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Salinity-dependent mechanisms of copper toxicity in the galaxiid fish, *Galaxias maculatus*

Chris N. Glover[a,b,*], Mauricio A. Urbina[a,c], Rachel A. Harley[a], Jacqueline A. Lee[a]

[a] School of Biological Sciences, University of Canterbury, New Zealand
[b] Faculty of Science and Technology, Athabasca University, Alberta, Canada
[c] Departamento de Zoología, Universidad de Concepción, Chile

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ABSTRACT

The euryhaline galaxiid fish, inanga (*Galaxias maculatus*) is widely spread throughout the Southern hemisphere occupying near-coastal streams that may be elevated in trace elements such as copper (Cu). Despite this, nothing is known regarding their sensitivity to Cu contamination. The mechanisms of Cu toxicity in inanga, and the ameliorating role of salinity, were investigated by acclimating fish to freshwater (FW), 50% seawater (SW), or 100% SW and exposing them to a graded series of Cu concentrations (0–200 $\mu g\,L^{-1}$) for 48 h. Mortality, whole body Cu accumulation, measures of ionoregulatory disturbance (whole body ions, sodium (Na) influx, sodium/potassium ATPase activity) and ammonia excretion were monitored. Toxicity of Cu was greatest in FW, with mortality likely resulting from impaired Na influx. In both FW and 100% SW, ammonia excretion was significantly elevated, an effect opposite to that observed in previous studies, suggesting fundamental differences in the effect of Cu in this species relative to other studied fish. Salinity was protective against Cu toxicity, and physiology seemed to play a more important role than water chemistry in this protection. Inanga are sensitive to waterborne Cu through a conserved impairment of Na ion homeostasis, but some effects of Cu exposure in this species are distinct. Based on effect concentrations, current regulatory tools and limits are likely protective of this species in New Zealand waters.

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1. Introduction

Copper (Cu) is an aquatic contaminant of significant concern. Occurring at trace levels in natural waters, anthropogenic sources such as mining, industrial, municipal, and agricultural effluents can elevate concentrations in aquatic environments (Eisler, 1998). In uncontaminated waters Cu levels are in the range of 1–8 $\mu g\,L^{-1}$, with this level rising to more than 7000 $\mu g\,L^{-1}$ in heavily contaminated watersheds (Eisler, 1998). In New Zealand freshwaters, levels in excess of 100 $\mu g\,L^{-1}$ have been measured in streams associated with acid mine drainage (Pope et al., 2010).

In freshwater (FW) Cu is known to cause toxicity by the impairment of sodium (Na) homeostasis (Grosell et al., 2002), despite these two metals belonging to different classes with distinct ligand-binding preferences (Nieboer and Richardson, 1980). Freshwater animals are hyperosmotic regulators, maintaining osmolality, and body ions such as Na, at levels higher than those of the environment. As a consequence, they are faced with the continuous loss of Na to the environment, and to maintain homeostasis Na must be actively taken up from their surroundings. In fish this is principally achieved by the gill. The sodium pump (sodium/potassium ATPase; NKA), translocates Na across the basolateral membrane and generates an electrochemical gradient that favours diffusive Na uptake across the apical membrane, via an epithelial Na channel (ENaC) functionally linked to a proton pump (H^+-ATPase), or via a sodium-hydrogen exchanger (NHE; Evans et al., 2005). Apical Na ion influx through either ENaC or NHE can be assisted by the actions of cytosolic carbonic anhydrase (CA), which generates protons through the hydration of intracellular carbon dioxide, facilitating Na ion influx by counter-ion exchange (Evans et al., 2005). The initial step in Cu toxicity involves the reduction of ionic copper (Cu^{2+}) by branchial reductases to Cu^+, which mimics Na^+ and enters the gill cell via the apical Na transporters mentioned above (Bury et al., 2003). Once in the cell, Cu causes toxicity by inhibiting NKA (e.g., De Boeck et al., 2001; Laurén and McDonald, 1987), and CA (de Polo et al., 2014; Zimmer et al., 2012), resulting in an inability of the fish to maintain adequate body Na levels, eventually leading to death.

* Corresponding author at: Faculty of Science and Technology, Athabasca University, 1 University Drive, Athabasca, Alberta T9S 3A3, Canada.
E-mail address: cglover@athabascau.ca (C.N. Glover).

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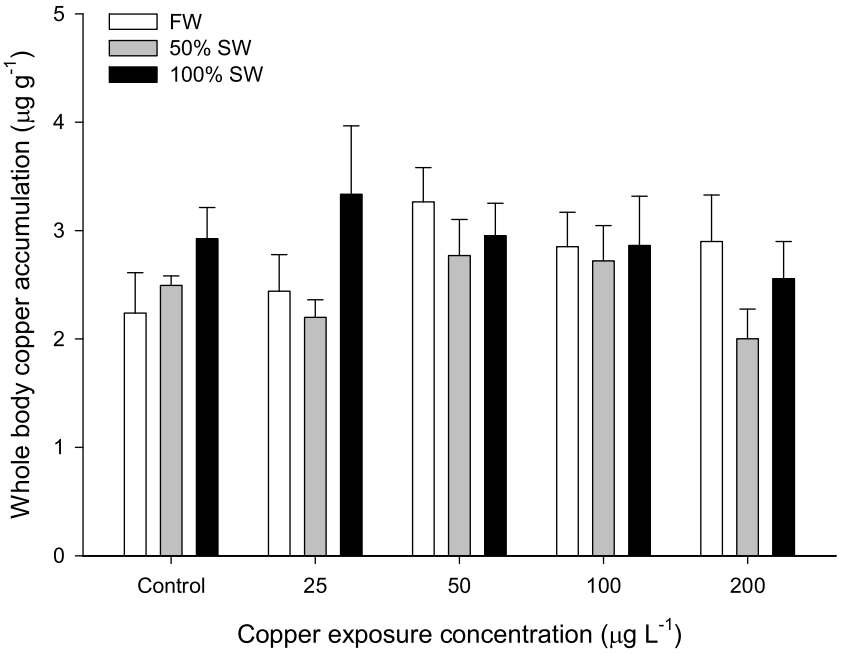

Fig. 1. Whole body Cu accumulation ($\mu g\,g^{-1}$) in inanga exposed to Cu (0, 25, 50, 100 or 200 $\mu g\,L^{-1}$) for 48 h in one of three salinities (FW, 50% SW, 100% SW). Plotted points represent the means ± SEM of 4–6 replicates. Statistical significance was determined by two-way ANOVA, followed by Tukey's post hoc test at $\alpha = 0.05$ (see Table 2).

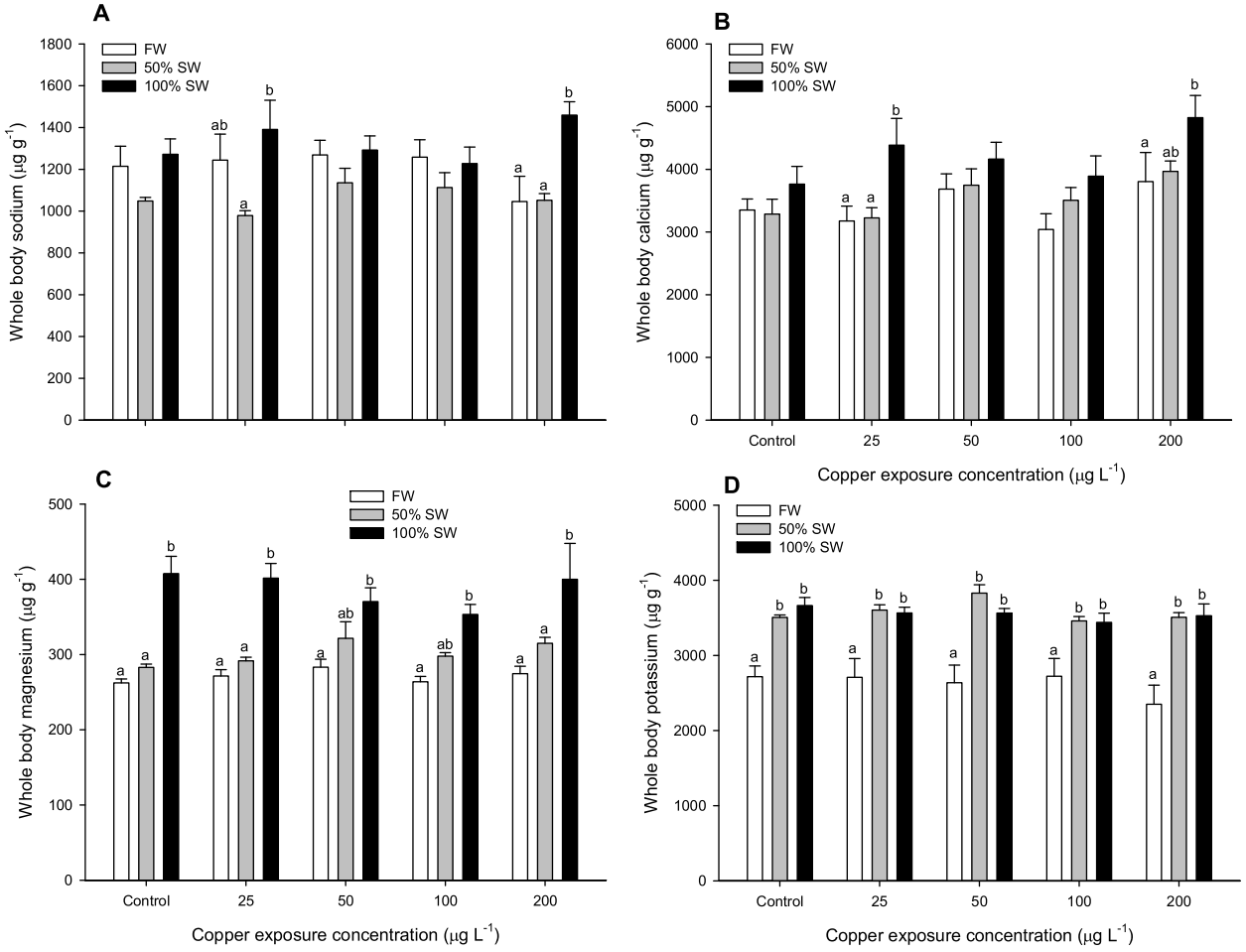

Fig. 2. Whole body Na (A), Ca (B), Mg (C) or K (D) concentrations ($\mu g\,g^{-1}$) in inanga exposed to Cu (0, 25, 50, 100 or 200 $\mu g\,L^{-1}$) for 48 h in one of three salinities (FW, 50% SW, 100% SW). Plotted points represent the means ± SEM of 4–6 replicates. Bars sharing letters are not significantly different relative to other salinities within a Cu exposure concentration. Statistical significance was determined by two-way ANOVA, followed by Tukey's post hoc test at $\alpha = 0.05$ (see Table 2). Exposure Cu concentrations where there were no significant differences with respect to salinity have no alphabetical indicators.

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