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Chronic toxicity of arsenic, cobalt, chromium and manganese to *Hyalella azteca* in relation to exposure and bioaccumulation

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Chronic toxicity of As, Co, Cr and Mn to Hyalella azteca can be described using a saturation-based mortality model in relationship to total-body or water metal concentration.

Abstract

Chronic toxicity of As, Co, Cr and Mn to *Hyalella azteca* can be described using a saturation-based mortality model relative to total-body or water metal concentration. LBC25s (total-body metal concentrations resulting in 25% mortality in 4 weeks) were 125, 103, 152 and 57,900 nmol g^{-1} dry weight for As, Co, Cr and Mn respectively. LC50s (metal concentrations in water resulting in 25% mortality in 4 weeks) were 5600, 183, 731, and 197,000 nmol L^{-1}, respectively. A hormesis growth response to As exposure was observed. Growth was a more variable endpoint than mortality for all four toxicants; however, confidence limits based on growth and mortality all overlapped, except Cr which had no effect on growth. Mn toxicity was greater in glass test containers compared to plastic. Bioaccumulation of As, Co, Cr, and Mn was strongly correlated with, and is useful for predicting, chronic mortality.

Keywords: *Hyalella*; Saturation mortality model; Toxicity; Bioaccumulation; Growth; Arsenic; Cobalt; Chromium; Manganese; Hormesis; Bioconcentration factors

1. Introduction

Since toxicity is based on the effect that a toxicant produces at a target site within an organism, establishing the relationship between the concentration of the substance at the target site and the subsequent toxic effect can provide a tool for predicting toxicity (Landrum et al., 1992). This is the primary toxicological principle generally referred to as ''dose–response'' or ''concentration–response'' in which the response of an organism is proportional to the dose or concentration of the substance at the target site (Connolly, 1985; McCarty, 1991). In many cases the target site is unknown, or measurement of the substance at the site is not possible. Instead, surrogate measures of the target site concentration have been used. A number of researchers have determined that the concentration of a substance in the organism (expressed as body concentration, critical internal concentration, tissue residue, tissue concentration or body burden) was a better predictor of effect than water concentration, sediment concentration, or equilibrium partitioning (Niimi and Kissoon, 1994; Connell, 1995; Driscoll and Landrum, 1997). The use of metal and metalloid body concentrations as a measure of bioavailability may negate complications that can arise from uncertainties such as, interactions with other ions or molecules that may hinder or enhance bioaccumulation, multiple compartments of exposure, multiple sources and pulsed exposures (Landrum et al., 1992; Hickie et al., 1995). Body concentrations of single elements have been

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shown to be useful indicators of toxic effects in aquatic invertebrates, even in the presence of various complexing agents (Borgmann et al., 1991) and can help identify the cause of biological effects in sediment assessments (Borgmann and Norwood, 1997, 1999; Borgmann et al., 2001a).

This research was undertaken to determine the toxicity of As, Co, Cr and Mn to the freshwater amphipod, *Hyalella azteca*. These elements are commonly found at metal contaminated sites and they are accumulated by *Hyalella.* However, unlike several other metals, their relative contribution to toxicity could not be assessed in previous sediment assessment studies because the relationship between bioaccumulation and toxicity was not known (Borgmann et al., 2001a). Norwood et al. (2006) recently demonstrated that the metalloid As and the metals Co, Cr and Mn demonstrate a clear relationship between exposure concentration and bioaccumulation. The present paper examines the relationship between chronic toxicity (mortality and growth effects) and the exposure and the bioaccumulation data from Norwood et al. (2006).

2. Theory

2.1. Metal toxicity

The simplest metal-toxicity paradigm is the allometric model. It has been used to describe the relationship between mortality rate and metal concentration, in water or tissue (Borgmann and Norwood, 1995; Borgmann et al., 2004) in which overall mortality rate m, is expressed as:

$$m = m' + aC^n \tag{1}$$

where m' is the control mortality rate, C is the water or tissue metal concentration and a and n are constants. If applied to both water and tissue concentrations, this model can only be mathematically correct if the toxicant bioaccumulation also follows an allometric relationship. The model cannot be mathematically correct when applied to both water and body concentrations if the relationship between water and body concentrations follows a saturation curve. However, saturation curves are mechanistically based and are often more useful than allometric models for describing metal bioaccumulation (Borgmann et al., 2004). A more appropriate mortality saturation model has been described (Borgmann et al., 2004) in which the allometric relationship $a^{(1/n)}C$ in eq. (1) is replaced with the saturation relationship; $\max''C(K'' + C)^{-1}$ such that

$$m = m' + \left[\max''_{\mathrm{W}}\, C_{\mathrm{W}}\left(K''_{\mathrm{W}} + C_{\mathrm{W}}\right)^{-1}\right]^{nw} \tag{2a}$$

and

$$m = m' + \left[\max''_{\mathrm{TB}X}\, C_{\mathrm{TB}X}\left(K''_{\mathrm{TB}X} + C_{\mathrm{TB}X}\right)^{-1}\right]^{nb} \tag{2b}$$

where $\max''_{\mathrm{W}}$ and $\max''_{\mathrm{TB}X}$ are the water and body concentrations when metal-induced mortality has reached a maximum, K''_W and $K''_{\mathrm{TB}X}$ are the concentrations when metal-induced mortality is half of the maximum, C_{W} is the background or control concentration in water, and $C_{\mathrm{TB}X}$ is the background-corrected body concentration. The max″ terms in eqs. (2a) and (2b) can be replaced with LC50 (water concentration resulting in 50% mortality) or $\mathrm{LBC50}_X$ (background-corrected. body concentration resulting in 50% mortality), which are of greater toxicological interest, giving:

$$m = m' + (\ln(2)/t)\left[C_{\mathrm{W}}(\mathrm{LC50}^{-1} + K''^{-1}_{\mathrm{W}})\left(1 + C_{\mathrm{W}}K''^{-1}_{\mathrm{W}}\right)^{-1}\right]^{nw} \tag{3a}$$

and

$$m = m' + (\ln(2)/t)\left[C_{\mathrm{TB}X}\left(\mathrm{LBC50}_X^{-1} + K''^{-1}_{\mathrm{TB}X}\right) \times \left(1 + C_{\mathrm{TB}X}\,K''^{-1}_{\mathrm{TB}X}\right)^{-1}\right]^{nb} \tag{3b}$$

where t is the exposure time corresponding to the LC50 and $\mathrm{LBC50}_X$. These equations are consistent with the saturation uptake models for As, Co, Cr, and Mn (Norwood et al., 2006).

2.2. Growth effects

The impact of the metals and metalloid on growth, expressed as final body size W (final wet weight after 4 weeks) was evaluated with a general growth model

$$W = W'\,(1 + aC^n)^{-1} \tag{4}$$

where W' is the control wet weight, C is the water metal concentration or background-corrected tissue metal concentration and a and n are constants (Borgmann et al., 1998). Since bioaccumulation was expressed as a saturation model in relation to water concentration, growth should also be expressed as a saturation model in relation to water or body concentrations to be mathematically consistent. However, saturation models, analogous to eqs. (2a) and (2b) for mortality, could not be satisfactorily fit to the final body size data for any of the four toxicants based on either water or body concentration. Therefore, the relationships of growth to water or body concentration were expressed with allometric models only. Due to this inconsistency, the IC25s (metal concentrations in water resulting in a 25% reduction in final body size) cannot be directly converted to $\mathrm{IBC25}_X$s (total-body metal concentrations resulting in a 25% reduction in final body size) with the bioaccumulation model for each toxicant.

In some cases growth was stimulated at low toxicant concentrations (hormesis) and the exposure-response relationship could be described using

$$W = W'\,(1 + bC^m)(1 + aC^n)^{-1} \tag{5}$$

in which the term $(1 + bC^m)$ describes low-exposure concentration stimulation of growth and the $(1 + aC^n)^{-1}$ term overrides the low-exposure term at higher concentrations.