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INDUCTION OF OXIDATIVE STRESS BY ACUTE ORAL EXPOSURE OF CADMIUM CHLORIDE IN KIDNEY OF ADULT RAT

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ABSTRACT
Cadmium is a most abundant toxic heavy metal and an environmental pollutant. Oral administration of cadmium chloride ($CdCl_2$) at a dose of 0.2 and 0.4% w/v with distilled water leads to significant ($p < 0.01$) increase in lipid peroxidation and significant decrease in superoxide-dismutase ($p < 0.001$), catalase ($p < 0.001$) and glutathione-peroxidase ($p < 0.01$) in the kidney of treated groups of rat as compared to control. Significantly higher level of lipid peroxidation and lower level of antioxidant enzymes (catalase, superoxide dismutase and glutathione-peroxidase) produces oxidative stress by disturbing the oxidative and antioxidative balance in the kidney of adult rats.

INTRODUCTION

Cadmium is a heavy metal, a well recognized environmental pollutant with numerous adverse health effects. Being widely used in industry, cadmium can affect human health through occupational and environmental exposure (Waisberg *et al*., 2003). The level of cadmium compounds in the environment have progressively increased as a consequence of industrial pollution which can be attributed to electroplating, stabilizers, pigments, plastics, semiconductors and batteries (Hisoyoshi *et al*., 1997). Another important source of cadmium to the atmosphere is cigarette (Akinloye *et al*., 2006). Cadmium compounds have been shown to exert toxic and carcinogenic effect in humans and experimental animals (Misra *et al*., 1998). Transition metals, act as a catalyst in the oxidative reaction of biological macromolecules, thus metal toxicities might be associated with oxidative tissue damage. Exposure of cadmium metal is known to induce the formation of reactive oxygen species (ROS) like superoxide radical, hydroxyl-ion and hydrogen-peroxide (Christopher *et al*., 2004). ROS may lead to cellular damage when the rate of its generation suppresses the rate of its decomposition by antioxidant defense system, such as catalase, superoxide-dismutase and glutathione-peroxidase (Mates *et al*., 1999; Datta *et al*., 2000). In the present study an attempt has been made to understand the dose related cadmium toxicity and induction of oxidative stress in the kidney of rats.

MATERIALS AND METHODS

Adult healthy male albino rats weighting 140-160 g were selected for the present study. These animals were given standard rat chow and tap water ad libitum and were housed at 25 ± 2°C on a 12h. dark/light cycle. For the experimental purpose the animals were divided into three groups each consisting of six rats. Group I (control group) received distilled water as sole drinking source. Group II (0.2% $CdCl_2$ group) and Groups III (0.4% $CdCl_2$ group) received cadmium chloride at dose of 0.2% and 0.4% w/v in distilled water respectively. After 5 weeks the animal of different groups were sacrificed under light anesthesia 1 day after the end of treatment. Tissue of kidney were minced and homogenized (10% w/v) in ice cold 50 mM potassium phosphate buffer (pH 7.5), 1mM EDTA. Homegenates were centrifuged at 4°C and the clear supernatants were used for the biochemical study. Lipid peroxidation was measured by thiobarbituric acid method (Ohkawa *et al*., 1979) that determines aldehyde formed by degradation of hydroperoxide, including malondialdehyde (MDA).The activity of catalase, superoxide-dismutase and glutathione-peroxidase was assayed by the method of Aebi (1984), Nishikimi *et al*., (1972) and Wendel (1980) respectively. Student t-test was applied for the test of significance.

RESULTS

Exposure of acute cadmium chloride at different doses on antioxidant profile of kidney tissue presented in Table 1. It was observed that the level of lipid peroxidation in the tissue homogenates of kidney increased in all cadmium exposed groups as compared to control group of rat. Lipid peroxidation level showed significantly higher level ($p < 0.01$) in treated

Table 1: Effect of Cadmium chloride ($CdCl_2$) on oxidative stress (biochemical parameters) in kidney of rats

Parameters	Group IControl group	Group II(0.2% $CdCl_2$)	Group III(0.4% $CdCl_2$)
Lipid peroxidation (n moles mg^{-1} tissue)	0.055 ± 0.003	0.093* ± 0.006	0.102** ± 0.003
Catalase(U mg^{-1} protein)	8.35 ± 0.45	7.53* ± 0.69	4.76*** ± 0.52
Super oxide dismutase(U mg^{-1} protein)	7.20 ± 0.56	6.12** ± 0.94	3.85*** ± 0.73
Glutathione peroxidase(U mg^{-1} protein)	4.13 ± 0.36	3.60* ± 0.51	2.12** ± 0.18

Values are M ± SE; *, **, *** indicates significance with control at 0.1, 0.01 and 0.001 level respectively

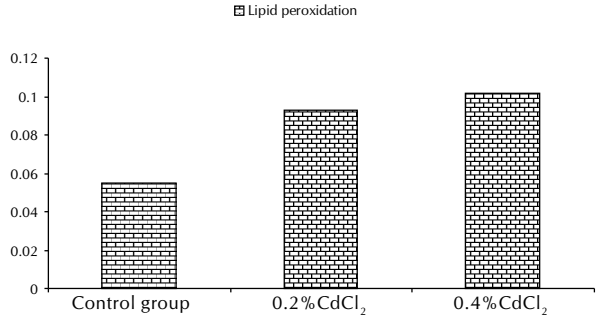

Figure 1a: Effect of cadmium chloride on lipid peroxidation in kidney of rat

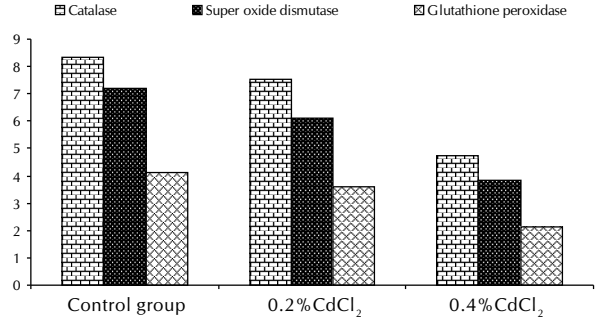

Figure 1b: Effect of cadmium chloride on catalase, superoxide-dismutase and glutathione-peroxidase in kidney of rat

group than the control (Table 1, Fig. 1a).The result also demonstrated that the activity of antioxidant enzymes decreased in the cadmium exposed groups as compared to the control (Table 1, Fig. 1b). Catalase activity in the kidney tissue showed significant decrease in group-II ($p < 0.1$) and group-III ($p < 0.001$) as compared to control group. In corollary to that superoxide-dismutase also showed significant decline in group-II ($p < 0.001$) and group III ($p < 0.001$) than the control group (group I) of rats. Similarly glutathione-peroxidase activity in kidney tissue also reduces significantly in group II ($p < 0.1$) and group III ($p < 0.01$) than the control.

DISCUSSION

The results of present study demonstrate that exposure of cadmium chloride leads to increase in lipid peroxidation. Increased level of lipid peroxidation indicates a decrease in the level of glutathione and change in the activities of antioxidant enzymes (Ei–Maraghy *et al*., 2001; Casalino *et al*., 2001). Excessive production of free radicals or reactive oxygen species (ROS) is mainly responsible for peroxidation of cell membrane lipids. Malondialdehyde (MDA) is terminal product of the lipid peroxidation process and determination of MDA levels provides a good measure of lipid peroxidation, which is among the chief mechanisms of cell damage leading to necrosis or apoptosis (Chlubek *et al*., 2003). The increased level of MDA in the present study were consistent with the finding of Ashraf *et al*. (2007) and suggest that cadmium chloride bring about oxidative damage to kidney by inducing lipid peroxidation in the kidney of rat. Catalase, superoxide-dismutase and glutathione-peroxidase are the antioxidant enzymes that provide cellular protection against the damage caused by free radicals and ROS (Ashraf *et al*., 2007; Patra *et al*., 2011). Present study reveals that treatment of cadmium chloride causes significant decrease in these three (catalse, superoxide-dismutase and glutathione-peroxidase) enzymes activity in kidney tissue of rat which corroborates the study of Ashraf *et al*., 2007. Increased level of lipid peroxidation indicates production of more free radical and ROS in the cadmium chloride treated rat tissue which is not scavenges by the reduced level of antioxidant enzymes in the kidney tissue of rat.Thus the increased level of lipid peroxidation and decreased level of antioxidant enzymes activity disturbing the oxidative and antioxidative balance in the kidney tissue which produces oxidative stress in the kidney of rats.

REFERENCES

Aebi, H. 1984. Catalase *in - vitro*. *Methods Enzymol.* **105:** 121-126.

Akinloye, O., Arowojolu, A. O., Shittu, O. B. and Anctor, J. I. 2006. Cadmium toxicity: A possible cause of male infertility in Nigeria. *Reprod. Biol.* **6:** 17-30.

Ashraf, M., Abdul-Moneim and Kamar, M. S. 2007. Acute effect of cadmium treatment on the kidney of rats : Biochemical and ultra-structure studies. *Pak. J. Biol. Sci.* **10(20):** 3497-3506.

Casalino, E., Calzaretti, G., Sblano, C. and Landriscina, C. 2001. Molecular inhibitory mechanisms of antioxidant enzymes in rat liver and kidney by cadmium. *Toxicol.* **178(1-2):** 37-50.

Chlubek, D., Grucka-Mamczar, E., Birkner, E., Polaniak, R., Stawiarska-Pieta, B. and Duliban, H. 2003. Activity of pancreatic antioxidative enzymes and malondialdehyde concentrations in rats with hyperglycemia caused by fluoride intoxication. *J.Trace. Elem-Med. Biol.* **17(1):** 57-60.

Christopher, O. I., Veera, L. B., Lambert, T. A. T., Lekan, M. L. and John, W. 2004. Response of antioxidant enzymes and redox metabolites to cadmium-induced oxidative stress in CRL-1439 normal rat liver cell. *Int. J. Mol. Med.* **14:** 87-92.

Datta, K., Sinha, S. and Chattopadhyay, P. 2000. Reactive oxygen species in health and disease. *Nat. Med. I. Ind.* **13:** 304-310.

Ei-Maraghy, S. A., Gad, M. Z., Fahim, A. T. and Hamdy, M. A. 2001. Effect of cadmium and aluminium intake on the antioxidant status and lipid peroxidation in rat tissue. *J. Biochem. Mol. Toxicol.* **15:** 201-214.

Hisoyoshi, O., Nakakita, M., Tanaka, H. and Yukioseki, H. Y. W. 1997. Induction of metallothionein like cadmium binding protein in

the testis by oral cadmium administration in rats. *Indus. Heal.* **35:** 96-103.

Mates, J. M., Perez-Gomez, C. and De-Castro, I. N. 1999. Antioxidant enzymes and human disease. *Clin. Biochem.* **32:** 595-603.

Misra, R. R., Smith, G. T. and Waalkes, M. P. 1998. Evaluation of direct genotoxic potential of cadmium in four different rodent cell lines. *Toxicol.* **126:** 103-114.

Nishikimi, M., Appaji, N. and Yagi, K. 1972. The occurance of superoxide anion in the reaction of reduced phenazine methosulphate and molecular oxygen. *Biochem. Biophys. Res. Commun.* **46:** 849-854.

Ohkawa, H., Ohishi, N. and Yagi, K. 1979. Assay for lipid peroxides in animal tissue by thiobarbituric acid reaction. *Anal. Biochem.* **95:** 351-358.

Patra, R. C., Rautray, A. K. and Swarup, D. 2011. Oxidative Stress in lead and cadmium toxicity and its amelioration. *Vet. Med. Int.* 1-9.

Waisberg, M., Joseph, P., Hale, B. and Beyersmann D. 2003. Molecular and cellular mechanisms of cadmium carcinogenesis. *Toxicol.* **192:** 95-117.

Wendel, A. 1980. Enzymatic basis of detoxification. *Vol. 1. Academic Press, NY.* 333.

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