Free Radicals and Antioxidants., 2023; 13(1):34-40.
https://www.antiox.org

Original Article

Influences of Ethanolic Extract of *Peltophorum pterocarpum* Leaves and Bark on Lipid Profile, Liver and Carbohydrate Metabolizing Enzymes on Toxin-induced Wistar Rats

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ABSTRACT

Background: The study was to identify the effects of ethanolic extract of *Peltophorum pterocarpum* leaves and bark on lipid profile, liver and carbohydrate enzymes on isoniazid and rifampicin induced Wistar rats. **Materials and Methods:** Ethanolic leaf and bark extract of *P. pterocarpum* freshly suspended in sterile water (300mg/kg BW), were administered to rats post-orally as a single dosage orally by intubation early morning for each day of the experimental period. Rats were separated into 10 groups with 6 rats in each group and maintained in isolated cages with proper ventilation. The samples were introduced to find the following parameters Total Cholesterol (TC), Triglycerides (TG), Low-Density Lipoprotein (LDL) cholesterol, High-Density Lipoprotein (HDL) cholesterol, Very-Low-Density Lipoprotein (VLDL) cholesterol, Phospholipids (PL), Free Fatty Acid (FFA), Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Alkaline Phosphatase (ALP), glucose, glucose-6-phosphatase, glucose-6-phosphate dehydrogenase and hexokinase, kidney markers such as urea, uric acid and creatinine. **Results:** The results highlighted that the leaves and bark extract of *P. pterocarpum* had a potential hepatoprotective effect against the toxin treated rats and may alter lipid profile, liver and carbohydrate metabolizing enzymes levels. **Conclusion:** This effect proves the hepatoprotective nature of both the leaves and barks of *P. pterocarpum*.

Keywords: *Peltophorum pterocarpum*, Liver enzymes, Lipid profile, Carbohydrate metabolizing enzymes, Isoniazid, Rifampicin.

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Received: 13-05-2023;
Revised: 28-05-2023;
Accepted: 14-06-2023.

INTRODUCTION

The liver, one of the largest and most important organs in the human body, which responsible for the most crucial body functions, like growth, immunity, metabolism nutrition, reproduction and excretion processes. The hepatic system also performs many hundreds of other metabolic and physiological processes. Liver diseases are causing severe complications, characterized as acute or chronic hepatitis, liver cirrhosis and hepatosis. It is inflicted by hepatotoxic agents (drugs like antibiotics and chemotherapeutics agents as well as toxins such as aflatoxins, carbon tetrachloride (CCl_4), peroxidised oil, chlorinated hydrocarbons, etc.,), excess intake of alcohol, autoimmune disorders and infections. In severe liver damages, most of the liver cells die or turn to the fibrotic stage resulting in the improper or non-functional liver. In this situation, the treatment must be included with the therapeutic agents which could stimulate the liver cell proliferation, to reverse the damaged liver cells to the normal state.[1] But synthetic drugs causes various adverse effects on our bodies. Because of the huge side effects and excessive cost of modern drugs, many commercial drugs are withdrawn from the market especially for their hepatotoxic side effects. Therefore, many polyherbal formulations are recommended nowadays for the treatment of various chronic diseases including diabetes mellitus, blood pressure, cardiovascular disease, liver disorders and cancer.

Peltophorum pterocarpum (family-Fabaceae) is a well-known ornamental tree grown on the roadsides. All the parts of the plant have ethnobotanical and Scientifically proven records for their medicinal values such as neuroprotective and memory enhancers, antimicrobial, antidiabetic, cardiotonic, antioxidant, anti-inflammatory, hypocholesterolemic,

DOI: 10.5530/fra.2023.1.6

antiarthritic, and anti-cancer.[2] The phytochemical studies in the plant *P. pterocarpum* revealed the presence of phenolic acids, monoterpenes, sesquiterpenes, valeronone, β-sitosterol, isosatvene, hexadecanoic acid, octadecanoic acid, flavanoids, (-)-epicatechol, (+)-leucocynidin, tannins, saponins, terpenoids, steroids, alkaloids, amino acids, carotenoids, coumarins, quercetin, berginin, vitamins, etc., are distributed in this plant.[3,4] Therefore, the present study aims to investigate the lipids profiles, liver enzymes and carbohydrate metabolism on the toxin-induced and *P. pterocarpum* treated rats.

MATERIALS AND METHODS

Plant collection and preparation of leaf and bark extract

The *P. pterocarpum* leaves and barks were collected in Tiruchirappalli, India. The species were identified, and a voucher specimen MJ 001 (Leaf) and MJ 002 (Bark) was deposited at the Herbarium of Rapinet at St. Joseph's College, Tiruchirappalli, India. The ethanol extract was arranged from plant material in the traditional method, as usually used. The dried leaves and barks were powdered mechanically then the extract was prepared by using the Soxhlet apparatus by using the ethanol as a solvent. The filtrate was evaporated and then dried in a vacuum and then the extracts were preserved in the refrigerator till further use and concentrations used in the experiments were based on the dry weight of the extract.

The ethical issue, animals, housing, and experimental design

The adult male Albino Wistar rats (with an age of 8 weeks and weight ranged from 150 to 165 g) were procured and maintained at the temperature 25±1°C with a 12 hr light/dark cycle with feed as a standard pellet diet consisting of protein, fat, fiber, balanced with carbohydrates, minerals, Vitamins and water *ad libitum*. All the experimental procedures were carried out at Srimad Andavan College of Arts and Science, Tiruchirappalli, India, (Reg. No: 790/03/ac/CPCSEA) the Institutional Animal Ethics Committee approved was obtained.

Ethanolic leaf and bark extract of *P. pterocarpum* freshly suspended in sterile water (300mg/kg BW), were given to the animals post-orally at a single dose orally in the early morning for each day up to the end of the experimental period. Rats were separated into 10 groups with 6 rats in each group and maintained in isolated cages with proper ventilation. Animal groups were categorized as control (basal diet, G1) were given saline water, isoniazid and rifampicin induced as negative control (G2), G2 supplemented with 100 mg/kg BW of leaf extract of *P. pterocarpum* (G3), G2 supplemented with 200 mg/kg BW of leaf extract of *P. pterocarpum* (G4), G2 supplemented with 400 mg/kg BW of leaf extract of *P. pterocarpum* (G5), G2 supplemented with 100 mg/kg BW of bark extract of *P. pterocarpum* (G6), G2 supplemented with 200 mg/kg BW of bark extract of *P. pterocarpum* (G7), G2 supplemented with 400 mg/kg BW of bark extract of *P. pterocarpum* (G8), G2 supplemented with 25 mg/kg BW of silymarin (G9) and G2 supplemented with both leaf and Bark extract of *P. pterocarpum* with 400 mg/ kg (G10).

Biochemical evaluation

The rats were sacrificed at the end of the study and the samples were collected with a standard procedure. The samples were introduced to find the following parameters Total Cholesterol (TC), Triglycerides (TG), Low-Density Lipoprotein cholesterol (LDL), High-Density Lipoprotein cholesterol (HDL), Very-Low-Density Lipoprotein cholesterol (VLDL), Phospholipids (PL), Free Fatty Acid (FFA), Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Alkaline Phosphatase (ALP), glucose, glucose-6-phosphatase, hexokinase and glucose-6-phosphate dehydrogenase, kidney markers such as urea, uric acid and creatinine are estimated at the end of the study. All the biochemical parameters are done using commercially available kits and were determined by a fully automated biochemical analyzer (Turbo Chem 100, CPC Diagnostics).

Statistical analysis

All data obtained in the biochemical parameters were measured for statistical significance using one-way Analysis of Variance (ANOVA) by Duncan's Multiple Range Test (SPSS® for Windows, version 17, Chicago, USA) and table values expressed as mean and standard deviation. The $p \leq 0.05$ was considered to be significant in all tests.

RESULTS AND DISCUSSION

The oxidative stress due to the toxin may play an important role in the pathogenesis of various chronic complications. The free radicals like superoxide anions, hydrogen peroxide, peroxynitrite and nitric oxide initiated the oxidative stress in toxin-induced animal models and these subsequently lead to cellular damages in the liver.[5]

Lipid Profile

Table 1 shows the results of lipid profiles. The level of lipid profiles (TC, TG, LDL, VLDL, PL and FFA) are noted to be increased and the levels of HDL are decreased in the toxin treated rats. After the treatment with the ethanolic leaf and bark extract of *P. pterocarpum*, the levels were significantly normalized. Lipids are the important components of biological membranes. The free radicals play a significant position in the maintenance of cellular functions and homeostasis in the human body. The liver contributes the lipids metabolism and it transports through the plasma by lipoproteins. The lipid metabolism is disturbed in the condition of severe liver damage.[6] The hormones plays a key role in fuel metabolism, especially the thyroid hormone involved in the fuel metabolism and is involved in both the anabolism and

Table 1: Effect of plant extract on various lipid parameters in the experimental animals.

Groups	Total Cholesterol (mg/dL)	Triglycerides (mg/dL)	High-density lipoprotein cholesterol (mg/dL)	Low-density lipoprotein cholesterol (mg/dL)	Very-low-density lipoprotein cholesterol (mg/dL)	Phospholipids (mg/dL)	Free fatty acids (mg/dL)
Group I	154.67±1.50[a]	70.50±0.76[a]	92.00±0.86[a]	48.57±1.08[a]	14.10±0.15[a]	64.5±1.1[a]	54.6±0.7[a]
Group II	408.83±1.54[b]	362.50±1.48[b]	41.67±0.88[b]	294.67±1.76[b]	72.50±0.30[b]	120.8±2.2[b]	104.8±1.1[b]
Group III	376.33±1.17[c]	320.17±0.83[c]	53.33±0.67[c]	258.97±1.79[c]	64.03±0.17[c]	101.9±1.5[c]	73.3±0.8[c]
Group IV	298.00±1.39[d]	271.50±1.23[d]	70.33±0.88[d]	173.37±1.78[d]	54.30±0.25[d]	80.2±0.8[d]	61.9±0.1[d]
Group V	222.00±1.06[e]	152.33±1.23[e]	81.33±0.76[e]	110.20±1.47[e]	30.47±0.25[e]	67.9±0.8[a]	58.0±0.6[a,d]
Group VI	382.50±0.72[c]	327.83±2.70[c]	49.67±0.56[c]	267.27±0.58[c]	65.57±0.54[c]	108.7±1.7[c]	77.1±0.5[c]
Group VII	303.83±0.87[d]	279.67±1.94[d]	67.67±1.48[d]	180.23±2.34[d]	55.93±0.39[d]	88.3±3.0[d]	67.2±0.7[c]
Group VIII	230.33±1.65[e]	160.67±1.05[e]	77.17±1.25[d]	121.03±2.08[e]	32.13±0.21[e]	72.5±2.0[a]	61.9±1.2[d]
Group IX	195.67±1.84[f]	129.17±0.95[f]	87.17±0.79[e]	82.67±2.37[f]	25.83±0.19[f]	65.7±0.6[a]	56.0±0.8[a]
Group X	161.83±1.78[a]	54.83±6.09[a]	97.17±0.48[f]	53.70±2.35 [a]	10.97±1.22[a]	63.0±0.4[a]	53.3±0.6[a]

Values are given in the table as means ± standard deviation for 6 rats.[a, b, c, d, e, f] means in the same row with different superscripts differ ($p < 0.05$).

catabolism of carbohydrate, fat and protein. Thyroid hormone induces the synthesis of fatty acids and cholesterol. The increasing levels of TC and TG may stimulate the degradation of cholesterol and fatty acids. The increased levels of lipids and proteins in the liver are associated with hyperthyroidism. These oxidized proteins and lipids may subsequently favour the production of more free radicals.[7]

In cells, the large number of unsaturated lipids favours the conversion of lipids into peroxidation. These peroxides may alter the membrane structure which may favour cell homeostasis and lipid fluidity (Rice Evans and Burdon, 1993).[8] Several chemical compounds that damage the liver, favours the excess accumulation of fat in the parenchymal cells. These activities may be due to the disproportion between the formation and degradation of TG in the circulation of blood.[9] In this study, the level of TC and TG are augmented, and the levels were significantly decreased in the treated leaf and bark treated groups.

In the liver, the metabolic process of lipogenesis and lipoprotein production occurs. Hence, liver damage alters the metabolism of lipids, lipogenesis, and lipoprotein production metabolism in a living organism. In the present study, isoniazid, and rifampicin 50:50mg/BW fed group damages the liver and causes necrosis in the liver. It favours the fat accumulation in the liver.[10] The deposition of fat in the liver favours the increasing concentration of cholesterol as well as alters the TG and PL is seen in the isoniazid and rifampicin 50:50 mg/BW rats in this investigation. The increasing levels of TG Can result in risk of CVD (Patel *et al.*, 2004).[11] The toxin-induced liver injury may influence the accretion of abnormal quantities of fats, particularly TG in the parenchyma cells.[12-15] TG accumulation can be thought of resulting due to the imbalance between the speed of synthesis and release of TG through the parenchymal cells into the circulation.[12,16] The mechanism might be involved the decreased activity of lipolytic enzymes[17] and this leads to the reduced level of removing capacity of TG from plasma. Hypertriglyceridemia is often observed in different degrees in tumour-bearing animals in blend with increased VLDL and decreased HDL is defective catabolism rather than elevated hepatic synthesis of TG rich lipoproteins.[18]

The PL is responsible for cell integrity. This compound is regulated and maintains cell permeability and signal transduction activity. In the present study, the isoniazid and rifampicin 50:50 mg/BW treated rats the PL level is raised because of cell damage due to the toxin. After the administration of the extract of *P. pterocarpum* leaves and barks, the level of PL is significantly reduced. This may be due to the productive activities of the plant extract on the cell membrane. A similar effect is identified by Halim *et al.*[19]

The present study demonstrated that the LDL and VLDL are raised and the HDL level is reduced in the toxin isoniazid and rifampicin 50:50 mg/BW treated rats. Reverse Cholesterol Transport (RCT) is an important mechanism for the transportation of cholesterol from cells to the liver for excretion. Generally, the HDL concentration is decreased in the liver disease condition. The higher concentration of cholesterol reduces the HDL level and increases the FA level in the liver this may due to the action of lysosomal acid triacylglycerol lipase activity.[20] The HDL level is indirectly proportional to the concentration of LDL and VLDL. After administration of the extract of *P. pterocarpum* leaves and

Table 2: Effect of plant extract on Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT) and Alkaline Phosphatase (ALP).

Groups	Aspartate aminotransferase (U/L)	Alanine aminotransferase (U/L)	Alkaline phosphatase (U/L)
Group I	55.05±0.85[a]	48.03±0.58[a]	100.18±1.45[a]
Group II	94.62±0.95[b]	97.40±0.79[b]	258.98±2.67[b]
Group III	74.69±1.09[c]	73.69±0.95[c]	203.18±2.22[c]
Group IV	67.02±0.09[c]	54.25±2.66[d]	171.49±1.75[d]
Group V	57.73±0.64[a]	49.70±0.24[a]	108.80±1.07[a]
Group VI	78.50±0.46[c]	77.57±0.45[c]	210.50±1.80[c]
Group VII	70.39±0.59[d]	59.27±0.42[d]	180.02±1.95[d]
Group VIII	63.03±0.79 [c]	53.59±0.46[a]	116.19±1.00[e]
Group IX	56.19±0.25[a]	48.75±0.17[a]	102.51±0.06[a]
Group X	55.88±0.04[a]	49.40±0.34[a]	101.62±1.21[a]

Values are given in the table as means ± standard deviation for 6 rats.[a, b, c, d, e, f] means in the same row with different superscripts differ ($p < 0.05$).

Table 3: Effect of plant extract on glucose and carbohydrate metabolizing enzymes of the experimental animals.

Groups	Glucose (mg/dL)	Glucose 6 phosphatase (µmole of Pi liberated/min/mg protein)	Glucose-6-Phosphate Dehydrogenase (µmole of NADP reduced/min/mg protein)	Hexokinase (µM of Glucose utilized/min/mg protein)
Group I	139.33±0.71[a]	39.58±0.76[a]	253.9±1.3[a]	101.20±1.10[a]
Group II	102.00±0.73[b]	90.97±1.16[b]	114.9±1.1[b]	24.75±1.41[b]
Group III	110.83±0.60[c]	76.39±1.39[c]	175.7±2.9[c]	42.90±3.41[c]
Group IV	122.50±0.76[d]	69.10±1.46[d]	200.6±2.1[d]	61.60±2.91[d]
Group V	129.67±0.61[d]	44.79±0.89[a]	236.4±4.6[e]	97.90±1.63[a,e]
Group VI	106.67±0.67[b]	80.90±1.97[c,e]	169.8±1.9[c]	40.15±1.79[c]
Group VII	117.17±1.01[a]	73.47±1.53[c]	193.1±5.0[d]	59.40±1.70[d]
Group VIII	123.33±0.88[d]	48.96±0.89[a,f]	221.1±4.1[f]	94.60±2.20[e]
Group IX	131.67±0.33[a,d]	41.32±0.64[a,d]	247.2±2.1[a]	99.00±0.85[a]
Group X	137.83±0.65[a]	38.54±1.40[a]	251.9±4.0[a]	99.55±2.47[a]

Values are given in the table as means ± standard deviation for 6 rats.[a, b, c, d, e, f] means in the same row with different superscripts differ ($p < 0.05$).

barks TC, TG, LDL, HDL, VLDL, PL and FFA to near normal. In the present study, there is no significant alterations are noted in the plant alone treated group when compared with the control group. This was evidence of the safety of plants. The maximum dose of both leaves and bark of *P. pterocarpum* extracts action is similar to the standard drug silymarin treated group.

Liver Marker Enzymes

Table 2 shows the treatment of isoniazid and rifampicin 50:50 mg/BW induce the liver damage indicated significant increase ($p<0.05$) of the levels of AST, ALT and ALP. The various concentration of the extract of *P. pterocarpum* leaves and barks reduces the abnormal level of these marker enzymes to the normal level. Treatment of standard drug (silymarin) (25mg/kg) also decreased the elevated level of the liver enzymes. In the liver large numbers of enzymes are present. It mainly contains cytoplasmic enzymes like AST, ALT and ALP. The administration of isoniazid and rifampicin 50:50 mg/BW damages the cells of the liver and favours the leakage of cytoplasmic enzymes in the blood. The increasing level of these Enzymes predicts the severity of the damage which occurs in the liver.

The level of AST, ALT and ALP are significantly raised ($p<0.05$) in toxin isoniazid and rifampicin treated rats due to hepatic damage. After the administration of the extract of *P. pterocarpum* leaves and barks, the levels were reversed back to normalcy. This result proves the liver protective activity of the leaves and barks of the plant extract. The ethanolic extracts of the leaves and bark of *P. pterocarpum* shows higher activity in a concentration- dependent manner. As well as the plant extract alone has a similar value when compared with the control rats. This may prove the safety efficiency of the plant. The possible mechanism of the extract of *P. pterocarpum* leaves and bark in a toxin-induced animal model has given in Figure 1.

Table 4: Effect of plant extract on Urea, Uric Acid and Creatinine of the experimental animals.

Groups	Urea (mg/dL)	Uric Acid (mg/dL)	Creatinine (mg/dL)
Group I	20.25±0.07[a]	1.70±0.02[a]	1.12±0.01[a]
Group II	68.53±0.59[b]	3.06±0.06[b]	2.68±0.05[b]
Group III	52.97±0.13[c]	2.68±0.03[c]	2.12±0.01[c]
Group IV	38.18±0.27[d]	2.03±0.05[d]	1.70±0.02[d]
Group V	22.97±0.17[a]	1.81±0.04[a]	1.19±0.01[e]
Group VI	57.43±0.79[e]	2.87±0.03[e]	2.31±0.06[f]
Group VII	43.11±0.57[f]	2.34±0.09[f]	1.96±0.05[g]
Group VIII	27.92±0.48[g]	2.03±0.04[d]	1.41±0.06[h]
Group IX	21.22±0.16[a]	1.74±0.01[a]	1.17±0.01[a,e]
Group X	20.15±0.05[a]	1.46±0.01[a]	0.83±0.01[a,i]

Values are given in the table as means ± standard deviation for 6 rats.[a, b, c, d, e, f] means in the same row with different superscripts differ ($p < 0.05$).

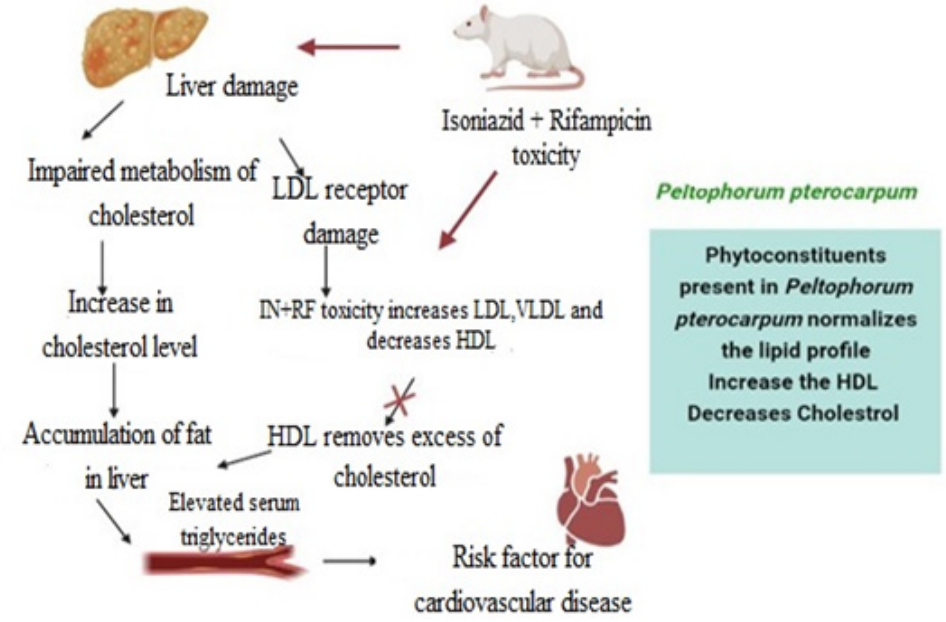

Figure 1: Schematic diagram of the mechanism of the extract of *P. pterocarpum* leaves and bark.

Glucose and carbohydrate metabolizing enzymes

Table 3 highlights the effects of ethanolic leaves and bark extract of *P. pterocarpum* on glucose as well as carbohydrate metabolizing enzymes such as glucose-6-phosphatase, glucose-6-phosphate dehydrogenase and hexokinase. The glucose-6-phosphatase and glucose-6-phosphate dehydrogenase is raised in isoniazid and rifampicin induced rats and the levels of glucose and hexokinase are reduced in the same. Administration of chlorpyrifos may alter the activities of glucose-6-phosphatase and glycogen phosphorylase, whereas, it caused a significant inhibition in the levels of hexokinase and glycogen content.[21] After the administration of the extract of *P. pterocarpum* leaves and bark significantly alters the level which is similar to the silymarin treated group. There are no remarkable changes were noted in the plant alone treated rats when compared to the control rats.

Glucose-6-phosphatase is playing a vital role in the breakdown of glycogen and the formation of glucose from non-carbohydrate sources.[22] The results of the present study noted the elevated level of glucose-6-phosphatase because of the liver damage due to the toxin. After the administration of the leaves and bark extract of *P. pterocarpum*, the enzyme level is reduced. The effect is maybe the liver protective effect and cell regeneration effect of the plant extract. The rising level of glucose-6-phosphatase may reduce the events of glycogenolysis and it subsequently leads to the reduction of glucose in the blood. Hexokinase is the first enzyme involved in the glycolysis process, catalyzing glucose phosphorylation by reducing ATP into glucose-6-phosphate. It is the enzyme that

controls the glycolysis process. The glucose 6-phosphate inhibits the action of hexokinase, hence if the further pathways are slow and if it inhibits phosphofructokinase, then glucose 6-phosphate will raise and subsequently inhibit the activity of hexokinase level in blood. Normally, the increasing concentration of glucose-6-phosphate reduces the hexokinase level.[23]

Glucose-6-phosphate dehydrogenase is an important carbohydrate metabolizing enzyme present in the liver. The deficiency of this enzyme is related to the risk of severe hemolytic anaemia due to the contact of the liver to oxidative stress.[24] The decreasing concentration of this enzyme is related to the increasing concentration of bilirubin in the blood. This is evident in this present study also. The results showed that the level of glucose-6-phosphate dehydrogenase is reduced because of liver damage due to oxidative stress. There is an increased level of this enzyme are noted after the administration of the ethanolic leaves and bark extract of *P. pterocarpum*.

Kidney Markers

Table 4 illustrates the levels of renal function markers (urea, uric acid and creatinine). These were found to be increased in the serum of toxin isoniazid and rifampicin induced rats. Oral administration of ethanolic leaves and bark extract of *P. pterocarpum* and silymarin resulted in a near normalization of these markers. There is no significant changes is noted in the plant extract alone treated group of the rats. This may prove the safety of the plant extract. Urea is the end product of protein metabolism. Excessive protein degradation may increase the urea level in blood. The excess amount of urea is excreted in the urine denotes the kidney disorder. This statement is proved in the present study also. In the present study, the level of urea is increased significantly ($p<0.05$) in isoniazid and rifampicin-induced hepatotoxic rats when compared to the control. After, the treatment of silymarin and ethanolic leaves and bark extract of *P. pterocarpum* significantly ($p<0.05$) restored the level when compared to the control. The decreasing protein degradation may reduce the urea level in blood. Uric acid is the major product of purine nucleotides; creatinine is endogenously produced and released into the body fluids and its clearance was measured as an indicator of glomerular filtration rate.[25,26] In the present study, the level of uric acid and creatinine is increased significantly ($p<0.05$) in toxin-induced rats when compared to the control. After, the treatment of ethanolic leaves and bark extract of *P. pterocarpum* significantly ($p<0.05$) decreases the level when compared to the standard drug silymarin treated group. In the present study, the increased urea, uric acid and creatinine level during isoniazid and rifampicin treated conditions may be impaired renal function which occurs as a minor event to reduce hepatocellular function.[27] After, the treatment of ethanolic leaves and bark extract of *P. pterocarpum* significantly alters the level when compared to the standard drug silymarin treated group. In the plant extract alone treated group there are no significant changes noted when compared to the control group. This may prove the safety efficacy of the plant extract.

CONCLUSION

In this study, the leaves and bark extract of *P. pterocarpum* have the potential hypolipidemic and hepatoprotective activity against the toxin treated rats. It also regulates the activity of liver enzymes, carbohydrate metabolizing enzymes and kidney marker levels. The results prove the beneficial nature of both the leaves and barks of *P. pterocarpum*. Further compound identification and molecular levels studies are warranted.

ACKNOWLEDGEMENT

The authors were grateful to their institutional authorities for their support.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

MJ: Maria Jerline; **RM:** Ramasamy Manikandan; **MS:** Muthukrishnan Saradhadevi; **BB:** Balasubramanian Balamuralikrishnan; **MY:** Mohd Younis; **AVA:** Arumugam Vijaya Anand.

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Cite this article: Jerline M, Manikandan R, Saradhadevi M, Balamuralikrishnan B, Younis M, Anand AV. Influences of Ethanolic Extract of *Peltophorum pterocarpum* Leaves and Bark on Lipid Profile, Liver and Carbohydrate Metabolizing Enzymes on Toxin-induced Wistar Rats. Free Radicals and Antioxidants. 2023;13(1):34-40.