Water hardness and the effects of Cd on oxygen consumption, plasma chlorides and bioaccumulation in *Tilapia sparrmanii*

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Abstract

Closed system respirometry was performed on captive juvenile (30 ± 8 g; mean ± S.E.M) *Tilapia sparrmanii* exposed for 96 h to low (1 mg·ℓ$^{-1}$) and high (20 mg·ℓ$^{-1}$) levels of cadmium in soft and hard water. In hard water (235 mg·ℓ$^{-1}$as $CaCO_3$), cadmium (Cd) applied as $CdCl_2$, precipitates completely out but in very soft water (16.5 mg·ℓ$^{-1}$as $Ca\text{-}CO_3$), 23% of Cd is in solution 96 h after it was dissolved. Cadmium complexation is not caused by the presence of chlorides but probably depends on carbonates and sulphates present in Mooi River water. Handling stress, that lasts for at least 6 h, increased the specific oxygen consumption rate of *T. sparrmanii* (MO_2) by more than 30% compared to resting oxygen consumption levels. In hard water no change in the $\dot{M}O_2$ was found when *T. sparrmanii* was exposed to 1, 5, 10, or 20 mg of Cd·ℓ$^{-1}$ of water. In soft alkaline water all fish died when exposed for 96 h in 20 mg Cd·ℓ$^{-1}$. For 10 mg Cd·ℓ$^{-1}$, the $\dot{M}O_2$ was reduced significantly ($p< 0.05$) by 30%. The percentage cadmium dissolved in hard water was, after 96 h, below 1%, 96 h after it had been dissolved. About 2 000 µg Cd·g^{-1} accumulates per gram dried gill mass when fish are exposed to 20 mg Cd·ℓ$^{-1}$ in hard water. For soft water the Cd accumulation is about twice as much. In liver tissue more than 60 µg Cd accumulates per gram dried liver mass in hard water. In soft water the accumulation was three times as much. Blood plasma chlorides decreased from a mean of 130 mmol to 60 mmol when exposed to 20 mg Cd·ℓ$^{-1}$ in soft water. The differences were statistically significant ($P< 0.05$). No decrease in blood plasma chlorides was found in hard water when fish were exposed to 20 mg Cd·ℓ$^{-1}$ of water. Fish handling reduces the oxygen consumption rate by 35% but $\dot{M}O_2$ returns to normal resting levels 6 h after handling stress. It is concluded from the results that $\dot{M}O_2$ and blood chlorides can be used as parameters for Cd toxicity in a 96 h exposure period provided that the precipitation of Cd in the water is known and the pH of the water is monitored.

Keywords: fish, Cd, $\dot{M}O_2$, body electrolytes, hard water, soft water

Introduction

The introduction of toxic metals such as Cd into the environment by anthropogenic sources is an important challenge to toxicologists and ecological management. The concentration of the metal, its geographical transportation, exposure to a target organism and the responses of the individual organism to a specific toxic metal should be known (Truhaut, 1974; Carpené et al., 1990). The peltic sediments adjacent to the gold mine slimes dams near Carletonville, South Africa, have Cd concentrations of between 0.18 and 0.86 µg·g^{-1} with an enrichment factor of 3 compared with standard South shale. These determinations were made before 1977 (Wittmann and Förstner, 1977). Since then no physical changes of the mine drainage around the slimes dams have taken place. A tributary of the Mooi River, receiving dolomitic water (Midgley et al*.,* 1990), but no drainage from the peltic sediments, from the mines, drains into the upper reaches of the Mooi River. It has been estimated that the global input of 9 400 t of Cd into the aquatic ecosytems per year is caused by anthropogenic activities where mining, smelting and refining contributes 41.5% of the total input (Nriagu and Pacyna, 1988).

Nothing is known what the effect of Cd is on the oxygen consumption rate of South African fishes. It has been shown that exposure of cadmium to fish affects the kidney and liver functions (Friberg et al., 1979). The use of gill ventilation, respiration or metabolic rate for individual fish to measure the responses of fish to toxic metals has been poorly studied (Anderson and D' Apollina, 1978; Kelly, 1988; Rand et al., 1995). According to Depledge (1990) individual physiological variability can be used as a tool to investigate toxicity effects above traditional methods such LC_{50} values especially for aquatic organisms. In this regard he strongly advocates the use of respiratory responses to pollutants. Furthermore, Klerks and Levinton (1989) have demonstrated that selection pressure has resulted in the evolution of resistance to Cd in an aquatic oligocheate.

Gill epithelial damage by metal toxicants in fish is often characterised by excessive excretion of mucus (Mallatt, 1985). Heath (1984), Felts and Heath (1984) and Heath (1987), working on the fish *Lepomis macrochirus,* disputed the view put forward by Burton et al. (1972), that heavy metals cause only tissue hypoxia due to excessive secretion of mucus on the gill surfaces. They provided evidence that heavy metals acted on the gill physiology, resulting in a decrease in the oxygen consumption because of ion-regulatory and acid-base balance disturbances (Goss and Wood, 1988).

An increase in the oxygen consumption rates by heavy metals has been described, effected by the stressor responses in fish. (Schreck and Lorz, 1978; Wendelaar-Bonga, 1997). These stressor responses induce a dramatic increase in adrenaline, resulting in increased oxygen uptake rate, initiated by higher gill ventilation rates. Furthermore, stimulation of the branchial blood flow and branchial oxygen diffusion capacity is enhanced. Most of these actions are caused by cathechol-amines acting through adrenergic mechanisms (Wendelaar-Bonga, 1997; Pelgrom et al*.,* 1994). It is

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