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Hepatoprotective potentials of *Butea Monosperma* (Lam) Taub leaves extract against carbon tetrachloride induced hepatotoxicity by non invasive method

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ABSTRACT

Carbon tetrachloride (CCl_4) pharmacological tool to produce liver damage reduced the urinary excretion of ascorbic acid in rats. Silymarin and extract of Butea monosperma (Lam) Taub (shown to be hepatoprotective substances) prevented the CCl_4 induced reduction of ascorbic acid excretion in urine. The results indicate that silymarin, Aqueous and Methanolic Extracts of leaves of Butea monosperma (Lam) Taub reduced the hepatic damage caused by i.p. administration of CCl_4 as compared to control based on the urinary ascorbic acid levels.

Key words: Urinary ascorbic acid, carbon tetrachloride, *Butea monosperma*(Lam) Taub, *Silymarin*, hepatoprotection.

INTRODUCTION

The traditional systems of medicine together with homoeopathy and folklore medicine continue to play a significant role largely in the health care system of the population. *Butea monosperma*(Lam) Taub (Palas) belonging to the family leguminoceae grown wildly in many parts of India. The plant is regularly used by the rural and tribal people in curing various disorders [1].

The bark of the plant is an appetiser, lessens inflammation, dysmenorrhoea used in liver disorders, fractures, and gonorrhoea, topically in piles and hydrocele purifies the blood. Leaf is appetiser, very astringent, carminative, anthelmintic, aphrodisiac, tonic, lessen inflammation and lumbago, cures boils and piles. Gum is acrid, astringent, aphrodisiac, tonic to the liver, used in the diseases of the chest and lungs useful in syphilis. The flower is bitter, aphrodisiac, expectorant, and tonic, emmenagogue, diuretic, astringent, and good in inflammation, burning

urine and gonorrhoea. The fruit and seeds are bitter and oily, anthelmintic, useful in piles, eye diseases and inflammation. The lye is useful in enlargement of spleen [2].

The present paper demonstrates hepatoprotective potential of the plant because it prevented CCl_4 induced reduction of ascorbic acid excretion in urine as a consequence of hepatotoxicity. Various biochemical parameters such as levels of SGOT, SGPT and serum bilirubin are also used to determine the hepatic function [3]. These methods require invasive blood sampling procedure and are coupled with expensive analytical techniques. A non-invasive method avoiding withdrawal of blood and employing a simple analytical procedure is very much preferred for regular screening to determine hepatoprotective activity of substances. The present work was done to find whether *Butea monosperma* (Lam) Taub extracts, possess hepatoprotective action against hepatotoxicity in CCl_4 treated rats.

MATERIAL AND METHODS

Collection of plant material:
Leaves of Butea monosperma were collected in month Sept. - Oct.2009 from local area of sangli and were authenticated by a Dr. Wadmare Dept. of botany KWC College, Sangli. A voucher specimen (No: 16587) was deposited at KWC College, and leaves are dried under shade, then sifting and sieving was done.

Preparation of Aqueous and Methanolic extract:
The aqueous extract was prepared by a simple decoction procedure & methanolic extract was prepared by solvent methanol and extracted by Soxhlet Apparatus. The extracts were used for further experimentation.

Administration: Silybon Tablets (*Silymarin* Micro Labs Ltd.) were purchased from the market and used as they were for oral administration to rats as suspensions in 5% gum acacia. Aqueous and methanolic extracts of *B. Monosperma* (Lam) Taub were also administered to rats orally as suspensions.

Animals:
Male albino rats of Wistar strain having weight range 120–150 g were used for the experiment. The animals were obtained from animal house of Dept. of Pharmacology, A.B.C.P. Sangli. Approval for the study was obtained from Animal Ethical committee and animals were housed in groups of 5 in separate cages in standard laboratory conditions of temperature ($25^0C \pm 2^0C$) and humidity (55 ± 5%) with 12 h light/dark cycles and provided with standard laboratory diet and water *ad libitum.*

Experimental design:
Animals were grouped into four groups of 5 animals each. They were kept in metabolic cages for collection of urine. They were supplied with standard diet & water *ad libitum* one week before & during experimental period.
The following administration regimen was per day for 7 days used for the different groups:
Group A: Control (CCl_4 0.5 ml/kg *i.p*[4].)
Group B: Received Aqueous Extract of *B. Monosperma* (Lam) Taub (500 mg/kg *p.o.*) one hour prior to CCl_4 dose.
Group C: Received Methanolic Extract of *B. Monosperma* (Lam) Taub (500 mg/kg *p.o.*) one hour prior to CCl_4 dose.

Group D: Received Silybon (Silymarin, 100 mg/kg *p.o.*) one hour prior to CCl_4 dose.

Before starting the actual experimentation twenty four hour urine samples were collected separately for each group for 5 days in 5% oxalic acid solution and analysed for ascorbic acid and their average values were taken as controls. Then the rats of groups A through to D were treated as per above mentioned regimen. The collection of 24 hour urine samples was continued for further period of 7 days for all groups and the samples were analysed for ascorbic acid by the method of Schaffert and Kingsley [5]. Statistical analysis was done by one way ANOVA test followed by Dunnett's Test and P values less than 0.05 were considered as significant.

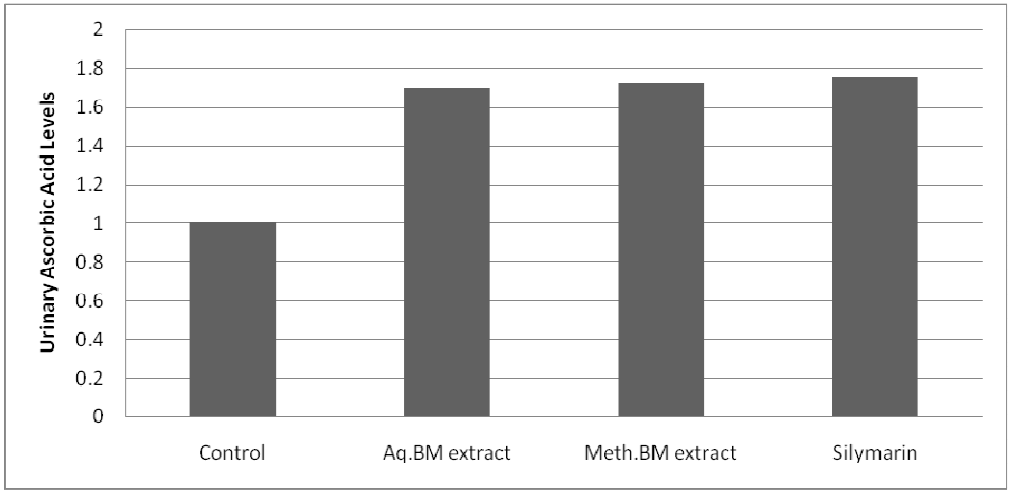
RESULTS

The normal urinary ascorbic acid levels for days 1 to 5 were taken without any treatment. The post treatment urinary excretion of ascorbic acid from days 6 to 12 was reduced in control rats whereas an initial dip and then restoration of urinary ascorbic acid levels was seen in rats treated with aqueous and methanolic extracts of *B. Monosperma* (Lam) Taub and those treated with Silybon (silymarin) was seen. This indicates the hepatoprotective potential of aqueous and methanolic extracts of *B. Monosperma* (Lam) Taub and Silybon (silymarin) is significant as compared to control as evident from the urinary ascorbic acid levels.

Table1. Urinary excretion of ascorbic acid (mg during 24 hrs)

Days	**Group**			
	A	**B**	**C**	**D**
1	2.07	1.29	1.59	1.80
2	1.41	1.56	1.80	1.77
3	1.74	1.86	2.01	1.98
4	1.56	1.68	1.26	1.56
5	1.63	1.48	1.49	1.61
Mean ± SEM	**1.722 ± 0.1133**	**1.574 ± 0.09558**	**1.63 ± 0.1287**	**1.744 ± 0.0761**
Treatment				
6	1.08	1.20	1.35	1.02
7	0.96	1.29	1.35	1.26
8	1.08	1.35	1.56	1.62
9	1.17	1.56	1.59	1.74
10	0.78	1.70	1.76	1.85
11	0.93	1.82	1.85	1.89
12	0.90	1.88	1.90	2.04
Mean ± SEM	**0.9857 ± .05004**	**1.5428 ± 1017**	**1.6228 ± 0.08468**	**1.6314 ± 0.1385**

Graph 1: Indicates urinary ascorbic acid levels in treated animals



DISCUSSION

Carbon tetrachloride is a pharmacological tool used to produce liver damage in animal models; its hepatotoxic action begins with changes in endoplasmic reticulum which results in loss of metabolic enzymes located in the intracellular structure [6-8]. Ascorbic acid is formed as a metabolite of glucose and galactose in rat liver microsomes *via* the glucuronic acid pathway and is excreted in urine. The enzyme UDP glucose dehydrogenase and UDP glucuronide transferase are responsible for its formation in the liver microsomes. Its formation and excretion is altered by several drugs and substances that affect the drug metabolising enzyme systems [9-11]. Our results showing reduction in ascorbic acid excretion in CCl_4 treated rats may reflect the inhibition of such enzymes. An earlier report that orally administered CCl_4 increased hepatic ascorbic acid level during 30-180 minutes and later reduced to minimum at 12 hours lends support for our observation [12]. Alteration in urinary ascorbic acid excretion appears to be reflecting ascorbic acid levels in liver. Hence, the reduction in urinary ascorbic acid excretion can be used as an index for CCl_4 produced hepatotoxicity. Prior administration of *silymarin* is reported to reverse CCl_4 induced prolongation of hexobarbital sleeping time in rats, by protecting hepatic metabolising enzymes from the effect of CCl_4. In our studies also it antagonised the $CC1_4$ induced reduction of ascorbic acid excretion in rats. So it might antagonise CCl_4 produced inhibition of enzymes responsible for ascorbic acid formation, the aqueous and methanolic extracts of *B. monosperma*(Lam) Taub leaves also antagonised CCl_4 effect on urinary ascorbic acid excretion similar to *silymarin. Silymarin* is a mixture of silybin, sildianin and silychristin which are flavanolignans. The phytochemical investigations of aqueous and methanolic extracts of *Butea monosperma* (Lam) Taub leaves showed the presence of flavonoids[13], polyphenols as well as flavonolignans which all are known to have hepatoprotective actions similar to silymarin.

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