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Evaluation of Hepatoprotective Activity of *Tabebuia Rosea* Linn

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ABSTRACT: Objective: This study screens the aqueous and ethanolic extracts of *Tabebuia Rosea* Linn for hepatoprotective activity in ethanol Swiss albino Wistar models in rats.

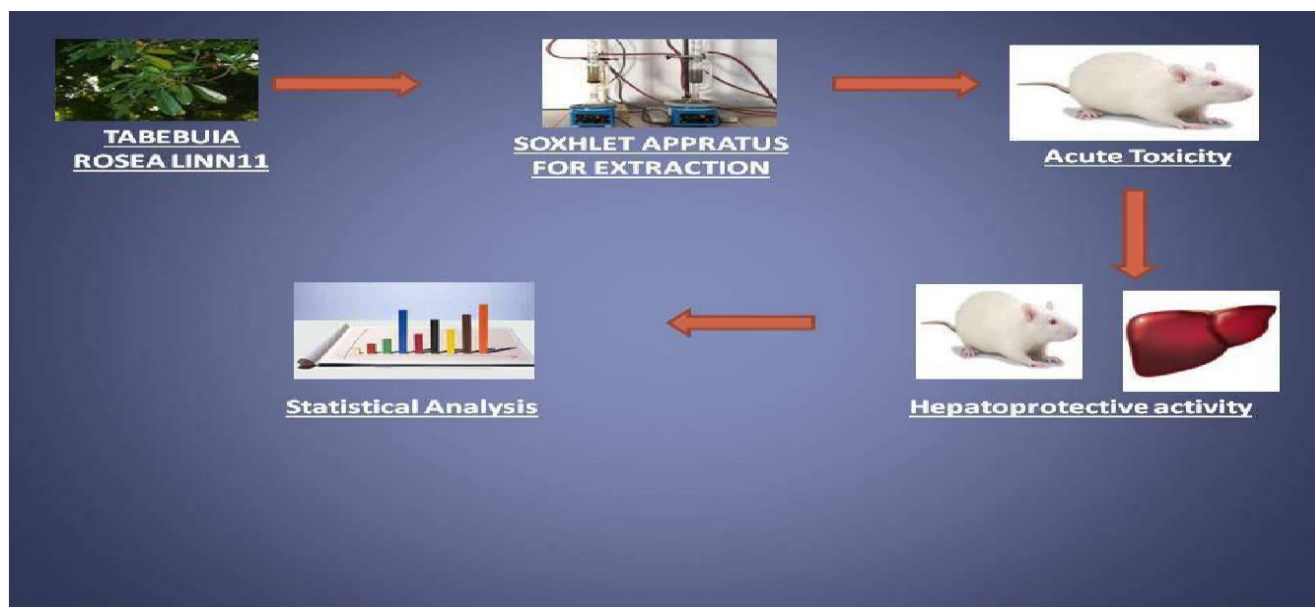
Materials and methods: Using the Soxhlet apparatus, the plant material from *Hepatotoxicity* Linn was extracted using a variety of solvents, including petroleum ether, chloroform, ethanol, and water. Various in vivo testing techniques were used to test the extracts for antiulcer activity. According to OECD recommendations, ethanol extract of *Tabebuia Rosea* and ethanol extract of *Tabebuia Rosea* oral acute toxicity studies were conducted in Swiss Wistar albino rats. In ethanolic rats, the hepatotoxic action of ethanol extract of *Tabebuia Rosea* 250 and ethanol extract of *Tabebuia Rosea* 500 doses of 100 and 200 mg/kg body weight were evaluated.

Results: According to a research on acute oral toxicity, ethanol extract of *Tabebuia Rosea* 250 and ethanol extract of *Tabebuia Rosea* 500 were safe up to 3000 mg/kg of rat body weight in terms of dosage. According to the findings of the hepatotoxicity activity investigation, *Tabebuia Rosea* Linn leaf extracts significantly increased hepatotoxicity activity when compared to control in a dose-dependent way. The hepatotoxicity effectiveness of ethanol extract of *Tabebuia Rosea*

250 & ethanol extract of *Tabebuia Rosea* 500 was further validated by histopathological examinations of treated stomach tissues.

Conclusion: Results suggested that the ethanol extract of *Tabebuia Rosea* 250 and ethanol extract of *Tabebuia Rosea* 500 may be used to prevent or treat hepatotoxicity. The antioxidant- potential phenolic/flavonoid content of ethanol extract of *Tabebuia Rosea* 250 and ethanol extract of *Tabebuia Rosea* 500 may be the cause of *Tabebuia Rosea* leaves' hepatotoxic effects.

Graphical Abstract



Keywords: *Tabebuia Rosea*, Hepatotoxicity, *Silymarin*

Abbreviation

ALT- Alanine aminoTranferase

CLD- Chronic Liver Disease

⁰C- Degree Centigrate

EHBA- Extra hepatic biliary atresia

GM- gram

IVC- Inferior vena cava

LFTs- Liver Function Test

METGF- Methanol Extract of tectona grandis

NASH- Non-alcoholic steatohepatitis

PELD- Pediatric end stage liver disease

transaminase

SGPT- Serum Glutamic pyruvate transamylase

AST- Aspartate aminotransferase

CCL4- Carbon tetrachloride

DILI- Drug induced Liver injury

GGT- Gama glutamyltransferase

HCC- Hepatocellular carcinoma

INR- International normalised ratio

MPT- Mitochondrial permeability Transtion

MELD- Model for end stage liver disease

PT- Prothrombin Time

SGOT- serum Glutamate oxaloacetic

STZ- Streptozotocin

I. INTRODUCTION

Hepatotoxicity is a word used to describe liver damage brought on by chemicals. The liver is sensitive to the toxicity from these substances and plays a crucial function in modifying and eliminating toxins. Absolute medicinal substances have the potential to harm the organ when consumed in excess and sporadically even when given within therapeutic parameters. Hepatotoxicity can also be caused by other chemical agents, including those found in industry, labs, natural compounds (such microcystins), and herbal medicines. It is a widely acknowledged cause for a medicine to be taken off the market, along with the 900 other pharmaceuticals that have been linked to liver damage[1]. Chemicals often result in subclinical liver damage, which is only seen in routine liver enzyme testing. 50 percent For 5% of all hospital admissions and 80% of all acute liver failures, the same medicine that damaged the liver is to blame.

Astringent, anti-inflammatory, bacterial, antifungal, diuretic, and laxative are some of the properties of *tabebuia rosea*.

Its ethanolic leaf extract is used to treat a variety of gram-positive and gram-negative bacteria because of its antibacterial properties. The Pink Trumpet Tree, also known as *Tabebuia Rosea*, may reach a height of 15 metres and is renowned for its exquisite blossom. In several European nations, wood is frequently utilised for carpentry and general building. The fruits are long, green, and bean-like, measuring 20–40 cm (8-16 inches). The fruit has flat, heart-shaped seeds with small wings that turn dark brown when they are fully mature. The tree provides medicinal benefits in addition to the elegant beauty of the leaves for treating eyes. The bark and leaves are used to make tea, which is known to lower fevers. [2,3]

Recent research has demonstrated that this plant contains flavonoids, tannins, saponins, alkaloids, and phenolic acids. The leaf and bark of *Tabebuia rosea* may be toxic to cells due to the presence of o-xylene (2.13 percent), 2, 4-dimethylhexane (1.03 percent), methyl cyclohexane (53.13 percent), methyl benzene (12.75 percent), and 3- Pentene-2-one (0.11 percent). According to reports, *Tabebuia rosea* has diuretic, antibacterial, antifungal, anti- inflammatory, and astringent properties. With regard to both gram positive and gram negative bacteria, Its leaf extract, which is ethanolic, is used as an antimicrobial.

II. MOLECULAR PHARMACOLOGY IN HEPATOTOXICITY CYTOKINES OF HEPATOTOXICITY

The liver is a significant target of drug toxicity and oxidative stress, and its particular metabolic link to the gastrointestinal system is one cause of hepatotoxicity. By promoting rapid transit from the cytoplasm to the plasma membrane, where self aggregation takes place to target death, cholestatic illness and endogenously produced bile acid cause hepatocellular apoptosis. Toxic damage is prolonged by neutrophils in filtration and kupffer-cell activation. Increased neutrophil visitation and activation was a result of kupffer cells releasing reactive oxygen species (ROS), cytokines, and chemokines. The liver expresses a large number of cytochrome P450 isoforms, including ethanol-induced CYP2E1. The primary mechanism through which ethanol induces oxidative stress is by the production of ROS by CYP2E1, which also activates several toxicologically significant substrates. Nitric oxide (NO) scavenges super oxide in acetaminophen toxicity to form peroxynitrite, which damages tissue and nitrates proteins. Microvascular steatosis, Non-alcoholic Steatohepatitis (NASH), and Cytolytic Hepatitis all entail dysfunctional mitochondria, impaired fatty acid -oxidation, suppression of mitochondrial respiration, and mitochondrial DNA damage. Hepatotoxicity continues to be a major factor in removal of a medication from clinical and pharmacological research. Induction of the mitochondrial permeability transition (MPT), which can result in necrosis from ATP depletion or caspase-dependent apoptosis if ATP depletion is inadequate, is another mechanism that causes mitochondrial failure.

Material and methods Animals

We bought Swiss albino Wistar rats from the national botanical research institution in Lucknow that weighed between 150 and 230 grammes. For a week prior to and throughout the tests, They were kept in the departmental animal house under typical circumstances (26 °C and relative humidity 30-35 percent), with 12 hour light cycles and 12 hour dark cycles, respectively. The rats were fed a typical mouse pellet diet and were given access to free water. The diet's macronutrient breakdown is as follows: 10% protein, 4% arachide oil, 1% fibre, 1% calcium, 1000 IU/g vitamin A, and 500 IU/g vitamin D are all present.

Drugs and chemicals

All the drugs and chemicals were provided by the national botanical research institute, Lucknow and these all were of L R grade.

Collection and authentication of plant

Fresh *Tabebuia rosea* leaves from the Bignoniaceae family were gathered from the National Botanical Research Institute's botanical garden in Lucknow, India. Before being stored for future use at the departmental herbarium of the National Botanical Research Institute in Lucknow, India, the leaves were examined by Dr. Vijay Kumar, a researcher in

the division of ethnopharmacology and pharmacognosy. The departmental herbarium of the National Botanical Research Institute in Lucknow houses the voucher specimen (NBP 2709). India.

Extraction of *tabebuia rosea*

The fresh leaves were washed by giving them a good rinse in clean water, and after drying in the sun for 24 hours, they were completely dried in a hot air oven. After drying, a mixer is used to grind the dried fragments into a coarse powder. The (2 kg) powdered leaves were first extracted with 50% ethanol for 3 days, and then using muslin. To get a 50% ethanolic extract, the filtrate is concentrated at decreased pressure in a rotator evaporator (R 110 Buchi, Switzerland) (EE5). The yield of extracted dried, which was kept in a tight container at a temperature of around 40°C until it was needed, was 21.56 percent (w/w). Further pharmacological research was done on the extracted materials.

Acute oral toxicity studies

According to OECD-423 recommendations, an acute oral toxicity test was conducted. Following an overnight fast with only water provided, albino mice were given *Tabebuia Rosea* ethanolic leaf extracts at a dose of 5 mg/kg of body weight for general behaviour and a 3000 mg/kg dose for acute toxicity as described in table 3.1.

Tabebuia Rosea leaf ethanolic extract was administered at a range of doses (250, 500, 2000, and 5000 mg/kg), given to pairs of mice at a dosage of 10 ml/kg body weight, dissolved in 1% carboxymethyl cellulose. The control mice are given 10 ml/kg of distilled water containing 1 percent carboxymethyl cellulose orally. The animal was observed for two hours, then periodically every four hours for another four hours. Finally, the animal was observed for a final observation of any mortality behaviour (gross behaviour, general motor activity, convulsion, response to tail pinching), pupil size, water intake, feeding behaviour, and sedation), for 72 hours, and the animals were kept for up to 14 days.

Experimental protocol

Assessment of hepatoprotective activity

***In-vitro* hepatoprotective activity of *Tabebuia Rosea* (leaf) against carbon tetrachloride- induced hepatotoxicity** (150-230 g) Swiss Albino wistar rats were used. Five groups were created at random from all the animals. Six animals made up each group, and they were given the following care:

Group I Saline was administered to normal control animals (1 ml/kg p.o.). Group II Animals were administered CCl₄/olive oil (1:1, 2 ml/kg s.c.).

Group III Silymarin (60 mg/kg p.o.) and CCl₄/Olive oil (1:1.2 ml/kg) were given to the animals.

Group IV The animals were received ethanolic extracts of *Tabebuia Rosea* leaf (250mg/kg p.o.) and CCl₄/ Olive oil (1:1, 2 ml/kg)

Group V Animals received ethanolic extracts of *Tabebuia Rosea* leaf (500mg/kg p.o.) and CCl₄/ Olive oil (1:1, 2 ml/kg)

According to prior discussions by xinpeng et al. 2011 and panigrati et al. 2010, the following procedure was used to cause liver damage in rats and cause hepatotoxicity. The blood from the rats was collected in plain tubes after they were killed via cervical decapitation. Centrifugation is used to get the serum (3000rpm). Their livers were weighed after bleeding, and a thin slice was kept in a buffered formalin solution for histological analyses.

Tabebuia Rosea (leaf) in-vitro hepatoprotective efficacy against paracetamol-induced hepatotoxicity***Tabebuia Rosea (leaf) in-vitro hepatoprotective efficacy against paracetamol-induced hepatotoxicity***

Albino (150-230 g) Wister rats were used. Five groupings of all the animals were created at random. Six animals make up each group, and they were given the following care:

Group I Saline was administered to normal control animals (1 ml/kg p.o.). Group II Animals were given a 1:1:2 s.c. mixture of paracetamol and olive oil.

Group III Silymarin (60 mg/kg p.o.) and Paracetamol/Olive oil (1:1.2 ml/kg) were administered to the animals.

Group IV Animals received ethanolic extracts of *Tabebuia Rosea* leaf (250mg/kg p.o.) and paracetamol / Olive oil (1:1, 2 ml/kg)

Group V Animals received ethanolic extracts of *Tabebuia Rosea* leaf (500mg/kg p.o.) and paracetamol / Olive oil (1:1, 2 ml/kg)

III. RESULTS AND DISCUSSION**Table 3.2: General behavior and acute toxicity study of ethanolic leaf extracts of *tabebuiaRosea* 3000 mg/kgdose.**

Gross Activity	Time after administration (Hours)					
	2	3	5	7	12	24
Respiration	-	-	-	-	-	-
Writhing	-	-	-	-	-	-
Convulsion	-	-	-	-	-	-
Sedation	-	-	-	-	-	-
CNS depression	-	-	-	-	-	-
Diarrohea	-	-	-	-	-	-
Skin irritation	-	-	-	-	-	-
Mortality	-	-	-	-	-	-
Salivation	-	-	-	-	-	-
Tremors	-	-	-	-	-	-

Percentage yield plant extract

500 gm leaf powder of *Tabebuia rosea* was macerated with ethanol. The percent yield of ethanol extracts of *Tabebuia rosea* was found to be 21.62%.

Pharmacological activity

Effects of *Tabebuia Rosea* leaf ethanolic extract on body weight and liver weight in rats exposed to carbon tetrachloride

The effect of *Tabebuia Rosea* on the bodyweight of CCl₄ intoxicated rats is shown in table 4.1.

According to the % daily weight changes, the CCl₄-treated group had shed more weight than the control group. During the trial, the *Tabebuia Rosea* treated group likewise saw a reduction in body weight.

Table 4.1 Effect of ethanolic leaf extracts of *Tabebuia Rosea* on the weight of liver and weight of the body in carbon tetrachloride intoxicated rats

Groups	Treatments	Dose mg/kg	Body weight (gm)	Liver weight (gm)
I	Control (normal saline)	10 ml, p.o.	238.03±5.82	8.42±1.0
II	CCl ₄ (negative control)	2 ml/kg, p.o.	226.34±3.83 ^a	9.05±0.13 ^a
III	<i>Silymarin</i> (standard drug)	100, p.o.	235.01±5.13 ^{ab}	10.1±1.0 ^{ab}
IV	EETR 250 (test drug- I)	250, p.o.	206.49±2.86 ^{ab}	9.30±1.0 ^{ab}
V	EETR 500 (test drug- II)	500, p.o.	209.05±3.66 ^{ab}	9.0±0.15 ^{ab}

The results are presented as the mean SEM of six rats from each group. When compared to control, a = p>0.001 and to CCl₄, b = p >0.001.

Effects of ethanolic leaf extract of *Tabebuia Rosea* on biochemical parameters (SGPT, SGOT, and ALP) of CCl₄ induced hepatotoxicity.

Serum trans-aminases (SGPT, SGOT, and ALP) were assessed to be at values of 112.56.47, 116.53.98, and 184.05.75, respectively (Table 4.2 & figure 4.1), however these levels considerably increased to 263.05.81 after the hazardous dosage of CCl₄ (2ml/kg) was administered. However, pretreatment of rats with 250 mg/kg and 500 mg/kg of *Tabebuia rosea* (SGPT, (p0.001) than the levels of toxic control.

Table 4.2 Effect of ethanolic extracts of *Tabebuia Rosea* leaf on a biochemical parameter of CCl₄ induced hepatotoxicity in rats.

Groups	Treatments	Dose (kg ⁻¹)	SGPT(U/L)	SGOT(U/L)	ALP(U/L)
I	Control (normal saline)	10 ml, p.o.	112.5±6.47	116.5±3.98	184.0±5.75
II	CCl ₄ (negative control)	2 ml, p.o.	263.0±5.81 ^a	427.0±4.66 ^a	535.8±5.28 ^a
III	<i>Silymarin</i> (standard drug)	100 mg, p.o.	131.2±3.12 ^{a,b}	138.5±2.88 ^{a,b}	226.3±3.23 ^{a,b}
IV	EETR 250 (test drug- I)	250, p.o.	186.0±3.61 ^{a,b}	381.5±5.00 ^{a,b}	371.2±4.12 ^{a,b}
V	EETR 500 (test drug- II)	500, p.o.	158.0±2.85 ^{a,b}	271.8±4.84 ^{a,b}	324.5±4.38 ^{a,b}

The results are presented as the mean ±SEM of six rats from each group. When compared to control, an is significantly different (p>0.001), and when compared to CCl₄, b is significantly different (p >0.001).

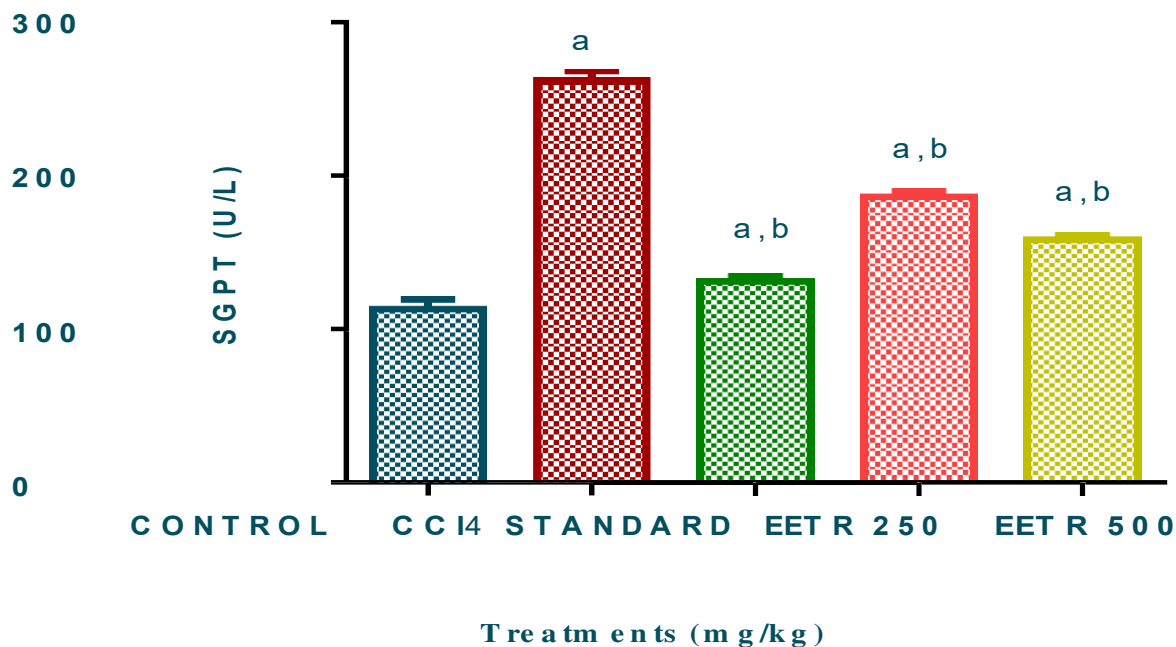


Fig- 1 Effect of ethanolic extract of *Tabebuia rosea* leaf on SGPT in CCl₄ induced hepatotoxicity in Wistar rats.

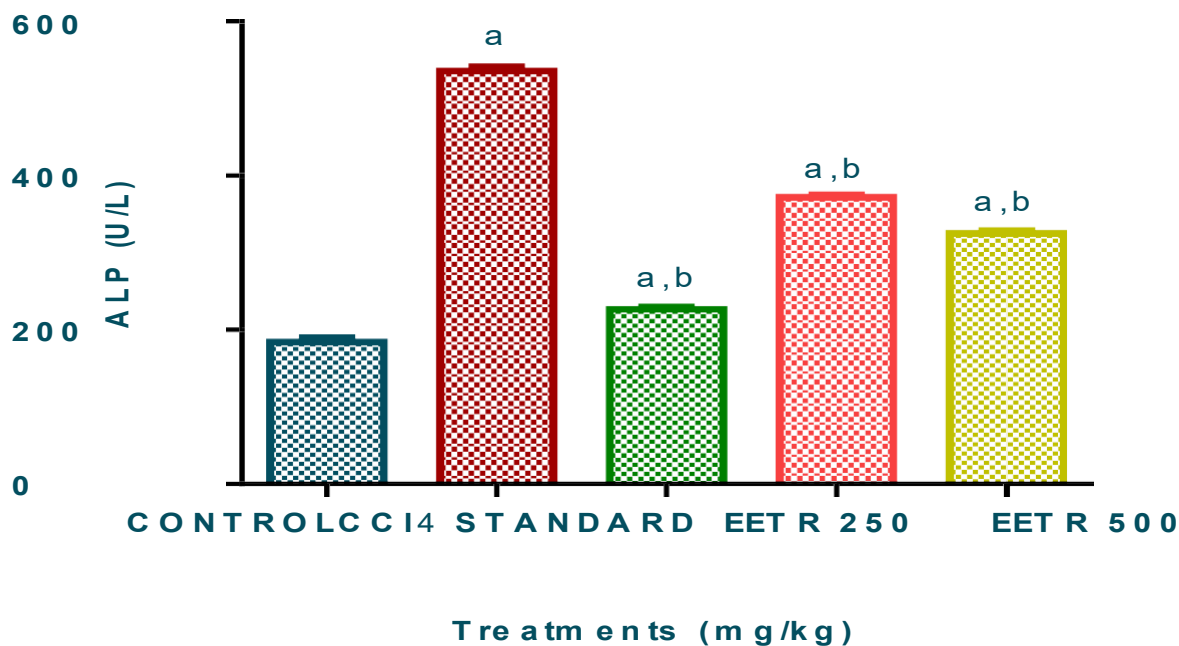


Fig – 2 Effect of ethanolic extract of *Tabebuia rosea* leaf extract on SGOT in CCl₄ induced hepatotoxicity in Wistar rats.

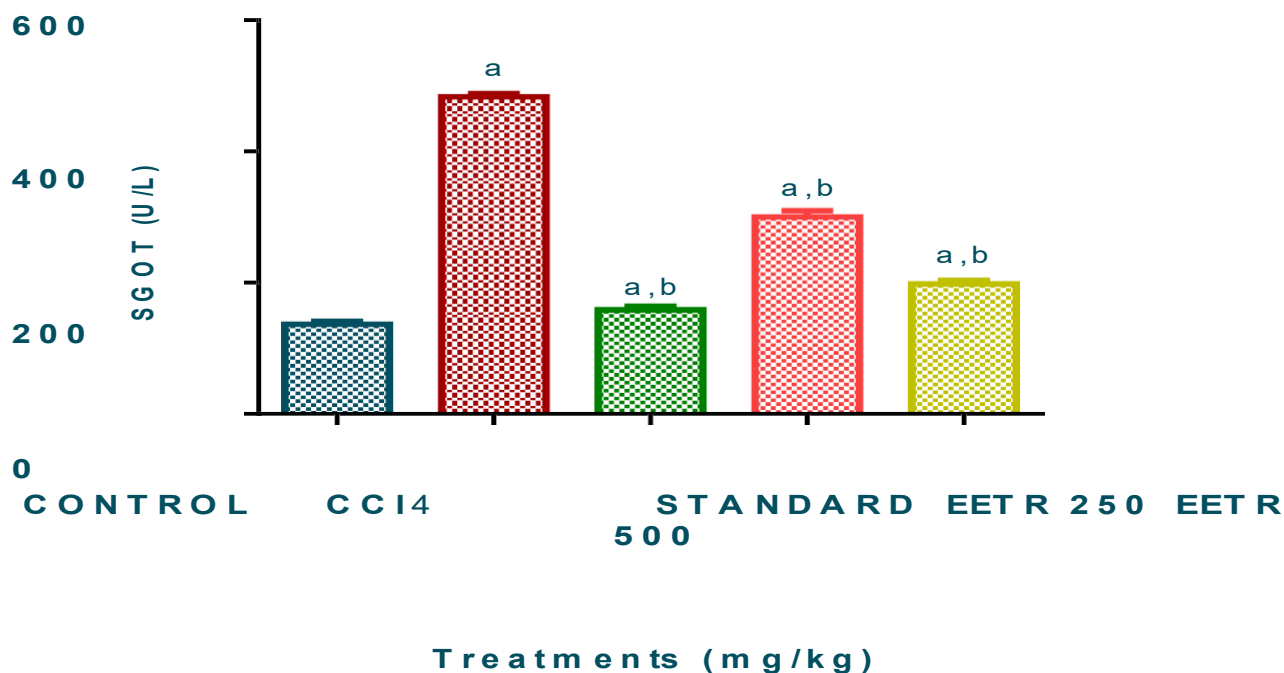


Fig- 3 Effect of ethanolic extract of *Tabebuia rosea* leaf on ALP in CCl₄ induced hepatotoxicity in Wistar rats.

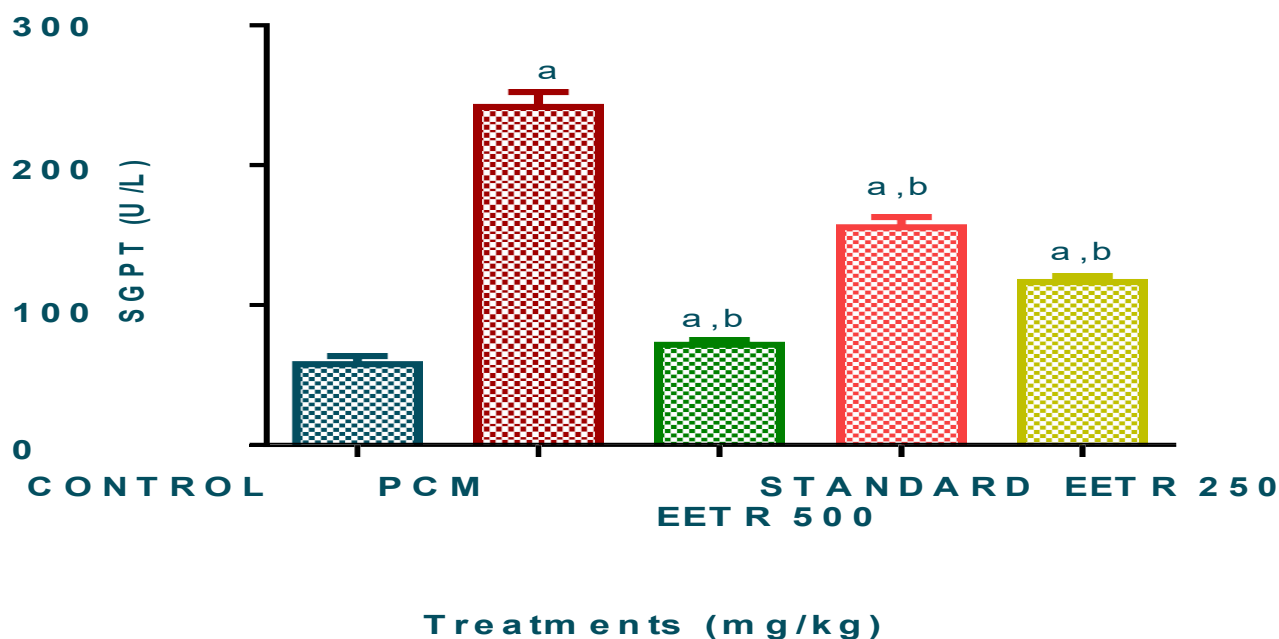


Fig – 4 Effect of ethanolic extract of *Tabebuia rosea* leaf on SGPT in Paracetamol induced hepatotoxicity in Wistar rats.

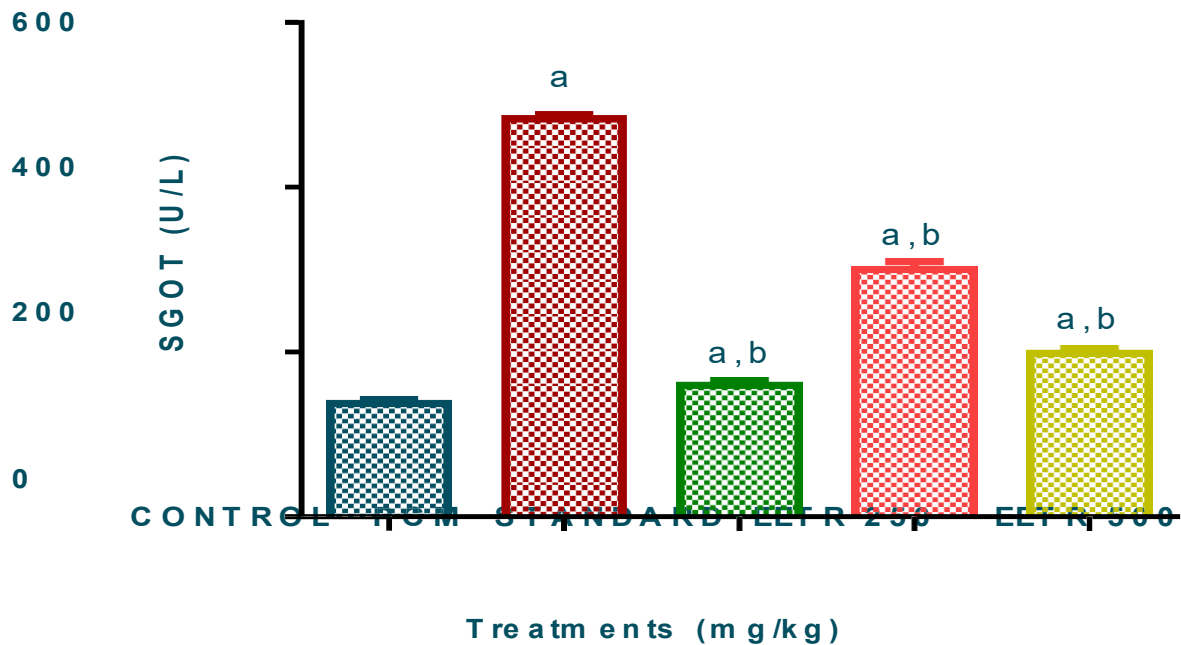


Fig – 5 Effect of *Tabebuia rosea* leaf extract on SGOT in Paracetamol induced hepatotoxicity in Wistar rats. Results were expressed as mean $\pm$ SEM with n=6 in each group.

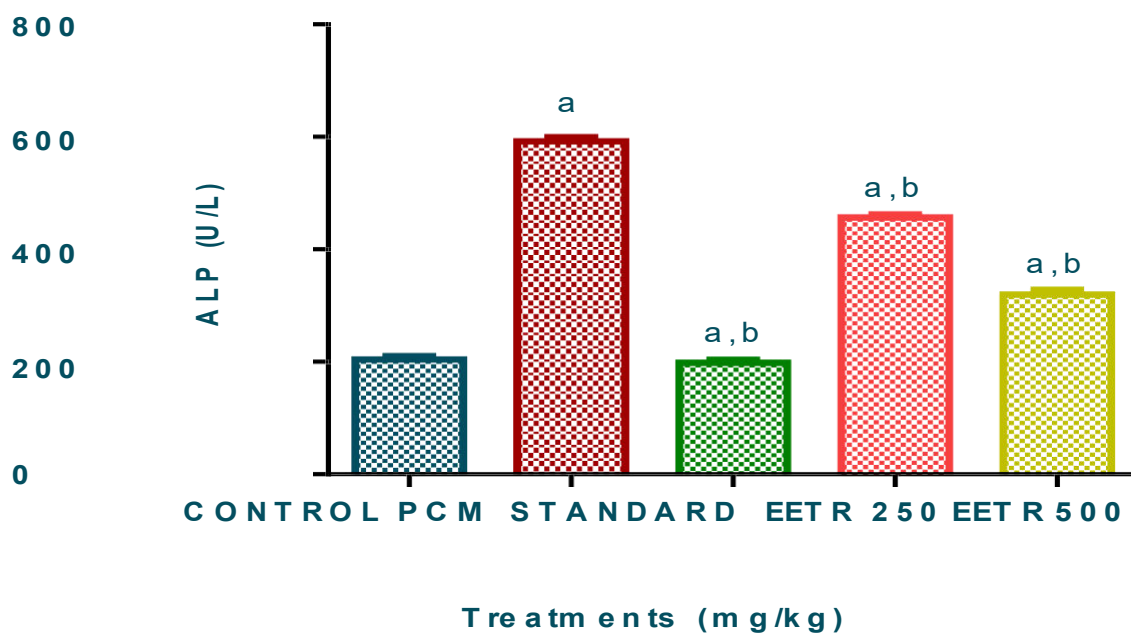
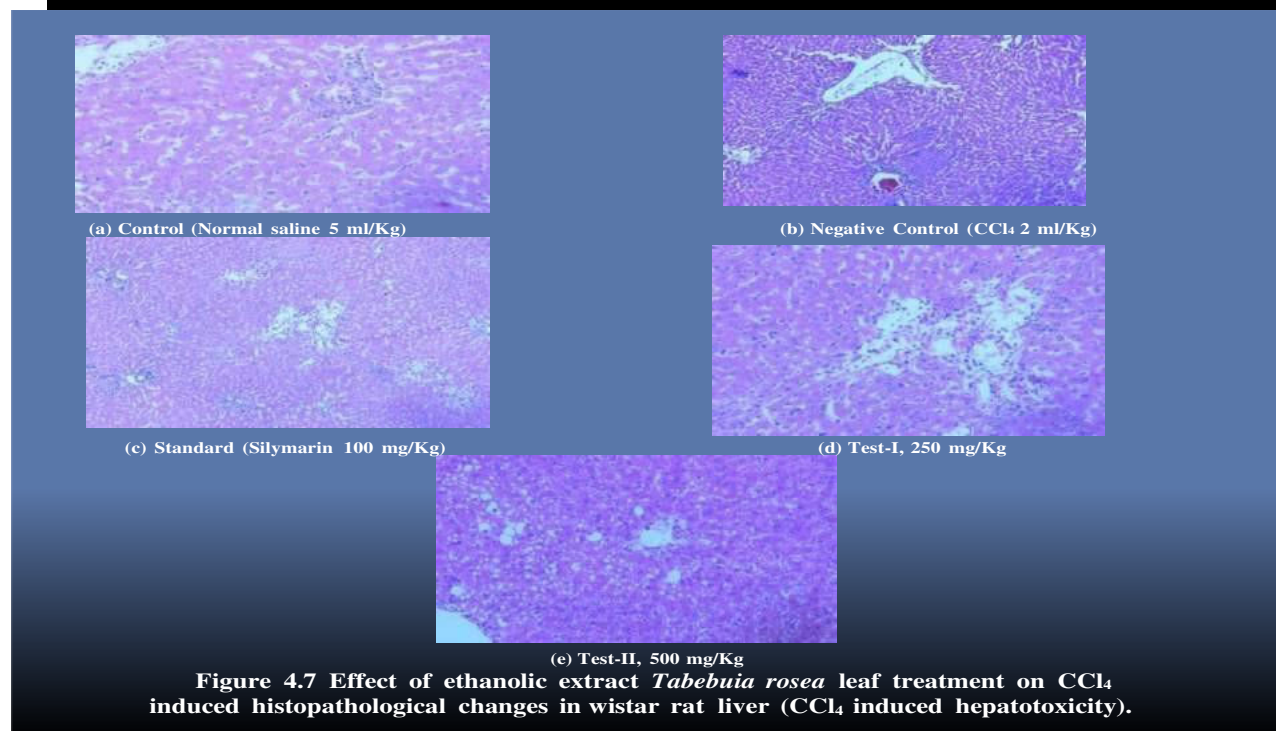
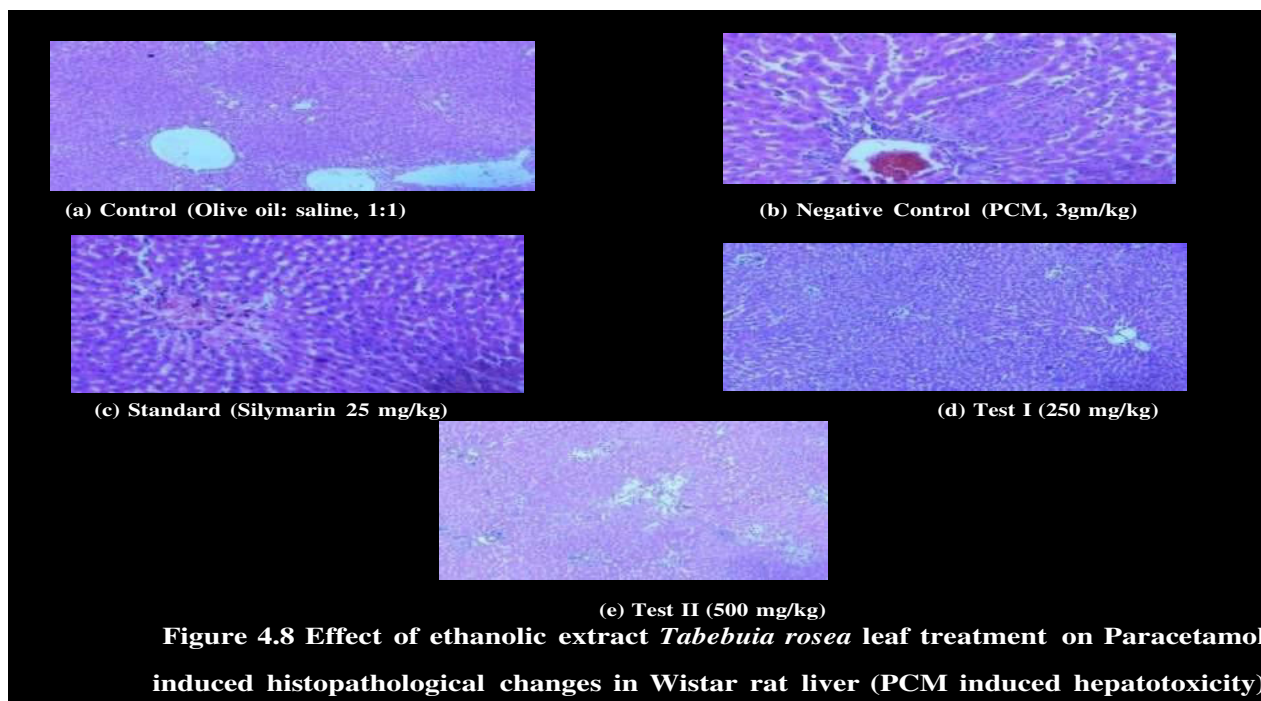


Fig – 6 Effect of *Tabebuia rosea* leaf extract on ALP in Paracetamol induced hepatotoxicity in Wistar rats. Results were expressed as mean $\pm$ SEM with n=6 in each group.



IV. DISCUSSION

The goal of the current investigation was to determine if an ethanolic extract of *Tabebuia Rosea* leaf might protect Wistar rats' livers against CCl₄ and paracetamol-induced damage. Paracetamol was shown to be the source of the liver's malfunction and damage; levels of total proteins, malondialdehyde, and glutathione were also examined. Paracetamol

treatment was observed to result in an increase in malondialdehyde, which *Tabebuia Rosea* leaf extract greatly reduced. After receiving treatment with *Tabebuia Rosea* leaf extract in ethanol, there was a discernible increase in total protein and a reduction in glutathione concentration in the liver. The effects of *Tabebuia Rosea* leaf extract in ethanol were similar to those of silymarin. Paracetamol-induced hepatic damage resulted in substantial alterations in glutathione levels, which were considerably greater in the paracetamol group than in the control group ($p < 0.001$). Malondialdehyde levels in the paracetamol group were substantially greater than those in the control group ($p < 0.05$), and glutathione levels were lower in the test groups (250 mg/kg and 500 mg/kg) compared to the paracetamol group ($p < 0.001$). When compared to the paracetamol group, malondialdehyde levels in the test groups at 250 and 500 mg/kg were lower ($p < 0.05$). Total protein was enhanced in the *Tabebuia Rosea* 250 mg/kg and 500 mg/kg test groups compared to the paracetamol-treated group ($p < 0.001$), despite the fact that it was considerably lower in the paracetamol group than the control group ($p < 0.01$) when compared to total protein in the control group.

Experimental animals' liver damage and dysfunction brought on by CCl₄ promote human viral hepatitis. Cytochrome P450 transforms CCl₄ into the extremely reactive poisonous free radical CCl₃O⁻, which is the cause of CCl₄'s harmful effects. Locally generated free radicals cause the membrane phospholipids' polyenic fatty acids to automatically oxidise. After interacting with oxygen, organic peroxide is created, which causes the smooth endoplasmic reticulum to enlarge and the ribosomes to separate from the rough endoplasmic reticulum. The cells' failure to produce lipoproteins from triglycerides and lipid acceptor proteins results in an accumulation of lipids, which causes the fatty liver. Furthermore, the plasma membrane is harmed by the production of lipid peroxidation byproducts. Progressive cell death swelling occurs after this. An obvious sign of cellular leakage and a loss of the functional integrity of the cell membrane was the increase in the levels of SGPT, SGOT, ALP, and total bilirubin.

The effectiveness of *Tabebuia Rosea* leaf extract as a hepatoprotective agent was further supported by comparing histological investigations of liver from various groups. In normal control rats, a section of the liver shows normal hepatic cells. In CCl₄ treated rats, their section of the liver shows centrilobular fatty degeneration, cloudy swelling, and necrosis of hepatic cells. In silymarin-treated rats, their section of the liver shows normally hepatic cells, central vein, and portal triad. Ethanolic extract *Tabebuia Rosea* leaf (250mg/kg) treated rats, a section of the liver shows improvement over the negative control group. *Tabebuia Rosea* leaf extract (500mg/kg) treated rats, section of the liver shows marked improvement over negative control and test 250mg/kg group. *Tabebuia Rosea* leaf extract (500mg/kg) treated rats, a section of the liver shows improvement over negative group control.

V. CONCLUSION

In conclusion, the information from the pharmacological research about the hydroalcoholic leaf extract of *Garuga Pinnata* will aid in distinguishing it from other species. The *Garuga Pinnata* plant contains a wide range of chemical compounds with medicinal activity, including carbohydrates, flavonoids, saponins, steroids, triterpenoids, and tannins. This provides a starting point for further research to pinpoint and isolate specific chemical constituents responsible for the plant's therapeutic activity. In conclusion, the co-administration of *Garuga Pinnata* normalised the effects of ethanol-induced ulcer, pylorus ligation, and cold restraint stress, suggesting a potential ulcer-curative role for *Garuga Pinnata*. Therefore, the traditional applications of the herb as reported in folk medicine are supported scientifically by our investigation.

Our results suggest that ethanolic extract of *Tabebuia Rosea* leaf possesses significant hepatoprotective properties. *Tabebuia Rosea* leaf extracts (250mg/kg, 500mg/kg b.w.) significantly decreased SGPT, SGOT, ALP and bilirubin in the animals treated with CCl₄ at 500 mg/kg. It shows a retrograde hepatoprotective effect. It is also significantly decreased malondialdehyde and protein levels and increased glutathione levels confirming its antioxidant activity in the animals treated with paracetamol. Similar results were obtained with standard drugs i.e. silymarin



CONFLICT OF INTEREST

The authors have no conflict of interest for the publication of this work.

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