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Hepato-reno protective effect of *lannea coromandelica* on cadmium chloride induced oxidative damage in rats

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ABSTRACT

In the present study to evaluate Hepato-reno protective effect of lannea coromandelica on cadmium chloride induced oxidative damage in rats. Animals were recorded and they were divided into three groups of six animals each as follows. Group I: Normal animal received with standard fed and water to allowed *ad libitum.* Group II: Animals received intraperitonial injection of cadmium chloride (2.5mg/kg body weight) daily for three consecutive days. Group III: Treatment group received cadmium chloride as group II. After 24 hours of last administration of cadmium chloride, treatment was started at a dose of 500mg/kg body weight of leaves of *Lannea Coromandelica* for five days. .*Lannea Coromandelica* treatment proved to be effective in improving the extent of DNA synthesis, decreases lipid peroxidation, improves the antioxidants, liver and kidney functions. Thus, the above results confirm that supplementation of *Lannea Coromandelica* preserve the genetic materials induced by cadmium. The potential hepatoreno protective activity of *Lannea Coromandelica* may be due to the presence of radical scavenging property of phenolic groups present in it. It can be concluded that use of *Lannea Coromandelica* has the capability to alleviate many of harmful effects of $CdCl_2$.

Keywords: *Lannea Coromandelica*, DNA synthesis, Lipid peroxidation, Cadmium chloride, Oxidative stress, Antioxidant

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INTRODUCTION

Metals particularly through their wide commercial use pose environmental problems, which may ultimately threaten human health (Friberg *et al.*, 1979). Heavy metals are persistent contaminants in the environment that come to the forefront of dangerous substances such as cadmium, lead, mercury, copper and zinc causing serious health hazard in humans and animals (Absernthy *et al.*, 1999; Authuman 2008). Many metals are considered essential trace elements and must be present in low concentration in the human body in order for normal cellular function. However altered concentration or transition states of metals in the body are thought to lead to a wide range of deleterious condition especially an increase in cancer incidence. Increased metal exposure in humans can occur via ingestion, inhalation dermal contact and occupational exposure (Churg *et al.*, 2003;

Gambelunghe *et al*., 2003). Although correlation between metal exposure and cancer are well documented more research is still needed in order to determine the exact mechanism of metal – induced carcinogenesis much of the metal toxicology and carcinogenicity data available are acquired in animal modal systems with few studies representing the effects of metals in humans.

Cadmium (Cd) is one of the most important toxic metals due to its increasing level in the environment as a result of industrial and agricultural practices and considered to be a main threat to human health together with other elements such as lead, mercury and arsenic (Jarup, 2003). Cd levels in environment vary widely and are dependent on the presence of industrial sites extensive agricultural activities or dumpsites. The first reports of severe health problems due to Cd intoxication arose in the 1940s in Japan where the itai-itai disease was endemic and major symptoms were bone and renal damage which were caused by eating Cd-polluted rice (Nomiyama and Nomiyama, 1998). Nowadays it is known that occupational and environmental Cd exposure can result in nephrotoxicity (Bernard, 2004) Hepatotoxicity (Gubrelay *et al.*, 2004) and skeletal damage (Akesson *et al.*, 2006) and several types of cancer in organs such as the urinary bladder, pancreas, breast, kidney, lungs and prostate. Cd administration produced damage to the entire kidney including proximal tubular cell degeneration interstitial inflammation and fibrosis glomerular swelling, atrophic and pyknotic nuclei interstitial edema, glomerular basement membrane swelling, mitochondria swelling, clear, vacuoles, apoptosis, necrosis, occasional segmental sclerosis and mesangial expansion in the glomeruli (Kaur *et al*., 2006).

A growing body of evidence indicates that transition metals act as catalysts in the oxidative deterioration of biological macromolecules and therefore the toxicities associated with these metals may be due to atleast in part to oxidative tissue damage. Recent studies have shown that metals such as Ir, Cu, Cd, Cr, Ld, Mu, Ni, Vn exhibit the ability to produce reactive oxygen species. Resulting in lipid peroxidation, DNA damage, depletion of sulfhydryls and altered calcium homeostasis.

Many metals are considered essential trace elements and must be present in low concentration in the human body in order for normal cellular function. However altered concentration or transition states of metals in the body are thought to lead to a wide range of deleterious condition especially an increase in cancer incidence. Increased metal exposure in humans can occur via ingestion, inhalation dermal contact and occupational exposure. Excessive concentrations of these elements are also toxic harmful. Some elements such as lead, cadmium and mercury have harmful effects on biological tissues at any concentration.

Indian medicinal plants and may herbal formulation belonging to the traditional system of medicine like ayurveda and siddha have been investigated as multi-organ protective drugs. Recent day research mainly focused of plant based drug need to cure various aliment instead of allopathic medicine which causes sever side effects. In the present study mercury chloride was chosen to induce hepato-renal damage for evaluation of *Lannea Coromandelica*. In order to evaluate protective effect of *Lannea Coromandelica* on cadmium chloride induced oxidative damage in rats.

MATERIALS AND METHODS

Animals:

Male albino rats of Wistar strain approximately weighing 100-125g were used in this study. They were healthy animals purchased from the Indian Institute of Science, Bangalore. The animals were housed in spacious polypropylene cages bedded with rice husk. The animal room was well ventilated and maintained under standard experimental conditions (Temperature 27 ± 2º C and 12 hour light/dark cycle) throughout the experimental period. All the animals were fed with standard pellet diet and water were provided *ad libitum*. They were acclimatized to the environment for one week prior to experimental use. The animal feed composition is crude protein (22.3%), crude oil (4.01%), crude fiber (4.02%), Ash (8.02%) and sand silica (1.02%).

Chemicals:

Thiobarbituric acid (TBA), 2,4, Dinitophenylhydrazine (DTNPH), reduced glutathione and cadmium chloride were purchased for sigma chemical company, Mumbai All other chemicals and reagents used in this study was of analytical grade with high purity and were obtained from Glaxo laboratories and Sisco Research laboratories, Mumbai, India.

Plant material and preparation of extract:

The leaf of *Lannea Coromandelica* was collected from Thanjavur, Tamil Nadu. The collected leaf of *Lannea Coromandelica* were cut into small pieces and shade dried at room temperature. The leaf of *Lannea Coromandelica* was soaked with ethanol (50%) for 48 hours. A semi solid extract was obtained after complete elimination of alcohol under reduced pressure. The extract was stored in refrigerator until used. The leaf of *Lannea Coromandelica* extract was dissolved in distilled water just before oral administration.

Experimental design:

Body weight of animals was recorded and they were divided into three groups of six animals each as follows. Group I: Normal animal received with standard fed and water to allowed *ad libitum.* Group II: Animals received intraperitonial injection of cadmium chloride (2.5mg/kg body weight) daily for three consecutive days. Group III: Treatment group received cadmium chloride as group II. After 24 hours of last administration of cadmium chloride, treatment was started at a dose of 500mg/kg body weight of leaves of *Lannea Coromandelica* for five days.

Collection of blood and preparation of serum sample:

At the end of the experimental period, the animals were anaesthetized using chloroform vapour prior to dissection. Blood was collected by cardiac puncture into serum separator tubes. The blood was allowed to clot by standing at room temperature for 30 minutes and then refrigerated for another 30 minute. The resultant clear part was centrifuged at 3000rpm for 10minutes, and then the serum (supernatant) was isolated and stored at refrigerated until required for analysis.

BIOCHEMICAL ESTIMATIONS

Reduced glutathione was estimated by method of Moron *et al.,* (1979). Malondialdehyde was estimated by the thiobarbituric acid assay method of Beuge and Aust (1978). Urea was estimated by the method of Natelson (1957). The serum GOT and GPT was estimated by the method of Reitman and Frankel (1957). Protein was estimated by the method of Lowry *et al.,*. (1951).

Statistical analysis:

The results were presented as mean ± SD. Data was statistically analyzed using student "t" test. P.values set as lower than 0.001,.01,.05 were considered as statistically significant.

RESULTS

The present study was carried out to evaluate hepatoreno protective effect of *Lannea Coromandelica* on cadmium chloride induced oxidative damage in rats. The observations made on different groups of experimental and control animals were compared as follows.

In the present study was carried out on the plant sample revealed the presence of medicinally active constituents. The phytochemical characters of the *Lannea Coromandelica* leaves investigated and summarized in **Table-1.** The phytochemical screening *Lannea Coromandelica* leaves showed that the presence of flavonoids, phenolics, steroids, tannin, saponins, glycosides, terpenoids, Steroid and alkaloids, pholobatannins amino acids were absent

Table 1 Qualitative Analysis of *Lannea Coromandelica*

Phytochemicals	Results
Tannin	+
Phlobatannins	–
Saponin	+
Flavonoids	+
Steroids	+
Terpenoids	+
Triterpenoids	+
Alkaloids	–
Carbohydrate	+
Amino acid	–
Anthroquinone	–
Polyphenol	–
Glycoside	+

(+) Presence (-) Absence

Table II represents the levels of MDA in serum of normal and experimental rats. Group II cadmium chloride intoxicated rats showed a significant increased in the level of MDA in serum when compared to Group I rats. Group III cadmium chloride intoxicated rats treated with *Lannea Coromandelica* significantly decreased in the level of MDA when compared to group II.

Table II Effect of *Lannea Coromandelica* on MDA in serum and liver of experimental rats

Parameters	Group I	Group II	Group III
MDA	1.96±0.742	6.96±0.98*	2.49±0.849**

Values were expressed as mean ± SD for six rats in each group.
* Significantly different from Group I ($P < 0.01$)
** Significantly different from Group II ($P < 0.01$)

Table III represents the levels of GSH in serum and liver of normal and experimental rats. Group II cadmium chloride intoxicated rats showed a significant decreased in the level of GSH in serum when compared to Group I rats. Group III cadmium chloride intoxicated rats treated with *Lannea Coromandelica* significantly increased in the level of GSH as compared to groupII.

Table III Effect of *Lannea Coromandelica* on GSH in serum and liver of experimental rats

Parameters	Group I	Group II	Group III
GSH	5.96±0.647	2.55±0.956*	6.06±0.386**

Values were expressed as mean ± SD for six rats in each group.
* Significantly different from Group I ($P < 0.001$)
** Significantly different from Group II ($P < 0.01$)

Table IV represents the activity of SGOT and SGPT in serum of normal and experimental rats. Group II cadmium chloride intoxicated rats showed a significant increased in the activity of SGOT when compared to Group I rats. Group III cadmium chloride intoxicated rats treated with *Lannea Coromandelica* significantly decreased in the activity of SGOT when compared to group II. Group II cadmium chloride intoxicated rats showed a significant increased in the activity of SGPT when compared to Group I rats. Group III cadmium chloride intoxicated rats treated with *Lannea Coromandelica* significantly decreased in the activity of SGPT as compared to groupII.

Table IV Effect of *Lannea Coromandelica* on SGOT and SGPT activities in serum of experimental rats

Parameters	Group I	Group II	Group III
SGOT	30.49±16.302	95.86±31.176*	30.49±30.808**
SGPT	25.2±10.476	71.86±21.372*	30.8±10.476**

Values were expressed as mean ± SD for six rats in each group.
* Significantly different from Group I ($P < 0.05$)
** Significantly different from Group I ($P < 0.01$)

Table V represents the levels of protein in serum of normal and experimental rats.Group II cadmium chloride intoxicated rats showed a significant increased in the level

of protein when compared to Group I rats. Group III cadmium chloride intoxicated rats treated with *Lannea Coromandelica* significantly decreased in the level of protein when compared to group II.

Table V Effect of *Lannea Coromandelica* on protein in serum of experimental rats

Parameters	Group I	Group II	Group III
Protein	5.86±0.997	10.15±3.555*	5.99±1.487**

Values were expressed as mean ± SD for six rats in each group.
* Significantly different from Group I ($P < 0.001$)
** Significantly different from Group II ($P < 0.001$)

Table V represents the levels of haemoglobin in blood and urea in serum of normal and experimental rats. Group II cadmium chloride intoxicated rats showed a significant decreased in the level of potassium when compared to Group I rats. Group III cadmium chloride intoxicated rats treated with *Lannea Coromandelica* significantly increased in the level of potassium when compared to group II. Group II cadmium chloride intoxicated rats showed a significant increased in the level of creatinine, urea and sodium when compared to Group I rats. Group III cadmium chloride intoxicated rats treated with *Lannea Coromandelica* significantly decreased in the level of creatinine, urea and sodium as compared to groupII.

Table V Effect of *Lannea Coromandelica* on Creatinine**, Urea,** Potasium**,** Sodium **and Cholesterol In Experimental Rats**

Parameters	Group I	Group II	Group III
Creatinine	0.94±0.134	1.45±0.178*	0.92±0.122**
Urea	18.56±4.04	27.85±4.925*	16.42±2.675**
Potasium	7.05±0.793	5.99±0.885*	7.07±0.746**
Sodium	139.28±40.236	253.56±62.717*	117.85±40.089**

Values were expressed as mean ± SD for six rats in each group.
* Significantly different from Group I ($P < 0.01$)
** Significantly different from Group II ($P < 0.05$)

DISCUSSION

Over 40 elements in the environment are classified as metals. Many, such as the alkaline earth group and some trace elements, are essential for life: other has great potential for toxicity. Macronutrients such as calcium, magnesium, iron, potassium and sodium are particularly important in sustaining life but may become toxic in excessive concentrations. Trace elements such as chromium, cobalt, copper, manganese, nickel, selenium and zinc are structurally part of important molecules and may serve as cofactors of enzymes in metabolic process. Excessive concentrations of these elements are also toxic harmful. Some elements such as lead, cadmium and mercury have harmful effects on biological tissues at any concentration (Satake *et al.,* 1997).

Cadmium (Cd) is a heavy metal of great environmental and human health concern. It is implicated in many industrial uses, such as in electroplating, paints, dyestuffs and mining industry and it is now a major threat to mans environment. In animals, cadmium was shown to be toxic to all tissues. It has been reported that Cd causes morphological and functional damage in hepatic (Horiguchi *et al.,* 2000) and renal tissues (El-Sharaky *et al.,* 2007), testicular necrosis (Lorico *et al.,* 2002), morphological and biochemical changes in lungs and gastrointestinal tract (Weisman, 1998).

Cadmium (Cd) induces a broad spectrum of toxicological effects and biochemical dysfunctions constituting a serious hazard to health. The observed increase in MDA level of serum in rats treated with Cd may indicate oxidative stress, which affects liver organelles. Cd participates in oxidation reactions associated with generation of some reactive species, which interact with membrane lipids of liver cells to produce lipid peroxides (El-Demerdash *et al.,* 2004; Ognjanovic *et al.,* 2003). Reactive oxygen species such as superoxide radical and hydroxyl radical provoke sever changes at cellular level leading to cell death because of their extreme reactivity. They attack essential cell constituents such as proteins, lipids and nucleic acids. Also, they induce peroxidation of fatty acids (Stadtman and Berlett, 2000). Lipid peroxides that accumulate due to peroxidation of lipids are known to be harmful to cells and tissues (Linden *et al.,* 2008). It was reported that subcutaneous administration of Cd to rats resulted in pronounced increase of lipid peroxidation in the liver accompanied by a depletion of hepatic GSH (Eybe *et al.,* 2006). Administration of *Lannea Coromandelica* significantly decreased (Table – I) the levels of MDA in Cd intoxicated rats as compared to control rats. This view supported by Bashandy and Alhazza (2008) studies.

The metal GSH conjugation process is desirable in that it results in the excretion of the toxic metal into the bile. As a result of the binding of Cd to glutathione and the subsequent elimination of intracellular glutathione, levels of reduced glutathione are lowered. In the present investigation it was that Cd intoxication significantly depletes the GSH content in serum and liver and thus reducing the antioxidant potential and accelerating the lipid peroxidation, resulting in hepatocytes damage (Eybe *et al.,* 2006). Depletion of GSH results in enhanced lipid peroxidation and excessive lipid peroxidation can cause increased GSH consumption as observed in the present study. Supplementation of *Lannea Coromandelica* significantly increased the levels of GSH in Cd intoxicated rats as compared to control rats, suggest that *Lannea Coromandelica* improve the GSH content.

All forms of Cd cause toxic effects in a number of tissues and organs, depending on the chemical form of Cd. Cd is known to have toxic effects on several biologic systems with the hematological (Ersteniuk *et al.,* 2004) and the liver, kidneys, and reproductive systems as the main

targets (Horiguchi *et al.,* 2000; El-Sharaky *et al.,* 2007). The functional activity of these organs may be impaired by Cd toxicity. In the present study, assess the various functional tests associated with kidney and liver.

Liver has shown to be target organ of acute Cd toxicity. The relation between the hepatic oxidative damage and elevation of the relevant serum enzymes in Cd toxicity is well documented (Vicente-Sanchez *et al.,* 2008). The observed increase in activities of serum protein, ALT and AST is likely due to lipid peroxidation of biomembranes, which causes leakage of cellular components (Iguchi *et al*., 1993). Exposure of hepatocytes to Cd stimulates cellular production of H_2O_2, which affect permeability barrier of plasma membrane (Koizumi *et al*.,., 1996). It seems that the increase in the liver enzymes of the present study may be due to accumulation of Cd in hepatic tissue that enhances formation of lipid peroxidation. Administration of *Lannea Coromandelica* to Cd intoxicated rats restored the level of protein, ALT and AST offering the maximum hepatoprotection with respect to different liver marker enzymes. This confirms the liver protective activity of *Lannea Coromandelica*. Further, *Lannea Coromandelica* has significantly increased the level of liver protein and serum ceruloplasmin, which indicates hepatoprotective activity. Stimulation of protein synthesis accelerates the regeneration process and the production of liver cells.

In the kidneys, the toxicity of Cd is related to its accumulation in the epithelial cells from the proximal tubules and with it's binding to intracellular sulfhydryl, carboxyl and phosphoryl groups (El-Sharaky *et al.,* 2007). Urea is an end product of protein catabolism. It is freely filtered by the glomerulus, passively reabsorbed in the both the proximal and distal nephron and excreted in high concentration in urine. The excretion of urea was recognized as an estimate of kidney function. The serum urea levels are used as an index of kidney function. In the present study, significantly increased (Table - V) level of urea in Cd intoxicated rats were observed as compared with control rats. The principal target of Cd toxicity is the *pars recta* (segment S3) from the proximal tubule, particularly the portion at the junction of the cortex and the outer medulla, which can reduce the glomerular filtration rate. Kidney is largely account for with Cd toxic actions. Supplementation of *Lannea Coromandelica* restored the increased level of urea in Cd intoxicated rats, suggest that restored the kidney functions.

Lannea Coromandelica treatment proved to be effective in improving the extent of DNA synthesis, decreases lipid peroxidation, improves the antioxidants, liver and kidney functions. Thus, the above results confirm that supplementation of *Lannea Coromandelica* preserve the genetic materials induced by cadmium. The potential hepatoreno protective activity of *Lannea Coromandelica* may be due to the presence of radical scavenging property of phenolic groups present in it. It ca be concluded that use of *Lannea Coromandelica* has the capability to alleviate many of harmful effects of $CdCl_2$.

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