A Literature Review of
Effects of Ammonia on Fish

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Introduction

The purpose of this paper is to summarize information from the scientific literature regarding the potential effects of ammonia on the environment, in particular on aquatic life near Bristol Bay, Alaska. Proposed mining of copper-sulfide ores near Bristol Bay, Alaska, could introduce ammonia to the environment.[1]

Ammonia in the Environment

- Blasting compounds used by mines usually result in ammonia contamination in the environment.

- When ammonia enters the body via breathing, ingestion, or skin contact, it can react with water to produce ammonium hydroxide, a highly corrosive contaminant that damages cells on contact.

- Ammonia toxicity in fish is based on numerous primary factors, most notably pH and temperature.

Current ammonia levels in the Bristol Bay area are generally below fish toxicity levels, but some areas have elevated ammonia levels that appear to be related to natural organic (humic) content (Zamzow 2009).[2] Mine proponents and regulators need to clearly understand natural background ammonia levels and ensure that mining activities do not increase ammonia concentrations to levels that could adversely impact fisheries.

This factsheet provides a description of ammonia's behavior in surface waters, the potential sources of ammonia that are relevant to proposed mining in Bristol Bay, Alaska, and the toxicity of ammonia to native fish.

Ammonia in the Environment

In natural surface waters, ammonia occurs in two forms: ionized ammonia, NH_4^+, and un-ionized ammonia, NH_3^0 (Francis-Floyd 2009).

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[1] As discussed below, ammonia can change chemical form/species which changes its impacts and behaviors, such as toxicity, availability, etc. On the surface ammonia may changes form, such as to nitrates or nitrites but is less likely to change forms in groundwater - and therefore ammonia itself is more likely to exist in groundwater (*see e.g.* Morin 2009; Forsyth 1995).

[2] The toxic form of ammonia (NH_3^0) never exceeded the EPA recommendation for fish propagation, but a shift in pH from 7.5 to 8 with an increase of $1^{\circ}C$ would cause some water to exceed ammonia criteria values (Zamzow 2009).
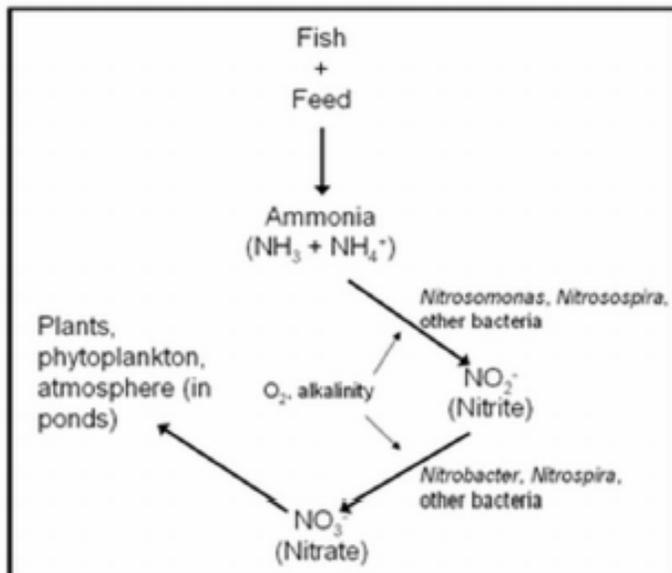
Ammonia can be produced naturally from the breakdown of organic matter and is excreted by fish as a nitrogenous waste product. In fish, ammonia is a byproduct of protein metabolism and is primarily excreted across the gill membranes, with a small amount excreted in the urine. As shown in Figure 1, ammonia produced by fish can be eliminated by bacterial conversion of ammonia to nitrite and nitrate. Nitrate can be used by plants and algae and is generally considered harmless to fish in natural waters (Francis-Floyd 2009).

Figure 1. Conversion of fish-generated ammonia in the environment (Francis-Floyd 2009).

Ammonia's aquatic toxicity is principally due to the un-ionized form, $NH_3{}^0$ (Arthur et al. 1987). As pH increases, the toxicity of ammonia increases because the relative proportion of unionized ammonia increases,[3] as shown in Figure 2 (Brinkman et al. 2009; Paley et al. 1993; EPA 1999).
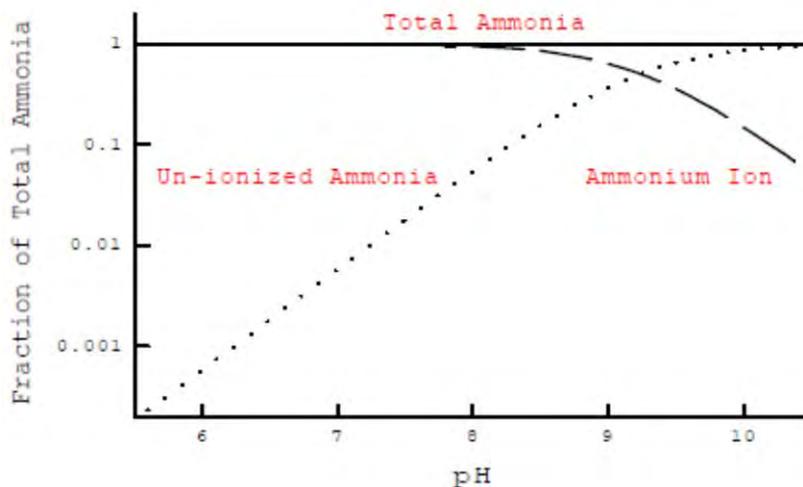

Figure 2. Chemical speciation of ammonia over a range of pH values (EPA, 1999).

Ammonia and Mining

The use of blasting agents in the excavation of underground or open-pit mines can cause a large increase in ammonia and nitrate concentrations in nearby groundwater and surface water. Ammonia is also contained to a lesser extent in drilling muds[4] (used in exploration drilling).

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[3] An increase in pH by 1 unit will increase unionized ammonia by a factor of ten (Paley et al. 1993).
[4] Halliburton EZ Mud Drilling by products MSDS; Accessed November 19, 2010.
http://www.muddirect.net/MSDS/PDFs/EZ-Mud.pdf

Ammonium nitrate and fuel oil (ANFO) is the most common blasting agent used at mines (Revey 1996, Forsyth et al. 1995). Commercial blasting agents are predominantly ammonium nitrate and contain <10 percent diesel fuel oil. Other additives are used but make up only a small percent of total weight or volume. ANFO is highly water- soluble and can contaminate surface water and groundwater either through leakage or dispersion of unexploded materials or explosion residues. (Zaitsev et al. 2008; Revey 1996, Forsyth et al. 1995).

At open-pit diamond mines in the Canadian Arctic, the largest potential source of ammonia in runoff water is explosives residues from blasting (AMEC 2004). Residual ammonia from ANFO can range from 0.1% (in dry rock) to 8 or 9% (in wet holes or misfires), and this residual ammonia is available for leaching (AMEC 2004). The primary sources of ammonia contamination at open-pit mines are waste rock dumps, open pits, and the walls of open pits (AMEC 2004).

At the Kensington Mine, an underground gold mine in Alaska, elevated nitrate and ammonia concentrations (above projected permit limits) coincided with the exploratory drilling period (USFS 2004). At that mine, the implementation of best management practices[5] for explosives reduced - but did not eliminate – the detection of ammonia (USFS 2004).

As an example of the quantities of explosives proposed to be used at a large mine, the NorthMet Project, an open-pit copper sulfide project in Minnesota, proposes to use over five million pounds of explosive materials during the life of the mine (NorthMet Project 2009). Even low leak and residual rates from such a large quantity of explosive material could raise ammonia concentrations for extended periods of time.

In addition to explosives, ammonia can derive from sewage at mine sites. The following section discusses the impacts of ammonia on fish.

Ammonia and Fish

Ammonia is a toxic compound that can adversely affect fish health. The nature and degree of toxicity depends on many factors, including the chemical form of ammonia, the pH and temperature of the water, the length of exposure, and the life stage of the exposed fish. Issues associated with toxicity testing and the consequences of ammonia exposure are also discussed.

Ammonia Speciation, Temperature, and pH

When dissolved in surface water, ammonia exists in two forms: NH_3^0 (unionized) and NH_4^+ (ionized). Ionized ammonia does not easily cross fish gills and is less bioavailable than the unionized form (Francis-Floyd 2009; EPA 1989). The unionized form (NH_3^0) can cross from water into fish, and once inside, some converts to the ionized form (NH_4^+), which then causes cellular damage (EPA 1989). The primary form of total body ammonia in fish at physiological pH (7.0–8.0) is ammonium, or NH_4^+; it is this chemical species that is responsible for toxic effects (NH_3^0 is the most toxic form to aquatic life, but NH_4^+ is the most toxic form *in the body*)

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[5] BMPs focused on the use of insoluble blasting agents and good housekeeping practices (USFS 2004).

(McKenzie et al. 2008; Smart 1976; Hillaby and Randall 1979). Although unionized ammonia is the more toxic form, toxicity is most commonly expressed as total ammonia - the sum of NH_3^0 and NH_4^+ in water.

Ammonia is more toxic to aquatic life at higher temperature and pH values. As pH increases, so does the fraction of unionized ammonia (see Figure 1) and the toxicity to fish (EPA 1999).[6] The ratio of NH_3^0 to NH_4^+ increases by 10 times for each one-unit rise in pH, and by approximately 2 times for each 10^0C rise in temperature from 0-30^0C (US EPA, 2009).

Ammonia toxicity is also a function of ionic strength, or the salinity of the water. Several studies have indicated that increasing the hardness of ambient water can in some cases decrease ammonia toxicity (Wicks et al. 2002; Soderberg and Meade, 1992). In general, seawater species are slightly more sensitive to ammonia toxicity than freshwater species (Randall et al. 2002).

EPA and State of Alaska Criteria and Issues with Testing

The US Environmental Protection Agency (EPA) is responsible for recommending methods for states to use when deriving water quality standards. EPA has recommended criteria for the protection of aquatic biota from ammonia in surface waters under the Clean Water Act. EPA protocols call for using measured pH and temperature values in surface water for calculating acute and chronic ammonia criteria values (EPA, 1999). Alaska's freshwater standard for ammonia is broken into three primary categories: Acute freshwater standards based on pH; chronic freshwater ammonia based on pH and temperature when early life stages of fish are present; and chronic freshwater ammonia based on pH and temperature when early life stages are absent (Alaska DEC 2003).

Acute toxicity studies for ammonia follow standard guidelines that include exposure of fish to ammonia under static conditions - using starved, resting, unstressed animals (Randall et al. 2002; Stephen et al. 1985). The standardization is important to facilitating comparisons between studies (Randall et al. 2002; Stephen et al. 1985). However, toxicity tests may underestimate the toxicity of ammonia in natural environments because they are conducted in artificial conditions that minimize stress and internal ammonia levels (Brinkman et al. 2009; Ip et al. 2001). Among other biological changes, stress in fish increases ammonia production - and ammonia toxicity (Randall et al. 2002). As a result, stressed fish are more sensitive to external ammonia than unstressed fish (although stressed fish may be more protected in the post-stress period) (Randall et al. 2002). In fish, internal ammonia levels increase after feeding, swimming, and stress - all conditions that are avoided when acute toxicity tests are carried out using EPA guidelines (Randall et al. 2002; Stephen et al. 1985). Fish have strategies to protect them from the internal ammonia pulse following feeding, and this also protects them from increases in external ammonia. Therefore, unfed fish may be more sensitive to external ammonia than fed fish (See

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[6] It makes sense that the unionized form would be more toxic: Unionized ammonia is a neutral molecule and can diffuse across cell membranes of aquatic organisms more easily than the ionized form (EPA 1999). As described below, ammonia is naturally produced by fish and their strategies to excrete the unionized form are largely dependent on passive diffusion across the gills - which would be much less efficient or impossible in waters with high external unionized ammonia (EPA 1999). In other words - water with higher unionized ammonia will reduce or reverse the diffusion gradient, causing fish to concentrate ammonia in their gills and blood (EPA 1999).

Randall et al. 2002). As a result, current EPA criteria are based on toxicity tests carried out on fish when the fish may be *least* sensitive to ammonia (Randall et al. 2002).

Further, the toxicity of ammonia may be affected by combinations of biotic factors (e.g., quiescent water in exposure tanks, feeding of test organisms, and other stressors) (Brinkman et al. 2009). Brinkman's study concluded that for the early life stage, the current EPA standards for ammonia are protective (Brinkman et al. 2009). But the results also indicate that more study is needed to determine the influences of other factors (such as quiescent exposure waters and combinations of other stressors) that may underestimate ammonia toxicity to fish in the wild (Brinkman et al. 2009).

Fish that are exercising and exposed to ammonia have decreased swimming performance and increased susceptibility to acute ammonia exposure (Wicks et al. 2002, *referring to* Beaumont et al. 1995; and Day and Butler 1996). It has been shown that swimming performance is reduced in Coho salmon exposed to ammonia levels higher than 0.04 mg per l NH_3[07] (Wicks et al. 2002). Salmonids swimming at 60% Ucrit[8] exposed to the ammonia levels promulgated by the EPA may not only reduce swimming performance, but could be lethal (Wicks et al. 2002). EPA's recommended standards may not protect swimming fish and may endanger annual migrations of anadromous fishes (Wicks et al. 2002).

Length of Exposure

The concentrations of ammonia that cause toxicity to fish depend on the length of exposure. Lower concentrations may not kill or adversely affect fish over short periods of time, but the same concentrations could kill or impair aquatic life under longer time frames. US EPA recommends an acute ammonia criterion of 2.9 or 5.0 mg N/L (for short-term exposure) and a chronic criterion of 0.26 or 1.8 mg N/L (for long-term exposure). Water with concentrations of less than 0.020 mg/L unionized ammonia is considered safe for fish reproduction (EPA 1989). As shown in Figure 3, lower and lower concentrations of ammonia are tolerated with increasing pH values under acute (short-term) exposure scenarios. The same is true under longer term exposures (Figure 4), and lower concentrations are tolerated under higher temperatures.

A single or simple concentration measurement of ammonia is not sufficient to evaluate and/or regulate ammonia contamination. The influence on the toxicity of ammonia to fish is impacted by both duration and frequency of exposure. Duration of exposure to ammonia has been demonstrated to be a critical factor affecting fish survival - and should therefore be incorporated into chronic and acute standards. The demonstrated influence of exposure frequency indicates the need to consider the return period of transient pollution events in deriving appropriate standards because the return period will fundamentally influence the degree of recovery following exposure and, consequently, the long-term viability of fish populations (Milne et al. 2000).

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[7] It has been shown that increased calcium can protect free-swimming rainbow trout fry exposed to elevated ammonia at pH of both 7.8 and 9.0 (Wicks et.al. 2002).
[8] This is about 2.2 body lengths per second - a speed often exceeded by migrating sockeye salmon whose swim speeds through river constrictions can range from 5.8 to 11.7 body lengths per second (Hinch and Bratty, 2000).

Acute toxicity of ammonia to rainbow trout has been extensively studied, and the results are largely consistent, indicating that ammonia can kill fish when levels exceed US EPA standards (Brinkman et al. 2009; EPA 1999; EPA 1989). In contrast, chronic toxicity data for rainbow trout are limited and demonstrate a wide range of toxicity thresholds (Brinkman et al. 2009). Many chronic toxicity studies have been based on relatively short exposure periods (less than 96 hours) (Brinkman et al. 2009). Therefore, conclusions about chronic toxicity should be made with caution (Brinkman et al. 2009).
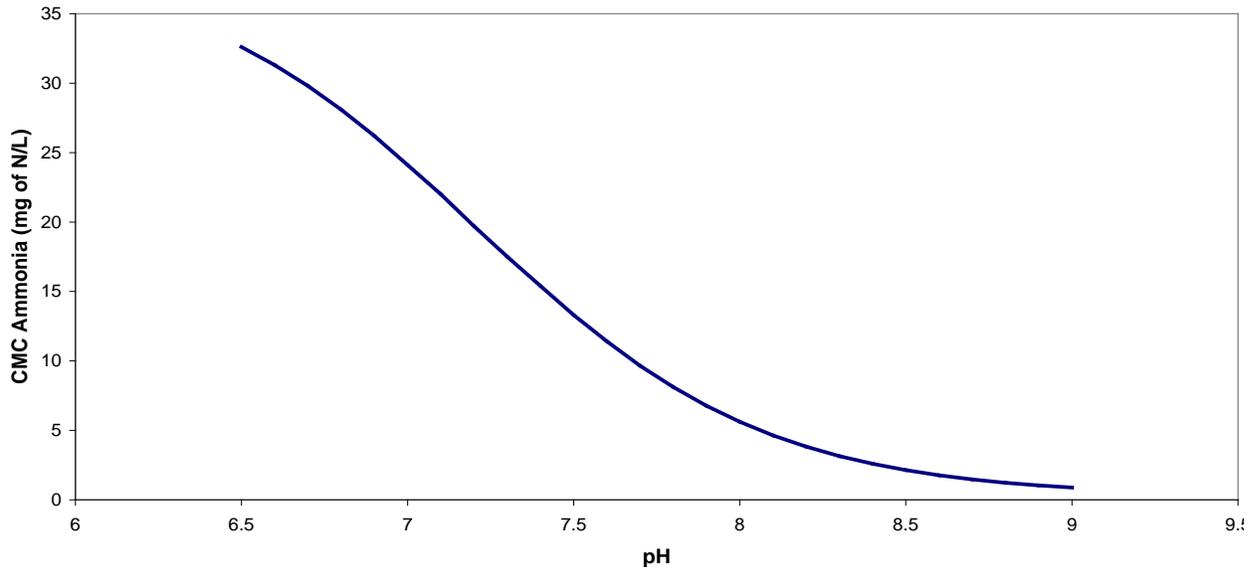

Figure 3. Acute ambient water quality criteria (criterion maximum concentration)[9] for ammonia (NH_3^0) toxicity, with salmonids present, across a range of pH values (EPA 1999).
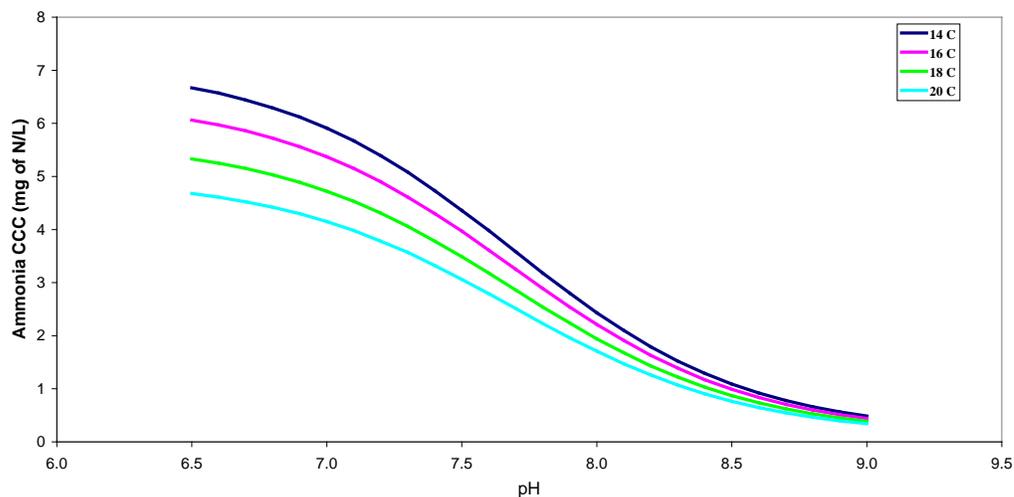

Figure 4. Chronic ambient water quality criteria (criterion continuous concentration)[10] for ammonia toxicity, with fish early life stages present, across a range of pH and temperature values (EPA 1999).

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[9] The Criteria Maximum Concentration (CMC) is an estimate of the highest concentration of a material in surface water to which an aquatic community can be exposed briefly without resulting in an unacceptable effect.

Toxicity and Life Stage

Acute toxicity susceptibility to ammonia varies by life stage of the fish. Susceptibility to ammonia decreases as fish develop from sac fry[11] to juveniles and increases with age of the fish after the juvenile stage. In rainbow trout, acute susceptibility decreases as the fish developed from sac fry to juveniles and increases thereafter (Thurston et al. 1983). Acute toxicity decreases in rainbow trout as temperature increases (over the range 12 to 19^{0}C), and no significant differences in toxicity were observed in tests with different ammonium salts (Thurston et al. 1983). Further, toxicity may increase for repeated exposures to ammonia, such that a second ammonia exposure during a fish's recovery period (from a prior exposure to ammonia, when it is unloading excess ammonia from the body) could be "extremely serious" for sac fry (alevins) (Paley et al. 1993). Rainbow trout embryos and sac fry are more resistant to the lethal effects of ammonia than are swim-up fry,[12] but development of sac fry to the swim-up stage is delayed by exposure to ammonia (Brinkman et al. 2009).

Consequences of Ammonia Exposure

Fish are exposed when ammonia enters a fish across its gill epithelium - in freshwater this may be almost exclusively via passive diffusion of ammonia as a gas in solution (McKenzie et al. 2008; Randall and Wright 1987; Ip et al. 2001; Randall and Tsui 2002; and Eddy 2005). Ammonia is also produced as an end product of metabolism and then excreted across the gill epithelium (McKenzie et al. 2008; Randall and Wright 1987; Wright 1995; Randall and Tsui 2002). Ammonia can accumulate in fish as a consequence of exposure to elevated water ammonia concentrations or when excretion of the endogenous metabolite is inhibited[13] (McKenzie et al. 2008).

Ammonia can be acutely toxic to fish mainly due to its effect on the central nervous system, because it causes "acute ammonia intoxication" - which includes convulsions and death (Randall et al. 2002). Concentrations of ammonia that are acutely toxic to fish may cause loss of equilibrium, hyperexcitability, increased breathing, cardiac output, and oxygen uptake, and, in extreme cases, convulsions, coma, and death (EPA 1989). Lower concentrations of ammonia can cause a reduction in hatching success, reduction in growth rate and morphological development, and pathologic changes in tissues of gills, livers, and kidneys (EPA 1989).

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[10] The Criterion Continuous Concentration (CCC) is an estimate of the highest concentration of a material in surface water to which an aquatic community can be exposed indefinitely without resulting in an unacceptable effect (EPA 2009).

[11] Newly hatched fish are called fry, or sometimes sac fry or alevins because they continue to feed on the yolk sac attached to their bellies (USFWS 2007).

[12] Hatching success and sac fry survival were unaffected by exposure to 16.8 mg NH_3^0, the highest concentration of ammonia used in those tests, and ammonia-related mortality did not occur until shortly after absorption of the yolk sac, which the authors concluded suggested that the swim-up fry stage is more sensitive than the embryonic and sac fry stages (Brinkman et al. 2009). Shortly after swim-up, fry are about 20 times more sensitive to ammonia than are eggs and sac fry (Brinkman et al. 2009).

[13] In freshwater, excretion occurs mostly by passive diffusion of NH_3^0 (Mckenzie et al. 2008), so ammonia contamination can harm fish by direct contamination as well as by limiting the ability to naturally diffuse ammonia.

Most fish species cannot tolerate high total ammonia concentrations (Randall et al. 2002). In fish, elevated ammonia levels in the environment either impair ammonia excretion or cause a net uptake of ammonia from the environment - with the end result that ammonia becomes elevated in the body and ultimately can lead to convulsions and death (Randall et al. 2002).[14]

Escape responses is a reflex that comprises a sudden and brief acceleration, typically in a direction away from the startling stimulus, such as a predator, and are used by fish as a defense mechanism (McKenzie et al. 2008; Domenici and Blake 1993). The success of an escape response for predator avoidance depends on both sensory and locomotory performance. (McKenzie et al. 2008). Many environmental factors influence the escape response, such as temperature, dissolved oxygen, and turbidity (McKenzie et al. 2008).

In response to sublethal concentrations of ammonia, some fish have been shown to suffer impaired performance of the startle-escape response and accumulations of ammonia in the water and in mullet tissues impaired various sensory and motor components of the response, often in a manner that appeared to be proportional to the ammonia concentrations. (McKenzie et al. 2008). In that study, the only two performance variables that were not affected by ammonia were responsiveness (an indicator of acoustic or visual sensitivity and motivation to escape) and directionality (an indicator of overall sensory performance) (McKenzie et al. 2008).[15]

Ammonia can act on a fish's central nervous system and cause effects such as hyperventilation, hyperexcitability, and loss of equilibrium (and, as described elsewhere, convulsions, coma, and death) (McKenzie et al. 2008; Olson and Fromm 1971; Hillaby and Randall 1979; Arillo et al. 1981; McKenzie et al. 1993). In rainbow trout (*Oncorhynchus mykiss*), sublethal concentrations of ammonia can cause hyperexcitability, resulting in fish crashing into the sides of the tank in response to any disturbance (McKenzie et al. 2008; Olson and Fromm 1971). Sublethal accumulations of ammonia in plasma (hyperammonemia) can impair the ability of salmonids to perform exercise in an incremental "critical swimming speed" protocol -- which can mean that fish are unable to escape predation or other threats because they can't recruit white muscle to power anaerobic burst swimming at relevant speeds (McKenzie et al. 2008; Beaumont et al. 1995; Shingles et al. 2001; Wicks et al. 2002).

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[14] Rainbow trout may be able to tolerate a slightly higher background ammonia level because they can excrete part of an ammonia load against high ambient ammonia levels in both freshwater and saltwater (Randall et al. 2002; Wilson and Taylor 1992; Randall et al. 1999).

[15] The study noted that the concentration of ammonia used was at various maximum concentration levels permitted by the EPA for waters without salmonids. It then suggested that higher numbers exist in the environment in spite of EPA regulations. (McKenzie et al. 2008). Further, the EPA regulations for waters containing salmonids are lower than the EPA regulations for waters containing non-salmonids. The study is presented here to underscore the need to protect fish from ammonia and to identify the need to examine the specific impacts and thresholds on salmonids such as those found in Alaska and in the Bristol Bay region. The study also noted that tissue loads, which will define toxic effects in fish, have been observed in salmonids following exposure to much lower ambient ammonia concentrations and that the venous ammonia concentrations measured in the mullet in this study were similar to those associated with impaired Ucrit exercise performance in salmonids in freshwater (McKenzie et al. 2008; Wicks and Randall 2002; Wicks et al. 2002; McKenzie et al. 2003). Further, the study authors noted that it has also been established that sublethal concentrations of other pollutants, such as water pH and heavy metals, can cause an accumulation of ammonia in the plasma of fish to levels that are similar to those measured in the mullet (McKenzie et al. 2008; Ye and Randall 1991; Butler et al. 1992; Beaumont et al. 1995a, 1995b; Day and Butler 1996).

A critical problem with sublethal ammonia toxicity is that increased latency of the response and reduced kinematic performance would presumably put the fish at greater risk of predation from predators such as birds and mammals that would not accumulate or be similarly impaired by ammonia exposure (McKenzie et al. 2008).

Avoiding Risks to Fish from Mining-Related Ammonia Contamination in Bristol Bay

The history of mining demonstrates that most if not all mines cause pollution - whether offsite or onsite (*see e.g*., Kuipers and Maest, 2006). Ammonia's behavior in the environment and toxicity can be largely site-specific based on major factors such as pH and temperature and biotic factors. Any mining-related regulatory standards for ammonia in the Bristol Bay ecosystem should be based on the worst-case scenario for temperature. Ammonia should be a regulated contaminant[16] in any mining permit, particularly an area as sensitive and important (biologically, culturally, economically, etc.) as Bristol Bay. To be effective and representative, the permit must require simultaneous measurement of both temperature and pH.

Predicting ammonia and nitrogen species contamination may not be reliable and therefore it may be that only after mining is underway that contamination is identified and quantified (Morin 2009). Therefore, it will be important for any mining activities in the Bristol Bay area to be preceded by a clear understanding of baseline/background ammonia concentrations and the causes of measured elevated (relative to other Alaskan waters) concentrations of ammonia in the vicinity of the lease area.

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 ammonia exposure. These evaluations should continue after mining commences.

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[16] There are some arguments that it is a secondary contaminant as compared to primary contaminants. However, because of the high amount of ammonia used in mining and frequency of ammonia directly resulting from blasting materials (and cyanide where that is degrading) it should be considered - and regulated as - a primary contaminant.

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