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Research Article

ANTIOXIDANT ROLE OF ZINC AND CALCIUM SUPPLEMENTATION AGAINST CADMIUM INDUCED OXIDATIVE STRESS IN FRESH WATER TELEOST *OREOCHROMIS MOSSAMBICUS* (TILAPIA)

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ABSTRACT

Cadmium (Cd) has the ability to accumulate in the tissues and disturb antioxidant defense system. In the present study, Fishes were exposed to 1/10th of LC_{50} / 48 hr. of Cd for 7, 15 and 30days to know the antioxidant role of zinc and calcium on cadmium toxicity. After 15d Cd exposure, fish were divided into three groups. 1st group was supplemented with Zinc (1 ppm), 2nd group received Calcium (1ppm) and 3rd group was supplemented with combination of both zinc and calcium at the above said doses for 7, 15 and 30days. After specific time intervals fish were sacrificed and selected tissues such as liver, kidney, brain, gill and muscle were isolated for the assay of oxidative stress enzymes like superoxide dismutase (SOD), catalase (CAT), glutathione-S-transferase (GST) and glutathione peroxidase (GPx). Simultaneously lipid peroxidation (LPO) levels were also measured. A significant elevation in LPO levels with decreased activity levels of CAT, SOD, GPx and GST were observed during Cd exposure. With zinc and calcium supplementation, a significant reversal in the activity levels of above said enzymes was observed. Our findings clearly envisage that the zinc and / or calcium supplementation is very effective in reducing the Cd toxicity.

INTRODUCTION

The contamination of fresh waters with a wide range of pollutants has become a matter of concern over the few decades (Dirilgen, 2001; Vutukuru, 2005; Kwong *et al.*, 2011). Natural aquatic resources are extensively contaminated with heavy metals like lead (Pb), cadmium (Cd), nickel (Ni) and copper (Cu) released from domestic, industrial and other man made activities. Among the heavy metals, Cd is one of the most toxic, non-essential heavy metal; known for its corrosive nature and is widely used in paints and dyes, cement and phosphate fertilizers (Jarrup, 2003). Cd occurs naturally in the environment in significant amounts but its release in the recent past is steadily increasing due to human activities causing pollution at considerably toxic amounts was reported by earlier workers in various aquatic ecosystems (Usha Rani, 2000; Siraj Bhasha and Usha Rani, 2003; Kiran *et al.,* 2006; Bhavani *et al.*, 2009; Jia *et al.,* 2011; Kim *et al.,* 2011; Obaiah and Usha Rani, 2012; 2013; 2014; 2015; 2016).

Cd has an extremely long half-life (20-30 Years) in the human body (Flora *et al.,* 2008) and is highly cumulative, especially in the liver and kidney (Hijova and Nistiar, 2005; Mahtap and Ethem, 2006; Nordberg *et al.,* 2007; Tim *et al.,* 2008).

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It is a ubiquitous toxic metal and induces oxidative damage by disturbing the prooxidant – antioxidant balance in the tissues (Ognjanovic *et al.,* 2008). Cd inhibits oxidative stress enzymes which protect tissues by either binding to sulfhydryl (-SH) groups essential for the enzymes, replace the bivalent metals like zinc (Zn), copper (Cu), selenium (Se), iron (Fe) and manganese (Mn) required for the enzymes (Eriyamremu *et al.,* 2008).

Cd like many other heavy metals is antagonistic to essential trace elements like Zn, Fe, Cu, Ca etc., (Sobha *et al*., 2007) and competes with these trace elements for binding sites as transport and storage proteins, metalloenzymes and receptors.
Zn is a ubiquitous essential trace element with numerous functions in biological systems. It occurs in all living cells as a constituent of metallo enzymes involved in major metabolic pathways. It plays a catalytic, inhibitory or accessory role in the regulatory enzymes such as kinases or phosphatases. Zn controls several enzymes of intermediary metabolism, DNA and RNA synthesis, gene expression, immune competence and plays a significant role in homeostasis of hormones (Brando *et al.,* 1995). It has been noted that Zn is a constituent of several enzymes (more than 300 enzymes) in the body and can prevent cell damage through activation of the antioxidant defense system (Ozturk *et al.,* 2003; Prasad *et al.,* 2004; Ozdemir and Inanc, 2005; Bashandy *et al.,* 2006; Amara *et al.,* 2008).