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Research Article

PHARMACOLOGICAL AND BIOCHEMICAL EVALUATION OF AQUEOUS PROP-ROOTS EXTRACT OF FICUS BENGALENSIS (APREFB) AGAINST NAFD INDUCED BY HFHF DIET IN ALBINO RATS

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Abstract

Fat accumulations in liver cells cause (NAFLD), a disorder linked to oxidative stress, metabolic syndrome, and obesity. It was examine if Ficus Bengalensis aqueous prop-roots extract (APREFB) protected rats from (HFHF) fatty liver disease. Five sets of thirty male rats were used: Group-1: control group with control diet), Group-2: HFHF group as a positive control (rats fed a HFHF diet) for 20 weeks, Group-3: HFHF+APREFB (250 mg/kg) group, Group-4: HFHF+ APREFB (500 mg/kg) group, and Group-5: HFHF+ OrlistaI120 mg/kg) group. To examine fasting blood glucose levels and pertinent metabolic markers, blood samples were drawn at various intervals. Giving tissue enzymes, hyperglycemia profile, oxidative stress indicators in liver tissue, and histological analysis were used to assess the course of NAFLD. Our findings demonstrated that 16 weeks of HFHF treatment resulted in hyperglycemia, hyperlipidemia, and an increase in liver enzymes. In addition, the HFHF GSH levels and higher hepatic MDA to the control group.

A histological analysis confirmed the findings, revealing pathological alterations in the HFHF group. However, when APREFB was taken orally, all of these alterations were better in comparison to HFHF+ OrlistaI120 mg/kg and positive control groups. The liver index, blood glucose level, and weight increase all significantly decreased ($P < 0.05$) as a result of APREFB. When compared to the group fed an HFHF diet alone, APREFB at all experimental doses significantly reduced the levels of triacylglycerol, cholesterol, low-density lipoprotein, and malondialdehyde in plasma and significantly raised the level of cholesterol ($P < 0.05$).

This study demonstrated that APREFB reduced the oxidant impact, hyperglycemia, and dyslipidemia linked to a high-fat, high-fructose diet. At last, we are able to Our results imply that APREFB ingestion might have protective effects against the advancement of NALFD. Further research on APREFB's prophylactic potential in humans is necessary since it should be viewed as an alternative natural prophylactic medication against this disease.

Keywords: Aqueous Prop-roots Extract of *Ficus Bengalensis* (APREFB), High fat–high Fructose (HFHF), Prophylactic Medication.

1. Introduction

One in three adults have this kind, which is extremely prevalent. NAFLD comes in two forms: Simple fatty liver: This indicates that although the liver contains fat, there may not be any inflammation or cell damage. NASH, or nonalcoholic steatohepatitis, NASH indicates inflammation in the liver. Liver cancer and other severe issues like cirrhosis and fibrosis, which are forms of liver scarring, can result from the inflammation and damage to the liver cells caused by NASH. NASH is present in about 20% of NAFLD patients[2].

A. Causes: Storage of fats in the liver as a result of eating too many calories. can also result from rapid weight loss, alcohol misuse, and starvation[3].

B. Risk Factors for Fatty Liver Disease: Enlarged liver or jaundice etc.

C. Diagnosis: Tests for liver function to identify and track liver damage and illness, Hepatitis C, hemoglobin A1c, which indicates how stable your blood sugar is, a lipid profile to assess blood lipids, such as cholesterol and triglycerides, an ultrasound, Liver biopsy and fibrosis.

2. Material & Methods

2.1 Plant Material

A. Drying and Size Reduction: The selected part of plant was dried for 15 days. Then different parts of selected medicinal herbal plants were powdered as well as stored at 35 ºc in air protected container.

B. Extraction of Plant Material: 500g of prop root powder was combined with distilled water after the powder was extracted using the aqueous extraction method in a Soxhlet device at temperatures between 35 and 400C. The mixture was filtered using a Buchner funnel, and it was then dried over a water bath until it was completely dry. The yield percentage was 9.1%[4].

2.2 Determination of Acute Lethal dose (LD50)[6]

Depending on the dosage, the crude extracts (200–2000 mg kg) caused physical symptoms such palpitations, depression, drowsiness, and even death. LD50 values in mice was used.

2.3 Procedure

Young adult in good health for this investigation, Swiss albino mice weighing between 30 and 35 grams of either sex were employed. The mice were starved before the following day's dosage experiment. Both the dose and the body weight of each starved mouse were calculated. For the purpose of administering 250, 500, 750, and 1000 mg/kg of each plant extract, 36 mice were split into six groups (n=6). Six groups of mice, each including five mice, received intraperitoneal injections of the research extract at varying doses after it had been dissolved in distilled water. Mortality after 24 hours was recorded in order to assess LD_{50}. Group 1: Mice were administered 50 mg/kg of extract; at this dosage, the mice exhibited typical behavior. Group 2: Mice were given 250 mg/kg of extract; at first, this amount caused discomfort, but occasionally, the mice returned to normal. Group 3: Mice were given 500 mg/kg of extract; at first, this amount caused discomfort, but occasionally, the mice returned to normal. Group 4: Mice were administered 1000 mg/kg of extract; at this dosage, 30 to 50% of the mice perished. Sixty to eighty percent of the mice in Group 5 died after receiving an extract dose of 1500 mg/kg. Group 6: Mice were administered extract dose of 2000 mg/kg; 90–100% of the mice perished at this dose. Individual mice were monitored for 30 minutes after medication, and changes in the mixed mice were assessed at several points during the first 24 hours. 1. Behavioral profile: Awareness: Passivity, Stereotypy, Visual Placement, and Alertness. 2. Mood: irritation, fear, restlessness, and grooming. 3. Neurological profile: Motor activity: corneal reflex, grip strength, tremor, touch reaction, pain response, and startle response. Defecation, Urination, and Writing Autonomic Profile respiration rate, heart rate, and pile erection.

Prior to determining probits, the percentage dead for 0 and 100 are adjusted as follows: Adjusted

percentage. The formula for both 0% and 100% mortality is: 100 for 0% dead (0.25/n) One hundred (n-0.25/n) dead The dose that corresponds to probit 5, or 50%. Dosage selection: For any further pharmacological activity, a treatment dose of 110 and 1/5 of the maximum tolerated dose was used. Analysis of statistics: The mean ± SD was used to describe the data.

2.4 Evaluation of Antifatty Liver Activity

A curative model was used to test the plant extract in rats. Five groups—the control group, the high-fat high-fructose (HFHF) group, two treatment groups, and one standard group—Six rats (n = 6) per group were used in all experiments. For nine weeks, the rats in the control group were given a regular chow diet. For nine weeks, all animals—aside from the control group (Group 1)—were given a high-fat diet that included 25% fructose (HFHF) in their drinking water.

At 0, 2, 5, and 9 weeks, the Accu-Chek glucometer and Test Strip (Infopia Co., Ltd., South Korea) were used to measure the blood glucose level via the tail vein. Animals fed an HFHF diet and having a glucose level of ≥110 mg/dl at the fifth week were split into five groups. Group 2 received an HFHF diet exclusively during the experiment, while groups 3, 4, and 5 received HFHF diets with X, Y, and Z mg/kg body weight of plant extract, respectively, throughout the experiment. Group 5 received HFHF diets with Z mg/kg body weight of a standard drug, administered orally via gavage during the final four weeks.

Blood samples were taken from the retro-orbital sinus at the conclusion of the trial in 2 ml Ependroff tubes in order to isolate serum. All of the liver and serum samples were promptly preserved for later use at -80°C[7].

2.5 Experimental Procedure

Induction of Liver Toxicity by HFHF

The liver toxicity in this model was induced by HFHF diet in animal by oral route of administration. The study period was of 9 weeks.

Design of the Experiment

The 3rd and 4th groups were studied for preventive regimen whereas 5th group is studied for standard regimen further histopathological study.

Heavy diet formula typically consists of a high percentage of calories coming from both fat and fructose (corn syrup), with a typical breakdown being around 40-50% of calories from fat and 20-30% from fructose, while the remaining calories would come from protein and other carbohydrates. High fat (45%) sources: Lard, coconut oil, butter, palm oil, High fructose (25%)source: High Fructose Corn Syrup (HFCS), Protein (15%) source: casein or soy protein, Carbohydrates (besides fructose): corn or wheat.

Assessment of Liver Protective Activity

A. Liver Homogenate Analysis

Each animal's liver was separated, and their fresh weight was noted. After that, it was thoroughly maintained at 4ºc as and mixed in cold potassium phosphate buffer (0.05 M, pH 7.4), and centrifuged for 15 minutes at 5000 rpm and 0°C16.The following were determined using the resultant supernatant: 1) Lipid peroxidation (LPO), 2) Catalase (CAT) 3) Glutathione S-transferase (GSH) 4) Superoxide Dismutase (SOD).

Table 1: Treatment protocol for a period of 09th weeks

S. No.	Animal groups	Treatment
1.	Control	Normal basal diet + distilled water (non-obesity control)
2.	Induced	HFHF for 9 weeks (induced obese rats)
3.	Test-1	HFHF + 250- mg/kg, p.o. of **APREFB**
4.	Test-2	HFHF + 500- mg/kg p.o. of **APREFB**
5.	Standard	HFHF + standard drug, orlistat (120 mg/kg body weight p.o.)

3. Results

3.1 Phytochemicalinvestigation

Phytochemical tests of **aqueous prop-roots extract of *Ficus Bengalensis APREFB*** showed the Anthocyanins were absent in ***APREFB.***

Acute Toxicity Study

At doses up to 250 mg/kg of plant extract, the test animals showed no discernible changes in physical appearance or behavioral pattern at the end of a 24-hour period of general observation compared to the control.

Following intraperitoneal administration, the LD50 of the APREFB extract in rats was determined to be 2000 mg/kg. The table 2 displays the outcomes of administering a plant extract intraperitoneally to rats. Animals given intraperitoneal injections experienced ataxia and contractions of the abdomen muscles that lasted for a few hours. They were sleepy and less responsive at the sixth hour. The dose level had an impact on how severe these effects were. The majority of survivors, however, had overcome these symptoms by the twenty-fourth hour.

Compared to dosages of plant extract for its pharmacological activity, the LD50 values shown here injection are 10–15 times higher.

Table 2 for 0% dead: 100(0.25/n), For 100% dead: 100(n-0.25/n).

Figure 1 for 0% dead: 100(0.25/n), For 100% dead: 100(n-0.25/n)

Table 2: The determination of the LD50 after intraperitoneal injection in rat (n=06)

Group No.	Dose (mg/kg) of *APREFB*	Log Dose of *APREFB*	No. of Deaths	% Deaths	*Correct ed %	Probits
1.	50	1.7	0/6	0	4.16	3.04
2.	250	2.4	0/6	0	4.16	3.04
3.	500	2.7	0/6	0	4.16	3.04
4.	1000	3	0/6	0	4.16	3.04
5.	1500	3.18	2/6	33.33	33.33	4.56
6.	2000	3.3	3/6	66.66	66.66	5.00

*Corrected % Formula: For 0 and 100 % deaths.

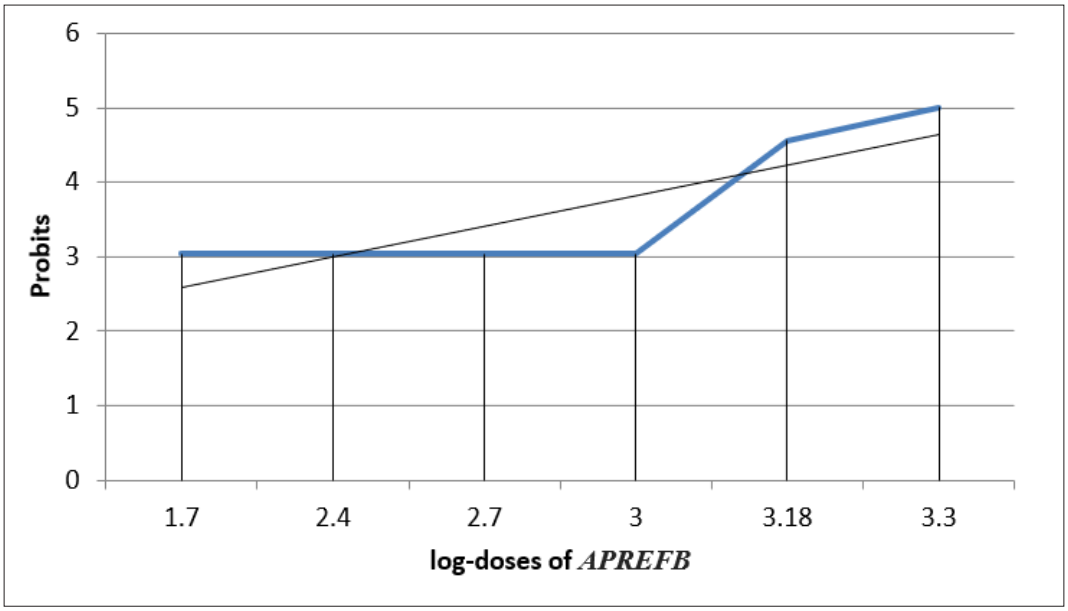

Figure 1: Plot of log-doses versus probits

Extract of ***APREFB*** was found relatively safe at the dose up to 150 to 500 mg/kg, b.w. i.p. to experimental animals. The extract was deemed safe for additional pharmacological screening at doses of 125, 250, and 500 mg/kg, b.w. i.p. The 1/10 or 1/5 of the LD50 was taken as the dose for the evaluation of anti-inflammatory activity because it did not cause any toxic symptoms of mortality in rats up to the dose level of 500 mg/kg body weight.

Finally it was observed that in-vitro anti-oxidant method for determining the in-vitro anti-oxidant activity as the concentration of ***APREFB*** was increased. When compared to the BHT by ANOVA, the ***APREFB*** was found to be less efficient ($P < 0.05$) in the DPPH radicals scavenging assay.

- mean ± SE
- control at $P < 0.05$
- HFHF at $P < 0.05$

HFHF + **APREFB** and HFHF groups at $P < 0.05$

The liver index was calculated by the equation (liver weight/body weight) × 100).

Table 3: The effect of HFHF and APREFB on (IBW), (FBW), (BWG), liver weight and liver index

Animal Groups	IBW (g)	FBW (g)	BWG	Liver weight (g)	Liver index %
Control	131.00 ± 1.4	316.33 ± 2.8	185.33 ± 11.8	6.82 ± 0.4	2.16 ± 0.5
Induced	129.00 ± 1.6	382.50 ± 4.5[a]	253.5 ± 14.3[a]	11.35 ± 0.4[a]	2.97 ± 0.3[a]
APREFB (250 mg/kg)	131.40 ± 0.44	375.00 ± 3.2[a]	243.6 ± 12.1[a]	8.79 ± 0.7[ab]	2.34 ± 0.4[a]
APREFB (500 mg/kg)	131.70 ± 0.67	359.17 ± 2.9[ab]	227.47 ± 12.9[ab]	8.28 ± 0.6[ab]	2.31 ± 0.2[ab]
Standard Orlistat (120 mg/kg)	130.00 ± 1.78	332.22 ± 2.2[a]	202.22 ± 13.4	8.18 ± 1.2[ab]	2.46 ± 0.3[ab]

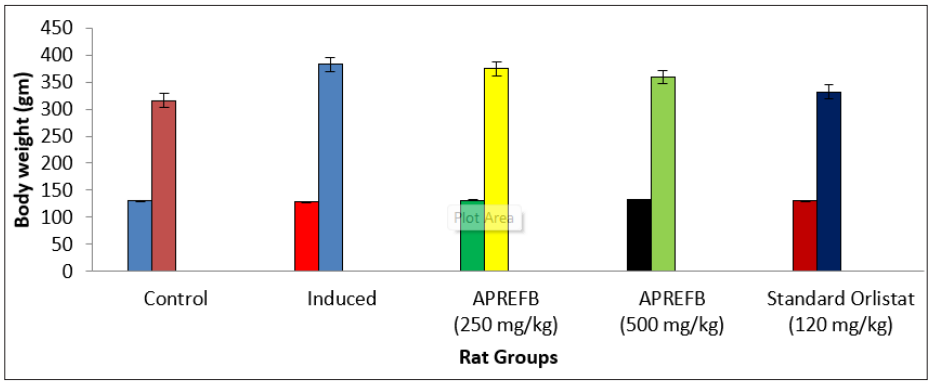

Figure 2: The effect of HFHF and APREFB on body weight

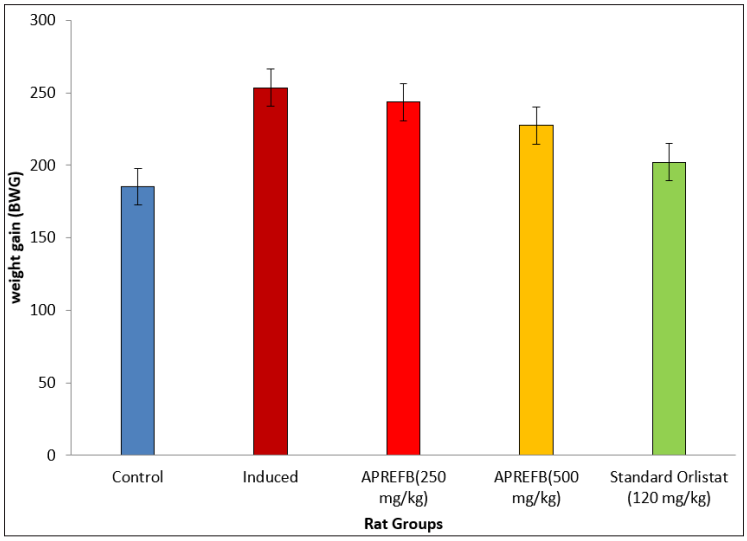

Figure 3: The effect of HFHF and APREFB on body weight gain (BWG)

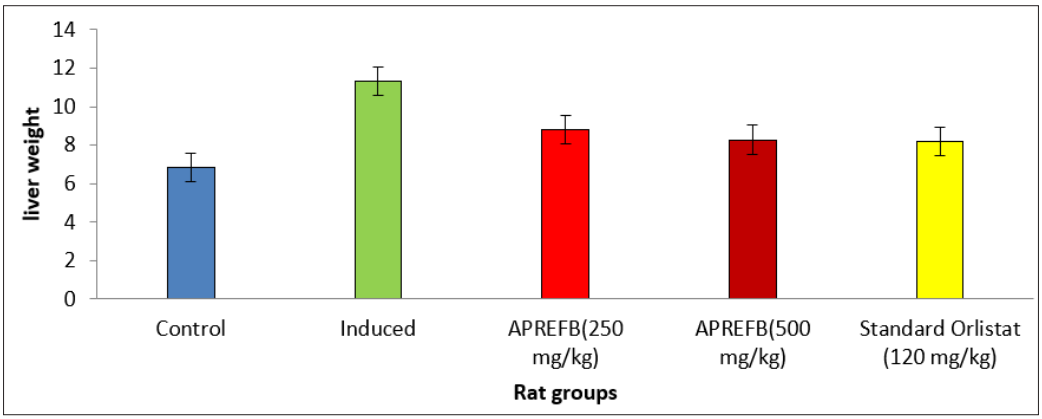

Figure 4: The effect of HFHF and APREFB on liver weight

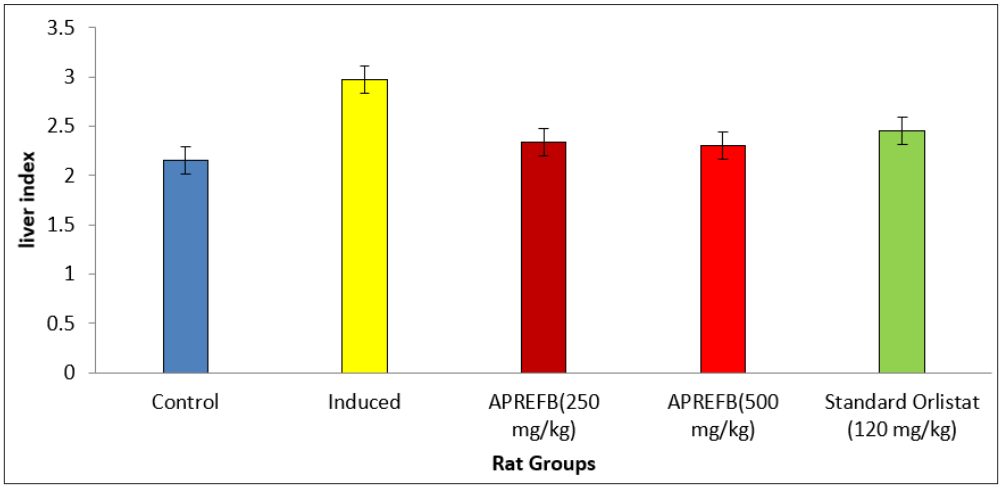

Figure 5: The effect of HFHF and APREFB on liver index

Table 4: The effect of HFHF and APREFB on serum liver enzymes

Animal Groups	Liver enzymes	
	SGPT (IU/L)	SGOT (IU/L)
Control	26.67±1.27	48.17±1.7
Induced	62.32±2.94[a]	79.01±2.1[a]
APREFB (250 mg/kg)	47.67±1.23[ab]	57.67±1.4[ab]
APREFB (500 mg/kg)	43.17±2.4[abc]	56.50±1.12[b]
Standard Orlistat (120 mg/kg)	41.17±1.23[ab]	50.22±1.2[bc]

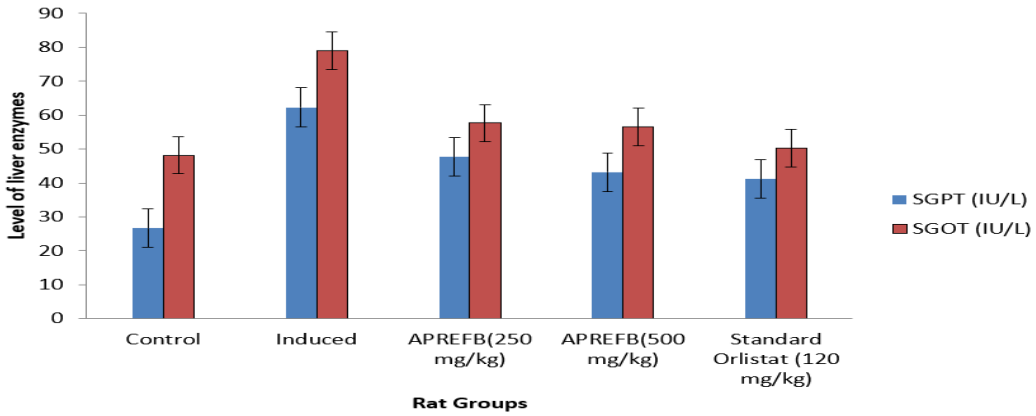

Figure 6: The effect of HFHF and APREFB on serum liver enzymes.

Effect of HFHF and APREFB on the activities of serum levels of SGPT and SGOT. Table 4 revealed that HFHF considerably increased serum SGPT and SGOT levels ($P<0.05$) when compared to the normal group, whereas the combination therapies significantly decreased SGPT and SGOT levels.

Table 5: The effect of HFHF and APREFB on serum glycemic profile

Animal Groups	Glycemic profile		
	FBG mg/dl	FBI Um/ml	HOMA-IR
Control	68.13±2.1	11.5±0.7	1.54±0.02
Induced	177.5±2.7[a]	8.8±0.2[a]	3.48±0.04[a]
APREFB (250 mg/kg)	153.5±2.3[a]	12.8±0.6[ab]	2.7±0.02[ab]
APREFB (500 mg/kg)	73.5±1.3[b]	11.9±0.5[b]	2.38±0.05[ab]
Standard Orlistat (120 mg/kg)	86.41±1.2[b]	9.5±0.4[ab]	2.51±0.07[ab]

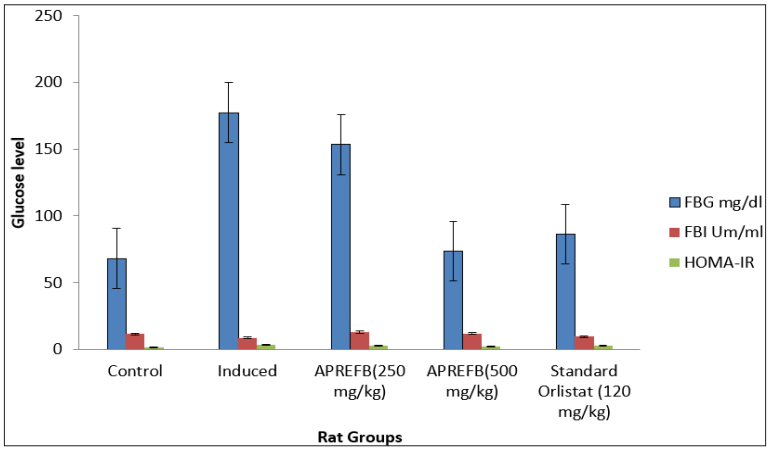

Figure 7: The effect of HFHF and APREFB on serum glycemic profile

Table 6: The effect of HFHF and APREFB on serum lipid profile

Groups	Lipid profile		
	S.CH (mg/dl)	S.TG (mg/dl)	HDL (mg/dl)
Control	165.37 ± 1.4	54.50 ± 2.32	29.51 ± 0.30
Induced	228.28 ± 2.2[a]	153.12 ± 1.2[a]	18.82 ± 0.12[a]
APREFB (250 mg/kg)	196.68 ± 1.6[ab]	126.23 ± 1.33[ab]	19.92 ± 0.13[a]
APREFB (500 mg/kg)	187.58 ± 1.14[ab]	65.44 ± 1.6[ab]	25.52 ± 0.13[ab]
Standard Orlistat (120 mg/kg)	169.25 ± 1.3[bc]	60.4 ± 1.1[b]	30.22 ± 016[bc]

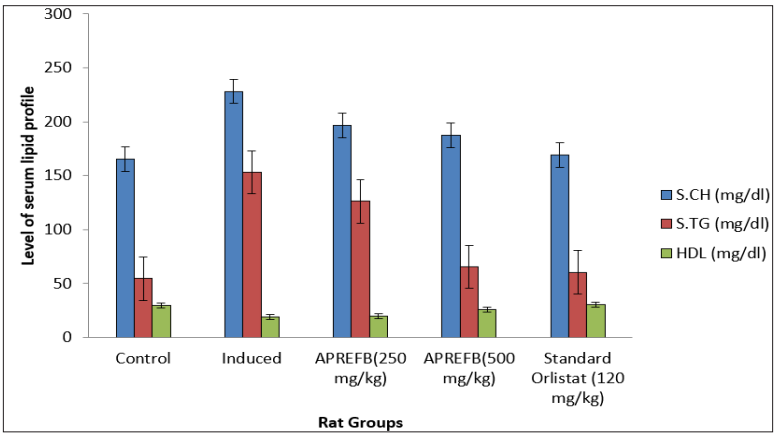

Figure 8: The effect of HFHF and APREFB on serum lipid profile

Table 7: The effect of HFHF and APREFB on serum Hepatic Oxidative stress profile

Groups	Hepatic Oxidative stress profile		
	MDA (µmol/g) wet. tissue	GSH (µmol/g) wet. tissue	SOD (U/g) wet. tissue
Control	1.52±0.03	26.7±0.24	1.59 ±0.06
Induced	2.51±0.1[a]	15.57±0.1[a]	1.36 ±0.23 [a]
APREFB (250 mg/kg)	1.81±0.03[ab]	18.44±0.09[ab]	1.51 ±0.16 [d]
APREFB (500 mg/kg)	1.78±0.07[b]	19.71±0.08[ab]	1.64 ±0.01 [d]
Standard Orlistat (120 mg/kg)	1.57±0.06[b]	22.22±0.12[ab]	1.62 ±0.03 [b, d]

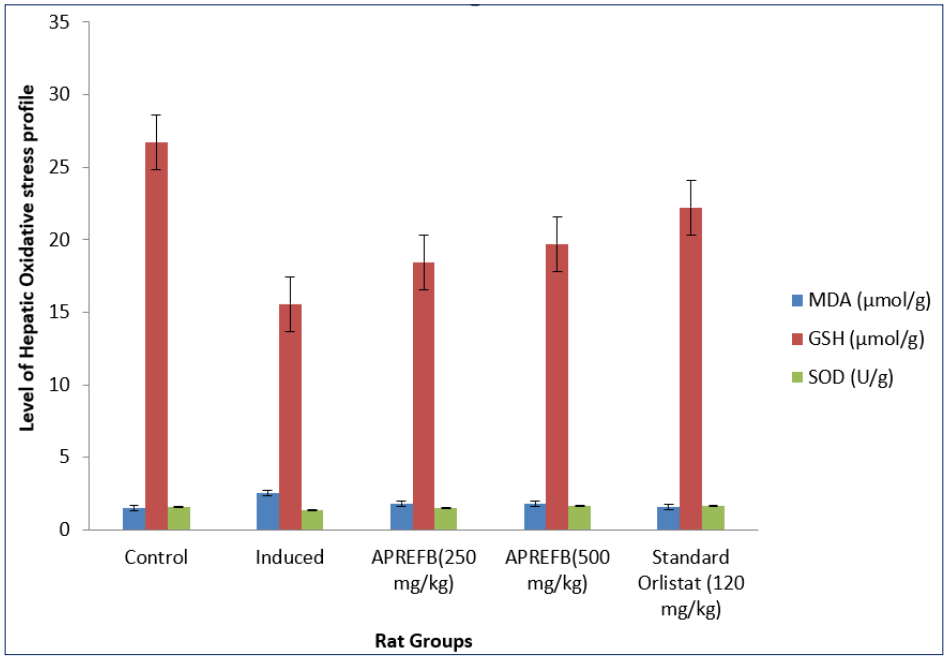

Figure 9: The effect of HFHF and APREFB on serum Hepatic Oxidative stress profile

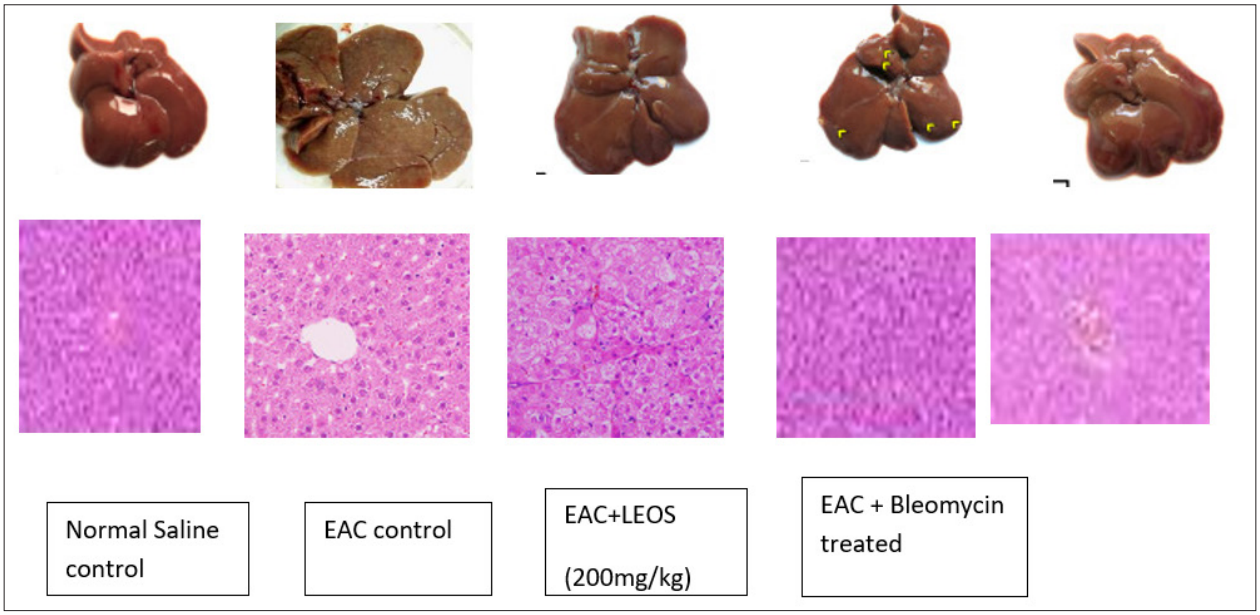

Figure 10 (a): Histopathological observations for liver of different groups

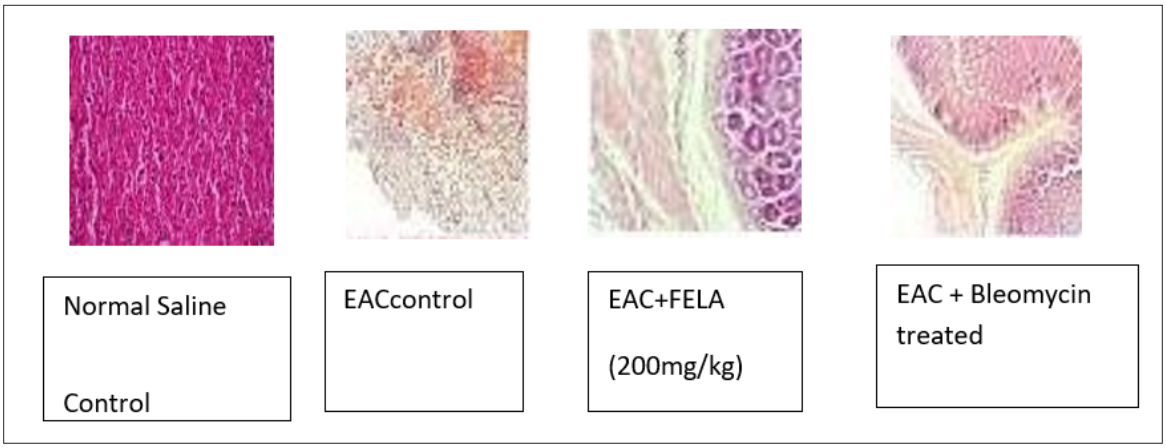

Figure 10 (b): Histopathological observations for liver of different groups in E.A.C. bearingmice: treated with root extract of Luffaacutangula.

4. Discussion

SThe impact of HFHF with APREFB on glycemic profile levels demonstrated a level ($P<0.05$) in rats fed the HFHF diet as opposed to the control group. APREFB, on the other hand, successfully decreased the glucose levels in rats fed an HFHF diet ($P<0.05$). Furthermore, APREFB-treated HFHF rats showed a recovery to nearly normal fasting blood glucose levels. Additionally, after 20 weeks, fasted animals' fasting blood insulin levels were assessed. According to the results of a one-way ANOVA, this levels significantly ($P<0.05$) decreased in comparison to the control group (Table 03).

Nonetheless, the APREFB-treated HFHF group reduce in fasting blood insulin than the HFHF group ($P<0.05$). Thus, in rats given HFHF treated with APREFB, the fasting blood insulin level returned to a range that was nearly normal. Additionally, as indicated in Table 03, the HFHF-fed rats' resistance in HOMA-IR was considerably ($P<0.05$) higher than that of the control group. Furthermore, as compared to rats given HFHF, the APREFB-treated HFHF groups showed significant changes ($P<0.05$).

Table 7 illustrates that a 16-week diet caused dyslipidemia, one of the NAFDs, as seen by a significant decrease in HDL levels and a considerable increase in blood levels of S.CH and S.TG when compared to normal control rats ($P<0.05$). Oral APREFB administration significantly increased HDL levels compared to rats fed HFHF diet ($P<0.05$) and effectively decreased with HFHF ($P<0.05$). Notably, all lipid profile indicators were reversed to values that were nearly normal, indicating that APREFB supplementation is superior in improving dyslipidemia.

Overall, the metrics, including MDA and GSH levels, were significantly different ($P<0.05$) across the groups, according to the data in Table 2. When compared to the control group, the HFHF group's MDA level was noticeably higher. In contrast, the identical group's GSH level is considerably lower ($P<0.05$). When compared to the HFHF group, oral APREFB administration significantly decreased the increase in MDA levels along with a significant increase in GSH levels ($P<0.05$).

4.1 Liver Histopathology

Administration of a fat diet resulted in the following: immune cell infiltration, binuclear hepatocytes, liver steatosis, severe ballooning degeneration of hepatocytes, glycogenated nuclei, which are primarily found in the vicinity of the portal tract, and Kupffer cells. There were a few resident lymphocytes in the portal connective stroma and normal histological architecture in the liver of control rats (Fig-10 a). Histopathological examination of the HFHF-treated group showed the presence of hepatocellular lesions, including necrosis, microvesicular steatosis, apoptosis, and hydropic degeneration (Fig. 10b).

5. Conclusion

The diet's high fructose and saturated fatty acid content promoted lipogenesis and inhibited insulin signaling. Additionally, a number of indicators of liver damage. The liver's tissue sustains metabolic damage as a result of the large flow of fructose within it. Rats in the current study were fed a high-fructose–high-fat diet (HFHF) for 16 weeks, according to the study's results, HFHF increased blood glucose, liver weight, liver index, total body weight gain, hypoinsulinemia, hyperlipidemia, and oxidative stress markers.

Researchers are attempting to treat a variety of herbal extracts that are less harmful than chemical medications because of the significant side effects of allopathic pharmaceutical treatments. This study uses APREFB as a medication for disease, taking into account its therapeutic potential on fatty liver caused by a high-fat diet (HFD) that causes non-alcoholic fatty liver.

Serum glucose levels in the group receiving APREFB in the current trial showed that it was contribute to APREFB's hypoglycemic effect.

According to this study, HFHF damaged the liver and showed a significant change in the serum activity of liver enzymes. Mice fed an HFHF diet develop obesity, which leads to NAFLD and increased transaminase activity. By lowering SGPT and SGOT levels, APREFB supplementation with an HFHF diet functions as a hepatoprotective, preventing fructose-induced hepatotoxicity and so minimizing liver injury. Thus, APREFB-restrained SGPT and SGOT levels in serum were demonstrated by our investigation. The study's data revealed that the group given high fructose concurrently with a high-fat diet had significantly higher levels of S.CH, S.TG, and LDL and significantly lower levels of HDL.

Because fructose is mostly metabolized in the liver, Fatty acids produced by fructose-induced DNL can subsequently be integrated into hepatic TGs or other lipid species. According to this study, APREFB increased HDL while significantly lowering S.CH, S.TG, and LDL levels.

Furthermore, reactive oxygen species (ROS) in the liver have been connected to the development of steatohepatitis from non-alcoholic steatosis. ROS cause harm to the liver tissue by increasing lipid peroxidation, damaging unsaturated lipids in the cell membrane, and lowering endogenous antioxidants. Furthermore, as previously described, fructose feeding has been linked to metabolic syndromes in rodents and has been demonstrated to increase oxidative stress. Our findings which show that rats fed the HFHFD had increased hepatic MDA, a sign of lipid peroxidation and decreased GSH as an antioxidant. As a result, APREFB's antioxidant quality aids in scavenging free radicals produced in a variety of circumstances linked to metabolic disorders.

The aforementioned findings suggest that sustained HFHF consumption may be a factor in the onset of NAFLD and raise the chance of developing NASH. Histopathological analysis of the liver, which shows the emergence of hepatic steatosis and hepatocyte damage, proves this. The detrimental effects of the HFHF diet were lessened by APREFB administration, which was also suggested as a viable dietary supplement to prevent the advancement of NAFLD. Lastly, in every parameter, APREFB shown ameliorative effects against the advancement of NALFD.

6. Conflict of Interest: None

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