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RESEARCH ARTICLE

CADMIUM INDUCED BIOACCUMULATION AND OXIDATIVE STRESS IN LIVER AND KIDNEY OF MALE ALBINO RAT

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ABSTRACT

The present study was carried out to know the cadmium (Cd) induced bioaccumulation and oxidative stress in liver and kidney of Cd treated rats. Wistar strain male albino rats were treated with cadmium chloride ($CdCl_2$) at a dose of $1/10^{th}$ LD_{50} / 48h i.e. 22.5 mg/kg body weight for 7, 15 and 30 days (d) long sojourn. After the specific time intervals, rats were decapitated and tissues like liver and kidney were isolated for the analysis of Cd bioaccumulation and assay of oxidative stress enzymes such as catalase (CAT), superoxide dismutase (SOD), glutathione -S-transferase (GST) and glutathione peroxidase (GPx). Simultaneously lipid peroxidation (LPO) levels were also measured. There was a significant elevation in Cd bioaccumulation in both the test tissues with increased period of Cd treatment. Maximum Cd accumulation wasfound in 30d Cd treated rat kidney. A significant elevation in LPO levels with decreased activity levels of CAT, SOD, GPx, and GST were observed during Cd intoxication. Our study clearly reveals that Cd intoxication can disturb the antioxidant defense system and increase the body burden of Cd in the tissues.

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INTRODUCTION

Cadmium (Cd) is one of the most toxic, non- essential heavy metal with many industrial uses that can contribute to a well-defined spectrum of diseases in animal models as well as in humans (Akeem *et al.,* 2011; Nobuhiko *et al.,* 2012). Cd has an extremely long half-life (20-30 Years) in the human body and is highly cumulative, especially in the liver and kidney (Flora *et al.,* 2008; Hijova and Nistiar, 2005; Mahtap and Ethem, 2006; Nordberg *et al.,* 2007; Tim *et al.,* 2008). Kidney is considered as the critical organ in long term low level exposure to Cd (Asagba, 2009). The main sources of Cd are storage batteries, electroplating, pigments, plastics, fertilizer industries and cigarette smoking. Although Cd is widely distributed throughout the body, most of it accumulates in the liver and kidney and alters biochemical and functional changes in the organs. Cd can cause Itai - Itai disease in humans as well as in animals (Nad *et al.,* 2005) and also it induces the onset of anemia, decreases red blood cell count and hemoglobin concentration (Ognjanovic *et al.,* 2003). Some of the toxic effects of Cd exposure are hepatic damage, renal dysfunction, hypertension, central nervous system injury and testicular atrophy (Usha Rani, 2000; Siraj Basha and Usha Rani, 2003; Haki *et al.,* 2005; Jeyaprakash and Chinnaswamy, 2005; Ognjanovic *et al.,* 2008). It is a ubiquitous toxic metal and induces oxidative damage by disturbing the prooxidant – antioxidant balance in the tissues (Ognjanovic *et al.,* 2008). Hence, the present study focused on the bioaccumulation of Cd and perturbations in the antioxidant defense system.

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MATERIALS AND METHODS

Chemicals

Cd as cadmium chloride ($CdCl_2$) was purchased from Merck (Dormstadt, Germany). All other chemicals which were used in the present study were obtained from the standard chemical companies like Sigma Chemical Co. (St Louis, MO, USA) and SD Fine Chemicals, India. The chemicals used in this study were of the highest purity.

Animals

Three-months-old Wistar strain male albino rats weighing 180 ± 20 g were chosen for the present study. The animals were obtained from Sri Venkateswara Traders, Bangalore, Karnataka, India and were kept in stainless steel mesh cages, housed under standard laboratory conditions (23 ± 2°C, 50 ±