Journal of Applied and Natural Science 2 (1): 66-69 (2010)

JANS

Effect of combinations of four trace elements on cadmium bioaccumulation in a few tissues of male albino rats

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Abstract: The present study is designed to investigate the accumulation of cadmium (Cd) in selected tissues such as liver, kidney, testis and small intestine of male albino rats treated for Cd and after combined supplementation with Zinc (Zn) + Iron (Fe) and Selenium (Se) + Copper (Cu). The wistar albino rats were treated with cadmium chloride($CdCl_2$)at a dose of 1/10th LD_{50} i.e. 22.5 mg / kg body weight for 7, 15 and 30 days(d) time intervals. After 15 d to Cd treatment, the rats were then supplemented with the above combination of two trace elements and then observed for accumulation of Cd at specific time intervals. These trace elements at a dosage of 1 mg/ kg body weight of Se, 16mg/kg body weight of Cu, 12 mg / kg body weight of Zn and 40 mg / kg body weight of Fe were given as supplements. There was significant Cd accumulation in liver and kidney among the selected tissues before to supplementation and there was significant decrease in the Cd accumulation levels in all the tissues after trace element supplementation. Moreover the 30d Zn + Fe supplemented rat kidney showed maximum decrease in Cd accumulation (8.327µg/g wet wt. of the tissue).

Keywords: Trace element supplements, Cadmium bioaccumulation, Rats

INTRODUCTION

As the civilization advances, growing environmental pollution brings the effect of various xenobiotics, including heavy metals, on the functioning of the living organisms (Moniuszko, 1999). Cadmium (Cd) is an environmental pollutant that has serious toxicity in humans and animals and causes Itai-Itai disease (Nad *et al.*, 2005) and also induces the onset of anemia, decreases red blood cell count and hemoglobin concentration (Ognjanovic *et al.*, 2003). Metals can not be destroyed metabolically. However, toxic metals may bind to organic molecules or compete with physiological metals, which may result in a metal-specific toxic effect. For example the Cd effect on Metallothionein (MT). Cd is an industrial and environmental pollutant arising primarily from battery, electroplating, pigment, plastics, fertilizer industries and cigarette smoking. The accumulation of Cd is consistently increased when a certain amount is ingested continuously (Shibutani *et al.*, 2001). When Cd enters the body, it reaches the liver within the first 6h and binds to metallothionein, which is a protein with a low molecular weight (6000-10,000 Da) and rich in cystein (Haki Kara *et al.*, 2005 and Chan and Cherian, 1993). The Cd-MT complex generated in liver was reported to mainly to be distributed to the kidney and other tissues and hence it causes damage in the tissues and organs causing many metabolic and histological changes, membrane damage, altered gene expression and apoptosis (Ognjanovic *et al.*, 2008; Haki Kara *et al.*, 2005; Sirja Basha and Usha Rani, 2003; Usha Rani, 2000 and Liu *et al.*, 1996). 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 (Tim *et al.*, 2008; Nordberg *et al.*, 2007; Mahtap and Ethem, 2006 and Hijova and Nistiar, 2005). The kidney is considered as the critical organ in long term low level exposure to Cd (Trzcinka *et al.*, 2004). Chronic exposure to Cd caused kidney damage and renal tubular dysfunction (Horiguchi *et al.*, 2005). It is shown that Cd is an inhibitor of enzymes with sulfhydral (-SH) groups and disrupts the pathways for the oxidative metabolism. Because of its carcinogenic properties, Cd has been classified as a#1 category human carcinogen by the International Agency for Research on Cancer of USA (IARC, 1993).

The complex inter-relationships between Cd and some essential trace elements have not been elucidated. Several essential trace elements like Selenium (Se), Copper (Cu), Zinc (Zn) and Iron (Fe) participate in controlling various metabolic and signaling pathways. In vitro studies suggests there is competition for transport mechanism between Cd and some essential trace elements like Zn and Cu (Hakan *et al.*, 2001), Se (Hijova and Nistiar, 2005) and Fe (Rodriguez- Matas *et al.*, 1998) in rats and Zn and Se in Japanese quails(Nad *et al.*, 2005) and calcium in suckling rats (Saric *et al.*, 2002). Se is involved in the metabolism of glutathione (GSH) which is related to reduce