PROTECTIVE ROLE OF VITAMIN C (ASCORBIC ACID) AND E (~-TOCOPHEROL) AGAINST CADMIUM INDUCED HEPATOTOXICITY IN RATS

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Asian Journal of Environmental Science, Vol. 3 No. 1 : 23-28 (June, 2008)

SUMMARY

The study was carried out to evaluate the effect of dietary vitamins C (ascorbic acid) and E (α-tocopherol) on serum hepatic marker enzymes, antioxidant and lipid peroxidation status in liver of cadmium intoxicated rats. Ral administration of cadmium chloride (5 mg/kg body wt./day) for 30 days resulted in a significant elevation of AST, ALT, ALP, LDH and the levels of lipid peroxidation markers (TBARS) in liver. Cadmium also caused a significant reduction in the activities of SOD, CAT, GPx and GSH level in liver. Prior oral administration of vitamins C and E (50 mg/kg body wt./day) along with cadmium significantly decreased the activities of serum, AST, ALT, ALP and LDH along with significant decrease in the level of lipid peroxidation in the liver. In addition to that, combined treatment of vitamins C and E significantly increased the glutathione level together with other hepatic antioxidant enzymes. The results suggest that combined treatment of vitamins C and E exhibited better antioxidant property and decrease the level of lipid peroxidation against cadmium induced oxidative stress in liver.

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Accepted : March, 2008

Key words : Cadmium, Lipid peroxidation, Antioxidant, vitamins, Rats.

Exposure of toxic heavy metals has become an increasingly recognized source of illness worldwide. Pollution due to heavy metals is of serious concern and among them cadmium merits the special attention. Cadmium is very toxic heavy metal and an important environmental pollutant that can be injested or inhaled from a variety of industrial and dietary sources. It causes severe damage to various tissues of humans and experimental animals, when exposed chronically (Yadav and Khandelwal, 2005).

Cadmium has a significant effect on hepatic and renal function and as a result, it alters bone mineralization, leading to osteoporosis and osteomalacia. Cadmium induced genotoxicity can also increase the risk of several cancers. The mechanism of cadmium induced damage include the production of free radicals that alter the mitochondrial activity and genetic information (Partrick, 2003).

Although the biochemical mechanisms involved in cadmium hepatotoxicity remain to be elucidated. One of the major concepts regarding the toxicity of cadmiums is attributed to its ability to generate reactive oxygen species which cause oxidative stress. Excessive production of oxygen radicals leads to altered enzyme activity, decreased DNA repair, impaired utilization of oxygen, lipid peroxidation (LPO) and protein oxidation. Some of these alterations induced by oxidative stress have been recognized to be characteristic features of necrosis and subsequently leads to organ damage by cadmium (Pande *et al.,* 2001).

Various studies connects cadmium with oxidative stress since this metal can alter the antioxidant defense system in several tissues of experimental animals, causing depletion in the levels of cellular reduced glutathione, as well as changes in the permeability of the cellular membrane through a process of lipid peroxidation (Bagchi *et al.,* 1997; Milton Prabu *et al.,* 2007b). Studies on mammals showed that cadmium stimulated the formation of ROS including superoxide, anion radical (Amoruso *et al.,* 1982), hydrogen peroxide (Wong *et al.,* 1990) and most probably hydroxyl radical (Ochi *et al.,* 1988).

Lipid peroxidation is essential to understand the extend of metal toxicity in animals. Increased lipid peroxidation is generally believed to be an important underlying cause of initiation of oxidative stress related to various tissue injury, cell death and further progression to many acute and chronic diseases (Basu, 2003). Oxygen free radicals can elicit wide spread damage to cells by the peroxidation of polyunsaturated membrane lipids (Pereira *et al.,* 1998). Manca *et al.* (1991) have also reported a significant increase of lipid peroxidation in the liver and kidney of cadmium intoxicated rats.

Superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) are the enzymes that provide cellular protection against the damage caused by free radicals and reactive oxygen species (ROS). Impairment in the function of these antioxidative enzymes leads to the accumulation of toxic oxidative free radicals and consequent degenerative changes in tissues