Cadmium can compete with Ca^{2+} at the gill surface (Niyogi et al. 2008; *see also* McGeer et al. 2000). Little is known about the effects of other potential competing ions, such as Mg^{2+} or Na^{+}, on cadmium accumulation and/or toxicity in fish (Niyogi et al. 2008). For cadmium's impacts on fish, however, the concentration of Ca has a greater influence than Mg (Hansen et al. 2002; Niyogi et al. 2008). The impacts of water hardness on cadmium toxiciy needs further assessment and analysis because alkalinity co-varied with hardness levels in many of the toxicity experiments (Niyogi et al. 2008). The results from at least one study suggested that calcium and dissolved organic content in water are the most protective factors against acute cadmium toxicity in freshwater fish, with calcium being the more important of the two (Niyogi et al. 2008).

Water pH also influences the toxicity of cadmium but is not considered in ambient water quality criteria (Hansen et al. 2002). As a result, water quality standards for a given location or stream must also consider the fish species present, water quality, and life stages (*See* Hansen et al. 2002). The effects of pH on acute cadmium toxicity are largley unknown (Niyogi et al. 2008; Playle et al. 1993). There are some indications that low pH may actually protect some fish against acute cadmium toxicity (Niyogi et al. 2008). It is important to understand the effects of pH independent of other surrogate variables (*See* Niyogi et al. 2008).

Cadmium and Fish Life Stage

Rainbow trout mortality from increased cadmium exposures appears to be lower for eggs or alevins and higher for swim-up fry (Mebane et al. 2007; Chapman, 1978b; Van Leeuwen et al. 1985).[4] In many fish tests, the life stage at which the tests were started appears to have major

[4] The USFWS summarizes early fish life stages as follows (USFWS 2007): When a fish hatches from its egg, it is called a yolk-sac fry, or alevins (because a tiny food supply - a sac of egg yolk - attached to its belly). Sac-fry stay

impacts on the test results and measured fish sensitivity and responses (Mebane et al. 2007). In other words, the life state at which the fish is exposed to contamination is an important factor in the fish's response and survival. Because mining in the Bristol Bay region would likely occur year-round, it will probably impact fish during all of their life stages - including those most sensitive to cadmium impacts.

The increased sensitivity of swim-up fry to metals likely results from their increased rates of respiration corresponding with the increased body activity as the fry turn from non-free-living organisms into a free-living stage and have to capture food (Mebane et al. 2007). Much greater water volumes move across gills to meet gas exchange needs in swim-up fry stage than sac fry stage (Mebane et al. 2007, Van Leeuwen et al. 1985).

Life stage should be an important consideration for determining water quality impacts to fish health and mortality. Acclimation to cadmium exposure does not imply or guarantee that fish are "healthy." Assessing actual health would require comparing the adapted population to a population living in an uncontaminated/undegraded environment. Copper-sulfide mining near Bristol Bay could adversely affect all freshwater life stages of salmon and all life stages of non-salmon, especially Dolly Varden and rainbow trout, and their food chains. The future of Bristol Bay fisheries requires adequate assessment of and protection from cadmium toxicity.

Sublethal Effects

Sublethal effects such as decreased growth, inhibited reproduction, and population alterations may occur after chronic exposures and can be pronounced or probable when cadmium concentrations exceed 3 □g/L (30 mg $CaCO_3$/L) in fresh water (Eisler 1985). However, some studies have concluded that sublethal cadmium exposure did not reduce growth but did cause alterations to appetite and metabolism (McGeer et al. 2001). Compared to pre-exposure conditions, fish exposed to cadmium consumed less food to achieve the same growth rate (McGeer et al. 2000). This is consistent with other studies that have observed hypoactivity from exposure to certain metals, suggesting that reduced activity permitted a greater proportion of consumed energy to be directed towards growth (McGeer et al. 2000; Wilson et al. 1994). Therefore, a sensitive measure of chronic sublethal effects may be measurement of behavior, particularly spontaneous and basal activity levels (McGeer et al. 2000). The comparison between Eisler's data and that of later studies suggests that further study is needed to ensure that fisheries are adequately protected from cadmium's sublethal toxicity.

Cadmium is an endocrine disrupter (Vetillard and Thierry Bailhache 2004). Cadmium has been shown to interfere with the formation of steroids, eggs, and sperm, in rainbow trout, and it alters hormone synthesis in testes. In carp (*Cyprinus carpio*) it inhibits steroid formation and ovarian maturation (Vetillard and Thierry Bailhache 2004; *see also* Szczerbik et al. 2006). Extensive (1.8 and 3.4 µg/L; *see* Brown et al. 1994[5]) cadmium exposure in trout causes delayed egg

in the protection of the gravel environment until the yolk is used up and are they are called "fry". They swim to the surface and gulp air to fill their swim bladders and begin to feed.

[5] Eggs obtained from rainbow trout exposed to 1.8 and 3.4 micrograms Cd liter-1 failed to develop to the fry stage. Brown trout exposed to 9.3 and 29.1 micrograms Cd liter-1 appeared to suffer delayed egg formation, but the eggs and fry that were produced developed normally after fertilization (Brown et al. 1994). Adult brown trout suffered "considerable mortality" when exposed to 29.1 micrograms Cd (Brown et al. 1994).

formation, and it has been shown to inhibit egg development into the fry stage (Vetillard and Thierry Bailhache 2004; *see also* Szczerbik et al. 2006). Direct exposure of rainbow trout embryos to cadmium induces premature hatching, mortality, and developmental abnormalities (Vetillard and Thierry Bailhache 2004).

Exposures to low levels of cadmium can cause DNA damage and stress in common carp (*Cyprinus carpio var. color*) (Jia et al. 2010). In Japan, Belgium, Sweden and China, cadmium exposure has caused bone and reproductive cancer in general population groups (Nordberg 2006).

Cadmium: from Fish to Humans

Fish provide an important subsistence food source for many families and people in the Bristol Bay watershed (*See e.g.* Krieg, et al, 2005). This is consistent with findings from the 1980s, which concluded that subsistence fishing activities have a long-historic importance to the Bristol Bay region; that fishing, hunting, gathering, and processing of wild resources are group activities participated in by relatives and friends; and that subsistence products are widely shared within and between communities (Wright, et al. 1985). Furthermore, fisheries-subsistence activities are important expressions of, and mechanisms for, maintaining values such as kinship, community, respect for elders, hospitality to visitors, and traditional ideological concepts (Wright, et al. 1985). In other words, there are multiple, complex values (e.g. ecological, social, cultural, health and diet) associated with subsistence fisheries.

Cadmium and Human Health

- Cadmium has no known beneficial function in the human body.
- Cadmium accumulates in animal tissue, and its toxicity can increase as accumulation increases.
- Cadmium causes cancer, birth defects, and genetic mutations.
- The greatest cadmium concentrations are found in the kidneys and the liver.
- Urinary cadmium excretion is slow; however, it is the primary mechanism by which the body eliminates cadmium.
- Due to slow excretion, cadmium accumulates in the body over a lifetime and its biologic half-life may be up to 38 years.

(ATSDR Fact Sheet: http://www.atsdr.cadmiumc.gov/csem/cadmium/cadmiumbiologic_fate.html)

For example, In the Kvichak River watershed of the Bristol Bay area, subsistence harvests of non-salmon freshwater fish contributed substantially to the annual food supply for the area's families (Krieg, et al. 2005). Moreover, the growth of a large, commercial, market economy within the Bristol Bay region has not supplanted the traditional reliance upon local, wild resources for subsistence use (Wright, et al. 1985). Historically in the Iliamna subregion, salmon, caribou, and moose made up from 75 to 90 percent of the pounds of food harvested for

domestic use (Wright et al. 1985; Behnke 1982; Gasbarro and Utermohle 1974; Wolfe et al. 1984).

The potential for mining copper-sulfide ores in an area near Iliamna may bring many new residents to the area, change access for recreational fishing, and create changing environmental and socioeconomic conditions that might affect subsistence harvests (Krieg et al. 2005).[6]

Given the many changing environmental conditions observed in the Kvichak River watershed, including those noted by local residents and failures of returns of sockeye salmon to that drainage, detailed and long-term perspectives are necessary for a more complete understanding of trends in fish populations and ecology (Krieg et al. 2005).

Consuming fish or other animals that have accumulated cadmium may pose a threat to human health. In one study, cadmium concentrations in catfish muscle tissue increased with increasing concentrations in their food (and significantly reduced fish growth) (Ruangsomboon and Wongrat, 2006). The study further demonstrated that a low level of cadmium in water may not indicate that fish living in those waters is safe for human consumption. The potential for mining copper-sulfide ores in the Bristol Bay, such as near Iliamna, could increase the risk of accumulation of cadmium in people who eat the contaminated fish.

Summary

The history of mining demonstrates that most if not all mines cause pollution - whether offsite or onsite (*see e.g*. Kuipers and Maest 2006). Cadmium's toxicity to freshwater fish can be altered by water chemistry variables, such as alkalinity, hardness, and natural organic matter.

Cadmium could pose a large threat to fish in the Bristol Bay area, where impacts are either unknown or reasonably likely because of Bristol Bay's geology and the presence of cadmium in rock that could be mined. Prior to permitting, mine proponents and regulators should evaluate and clearly convey to the public the potential for acute and chronic impacts to aquatic life from cadmium exposure. These evaluations should continue after mining commences.

References

Agency for Toxic Substances and Disease Registry (ATSDR), 2008. ATSDR Toxicological Profile for Cadmium. Draft for Public Comment; Public Comment Period Ends on February 26, 2009; Accessed November 19, 2010.
http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=48&tid=15.

[6] In Wright et al's 1985 study, it is interesting to note that "One of the greatest concerns was for the future of subsistence uses in the face of potential development and growth within the region. Oil and gas developments, and also mining and hydropower proposals, were a primary concern because of their potential for affecting the extremely valuable fish resources of Bristol Bay …. In general, when considering the future of the Bristol Bay region the primary issue was the continued availability of fish and wildlife resources which form the basis of the regional subsistence and commercial economies. Salmon, in particular, was recognized by most residents of Bristol Bay as their most important resource" (Wright et al. 1985).

Agency for Toxic Substances and Disease Registry (ATSDR), 2008. ATSDR Medical Fact Sheet; Division of Toxicology and Environmental Medicine ToxFAQs; Accessed November 19, 2010. http://www.atsdr.cdc.gov/tfacts5.pdf.

Agency for Toxic Substances and Disease Registry (ATSDR), ____. ATSDR Fact Sheet; Case Studies in Environmental Medicine (CSEM) Cadmium Toxicity What Is the Biological Fate of Cadmium in the Body? Accessed November 19, 2010. http://www.atsdr.cdc.gov/csem/cadmium/cdbiologic_fate.html.

Alaska, 2003. Alaska Water Quality Criteria Manual for Toxic and Other Deleterious Organic and Inorganic Substances, May 15, 2003. Accessed November 19, 2010. http://www.epa.gov/waterscience/standards/wqslibrary/ak/ak_10_toxics_manual.pdf

Bandara JM, Senevirathna DM, Dasanayake DM, Herath V, Bandara JM, Abeysekara T, and Rajapaksha KH , 2008. Chronic renal failure among farm families in cascade irrigation systems in Sri Lanka associated with elevated dietary cadmium levels in rice and freshwater fish (Tilapia). Environ Geochem Health. 2008 Oct;30(5):465-78. Epub 2008 Jan 17.

Borgmann, U., Couillard, Y., Doyle, P., and Dixon, D.G., 2005, Toxicity of sixty-three metals and metalloids to *Hyalella azteca* at two levels of water hardness: Environmental Toxicology and Chemistry, v. 24, no. 3, p. 641–652.

Brown V, Shurben D, Miller W, and Crane M, 2004. Cadmium toxicity to rainbow trout Oncorhynchus mykiss Walbaum and brown trout Salmo trutta L. over extended exposure periods. Ecotoxicol Environ Saf, 29:38-46.

M.J. Chowdhury, D.G. McDonald, and C.M. Wood, 2004. Gastrointestinal uptake and fate of cadmium in rainbow trout acclimated to sublethal dietary cadmium. Aquatic Toxicology 69, 149–163.

Clark, R.B., 1992. The effects of metal contamination in aquatic systems are of particular concern due to their persistence and toxicity. Unlike organic contaminants whose toxicity decreases with biodegradation, metals cannot be degraded further and their toxic effects can be long lasting from Marine Pollution, Oxford, U.K: Clarendon Press, pp. 61- 79.

Reinhard Dallinger, Margit Egg, Giinther K&k, and Rudolf Hofer, 1997. The role of metallothionein in cadmium accumulation of Arctic char (*Salvelinus alpinus*) from high alpine lakes. Aquatic Toxicology 38, 47-66.

Das, S. and B. B. Jana, 2004. Distribution pattern of ambient cadmium in wetland ponds distributed along an industrial complex. Chemosphere 55(2): 175-185.

Robert Edmonds and Dan Peplow, 2000. Environmental Impacts of Hardrock Mining in Eastern Washington, Fact Sheet #8. University of Washington College of Forest Resources and College of Ocean and Fishery Sciences Center for Streamside Studies November 2000.

Eisler, R. 1985. Cadmium Hazards To Fish, Wildlife , And Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service Biological Report 85 (1.2), Contaminant Hazard Reviews Report No. 2. 46 pp.

Jia X, Zhang H, and Liu X, 2010. Low levels of cadmium exposure induce DNA damage and oxidative stress in the liver of Oujiang colored common carp Cyprinus carpio var. color. Fish Physiol Biochem. 2010 Jul 25.

James A. Hansen, Paul G. Welsh, Josh Lipton, Dave Cacela, And Anne D. Dailey, 2002. Relative Sensitivity Of Bull Trout (*Salvelinus Confluentus*) And Rainbow Trout (*Oncorhynchus Mykiss*) To Acute Exposures Of Cadmium And Zinc. Environmental Toxicology and Chemistry, Vol. 21, No. 1, 67–75.

Krieg, T., M. Chythlook, P. Coiley-Kenner, D. Holen, K. Kamletz, and H. Nicholson. 2005. Subsistence Fisheries Assessment: Kvichak River Watershed Resident Species. Federal Subsistence Fishery Monitoring Program, Final Project Report No. FIS 02-034. U. S. Fish and Wildlife Service, Office of Subsistence Management, Fisheries Resource Monitoring Program, Fishery Information Service, Anchorage, Alaska.

Christopher A. Mebane, Daniel P. Hennessy, and Frank S. Dillon, 2009. Incubating Rainbow Trout in Soft Water Increased Their Later Sensitivity to Cadmium and Zinc. Water, Air, & Soil Pollution Volume 205, Numbers 1-4, 245-250.

Christopher A. Mebane, Daniel P. Hennessy, and Frank S. Dillon, 2008. Developing Acute-to-chronic Toxicity Ratios for Lead, Cadmium, and Zinc using Rainbow Trout, a Mayfly, and a Midge. Water Air Soil Pollution, 188:41–66.

Christopher A. Mebane, 2006. Derivation and Validation of Low-Effect Criteria Values using Laboratory and Field Studies. Scientific Investigations Report 2006–5245
Version 1.1, December 2006, U.S. Department of the Interior, U.S. Geological Survey.

Som Niyogi, Rebecca Kent, and Chris M. Wood, 2008. Effects of water chemistry variables on gill binding and acute toxicity of cadmium in rainbow trout (*Oncorhynchus mykiss*): A biotic ligand model. Comparative Biochemistry and Physiology, Part C 148 (2008) 305–314.

Olsson IM, Eriksson J, Oborn I, Skerfving S, and Oskarsson A., 2005. Cadmium in food production systems: a health risk for sensitive population groups. Ambio. 2005 Jun;34(4-5):344-51.

Daniel Peplow and Robert Edmonds,' 2004. Health Risks Associated with Contamination of Groundwater by Abandoned Mines Near Twisp in Okanogan County, Washington, USA. Environmental Geochemistry and Health, Volume 26, Number 1.

Rebagliati, CM, J Lang, E Titley, D Rennie, L Melis, D Barratt, and S Hodgson. 2009. Technical Report on the 2008 Program and Update on Metallurgy and Resources on the Pebble Copper-

Gold-Molybdenum Project, Iliamna Lake area, Southwestern Alaska, USA. for Northern Dynasty Minerals, Ltd.

Rebagliati, CM and JG Payne. 2005. Summary Report on the Pebble Porphyry Gold-Copper Project. for Northern Dynasty Minerals, Ltd. March 31.

Ruangsomboon S and Wongrat L., 2006. Bioaccumulation of cadmium in an experimental aquatic food chain involving phytoplankton (Chlorella vulgaris), zooplankton (Moina macrocopa), and the predatory catfish Clarias macrocephalus x C. gariepinus. Aquat Toxicol. 2006 Jun 10;78(1):15-20.

Michael K. Saiki, Barbara A. Martin1, Larry D. Thompson And Daniel Welsh, 2000. Copper, Cadmium, And Zinc Concentrations In Juvenile Chinook Salmon And Selected Fish-Forage Organisms (Aquatic Insects) In The Upper Sacramento River, California. Water, Air, & Soil Pollution Volume 132, Numbers 1-2, 127-139.

Laurel A. Schaider, David B. Senn, Daniel J. Brabander, Kathleen D. McCarthy,and James P. Shine, 2007. Characterization of Zinc, Lead, and Cadmium in Mine Waste: Implications for Transport, Exposure, and Bioavailability. *Environ. Sci. Technol.*, 41 (11), pp 4164–4171.

Sunila, I., 1986. Chronic histopathological effects of short-term copper and cadmium exposure on the gill of the mussel, Mytilus edulis. Journal of Invertebrate Pathology 47(2): 125-142.

P. Szczerbik, T. Mikoajczyk, M. Sokoowska-Mikoajczyk, M. Socha, J. Chyb, and P. Epler, 2006. Influence of long-term exposure to dietary cadmium on growth, maturation and reproduction of goldfish (subspecies: Prussian carp *Carassius auratus gibelio B.*). Aquatic Toxicology 77, 126–135.

Cheryl Szebedinszky, D. Gordon McDonald, Chris M. Wood, 2000. 243 Effects of chronic sublethal exposure to waterborne Cu, Cd or Zn in rainbow trout. Aquatic Toxicology 50, 231.

US Environmental Protection Agency (EPA), 2000. Cadmium Compounds Hazard Summary-Created in April 1992; Revised in January 2000. Accessed November 19, 2010. http://www.epa.gov/ttn/atw/hlthef/cadmium.html.

U.S. Fish and Wildlife Service (USFWS), 2007. Brochure: Salmon of the Pacific Coast. Available at: http://www.fws.gov/dpps/visualmedia/printingandpublishing/pdf /pubpricelistnov09.pdf; Accessed November 19, 2010.
http://www.krisweb.com/aqualife/lifecycle_early.htm.
Angelique Vetillard and Thierry Bailhache, 2004. Cadmium: An Endocrine Disrupter That Affects Gene Expression in the Liver and Brain of Juvenile Rainbow Trout. Biology of Reproduction 72, 119–126.

John M. Wright, Judith M. Morris, and Robert Schroeder, 1985. Bristol Bay Regional Subsistence Profile Technical Paper No. 114, Alaska Department of Fish and Game Division of Subsistence, Dillingham, Alaska.

Paul L. Younger, Steven A. Banwart and Robert S. Hedin, 2002. Mine Water Hydrology, Pollution, Remediation. Vol. 5 in the Environmental Pollution Series by Kluwer Academic Publishers, 442 pp.

Yana Yudkovski, Adelina Rogowska-Wrzesinska, Irena Yankelevich, Edna Shefer, Barak Herut, and Moshe Toma, 2008. Quantitative Immunochemical Evaluation Of Fish Metallothionein Upon Exposure To Cadmium. Marine Environmental Research 65, 427–436.