The effects of arsenic of the Na^+/K^+ ATPase transporter in the gills of the freshwater crayfish, *Cherax destructor*

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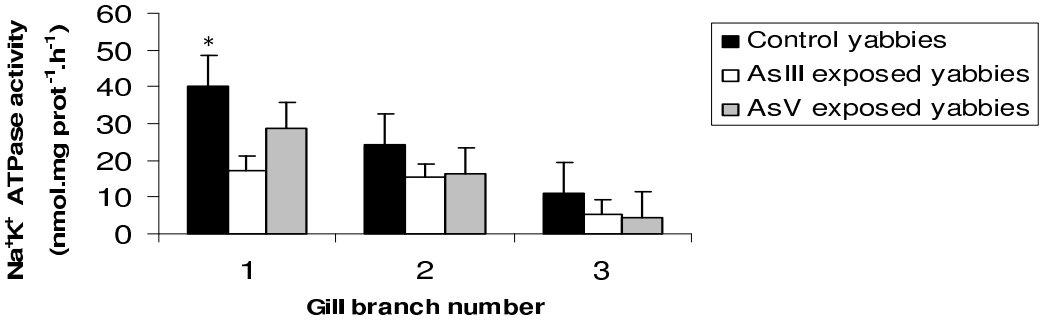
Arsenic is a ubiquitous element found in varying concentrations throughout the environment. It is a known carcinogen found at high concentrations in association with gold and other metals. Several anthropogenic activities, such as mining and agriculture can lead to elevated arsenic concentrations in the environment. Arsenic is found in the environment mainly as inorganic arsenic, arsenite (As(III)) and arsenate (As(V)) which are the most toxic and bioaccessible arsenic compounds.

Contamination in the environment can often lead to high levels of arsenic in surrounding water bodies. Thus aquatic animals are more highly exposed and affected than terrestrial animals as they are immersed in this toxin. Exposure to metal contamination has been shown to affect the function of a number of organs in a range of aquatic animals. Marine species are generally found to be more highly affected by metal exposure; however effects of metal contamination can vary amongst freshwater species.

The freshwater crayfish, *Cherax destructor* (Decapoda, Parastacidea) (yabbies), is the most widespread genus of Australian crayfish. Yabbies can accumulate significant amounts of arsenic within their tissues (Williams *et al*., 2008) and exposure to arsenic in their water results in reduced appetite and increased mortality post-moult.

This study aims to investigate the effects of arsenic exposure on the activity of the Na^+/K^+-ATPase transporter responsible for actively pumping sodium ions from the gill epithelium in yabbies exposed to 3ppm As(V) or As(III) in their water. Three gill filaments are attached to each walking leg. The three gill filaments on each of the four walking legs were used in the analysis. The activity of the Na^+/K^+ATPase transporter was measured using the method of McCormick & Bern (1989). The activity of this transporter was significantly different between the gill branches with the largest filament (22.3 nmol·mg $prot^{-1}·h^{-1}$) showing the highest activity in animals from the intermoult (8.3 nmol·mg $prot^{-1}·h^{-1}$ smallest gill). Activity was also significantly higher ($p < 0.05$) at different stages of the moult cycle in control animals (40.0 nmol·mg $prot^{-1}·h^{-1}$ – postmoult; 22.2 nmol·mg $prot^{-1}·h^{-1}$ - intermoult). Exposure to As(III) caused a significant reduction in transporter activity, but only after 12 weeks exposure from 40.0 nmol·mg $prot^{-1}·h^{-1}$ in the control animals to 17.4 nmol·mg $prot^{-1}·h^{-1}$ in the As(III) exposed animals (see Figure).



These results may explain the increased mortality seen in post-moult animals exposed to arsenite in their water. As new gill epithelium is laid down during the premoult, arsenic may alter the tissue structure such that their is reduced number of transporters or the level of activity of the Na^+/K^+-ATPase enzyme within the transporter is affected.

McCormick SD, Bern HA (1989) *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology* **256**: 707-715.

Williams G, West JM, Snow ET (2008) *Environmental Toxicology and Chemistry* **27**:1332-1342.