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

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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 140-160g 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% CdCl₂ group) and Groups III (0.4% CdCl₂ group) received cadmium chloride at dose of 0.2% and 0.4% w/v in distilled water respectively. After 5 weeks the animals 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...
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) as compared to control group.

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 (catalase, 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 scavenged 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 rat.

REPRESENTATIONS

Table 1: Effect of Cadmium chloride (CdCl₂) on oxidative stress (biochemical parameters) in kidney of rats

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group I (Control)</th>
<th>Group II (0.2% CdCl₂)</th>
<th>Group III (0.4% CdCl₂)</th>
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<tr>
<td>Lipid peroxidation (in moles mg⁻¹ tissue)</td>
<td>0.055±0.003</td>
<td>0.093*±0.006</td>
<td>0.102**±0.003</td>
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<td>Catalase (U mg⁻¹ protein)</td>
<td>8.35±0.43</td>
<td>7.53*±0.69</td>
<td>4.76***±0.52</td>
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<tr>
<td>Superoxide dismutase (U mg⁻¹ protein)</td>
<td>7.20±0.56</td>
<td>6.12***±0.94</td>
<td>3.85***±0.73</td>
</tr>
<tr>
<td>Glutathione peroxidase (U mg⁻¹ protein)</td>
<td>4.13±0.36</td>
<td>3.60*±0.51</td>
<td>2.12***±0.18</td>
</tr>
</tbody>
</table>

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

REFERENCES

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


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