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# TEPHROSIA PURPUREA (LINN.) PERS: A FOLK MEDICINAL PLANT AMELIORATES CARBON TETRACHLORIDE INDUCED HEPATIC DAMAGE IN RATS

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## ABSTRACT

Herbal drugs are traditionally used in various parts of the world to cure different diseases. The present study has been conducted to evaluate the protective role of the ethanolic extract of the root of *Tephrosia purpurea*; an important Indian medicinal plant widely used in the preparation of ayurvedic formulations, on $CCl_4$ induced oxidative damage and resultant dysfunction in the liver of rats. The experiments were performed using five groups of animals. The experimental animals were administered with 30% $CCl_4$ in liquid paraffin (1ml/kg bw) for 10 days at 72 hr intervals and the fine crude plant root powder ethanolic extract (EETP) and Silymarin a standard drug, 25 mg/kg bw were fed to the $CCl_4$ treated animals. The effect of EETP and silymarin on Total protein, albumin, bilirubin, cholesterol and glycogen were measured. Further, the effects of the extract on hepatospecific enzymes such as, aspartate transaminase (AST), alanine transaminase (ALT), acid phosphatase (ACP), alkaline phosphatase (ALP), lactate dehydrogenase (LDH) and 5' nucleotidase (5'NT) were estimated. The EETP and Silymarin produced significant effect by decreasing the serum levels of bilirubin and cholesterol whereas Total protein, albumin, glycogen and hepatospecific enzymes were significantly increased. From these results, it was suggested that *Tephrosia purpurea* protects the liver against $CCl_4$ induced oxidative damage probably by increasing antioxidative defense activities.

## KEYWORDS

*Tephrosia purpurea*, hepatoprotective, biochemical study, carbon tetrachloride

## INTRODUCTION

The liver is the key organ regulating homeostasis in the body. It is involved with almost all the biochemical pathways related to growth, fight against disease, nutrient supply, energy provision and reproduction [1]. The liver is expected not only to perform physiological functions but also to protect against hazards of harmful drugs and chemicals. Inspite of tremendous scientific advancement in the field of hepatology in recent years, liver problems are on the rise [2].

Hepatotoxicity is one of the very common ailment resulting in to a serious debilities ranging from severe metabolic disorders to even mortality. Hepatotoxicity in most cases is due to free

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radicals. Free radicals generated by the metabolism of toxicants initiate the toxicity cascade [3]. In view of severe undesirable side effects of synthetic agents, there is growing focus to follow systematic research methodology and to evaluate scientific basis for the traditional herbal medicines that are claimed to possess hepatoprotective activity. Traditional medicine is widespread and plants still presents a large source of natural antioxidants that might serve as leads for the development of novel drugs. Several anti-inflammatory, digestive, anti-necrotic, neuroprotective, and hepatoprotective drugs have recently been shown to have an antioxidant and/or anti-radical scavenging mechanism as part of their activity [4]. Conventional medicine is now pursuing the use of natural products such as herbs to provide the support that the liver needs on a daily basis [5]. Herbal drugs are frequently considered to be less toxic and free from side effects than synthetic drugs.

*Tephrosia purpurea* (Linn) Pers, (Leguminasae) is a polymorphic, much branched sub erect perennial herb popularly known as “Sarapunkha” in Sanskrit, “Purple Tephrosia” in English and “Kaattukolingi”in Tamil. It is a highly branched, sub – erect perennial herb [6]. Its aerial parts and roots are used in bronchial asthma, hepatic ailments [7], cutaneous toxicities, pain and inflammation. Due to the wide spread use of this plant by the rural communities to treat several diseases the objective of the present study was framed to determine the effect of ethanolic root extract of *Tephrosia purpurea* on $CCl_4$ induced hepatotoxicity in rats.

## MATERIALS AND METHODS

### *Drugs and chemicals*

All the drugs and chemicals used were of analytical grade.

### *Plant material and Extraction:*

The plant *Tephrosia purpurea* belongs to the family Leguminasae was collected from Thirumurthy hills area, Udumalpet, Tirupur District, Tamilnadu, India and was authenticated by Dr.V.S.Ramachandran, Associate Professor, Department of Botany, Bharathiar University, Coimbatore, Tamilnadu, India. The roots of the plants were collected, shade dried and powdered to coarse size. They were extracted with ethanol in Soxhlet apparatus. The solvents were evaporated in a rotavapour at 40 – 50ºC, under reduced pressure. A dark semisolid material (EETP) obtained, was stored at - 4 ºC, until use.

### *Experimental Animals*

Studies were carried out using female Wistar albino rats (175-200g). They were obtained from Small Animals Breeding Centre of Kerala Agricultural University, Mannuthy, Thrissur. The animals were grouped and housed in polyacrylic cages with not more than six animals per cage and maintained under standard laboratory conditions with dark and light cycle. They were allowed free access to standard pellet diet and water *ad libitum.* The rats were acclimatized to the laboratory conditions for 10 days before the commencement of the experiment. All procedures were reviewed and approved by Institutional Animal Ethical Committee (IAEC).

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***Hepatoprotective activity***

***$CCl_4$ induced liver damage in rats***

Healthy female Wistar albino rats weighing in the range of 175 – 200 g were divided into 5 groups each containing six animals. Group I: normal control rats. Group II: rats induced with 30% $CCl_4$ in liquid paraffin (1ml/kg bw, i.p) for 10 days at 72 hr intervals. Group III: rats induced with 30% $CCl_4$ in liquid paraffin (1ml/kg bw, i.p) and received 500 mg/kg bw of EETP once in a day. Group IV: rats induced with 30% $CCl_4$ in liquid paraffin (1ml/kg bw, i.p) and received standard drug Silymarin (25 mg/kg bw) once in a day. Group V: received 500 mg/kg bw of EETP once in a day. Treatment duration was 10 days and the dose of $CCl_4$ was administered every 72 hr [8]. Animals were sacrificed 24 hr after the last injection. Blood was collected, allowed to clot and serum was separated. The liver was dissected out and used for various biochemical studies.

***Biochemical Studies:***

Blood was collected on cervical decapitation and was allowed to clot for 45 mins at room temperature. Serum was separated by centrifugation at 2500 rpm for 15 mins and utilized for various biochemical parameters namely total protein & albumin [9], bilirubin [10] and cholesterol [11].

After collection of blood samples rats were sacrificed and their livers were excised, rinsed in ice cold saline followed by 0.1M phosphate buffer (pH 7.4), blotted dry and weighed. A 10% w/v of homogenate was prepared in 0.1M phosphate buffer and processed for the various estimations like aspartate transaminase (AST) [12], alanine transaminase (ALT) [12], acid phosphatase (ACP) [13], alkaline phosphatase (ALP) [14], lactate dehydrogenase (LDH) [15] and 5' nucleotidase (5'NT) [16].

**Statistical Analysis**

The results obtained were reported as mean ± SD. One Way Analysis Of Variance (ANOVA) was performed to analyze statistical significance of the data using Agres statistical package.

## RESULTS AND DISCUSSION

Liver is an important organ actively involved in metabolic functions and is a frequent target of number of toxicants. One of the major functions of the liver is detoxification of xenobiotics and toxin [17]. Because liver performs many vital functions in the human body, damage of liver causes unbearable problems [18]. The involvement of free radicals in the pathogenesis of liver injury has been investigated for many years by using acute poisoning with $CCl_4$ [19]. It is well documented that $CCl_4$ is biotransformed under the action of cytochrome P450 in the microsomal compartment of liver to trichloromethyl radical which readily reacts with molecular oxygen to form trichloromethyl peroxy radical [20]. The hepatotoxic effects of $CCl_4$ are largely due to its active metabolite trichloromethyl radical and trichloromethyl peroxy radical [21]. Both the radicals can bind covalently to the macromolecules and induce peroxidative degradation of the membrane lipids of endoplasmic

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reticulum rich in polyunsaturated fattyacids [22]. This leads to the formation of lipid peroxides followed by various changes in biochemical parameters.

The levels of Total protein and albumin in both serum and liver of control and experimental animals are illustrated in Table 1. It is evident from the table that there was a significant decline in protein and albumin in toxicity induced group than control. On treating with EETP and silymarin the serum and liver levels were found to be significantly increased than Group II. There was no significant difference between the EETP treated group and the control group.

**Table 1.**
***Effect of EETP on Total protein and albumin levels in Control and Experimental rats***

| Groups | Serum (g / dl) | | Liver (mg / gm tissue) | |
|---|---|---|---|---|
| | Total Protein | Albumin | Total Protein | Albumin |
| Group I | 5.41 ± 0.14 | 3.45 ± 0.71 | 7.35 ± 0.11 | 4.17 ± 0.09 |
| Group II | 3.14 ± 0.08a** | 1.28 ± 0.68a** | 4.59 ± 0.12a** | 2.18 ± 0.11a** |
| Group III | 4.70 ± 0.10b** | 2.76 ± 0.66b** | 6.77 ± 0.09b** | 3.64 ± 0.11b** |
| Group IV | 4.74 ± 0.11c$^{ns}$ | 3.05 ± 1.03c** | 6.84 ± 0.08c$^{ns}$ | 3.87 ± 0.08c$^{ns}$ |
| Group V | 5.59 ± 0.09d$^{ns}$ | 3.27 ± 0.95d$^{ns}$ | 7.56 ± 0.12d$^{ns}$ | 4.04 ± 0.09d$^{ns}$ |

Values are mean ± SD of six samples
**Groups comparison:** a – Group I vs Group II; b – Group II vs Group III;
c – Group III vs Group IV; d – Group V vs Group I
**Statistical significance:** ** - $p<0.01$ ns – not significant

Formation of lipid peroxides by $CCl_4$ intoxication depresses the protein synthesis. The decrease in total protein observed in $CCl_4$ treated rats may be associated with the decrease in the number of hepatocytes which in turn, may result in to the decreased hepatic capacity to synthesize protein [23]. The lowered level of total proteins recorded in the serum as well as in liver of $CCl_4$ treated rats suggests the severity of hepatopathy [24]. The reduction is attributed to the initial damage produced and localized in the loss of $p_{450}$ leading to its functional failure with a decrease in protein synthesis and accumulation of triglycerides leading to fatty liver [25]. The site-specific oxidative damage in some susceptible aminoacids of proteins is now regarded as the major cause metabolic dysfunction during pathogenesis [26]. Restoration of the level of total protein and albumin after the administration of EETP may be due to the presence of flavonoids and polyphenols in the plant.

Table 2, show the levels of bilirubin and cholesterol in serum of control and experimental animals. The concentration of bilirubin and cholesterol were significantly increased in toxicity

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induced group than control. On treating with EETP and silymarin the levels were found to be significantly ($p<0.01$) decreased in group III and group IV respectively. There was no significant difference between plant treated group and the control group.

**Table 2.**
***Effect of EETP on serum Bilirubin and Cholesterol in Control and Experimental animals***

| Groups | Bilirubin (mg/dl) | Cholesterol (mg/dl) |
|---|---|---|
| Group I | 0.63 ± 0.07 | 89.44 ± 5.04 |
| Group II | 1.25 ± 0.09a** | 143.47 ± 7.76a** |
| Group III | 0.88 ± 0.06b** | 105.09 ± 6.46b** |
| Group IV | 0.83 ± 0.09c$^{ns}$ | 101.98 ± 5.69c$^{ns}$ |
| Group V | 0.64 ± 0.07d$^{ns}$ | 93.27 ± 5.77d$^{ns}$ |

Values are mean ± SD of six samples
Groups comparison: a – Group I vs Group II; b – Group II vs Group III;
c – Group III vs Group IV; d – Group V vs Group I
Statistical significance: ** - $p<0.01$ ns – not significant

Bilirubin is the conventional indicator of liver diseases [27]. Hyperbilirubinemia is a very sensitive test to substantiate the functional integrity of the liver and severity of necrosis which increases the binding, conjugating and excretory capacity of hepatocytes that is proportional to the erythrocyte degeneration rate [28].

Depletion of the elevated bilirubin level in the serum of rats treated with EETP, suggests the possibility of the plant being able to stabilize biliary dysfunction and also an early improvement in the secretory mechanism of the hepatic cell. Decreased bilirubin level after the administration of EETP could be correlated with an earlier study which reported that the ethanolic extract of *Hibiscus hispidissmus* Griffith offered protection against paracetamol and $CCl_4$ induced hepatotoxicity [29].

Inhibition of bile acids synthesis from cholesterol in liver, leading to increase in cholesterol levels was also resulted due to $CCl_4$ intoxication. Suppression of cholesterol level by the extract suggests the bile acids synthesis inhibition was reversed. This may be due to the presence of flavonoids and polyphenols in the plant extract.

Fig.1 shows the effect of EETP on glycogen in liver of control and experimental animals. Induction of $CCl_4$ induced a marked decrease in the level of glycogen significantly ($p<0.01$) as compared to the control group. Where as treatment with EETP showed significant ($p<0.01$)

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increased level against $CCl_4$ intoxicated rats. Treatment with silymarin also significantly ($p<0.01$) increased the level than $CCl_4$ treated group. There is no significant difference between the plant treated group and the control group.

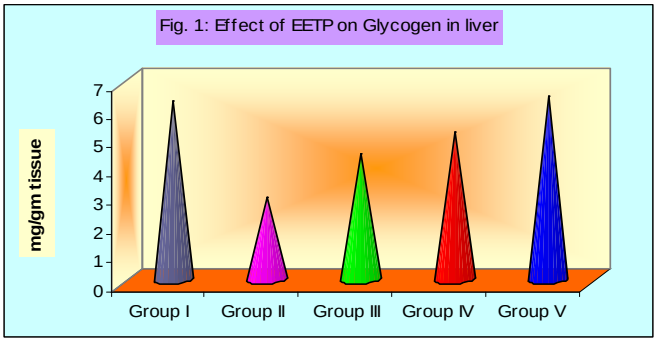


**Fig. 1**

***Effect of Glycogen in liver of Control and Experimental animals***

A reduction in the liver glycogen (Fig.1) observed in $CCl_4$ treated animals may be associated with the decrease in the number of hepatocytes which in turn may result into decreased hepatic capacity to synthesise glycogen [30]. Supplementation with EETP significantly prevented the glycogen depletion indicating the membrane stabilizing activity. This reveals that *Tephrosia purpurea* helped to resist the damage caused by $CCl_4$ and could be attributable through prevention of glycogenolysis and promotion of glucoronidation effect.

The hepatic marker enzymes in liver has been depicted in Fig. 2 & 3. A significant ($p<0.01$) decrease in the activity of the enzymes AST, ALT, ACP, ALP, LDH and 5'NT in liver were seen in the group II, $CCl_4$ intoxicated animals. These enzymes were increased significantly ($p<0.01$) to near normal levels in group III & IV, animals treated with EETP and Silymarin respectively. There is no significant difference between the plant treated group and the control group.

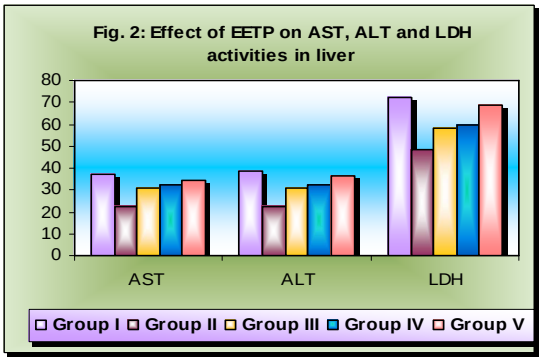


**Fig.2**

**Effect of EETP on AST, ALT and LDH activities in liver of Control and Experimental animals**

Units:

AST, ALT, LDH: nmoles of phosphate liberated / min / mg protein

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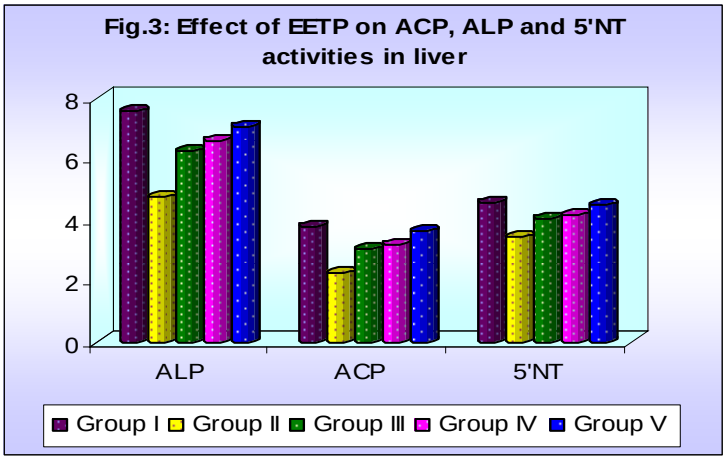


**Fig.3**
***Effect of EETP on ACP, ALP and 5'NT activities in liver of Control and Experimental animals***

Units:

ALP, ACP - nmoles of phenol liberated / min / mg protein
5'NT - nmoles of phosphorus liberated / min / mg protein

Transaminases activity is closely related to the liver function in liver disease, large quantities of transaminase usually enter in to the blood compartment [31]. It is reported that toxic damage to liver results in extreme hypertransaminasemia [32]. $CCl_4$ induced fall of AST and ALT in liver associated with rise in plasma suggests the extent of liver damage and release of these enzymes from the damaged liver cells and disruption of cellular integrity. The decrease in the liver AST and ALT supports the hypothesis of hepatocellular necrosis. In group III and IV, there is a significant increase in the levels compared to $CCl_4$ intoxicated animals. This increase may be due to the presence of flavonoids in the plant and the standard drug. This indicates the beneficial effect of *Tephrosia purpurea* as protective agent against $CCl_4$ induced hepatotoxicity.

The release of LDH reflects a non-specific alteration in the plasmamembrane integrity and /or permeability as a response to $CCl_4$. Administration of EETP has altered the membrane integrity and thus indicates the effective nature of the plant.

Visen *et al.*, (1996) showed decreased hepatic AST, ALT and LDH levels in ethanol induced toxicity of rat hepatocytes and its increment by treatment with picroliv isolated from *Picrrorhiza kurroa* [33].

Due to liver injury there is a disturbance in the transport function of the hepatocytes resulting in leakage due to changed permeability of membrane [34]. This results in the decreased level of enzymes in the hepatic cells and raised levels in serum.

ACP is regarded as a key lysosomal enzyme involved in autolytic degradation of tissues. It is used to monitor cell death and lysis. ALP on the

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other hand is related to the functioning of hepatocytes. $CCl_4$ induced fall of ACP and ALP levels in liver suggest some alteration of lysosomal enzyme activities in hepatic tissues.

In liver injury, damaged to lysosomal membrane leads to liberation of the degradative enzyme followed by cell destruction. Significant decreases in hepatic lysosomal enzyme activities are reported at the later stage of the liver injury when necrosis is well established [35]. The decreased activity of ACP and ALP in liver of $CCl_4$ treated rats could be due to damage to the cell membrane of tissues, where these enzymes are firmly attached to cell membrane and the damage releases these enzymes from the membrane joining the biliary canalicules and sinusoidal border of parenchymal cells. The recoupment of ACP and ALP to near normal levels in EETP and silymarin treated rats is observed. Supplementation of EETP rectifies the lysosomal membrane damage indicating the protective nature of the plant.

There are reports indicating elevation in the activities of depleted hepatic ACP and ALP on aspirin toxicity by the administration of ascorbic acid [36].

In liver injury and its protection by hepatoprotectives, several enzymes of liver related to subcellular fractions such as plasmamembrane 5' nucleotidase is affected [37].

5'NT is a plasmamembrane marker enzyme. This enzyme is localized in the cytoplasmic membrane of the cell in which it occurs. Decreased activity of 5' nucleotidase in $CCl_4$ induced liver damage was found to be elevated by the administration *Tephrosia purpurea*, proves its hepatoprotective action.

In conclusion, EETP afforded protection against $CCl_4$ induced liver damage. The protection against liver damage by EETP was found comparable to silymarin. Possible mechanism that may be responsible for the protection against $CCl_4$ induced liver damage by EETP may be it could act as free radical scavenger intercepting those radicals involved in $CCl_4$ metabolism by microsomal enzymes. This might be due to the presence of flavonoids and polypenols. Antioxidant property is claimed to be one of the mechanism of Hepatoprotective drugs. Further flavonoids and polypenols have been suggested to act as antioxidants by free radical scavenging. Thus the Hepatoprotective activity of EETP may be attributed to the presence of flavonoids and polypenols.

## REFERENCES


1. Ward FM, Daly MJ, Hepatic disease. *In Clinical Pharmacy and Therapeutics*. Walker R, Edward C, Eds, Churchill Livingston, Newyork, 1999, 195-212.
2. Pangs, Xinx, Stpierre MV, Determinants of metabolic disposition. Ann Rev Pharmacol Toxicol, 32: 625-626, (1992).
3. Zimmerman HJ, Kendler J, Libber S, Free radical activity its importance and role in disease. Biochem Pharmacol, 23: 2187-2189 (1974).
4. Linn CC, Huang PC, Antioxidant and Hepatoprotective effects of *Acanthopanax senticosus*. Phytother. Res, 14: 489-494, (2002).
5. Sherlock S, Dodey J, Diseases of liver and biliary system. 11th Edition. Oxford: Blackwell Scientific Publications. 2002, 332-356.
6. Kritikar KR, Basu BD: Indian Medicinal Plants. India: Lalif Mohan Basu, vol 1, 249 (1956).
7. Upadhyay YN, Shukla KP, Shankaran PS, Pathak SN, J. Med. Sci. Banaras Hindu University, 5: 97, (1964).
8. Manoj B, Aqueed K, Protective effect of *Lawsonia alba* Lam., against $CCl_4$ induced

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hepatic damage in albino rats. Ind J Expt Biol, 41: 85-87, (2000).

9. Wolfson. Estimation of albumin – globulin ratio by Biuret method. In: *Harold. Practical Clinical biochemistry*, 4th edition, CBS, Publishers, 236-247.
10. Malloy HT, Evelyn KA, The determination of bilirubin with the photoelectric colorimeter. J. Biol. Chem, 119: 481, (1937).
11. Parkeh AC, Jung DH, Cholesterol determination with ferric chloride uranium acetate and sulphuric acid ferrous sulphate reagents. Anal. Chem, 42: 1423-1427, (1970).
12. Reitman S, Frankel S, A Colorimetric method for the determination of serum glutamate oxaloacetate and serum glutamate pyruvate transaminases. Am. J. Clin. Pathol, 28: 56-63, (1957).
13. King J, The hydrolases – acid and alkaline phosphatases, In: *Practical Clinical Enzymology* (Ed. Van, D.) London, Nostrond Company Limited. 191, (1965 a).
14. King EJ, Armstrong AR, Determination of serum and bile phosphatases activity. Canad. Med. Ass. J, 131: 376, (1934).
15. King J, The dehydrogenase or oxidoreductase – lactate dehydrogenase, In: *Practical Clinical Enzymology* (Ed. Van, D.) London, Nostrond Company Limited, 83, (1965 b).
16. Campbell DM, Determination of 5'Nucleotidase in serum. Biochem. J. 84: 34, (1962).
17. Mitra SK, Venkataranganna MV, Sundaram R, Gopumadhavan S, Protective effect of HD-03 a herbal formulation, against various hepatotoxic agents in rats. J. Ethanopharmacol, 63:181-186, (1998).
18. Chattopadhyay RR, Possible mechanism of hepatoprotective activity of *Azadirachta indica* leaf extract: part II. J. Ethnopharmacol, 89: 217-219, (2003).
19. Recknagel RU, Glender EA Jr, Dolak JA, Walker RL, Mechanisms of carbon tetrachloride toxicity. Pharmacol. Ther, 43: 139-54, (1989).
20. Raucy JL, Kraner JC, Lasker. J, Bioactivation of halogenated hydrocarbons by cytochrome P450 E1. Crit. Rev. Toxicol, 23: 1-20, (1993).
21. Srivastava SP, Chen NO, Holtzman JL, The in vitro NADPH – dependent inhibition by carbon tetrachloride of the ATP- dependent calcium uptake of hepatic microsomes from male rats. Studies on the mechanism of inactivation of the hepatic microsomal calcium pump by the $CCl_3$ radical. J. Biolchem, 265: 8392-9, (1990).
22. Recnagel R, Carbon tetrachloride hepatotoxicity. Pharmacological Review, 19: 145-196, (1967).
23. Shahjahan M, Sabitha KE, Mallika J, Shymala Devi CS, Effect of *Solanum trilobatum* against carbon tetrachloride induced hepatic damage in albino rats. Indian J Med Res, 120: 194-198, (2004).
24. Aniya Y, Koyama T, Miyagi C, Miyahira M, Inomata C, Kinoshita S, Ichiba T, Free radical Scavenging and hepatoprotective actions of the medicinal herb, *Crassocephalum crepidioides* from the Okinawa Islands. Biol. Pharm. Bull, 28: 19-23 (2005).
25. Recnagel RO, Carbon tetrachloride hepatotoxicity. Pharmacological Reviews, 19(2): 145-208, (1967).
26. Bandyopadhyay U, Dipak D, Ranjit BK, Reactive oxygen species: Oxidative damage and pathogenesis. Curr. Sci, 5: 658 (1999).
27. Girish S, Achliya, Sudhir, Wadodkar G, Avinash, Dorle K, Evaluation of hepatoprotective effect of Amalkadi Ghrita

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against carbon tetrachloride induced hepatic damage in rats. J. Ethnopharmacol, 90: 229-232, (2004).

28. Rajesh MG, Latha MS, Preliminary evaluation of the antihepatotoxic activity of Kamilari, a polyherbal formulation. J Ethanopharmacol., 91: 99-104, (2004).
29. Krisnakumar NM, Latha PG, Suja SR, Shine VJ, Shyamal S, Anuja GI, Pradeep S, Shikha P, Somasekharan PK, Rajasekharan S, Hepatoprotecive effect of *Hibiscus hispidissimus* Griffith, ethanolic extract in paracetamol and carbon tetrachloride induced hepatotoxicity in wistar rats. Indian Journal of Experimental Biology. 46: 653-659, (2008).
30. Ajay K, Havagiray C, Sujatha K, Neelam M, Hepatoprotective activity of *Rauwolfia serpentina* Rhizome in paracetamol intoxicated rats. Journal of Pharmacology and Toxicology, 1(1): 82-88, (2006).
31. De Ritis F, Coltorti M, Giusti G, Serum and Liver transaminase activities. In experimental hepatitis in mice. Science, 124: 32, (1956).
32. Wroblenski F, La due JS, Serum glutamic pyruvate transaminase activity in hepatic disease preliminary report. Ann Int. Med, 45: 8, (1956).
33. Visen PKS, Saraswat B, Patnaik GK, Agarwal DP, Dawan BN, Protective activity of piroliv isolated from *Picrrorhiza kurroa* against ethanol toxicity in isolated rat hepatocytes. In. J. of Pha, 28: 98-101, (1996).
34. Zimmerman HJ, Seef LB, Enzymes in hepatic disease In: *wodely L Diagnostic Enzymology*, Lea and Febiger, Philadelphia, 1970, 1-38.
35. Saxena AK, Singh B, Anand KK, Hepatoprotectve effects of *Eclipta alba* on subcellular levels in rats. J Ethanopharmacol, 40: 155-161, (1993).
36. Das KK, Dasgupta S, Influence of ascorbic acid on acid and alkaline phosphatase activities in some metabolically active tissues of aspirin treated rats. Ind. J. Physiol. Pharmaco, 41(4): 421-423, (1997).
37. Dwivedi Y, Rastogi R, Chander R, Sharma SK, Kapoor NK, Garg NK, and Dhawan BN, Hepatoprotective activity of picroliv against carbon tetrachloride induced damage in rats. Ind J. Med. Res, 92: 195-200, (1990).