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# TRACE ELEMENT SUPPLEMENTATION AGAINST CADMIUM INDUCED TOXICITY IN MALE ALBINO RATS : PROTECTIVE ROLE OF ZINC AND IRON

OBAIAH J, A USHA RANI* and D KUMAR BABU
Division of Environmental Biology, Department of Zoology, S.V.University, Tirupati – 517 502, A.P.

## ABSTRACT

Cadmium (Cd) is one of the most toxic substances in the environment with a wide range of organ toxicity and long elimination half-life. The present study is designed to investigate the accumulation of Cd in selected tissues such as liver, kidney, testis and small intestine of male albino rats treated for Cd and after supplementation with Zinc (Zn) and Iron (Fe). The wistar albino rats were treated with cadmium chloride($CdCl_2$)at a dose of $1/10^{th}$ $LD_{50}$ i.e. 22.5 mg / kg body weight for 7, 15 and 30 days(d) time intervals. After 15 d Cd treatment, the rats were supplemented with Zn and Fe individually and also in combination for 7, 15 and 30d duration. A dosage of 12 mg / kg body weight of Zn and 40 mg / kg body weight of Fe were given as supplements. After the specified time intervals, rats were sacrificed and the tissues were analysed for Cd accumulation by Atomic Absorption Spectrophotometer (Shimadzu – AA 6300) before and after supplementation with Zn and Fe. Results revealed significantly high Cd accumulation in liver and kidney with a significant level of decrease in Cd accumulation in all the selected tissues after supplementation with trace elements like Zn and Fe. In 30d Fe supplemented rats, liver tissue showed maximum decrease in accumulation of Cd. However both Zn and Fe supplementation eliminated the Cd burden of kidney significantly (8.327µg/g wet wt. of the tissue) than that of liver suggesting Fe and Zn supplementation in combination to be more effective in elimination of Cd body burden from the tissues of rats.

**Key words**: Cadmium toxicity, Trace element supplementation, Rats.

## INTRODUCTION

Cadmium (Cd), a non essential highly toxic heavy metal, still attracts attention because it is often detected in the air, water and food products extending the maximum allowable limits[1,2]. Cd toxicity may be more common among natural populations of vertebrates that has been appreciated to date and that Cd toxicity may often go undetected or unrecognized[3]. Cd causes poisoning in various tissues of humans and animals[4]. Cd has an estimated elimination half- life of 10-30 years and accumulates in the human body particularly in liver and kidney[5,6]. Chronic exposure to inorganic Cd results in accumulation of the metal mainly in the liver and kidneys, as well as in other tissues and organs causing many metabolic and histological changes, membrane damage, altered gene expression and apoptosis[7,8,9,10].

In long- term chronic occupational exposure to Cd, the kidneys are usually the most critically affected organs[6,11]. The kidney is well known to be a major target organ of Cd in animals and humans. During chronic exposure the metal accumulates in renal cortex upto what appears to constitute a critical level at which the incidence of overt malfunction in a human population at risk begins to increase and Cd exposure leads to renal tubular dysfunction[3]. This is primarily a re-absorption defect in the proximal tubules and the critical effect o Cd.

Several essential transitional metals like copper, zinc, iron and manganese participate in controlling various metabolic and signaling pathways. Cd can replace Zn in key members of nucleotide and base excision pathways[12]. Zn is an essential trace element for living organisms and more than 300 enzymes require Zn for