

Key Notes: Session 3

[KN 3.1]

Effects of Exposure – Water Chemistry on Acute Metal Toxicity to Fish

Russell J. Erickson

Mid-Continent Ecology Division, National Health and Environmental Effects Laboratory, U.S. Environmental Protection Agency, 6201 Congdon Boulevard, Duluth, MN, USA, 55804; e-mail: erickson.russell@epa.gov

The toxicities of cationic metals to fish vary widely as a function of various physicochemical characteristics of exposure water, including pH, temperature, suspended solids, dissolved organic matter, and various inorganic cations and anions. For example, acute copper toxicity to fathead minnows varies by more than two orders of magnitude over a range of chemistry typical of natural freshwaters. Effective regulation of metal pollution requires that such effects be understood and predicted. United States water quality standards for metals are often an empirical function of water hardness, but such adjustments are an incomplete description of the total effects of exposure water chemistry.

Past research has provided a basis for more comprehensive and mechanistic models for the effects of water chemistry on metal toxicity. Research with marine invertebrates and phytoplankton on the effects of organic ligands generally has demonstrated a close correlation of metal toxicity with the chemical activity of 'free' metal (the aquo-complex of the metal cation). In these studies, metal complexes with the organic ligands apparently contribute little or nothing to toxicity. Some research with plankton has also suggested antagonism between metal toxicity and hydrogen ion. Research with fish likewise suggests considerable importance of the free metal, but also importance of other factors. Most notably, increasing water hardness reduces the toxicity of many metals, and complexes of metal with certain small ligands appear to contribute somewhat to toxicity.

These observations suggest certain attributes of the interaction of metals with organisms (passage through membranes; reactions with macromolecules). First, only free metal (or small metal complexes) will penetrate membranes and/or react with macromolecules. Second, other cations compete or interfere in some manner with the uptake of the metal and/or its reactions. Simple models incorporating these attributes assume that a certain level of toxicity requires a specific amount of metal accumulation, that metal uptake depends in a first-order manner on free metal concentration in exposure water, and that other cations compete in a first-order manner with metal uptake. This would be true, for

example, if the rate-limiting step in metal uptake involves the metal forming a complex with a macromolecule on the surface of an organism, with other cations also forming complexes with this macromolecule and thus reducing uptake. Such a model has been called the Gill Surface Interaction Model, the Free Ion Activity Model, and the Biotic Ligand Model by different authors.

Although this model is extremely simplified, it is generally consistent with many reported effects of water chemistry on metal toxicity. Moreover, some model assumptions regarding metal uptake and its relationship to toxicity have been directly demonstrated. Complexation of metal by various ligands does result in less accumulation onto fish gills, consistent with these complexes having little or no bioavailability. Increased water hardness does reduce metal accumulation, consistent with assumptions regarding competition. Over a range of water chemistries, the level of metal accumulation in gills associated with toxicity is much more constant than the water concentrations associated with this toxicity. This suggests that the effect of water chemistry on metal toxicity does involve complexation/competition reactions which regulate metal uptake.

However, this model is still poorly validated and needs further development. Direct demonstration of model assumptions and relationships is limited and uncertain. Some effects of water chemistry on metal toxicity are consistent with alternate explanations. Some important factors are not addressed in the model, most notably the issue of chemical microenvironments near biological membranes. Detailed analysis of a large dataset for acute copper toxicity to fathead minnows demonstrates that, while general relationships predicted by the model are approximately followed, there are definite deviations and shortcomings which create uncertainties in model applications. In particular, the consequences of reduced pH at gill surfaces and alternate mechanisms for calcium and sodium effects need to be better considered. Data for silver toxicity show the potential importance of species other than free metal in metal uptake and toxicity.