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Acute waterborne cadmium toxicity in the estuarine pulmonate mud snail, *Amphibola crenata*

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ABSTRACT

Freshwater pulmonate snails are sensitive to trace metals, but to date, the sensitivity of estuarine pulmonate snails to these important environmental toxicants is undescribed. Using the estuarine mud snail *Amphibola crenata*, effects of a 48-h exposure to waterborne cadmium (Cd) were investigated. The 48-h median lethal concentration (LC_{50}) was 50.4 mg L^{-1}, a value higher than that previously reported for any gastropod mollusc. Cadmium levels in the tissues of mud snails were highest in the viscera (digestive gland and gonad), with the foot muscle and remaining tissue compartment (kidney, mantle, remaining digestive tissues and heart) displaying significantly lower concentrations. Over a Cd exposure concentration range of 0–32 mg L^{-1}, *Amphibola* exhibited reduced oxygen consumption and elevated ammonia excretion in response to increasing Cd, the latter effect likely reflecting a switch to protein metabolism. This finding was supported by a declining oxygen: nitrogen ratio (O:N) as exposure Cd concentration increased. Other energy imbalances were noted, with a decrease in tissue glycogen (an effect strongly correlated with Cd burden in the viscera and foot muscle) and an elevated haemolymph glucose observed. An increase in catalase activity in the visceral tissues was recorded, suggestive of an effect of Cd on oxidative stress. The magnitude of this effect was correlated with tissue Cd burden. The induction of antioxidant defence mechanisms likely prevented an increase in levels of lipid peroxidation, which were unchanged relative to Cd exposure concentration in all measured tissues.

1. Introduction

Estuaries are demanding environments, exposing the biota therein to extreme fluctuations in salinity, dissolved oxygen, temperature, and nutrients (e.g. Hubertz and Cahoon, 1999), all factors that threaten organism homeostasis. Estuaries are also sinks for contaminants, which further challenge health and survival. This is especially true of estuaries located near urban centres, which may receive domestic, industrial and agricultural effluents, either through direct inputs, or via the river systems that drain into them (Matthiessen and Law, 2002). Furthermore, a variety of physical and chemical factors can effectively trap contaminants within estuaries (Ridgway and Shimmield, 2002), exposing estuarine biota to potentially harmful concentrations of a diverse range of toxicants.

Among the contaminants of particular concern in estuarine settings are trace metals. These are environmentally persistent and can cause a variety of toxic effects in the exposed animal (e.g. Chandurvelan et al., 2015). Sub-lethal (e.g. biochemical and physiological) effects can lead to changes in organism health and fitness, eventually resulting in changes at the population, community and ecosystem level (e.g. Lagadic et al., 1994; Marsden and Swinscoe, 2014). Among trace metals, cadmium (Cd) is of significant interest. This highly toxic, non-essential trace element, is distributed ubiquitously in aquatic environments. It is released into the environment by both anthropogenic (e.g. municipal wastewater, fossil fuel combustion, metal processing, agricultural runoff; Eisler, 1985; Butler and Timperley, 1996) and natural sources, such as weathering of Cd-rich geology and volcanism (Eisler, 1985). Although the average concentration of Cd in seawater is around 0.1 μg L^{-1} (Korte, 1983), marine sediment concentrations can be as high as 80 μg g^{-1} (Böning et al., 2004). Estuarine Cd contamination has received significant recent attention in New Zealand (e.g. Chandurvelan et al., 2015, 2016), owing in part to concerns regarding Cd contamination of superphosphate fertilisers applied extensively to near-coastal lowland streams (Butler and Timperley, 1996; McDowell, 2010), and run-off from mine tailings that can result in river Cd concentrations as high as 800 μg L^{-1} (Craw et al., 2005). Cadmium can

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cause a wide variety of toxicological impacts on aquatic organisms including changes in biochemical pathways associated with oxidative stress, altered energy metabolism, impaired calcium homeostasis, perturbed growth and reproduction, and at sufficiently high exposure concentrations, death (e.g. Eisler, 1985; McGeer et al., 2012).

One group of aquatic organisms that have been reported as displaying high sensitivity to trace metals (Grosell et al., 2006; Brix et al., 2011), including Cd (Das and Khangarot, 2010), are the freshwater pulmonate gastropods. For example, *Lymnaea stagnalis* is highly susceptible to copper toxicity, stemming from impairments in ion regulation that affect shell development (Brix et al., 2011), while inhibition of feeding and growth are observed at Cd concentrations as low as 32 μg L^{-1} in *L. luteola* (Das and Khangarot, 2010). It is, however, important to note that not all studies support the conclusion of high sensitivity of freshwater pulmonate snails to trace metals. A study on copper toxicity comparing pulmonate snails with hydrobiid snails, found that toxicity was similar in both groups (Besser et al., 2016). The difference in the sensitivity of pulmonate snails in this study relative to other work, was attributed to the authors use of a distinct age class of animals (Besser et al., 2016). However, this finding of relatively high tolerance is consistent with Cd toxicity studies in terrestrial pulmonate snails (Chabicovsky et al., 2004). It is intriguing that, irrespective of sensitivity, both freshwater and terrestrial pulmonates have an excellent capacity for Cd bioaccumulation (Chabicovsky et al., 2004; Das and Khangarot, 2010). For example, bioconcentration factors of more than 6000 have been reported in *L. palustris* exposed to 160 μg L^{-1} waterborne Cd for four weeks (Das and Khangarot, 2010). However, we are unaware of any published reports that detail the sensitivity of marine or estuarine pulmonate snails to Cd, or that delineate their capacity for Cd bioaccumulation.

In the current study, the mortality, bioaccumulation, biochemical and physiological impacts of waterborne Cd exposure were examined in the estuarine pulmonate mud snail, *Amphibola crenata*. This species is widely distributed throughout New Zealand and is found in both relatively clean and contaminated areas (Marsden and Baharuddin, 2015). It has a sedentary lifestyle, is abundant year-round, easy to collect, and is highly tolerant to extremes of temperature, aerial exposure and desiccation (Shumway, 1981; Shumway and Marsden, 1982). Little is known regarding the sensitivity of this species to metal contaminants, however it has been shown that those snails inhabiting settings with high sediment metals display a decreased condition index (Marsden and Baharuddin, 2015).

Our studies were conducted in the laboratory under acute exposure conditions. Laboratory-based testing overcomes some of the practical difficulties in determining the effects of individual stressors in the field, and thus is an essential tool for understanding the mechanisms of contaminant impacts in organisms. An understanding of toxic mechanisms is itself essential for development of predictive models, allowing extrapolation between different species on the basis of shared pathways of impact. Mechanistic data are also increasingly important for the development of regulatory tools (e.g. Biotic Ligand Model), which facilitate site-specific determination of toxicity based on knowledge of water chemistry, and mechanisms of uptake and toxicity (Di Toro et al., 2001). Studies performed over acute time-frames provide rapid and reproducible estimates of the effects of contaminants on biota, and are critical to the development of acute-to-chronic toxicity ratios, which are the basis of many water quality criteria (e.g. Shuhaimi-Othman et al., 2013). Moreover, when coupled with measurement of mechanistic endpoints, these tests provide an overview of toxic responses in an organism (Chandurvelan et al., 2012), and may help in the future selection of biomarkers and bioindicator species for environmental monitoring (e.g. Shuhaimi-Othman et al., 2012).

In the current study, biochemical and physiological endpoints were used to characterise the mechanisms by which Cd exerts its effects. These included physiological indices of metabolic impairment such as oxygen consumption, ammonia excretion and the oxygen to nitrogen ratio (O:N), which have previously been shown to be impacted by Cd in aquatic biota (e.g. Barbieri and Paes, 2011; Chandurvelan et al., 2012, 2017). In addition, biochemical correlates of energy use (tissue glycogen, haemolymph glucose) were measured. Biochemical analysis also included measures of oxidative stress, based on the ability of Cd to induce reactive oxygen species (ROS), likely via displacement of redox active metals from cofactor binding sites (Nair et al., 2013). Cadmium has also been shown to impair the function of enzymes that scavenge ROS, and thus generate oxidative damage indirectly (e.g. Chandran et al., 2005; Chandurvelan et al., 2013; McRae et al., 2018). Overall, the objectives of the present study were to determine toxicological, physiological and biochemical responses of *A. crenata* to acute Cd exposure, and to investigate Cd bioaccumulation in different tissues as a function of exposure levels.

2. Materials and methods

2.1. Sample collection and maintenance

Adult mud snails (> 18 mm) were collected during December 2015 from the mouth of the Avon-Heathcote Estuary/Ihutai (S 43°33.136', E 172°44.709') in Canterbury, New Zealand. Recently measured sediment Cd concentrations near the site of collection were 0.1 μg g dry weight $^{-1}$ (Chandurvelan et al., 2016). Snails were then transported to the aquarium at the University of Canterbury, where they were washed thoroughly with filtered natural seawater, and attached algae and mud was scraped from the shells. Snails were then transferred into a holding tank containing 2 L of 20 ppt filtered natural seawater (pH 7.6; Na 325 mmol L^{-1}, Ca 9 mmol L^{-1}; Mg 37 mmol L^{-1}; K 8 mmol L^{-1}; Cl 360 mmol L^{-1}; dissolved organic carbon 0.3 mg C L^{-1}) made by diluting Lyttelton Harbour seawater with City of Christchurch artesian well water, and acclimated for 48 h prior to the experiments, under constant temperature (15 ± 0.5 °C) and photoperiod (12 h light: 12 h dark). Snails were not fed during acclimation.

2.2. Determining median lethal concentration (LC_{50})

Toxicity testing was carried out in constantly-aerated acid-washed 1.5 L polypropylene containers at 15 °C and 20 ppt salinity (natural SW as described above) at six Cd exposure concentrations (nominally: 0, 8, 16, 32, 64, and 128 mg Cd L^{-1}), achieved by the dilution of a stock solution of 10 g L^{-1} Cd (as $CdCl_2.2½H_2O$). Each exposure concentration was replicated 6 times, with 15 individual mud snails per replicate. Snails were assigned randomly to the different treatments, and were not fed during the 48-h exposure period. A 15-mL water sample was taken from each treatment at time 0 and 48 h, by filtering through a Millex 0.45 μm filter (Millipore Ltd, Cork, Ireland), with these samples subsequently acidified to pH < 2 using 70% ultrapure HNO_3. Acidified samples were then diluted 30 × with milli-Q water and stored at 4 °C until analysed by Atomic Absorption Spectroscopy (see below). Mortality was assessed every 12-h throughout the experiment period, with death defined as the point when immobile mud snails failed to respond to probing using forceps. The LC_{50} values were calculated based on measured Cd exposure concentrations.

2.3. Bioaccumulation, biochemistry and physiology

Surviving individuals from the 0, 8, 16 and 32 mg Cd L^{-1} exposure concentrations used to determine the LC_{50} were then used for assessment of tissue Cd content, and physiological and biochemical responses to Cd exposure. While these exposure concentrations are significantly elevated relative to those likely to be experienced in natural settings, they provide a means of exploring the mechanisms by which Cd exerts toxic effects on *Amphibola*.

Immediately following the 48-h exposure, two groups of snails were subjected to physiological assays (see below; each n = 6), while a

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