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RESEARCH ARTICLE

Anti-hyperlipidemic Activity of Epipremnum aureum Root Extract in High- Fat Diet-Induced Hyperlipidemic Wistar Rats.

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Abstract: ***Background***: Hyperlipidemia, defined by elevated cholesterol and triglyceride levels, is a major contributor to cardiovascular and metabolic diseases worldwide. Sedentary lifestyles, dietary changes, and genetic factors have increased its global prevalence, particularly in developing countries. Although statins such as atorvastatin remain the standard therapy, their adverse effects—including hepatotoxicity and myopathy—have prompted interest in safer plant-based alternatives. Epipremnum aureum (Golden Pothos) contains flavonoids, alkaloids, tannins, and saponins known for antioxidant and lipid-lowering properties. While traditionally used for detoxification and wound healing, its roots have not been previously studied for anti-hyperlipidemic effects. This study therefore evaluated the lipid-modulating potential of Epipremnum aureum root extract (REEA) in a high-fat diet (HFD) rat model. Wistar albino rats were fed a modified HFD for four weeks to induce hyperlipidemia, followed by four weeks of treatment. Hydroalcoholic root extract was prepared using Soxhlet extraction, yielding approximately 10%. Thirty rats were divided into five groups: normal control, HFD control, REEA 100 mg/kg, REEA 200 mg/kg, and atorvastatin 10 mg/kg. Blood samples were collected at baseline, week 4, and week 8 for lipid profiling. Secondary assessments included body weight, abdominal circumference, BMI, and histopathological evaluation of liver, aorta, kidney, and adipose tissues. Data were analyzed using one-way ANOVA with $p<0.05$ considered significant.
The HFD control group showed marked elevations in total cholesterol, triglycerides, LDL-C, and VLDL-C, with reduced HDL-C, confirming successful hyperlipidemia induction. REEA produced dose-dependent lipid improvements. The 100 mg/kg dose caused mild reductions, whereas 200 mg/kg significantly decreased total cholesterol, triglycerides, LDL-C, and VLDL-C while elevating HDL-C. Atorvastatin produced the greatest overall effect but REEA 200 mg/kg showed meaningful improvement. Anthropometric parameters followed a similar pattern: BMI and abdominal circumference increased in the HFD group but decreased modestly with REEA, especially at 200 mg/kg. Histopathology supported these biochemical findings. HFD rats exhibited hepatic steatosis, aortic lipid deposition, and increased visceral fat. REEA at 200 mg/kg reduced hepatic fat accumulation, attenuated early atherosclerotic changes, and lowered mesenteric and perirenal fat deposition, without evidence of nephrotoxicity. In ***conclusion***, Epipremnum aureum root extract demonstrates significant anti-hyperlipidemic and mild anti-obesity effects in HFD-induced rats, particularly at 200 mg/kg. While less potent than atorvastatin, its favorable safety profile and phytochemical richness highlight its potential as a complementary therapeutic option. Further studies should explore its mechanisms and clinical applicability.

Keywords: Hydronephrosis, Renal Function, Ureteric Obstruction.

INTRODUCTION

Hyperlipidemia, characterized by elevated blood lipids like cholesterol and triglycerides, is a major risk factor for cardiovascular diseases (CVDs) and metabolic disorders.[1] The global prevalence of hyperlipidemia has risen due to sedentary lifestyles, poor diet, and genetic factors, contributing to a significant portion of worldwide deaths.[2] Developing nations, in particular, face a growing burden from this condition as a result of urbanization and lifestyle changes.[3,4] Despite the widespread use of statins like Atorvastatin, their associated side effects, such as hepatotoxicity and myopathy, necessitate the exploration of alternative treatments.[5] Consequently, there is a growing interest in plant-based medicines with fewer side effects. Medicinal plants containing bioactive compounds like flavonoids, alkaloids, and saponins have shown potential in managing hyperlipidemia through their antioxidant and lipid-modulating properties. Among these, *Epipremnum aureum* (Golden Pothos) has been historically recognized for its medicinal properties and warrants further investigation for its lipid-lowering potential.[6]

Phytochemical studies have identified bioactive compounds, such as flavonoids, alkaloids, saponins, and tannins, in various parts of the plant, which are likely responsible for its pharmacological properties. Although the plant has a history of use in folk medicine for general detoxification and wound healing, there is no documented traditional use for hyperlipidemia. However, the increasing interest in plant-based therapies for metabolic disorders has led to

scientific research into its potential. The roots of *Epipremnum aureum*, in particular, contain phytoconstituents with known lipid-lowering and antioxidant effects that can modulate lipid metabolism and oxidative stress, both of which are central to the development of hyperlipidemia.[7,8]

Despite the growing demand for plant-based treatments for dyslipidemia, there are currently no published studies that conclusively demonstrate the anti-hyperlipidemic effects of *Epipremnum aureum* root. This gap in the literature highlights a significant need for a systematic investigation into its therapeutic potential. This study aims to address this need by evaluating the anti-hyperlipidemic activity of an *Epipremnum aureum* root extract in a high-fat diet-induced hyperlipidemia model in Wistar albino rats. Further, the research focussed on comparing the extract's effects on key lipid markers—total cholesterol, triglycerides, HDL, and LDL—against those of the standard drug, Atorvastatin. Additionally, the study will assess any secondary anti-obesity effects of the extract.

MATERIAL AND METHODS

This experimental study employed a comparative study using a high-fat diet-induced hyperlipidemia model in Wistar albino rats to evaluate the anti-hyperlipidemic activity of *Epipremnum aureum* root extract (REEA). The study was conducted over an 8-week period with a 4-week induction phase followed by a 4-week treatment phase. All experimental procedures were reviewed and approved by the Institutional Animal Ethics Committee of Mahatma Gandhi Medical College & Research Institute, Puducherry (07/IAEC/MG/04/2023-I), ensuring compliance with the Committee for Control and Supervision of Experiments on Animals (CCSEA) guidelines.

Plant Material Collection and Authentication

Epipremnum aureum plants were procured from a certified nursery in Puducherry, India. Plant authentication was performed by a qualified botanist from the local science college, and a voucher specimen was deposited for future reference. Approximately 10 kg of fresh roots were collected. Diseased, decayed, or otherwise unsuitable portions are discarded prior to processing.

Extract Preparation and Standardization

The collected roots were thoroughly cleaned, shade-dried, and pulverized into fine powder using appropriate grinding equipment. The powder was stored in airtight containers under controlled conditions until extraction. Hydroalcoholic extraction was performed using the Soxhlet method as described by Mahanta & Mukherjee. Briefly, 45g of root powder was loaded into the Soxhlet apparatus and continuously extracted using 80% ethanol at 50-70°C. The resulting extract was collected in porcelain vessels and dried at room temperature (36- 39°C) for 72 hours, followed by lyophilization. The extraction yield was approximately 10% (~4.5g per cycle).

Animals and Housing Conditions

Thirty healthy male Wistar albino rats, weighing 180±10 g, were acquired from a CCSEA-approved vendor for this study. Following a seven-day acclimatization period in the Central Animal House facility of tertiary care hospital, the rats were housed in polypropylene cages with paddy husk bedding under controlled conditions of a 12-hour light/dark cycle and a temperature of 27∘C±2∘C. They were given *ad libitum* access to water and a standard pellet diet. For all experimental procedures, the animals were divided into groups of six.

Dose Selection and Safety Assessment

Building on prior acute toxicity studies by Abhinaiyani et al., which established the safety of *E. aureum* extracts with an LD50 greater than 2000 mg/kg, two doses were selected for this study: 100 mg/kg and 200 mg/kg, corresponding to 1/20th and 1/10th of the established LD50, respectively.[8]

Atorvastatin at 10 mg/kg was used as a positive control, representing the standard therapeutic intervention. All other reagents are of analytical grade and procured from, --.

High-Fat Diet Formulation

The hyperlipidemia-inducing diet was prepared by modifying Blank's method, combining 92% standard pellet diet with 2.0% cholesterol, 1.0% cholic acid, and 5.0% coconut oil. This diet was administered to the experimental groups (Groups 2 to 5) for 60 days, while the normal control group (Group 1) was maintained on a standard chow diet for the duration of the study.

RESULTS

Experimental Design and Group Allocation

Group	Treatment	Diet
Group 1	Normal Control	Standard Rat Chow
Group 2	HFD Control	High-Fat Diet (HFD) + 1% CMC
Group 3	REEA 100	HFD + REEA (100 mg/kg) in 1% CMC
Group 4	REEA 200	HFD + REEA (200 mg/kg) in 1% CMC
Group 5	Standard Control	HFD + Atorvastatin (10 mg/kg) in 1% CMC

Outcome Measures and Sampling Schedule

Primary outcomes included an assessment of the serum lipid profile, specifically total cholesterol, triglycerides, and high-density, low-density, and very low-density lipoprotein cholesterol levels. Secondary outcomes encompassed changes in anthropometric measurements, such as body weight, abdominal circumference, and body mass index, as well as the histopathological evaluation of the liver, aorta, kidney, and both mesenteric and perirenal adipose tissues. Blood samples for lipid analysis were drawn at baseline (day 0) and at bi- weekly intervals (days 14, 28, 42) throughout the study period.

Sample collection and terminal procedures:

Blood samples were collected from the tail vein of the animals in heparinized tubes at three distinct time points: baseline (Day 0), post-induction and pre-treatment (Week 4), and post-treatment (Week 8). At the end of the study (Week 8), all animals were humanely euthanized via a carbon dioxide chamber. A necropsy examination was then performed, and various adipose tissues, including mesenteric, subcutaneous, epididymal, and perirenal fat, were collected, weighed, and recorded.

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Biochemical Analysis

All biochemical analyses were performed at the Central Laboratory of MGMCRI using standardized clinical chemistry methods. Blood samples were collected from each animal via retro-orbital puncture, and the serum was separated by centrifugation at 3000 rpm for 15 minutes. The serum was then stored in sterile vials at -80°C to minimize degradation. All samples were analyzed in a single batch at the end of the study to eliminate inter-assay variability. Serum lipid profiles were analyzed using automated clinical chemistry analyzers following manufacturer protocols and quality control procedures.

Histopathological Examination

Tissue specimens from the aorta, mesenteric fat, perirenal adipose tissue, liver, and kidney were collected immediately after sacrifice, washed in normal saline, and fixed in 10% neutral buffered formalin. Fixed tissues were processed using standard histological techniques including paraffin embedding, sectioning at 5µm thickness using a microtome, and staining with hematoxylin and eosin (H&E). Histological evaluation was performed under light microscopy at both low power (10×) and high power (40×) magnification.

Statistical Analysis

Data were entered in Microsoft Excel and analysed using IBM SPSS (ver 16). Results are presented as mean ± standard deviation (SD). Statistical comparisons between groups were performed using one-way analysis of variance (ANOVA) followed by post hoc Bonferroni correction for multiple comparisons. Statistical significance was defined as $p<0.05$ for significant differences. All statistical tests were two-tailed.

Results

Induction of Hyperlipidemia

The high-fat diet successfully induced hyperlipidemia in all experimental groups (Groups 2 to 5) by the end of the 4-week induction period. All groups receiving the high-fat diet showed significant increases in serum total cholesterol, triglycerides, LDL-C, and VLDL-C levels, with concurrent decreases in HDL-C levels compared to the normal control group, confirming the establishment of the experimental hyperlipidemia model.

Lipid Profile Parameters Total Cholesterol

The HFD control group demonstrated a progressive increase in total cholesterol levels throughout the study period, rising from 44.6 ± 3.9 mg/dl at baseline to 66.8 ± 4.3 mg/dl at week 4, and further increasing to 84.0 ± 4.7 mg/dl at week 8, representing an 88% increase from baseline.

Treatment with REEA at 100 mg/kg resulted in a modest but statistically significant reduction in total cholesterol from 73.5 ± 9.3 mg/dl at week 4 to 70.6 ± 10.4 mg/dl at week 8 ($p < 0.05$ vs. HFD control). The higher dose of REEA (200 mg/kg) produced a more pronounced reduction from 74.1 ± 9.1 mg/dl to 68.1 ± 7.8 mg/dl ($p < 0.01$ vs. HFD control). Atorvastatin 10 mg/kg demonstrated the most significant cholesterol-lowering effect, reducing levels from 76.3 ± 6.7 mg/dl to 62.8 ± 10.7 mg/dl ($p < 0.01$ vs. HFD control).

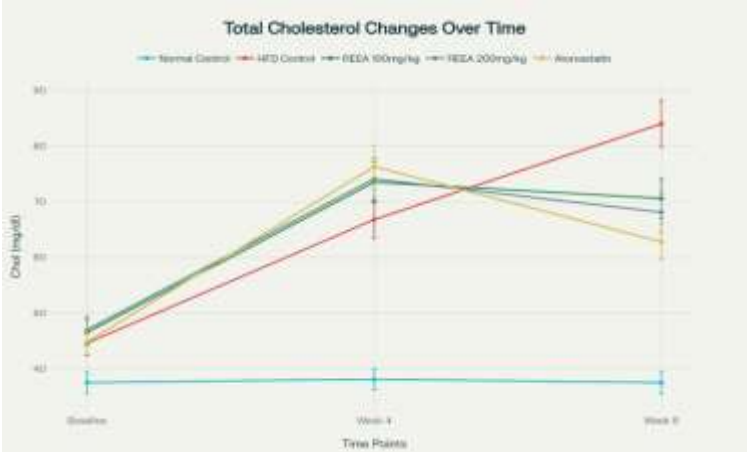

Figure 1: Total Cholesterol changes over time

Triglycerides

Triglyceride levels in the HFD control group increased progressively from baseline to week 4 and week 8, representing a 34% increase from baseline.

REEA at 100 mg/kg showed a non-significant reduction in triglycerides from 94.1 ± 5.7 mg/dl to 87.0 ± 12.2 mg/dl. The 200 mg/kg dose achieved a statistically significant reduction from 90.0 ± 8.6 mg/dl to 82.5 ± 7.6 mg/dl ($p < 0.05$ vs. HFD control). Atorvastatin produced the most substantial triglyceride reduction from 91.8 ± 7.5 mg/dl to 76.8 ± 8.8 mg/dl ($p < 0.01$ vs. HFD control).

High-Density Lipoprotein Cholesterol (HDL-C)

HDL-C levels in the HFD control group remained consistently low throughout the study, declining from 23.8 ± 4.1 mg/dl at baseline to 23.0 ± 2.5 mg/dl at week 8.

REEA at 100 mg/kg showed a non-significant increase in HDL-C from 26.0 ± 6.0 mg/dl to 36.0 ± 6.3 mg/dl. The 200 mg/kg dose resulted in a significant elevation from 27.8 ± 3.4 mg/dl to 43.0 ± 6.3 mg/dl ($p < 0.05$ vs. HFD control). Atorvastatin achieved the highest HDL-C increase from 25.1 ± 4.6 mg/dl to 52.0 ± 7.2 mg/dl ($p < 0.05$ vs. HFD control).

Low-Density Lipoprotein Cholesterol (LDL-C)

LDL-C levels in the HFD control group increased dramatically from baseline (10.5 ± 3.2 mg/dl) to week 4 (65.6 ± 1.9 mg/dl) and continued to rise to 69.0 ± 2.6 mg/dl at week 8, representing a 557% increase from baseline.

REEA at 100 mg/kg produced minimal LDL-C reduction from 67.0 ± 7.0 mg/dl to 66.0 ± 7.3 mg/dl (non-significant). The 200 mg/kg dose achieved a substantial reduction from 65.1 ± 2.8 mg/dl to 49.0 ± 4.8 mg/dl ($p < 0.01$ vs. HFD control). Atorvastatin demonstrated comparable efficacy with a reduction from 64.1 ± 5.0 mg/dl to 45.6 ± 5.9 mg/dl ($p < 0.01$ vs. HFD control).

Very Low-Density Lipoprotein Cholesterol (VLDL-C)

VLDL-C levels in the HFD control group increased from baseline (10.3 ± 1.0 mg/dl) to 29.1 ± 2.1 mg/dl at week 4 and 38.0 ± 1.2 mg/dl at week 8.

REEA at 100 mg/kg showed minimal reduction from 28.8 ± 0.9 mg/dl to 26.0 ± 1.7 mg/dl (non-significant). The 200 mg/kg dose achieved a significant reduction from 27.3 ± 1.7 mg/dl to 22.0 ± 1.4 mg/dl ($p < 0.01$ vs. HFD control). Atorvastatin produced the most pronounced VLDL-C reduction from 28.8 ± 0.75 mg/dl to 20.0 ± 0.89 mg/dl ($p < 0.01$ vs. HFD control).

Table 1: Complete Lipid Profile Throughout Study Period

Parameter	Group	Baseline (Week 0)	Week 4	Week 8	p-value
Total Cholesterol (mg/dl)	Normal Control	37.5 Â± 6.2	38.1 Â± 6.1	37.5 Â± 7.2	NS
	HFD Control	44.6 Â± 3.9	66.8 Â± 4.3	84.0 Â± 4.7	-
	REEA 100mg/kg	46.5 Â± 4.0	73.5 Â± 9.3	70.6 Â± 10.4	*p < 0.05
	REEA 200mg/kg	47.0 Â± 3.3	74.1 Â± 9.1	68.1 Â± 7.8	**p < 0.01
	Atorvastatin	44.8 Â± 2.6	76.3 Â± 6.7	62.8 Â± 10.7	**p < 0.01
HDL-C (mg/dl)	Normal Control	29.0 Â± 2.4	28.3 Â± 2.2	30.8 Â± 4.2	NS
	HFD Control	23.8 Â± 4.1	25.0 Â± 4.0	23.0 Â± 2.5	-
	REEA 100mg/kg	29.3 Â± 6.6	26.0 Â± 6.0	36.0 Â± 6.3	NS
	REEA 200mg/kg	31.5 Â± 3.3	27.8 Â± 3.4	43.0 Â± 6.3	*p < 0.05
	Atorvastatin	29.0 Â± 5.1	25.1 Â± 4.6	52.0 Â± 7.2	*p < 0.05
LDL-C (mg/dl)	Normal Control	8.5 Â± 4.2	11.3 Â± 2.4	10.6 Â± 3.2	NS
	HFD Control	10.5 Â± 3.2	65.6 Â± 1.9	69.0 Â± 2.6	-
	REEA 100mg/kg	12.8 Â± 4.7	67.0 Â± 7.0	66.0 Â± 7.3	NS
	REEA 200mg/kg	12.3 Â± 4.1	65.1 Â± 2.8	49.0 Â± 4.8	**p < 0.01
	Atorvastatin	12.0 Â± 5.0	64.1 Â± 5.0	45.6 Â± 5.9	**p < 0.01

Anthropometric Parameters

Abdominal Circumference

The HFD control group showed progressive increases in abdominal circumference from 16.1 ± 0.98 cm at baseline to 17.3 ± 1.3 cm at week 4 and 19.3 ± 0.8 cm at week 8.

Both REEA doses demonstrated reductions in abdominal circumference, though statistical significance was not achieved. REEA 100 mg/kg reduced circumference from 18.5 ± 1.0 cm to 18.1 ± 1.6 cm, while REEA 200 mg/kg showed reduction from 17.8 ± 0.75 cm to 17.6 ± 1.7 cm. Atorvastatin achieved a notable reduction from 19.1 ± 0.4 cm to 17.6 ± 0.8 cm.

Body Mass Index (BMI)

BMI in the HFD control group increased progressively from baseline (0.46 ± 0.04 g/cm²) to week 4 (0.58 ± 0.08 g/cm²) and week 8 (0.62 ± 0.03 g/cm²).

REEA at 100 mg/kg reduced BMI from 0.65 ± 0.1 g/cm² to 0.53 ± 0.06 g/cm² (non-significant). REEA at 200 mg/kg achieved a significant reduction from 0.63 ± 0.05 g/cm² to 0.47 ± 0.07 g/cm² ($p < 0.05$ vs. HFD control). Atorvastatin produced the most substantial BMI reduction from 0.61 ± 0.12 g/cm² to 0.43 ± 0.04 g/cm² ($p < 0.01$ vs. HFD control).

Table 2: Anthropometric Parameters and Metabolic Outcomes

Parameter	Group	Week 4	Week 8	% Change	Significance
BMI (g/cmÂ²)	HFD Control	0.58 Â± 0.08	0.62 Â± 0.03	34.80%	-
	REEA 100mg/kg	0.65 Â± 0.10	0.53 Â± 0.06	-18.50%	NS
	REEA 200mg/kg	0.63 Â± 0.05	0.47 Â± 0.07	-25.40%	*p < 0.05
	Atorvastatin	0.61 Â± 0.12	0.43 Â± 0.04	-29.50%	**p < 0.01
Abdominal Circumference (cm)	HFD Control	17.3 Â± 1.3	19.3 Â± 0.8	11.60%	-
	REEA 100mg/kg	18.5 Â± 1.0	18.1 Â± 1.6	-2.20%	NS
	REEA 200mg/kg	17.8 Â± 0.75	17.6 Â± 1.7	-1.10%	NS
	Atorvastatin	19.1 Â± 0.4	17.6 Â± 0.8	-7.90%	NS

Histopathological Findings

Histopathological examination revealed dose-dependent protective effects of REEA across multiple organ systems. In hepatic tissue, the HFD control group exhibited microvesicular steatosis and periportal inflammation consistent with non-alcoholic fatty liver disease. REEA treatment at 100 mg/kg showed moderate steatosis with reduced inflammation, while the 200 mg/kg dose demonstrated mild steatosis and minimal inflammation, indicating hepatoprotective activity.

Cardiovascular tissue examination revealed that the HFD control group developed aortic lipid deposits and myocardial necrosis indicative of early atherogenic changes. Both REEA treatment groups showed attenuated atherosclerotic and myocardial alterations compared to the HFD control. Adipose tissue analysis demonstrated significant accumulation in the HFD control group, with both REEA doses showing reduced perirenal and mesenteric fat deposition. No nephrotoxic effects were observed in any treatment group.

DISCUSSION

Despite the widespread availability of antihyperlipidemic drugs, a significant portion of patients fail to reach their lipid targets, largely due to medication side effects, poor adherence, or statin intolerance. This contributes substantially to the global burden of cardiovascular diseases, which remain the leading cause of mortality. Consequently, there is an ongoing need for more effective and better-tolerated therapeutic agents.

In our study, the control group on a high-fat diet showed a progressive increase in total cholesterol over eight weeks, confirming a successful induction of hyperlipidemia. This observation is consistent with established rodent models. Treatment with REEA at a dose of 100 mg/kg produced a mild reduction in cholesterol, while a 200 mg/kg dose resulted in a moderate decrease.9

Although atorvastatin demonstrated a more pronounced effect, the cholesterol- lowering potential of REEA is considered clinically meaningful and aligns with findings from similar studies. Epipremnum aureum extract has demonstrated significantly lower total cholesterol and improved lipid profiles in rat models, possibly by modulating key pathways such as HMG-CoA reductase and LDL receptor expression. This biological plausibility supports our observed effects and justifies further investigation into REEA's therapeutic potential. Similarly, ethanolic extract of Epipremnum aureum has been shown to significantly lower TC—and improve lipid profiles—in high-fat diet or

streptozotocin- induced hyperlipidaemic and diabetic downregulation of HMG-CoA reductase and upregulation of LDL receptor expression.10 This aligns with the observed cholesterol-lowering effect of REEA and strengthens its biological plausibility.

Phytochemical analysis of Epipremnum aureum roots revealed significant levels of phenolics and flavonoids, including catechins, quercetin, and rutin, which are known to inhibit cholesterol biosynthesis and enhance bile acid excretion.11,12 The cholesterol- lowering effect observed in our study, particularly with a 200 mg/kg dose of the root extract, is consistent with prior research showing that quercetin supplementation can reduce total cholesterol in hyperlipidemic models.13

In this study, high-fat diet (HFD) controls showed a progressive increase in plasma triglycerides (TG), a finding that aligns with documented trends in diet-induced hyperlipidemia models.14,15 Although atorvastatin demonstrated a more pronounced TG-lowering effect, the moderate reduction seen with the root extract is promising. The mechanism behind this effect is likely related to the extract's bioactive compounds, such as flavonoids and triterpenoids, which are known to activate lipoprotein lipase and inhibit hepatic diacylglycerol acyltransferase (DGAT).16,17 While the higher dose of the extract did not outperform atorvastatin, its efficacy, coupled with a potentially more favorable side- effect profile, warrants further investigation.18

Low-density lipoprotein (LDL) is a central factor in the development of atherosclerosis, with elevated levels directly linked to cardiovascular disease. Consistent with established literature on diet-induced dyslipidemia, this study found a significant increase in LDL levels in rats fed a high-fat diet (HFD).19 While the 100 mg/kg dose of REEA produced only a modest reduction in LDL, the 200 mg/kg dose demonstrated a more pronounced decrease, suggesting a dose-dependent lipid-lowering effect. However, it is important to note that Atorvastatin (10 mg/kg) proved to be more potent in reducing LDL levels than either dose of REEA.

The mechanism behind REEA's effect is likely related to its rich content of phytosterols and polyphenols. Phytosterols are known to compete with cholesterol for absorption in the intestine, while polyphenols may upregulate hepatic LDL receptors and downregulate HMG-CoA reductase, an enzyme targeted by statins.20 While not as potent as Atorvastatin, REEA could be a valuable complementary agent for patients who prefer phytomedicine or as part of a multi-drug regimen, offering additional benefits such as

promoting reverse cholesterol transport and providing anti-inflammatory, antioxidant, and endothelial-stabilizing effects.21,22

rat models, likely via mechanisms such as This study confirms the atherogenic effect of a high-fat diet, which significantly reduced HDL levels in rats compared to a normal control group. While both doses of REEA increased HDL, the 200 mg/kg dose produced a notable rise, whereas the 100 mg/kg dose resulted in a non-significant change. Similarly, VLDL levels were significantly elevated in the high-fat diet group. The 200 mg/kg dose of REEA significantly lowered VLDL, while the 100 mg/kg dose had a minor effect. Atorvastatin again produced the greatest reduction. These results suggest REEA has a dual mechanism of action: it appears to reduce VLDL levels, likely by inhibiting lipid synthesis or secretion, while also promoting HDL formation. This is possibly due to its antioxidant and polyphenolic content. Although less potent than atorvastatin, the effects of REEA at the higher dose are comparable to other plant extracts known for their antihyperlipidemic properties, reinforcing its potential as a cardioprotective agent.23,24

Consumption of a high-fat diet (HFD) is associated with an increase in visceral adiposity and excessive weight gain, both of which elevate BMI and abdominal circumference (AC). These are established surrogate markers for cardiovascular risk and metabolic syndrome. In our study, rats on an HFD showed a mild, yet consistent, elevation in both BMI and AC compared to control groups, which aligns with existing literature on chronic HFD-induced adiposity. Treatment with the REEA at a 100 mg/kg dose led to a minimal reduction in these anthropometric indices. Notably, this effect was comparable to the reduction observed with atorvastatin, a statin with known anti-obesity properties. The observed reduction in anthropometric indices with REEA suggests that its mechanism of action may involve interfering with lipid absorption or adipogenesis. This effect is likely mediated by its rich phytochemical composition, including saponins, polyphenols, and alkaloids, which have been shown to inhibit pancreatic lipase, reduce fat deposition, and enhance leptin sensitivity.25,26

Histopathological analysis of various tissues—liver, aorta, mesenteric fat, perirenal adipose tissue, and kidney—revealed significant HFD-induced damage and demonstrated the protective effects of the root extract of Epipremnum aureum (REEA). In the liver, Group 1 (normal control) had a normal structure, whereas Group 2 (HFD control) showed pronounced microvesicular steatosis and periportal inflammation, indicative of non-alcoholic fatty liver disease (NAFLD). Groups 3 and 4, treated with low- and high-dose REEA respectively, exhibited a dose-dependent reduction in steatosis and inflammation, suggesting the extract's hepatoprotective potential. The positive control group (Group 5, Atorvastatin) also displayed hepatoprotective effects with only mild steatosis.27,28

In the adipose tissue, HFD exposure (Group 2) led to significant accumulation in both perirenal and mesenteric fat depots. REEA treatment in Groups 3 and 4 dose-dependently limited this accumulation, suggesting a role in reducing visceral adiposity. Atorvastatin showed a similar but milder effect. Examination of aortic and myocardial sections in Group 2 revealed early atherogenic changes, including lipid deposits and myocardial necrosis, which were significantly attenuated in the REEA-treated groups, with Group 4 showing near-normal histology. The Atorvastatin group maintained a normal cardiovascular profile, reinforcing the known cardioprotective effects of statins. The protective effects of REEA on these tissues are likely due to its antioxidant and anti-inflammatory properties, which counteract the oxidative stress central to atherogenesis.29,30 Importantly, no significant

histological changes were observed in the kidneys across any of the groups, indicating that neither the high-fat diet nor the treatments induced nephrotoxicity.
In summary, the histopathological findings confirm that REEA, particularly at the higher dose, effectively ameliorates hepatic steatosis, reduces adipose tissue accumulation, and protects cardiovascular tissues in HFD-induced hyperlipidaemic rats. While its effects were slightly less potent than those of Atorvastatin, these findings support the potential of REEA as a safe therapeutic agent for metabolic disorders.

This study used a well-established high-fat diet model in Wistar rats, which ensures the relevance and reproducibility of the findings. The research provides a comprehensive assessment by measuring a range of lipid parameters and anthropometric data, complemented by histopathological analysis to assess organ-level effects. The inclusion of both negative and positive controls, as well as two different dose levels, allows for a robust comparison of the extract's efficacy against a standard drug. Despite its strengths, the four-week duration and small sample size may limit the evaluation of long-term effects detection of subtle effects. Furthermore, the study lacks a detailed mechanistic investigation into the extract's action, and potential batch-to-batch variability in the extract's preparation could affect reproducibility.

CONCLUSION

This study provides compelling evidence that the root extract of Epipremnum aureum possesses significant anti-hyperlipidemic properties. In a diet-induced hyperlipidemia model, REEA demonstrated a dose-dependent ability to lower lipid levels, with the 200 mg/kg dose leading to a statistically significant improvement in total cholesterol, LDL-C, VLDL-C, and HDL-C. While its efficacy was moderate compared to Atorvastatin, its potential as a complementary or alternative therapy is notable, particularly given the absence of observed toxicity. The histopathological findings suggest that REEA may also offer additional benefits, such as hepatoprotection and a reduction in adipose tissue accumulation. These findings collectively support the need for further research, including more comprehensive preclinical studies, an investigation into the underlying mechanisms of action, and the eventual isolation of active compounds for the development of standardized therapeutic preparations. The study establishes a strong foundation for the continued exploration of Epipremnum aureum as a promising botanical agent for the management of dyslipidemia.

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