

DEVELOPMENT AND EVALUATION OF ANTI-OBESITY POLYHERBAL FORMULATION CONTAINING BRYONIA LACINIOSA AND FICUS RACEMOSA: A COMPREHENSIVE IN VITRO AND IN VIVO INVESTIGATION

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ABSTRACT

Obesity represents a global pandemic requiring safe and effective therapeutic interventions. Traditional medicinal plants offer promising alternatives to synthetic anti-obesity drugs with potentially fewer adverse effects. This study investigated the anti-obesity potential of a novel polyherbal formulation combining *Bryonia laciniola* and *Ficus racemosa* extracts. Ethanolic extracts of *B. laciniola* seeds and *F. racemosa* bark were prepared and characterized through phytochemical screening. In vitro antioxidant activity was assessed using DPPH and FRAP assays, while pancreatic lipase inhibition was evaluated spectrophotometrically. Acute toxicity studies followed OECD guidelines. Anti-dyslipidemic effects were evaluated in Triton X-100-induced hyperlipidemic rats. A polyherbal tablet formulation was developed and optimized, followed by anti-obesity evaluation in high-fat diet-induced obese rats. Both extracts demonstrated significant antioxidant activity (IC_{50} : *B. laciniola* $96.85 \pm 0.04 \mu\text{g/mL}$; *F. racemosa* $115.82 \pm 0.06 \mu\text{g/mL}$) and pancreatic lipase inhibition (IC_{50} : 27.70 ± 0.15 and $36.88 \pm 0.14 \mu\text{g/mL}$, respectively). In hyperlipidemic rats, treatment significantly reduced total cholesterol, triglycerides, LDL, and VLDL while increasing HDL levels ($p < 0.01$). The optimized polyherbal tablet (PHT4) demonstrated superior anti-obesity effects in high-fat diet-induced obese rats, significantly reducing body weight, serum lipids, and hepatic lipid accumulation compared to disease controls ($p < 0.01$). The polyherbal formulation combining *B. laciniola* and *F. racemosa* extracts exhibits significant anti-obesity and anti-dyslipidemic properties through multiple mechanisms including pancreatic lipase inhibition and antioxidant activity. These findings support the potential clinical application of this formulation for obesity management.

Keywords: Anti-obesity, *Bryonia laciniola*, *Ficus racemosa*, polyherbal formulation, pancreatic lipase inhibition, dyslipidemia.

INTRODUCTION

Obesity has emerged as one of the most pressing global health challenges of the 21st century, with the World Health Organization classifying it as a pandemic affecting over 650 million adults worldwide¹.

The condition is characterized by excessive accumulation of adipose tissue resulting from chronic energy imbalance, leading to increased morbidity and mortality associated with cardiovascular disease, type 2 diabetes mellitus, and metabolic syndrome^{2,3}. The economic burden of obesity-related healthcare costs represents approximately 25% higher expenditure compared to normal-weight individuals⁴.

Current pharmacological interventions for obesity management include pancreatic lipase inhibitors, appetite suppressants, and combination therapies⁵. However, these synthetic drugs are often associated with significant adverse effects including gastrointestinal disturbances, cardiovascular complications, and hepatotoxicity^{6,7}. Consequently, there is an urgent need for safe and effective alternative therapeutic approaches.

Traditional medicinal plants have gained considerable attention as potential sources of anti-obesity compounds due to their multi-target mechanisms, lower toxicity profiles, and synergistic effects^{8,9}. Plant-derived bioactive compounds can modulate obesity through various pathways including inhibition of pancreatic lipase, suppression of adipogenesis, enhancement of lipolysis, and improvement of metabolic homeostasis^{10,11}.

Bryonia laciniosa Linn. (Cucurbitaceae), commonly known as Shivlingi, is a traditional Ayurvedic medicinal plant historically used for treating metabolic disorders¹². The plant contains diverse phytochemicals including flavonoids, alkaloids, saponins, and phenolic compounds with demonstrated antioxidant, anti-inflammatory, and metabolic regulatory properties^{13,14}. *Ficus racemosa* Linn. (Moraceae), known as cluster fig, has been traditionally employed for managing digestive and metabolic disorders¹⁵. Its bark contains tannins, phenolic acids, and flavonoids with reported hypolipidemic and anti-inflammatory activities^{16,17}.

Despite their traditional use and promising phytochemical profiles, comprehensive scientific evaluation of these plants' anti-obesity potential, particularly in combination, remains limited. The present study aimed to develop and evaluate a novel polyherbal formulation combining *B. laciniosa* and *F. racemosa* extracts for anti-obesity activity through systematic in vitro and in vivo investigations.

Materials and Methods

Plant Material Collection and Authentication

Seeds of *B. laciniosa* and bark of *F. racemosa* were collected from Dhana region, Sagar district, Madhya Pradesh, India, during October 2024. Taxonomic identification and authentication were

conducted by Dr. S. N. Dwivedi, Head, Department of Botany, Janata PG College, A.P.S. University, Rewa (Voucher no: JBOT/H31/11/01/2025). Plant materials were processed, cleaned, and dried under shade conditions before pulverization.

Extract Preparation

Coarse plant powder (500 g) was initially defatted using petroleum ether. Sequential extraction was performed using 70% ethanol (1:3 w/v) through maceration method for 72 hours with intermittent stirring. Extracts were filtered through Whatman No. 1 filter paper and concentrated under reduced pressure at 40-45°C using rotary evaporator. Dried extracts were stored at 4-8°C until analysis.

Phytochemical Screening

Preliminary qualitative phytochemical analysis was performed following standard protocols¹⁸. Tests for alkaloids, phenolics, saponins, tannins, flavonoids, carbohydrates, amino acids, terpenoids, steroids, and glycosides were conducted using established reagents and procedures.

In Vitro Antioxidant Activity

DPPH Radical Scavenging Assay: Antioxidant potential was evaluated using 2,2-diphenyl-1-picrylhydrazyl (DPPH) assay¹⁹. Extract concentrations of 25-250 µg/mL were prepared in DMSO. Three milliliters of each concentration was mixed with 1 mL DPPH solution (0.1 mM in methanol), incubated at 37°C for 30 minutes, and absorbance measured at 517 nm. Ascorbic acid served as standard reference.

Ferric Reducing Antioxidant Power (FRAP) Assay: Reducing power was determined using potassium ferricyanide-ferric chloride method²⁰. Extract solutions (1 mL) were mixed with phosphate buffer (2.5 mL, 0.2 M, pH 6.6) and potassium ferricyanide (2.5 mL, 1%), incubated at 50°C for 20 minutes, followed by trichloroacetic acid addition and centrifugation. Absorbance was measured at 700 nm.

Pancreatic Lipase Inhibitory Activity

Anti-lipase activity was assessed using p-nitrophenyl butyrate (p-NPB) as chromogenic substrate²¹. Extract concentrations (50-400 µg/mL) were mixed with porcine pancreatic lipase solution (0.1 mL, 1 mg/mL) in Tris-HCl buffer (pH 7.4), incubated at 25°C for 15 minutes, followed by p-NPB addition and further incubation at 37°C for 30 minutes. Absorbance was measured at 405 nm. Orlistat served as positive control.

Acute Oral Toxicity Studies

Acute toxicity evaluation was conducted following OECD Guideline 423²². Female Wistar rats (150-200 g) were administered single oral doses of 300, 2000, and 5000 mg/kg body weight. Animals were observed for 14 days for clinical signs, mortality, and gross pathological changes. All animal studies were approved by the Institutional Animal Ethics Committee (Approval No.

1546/PO/Re/S/11/CCSEA).

Anti-dyslipidemic Activity Study

Fifty-four male Wistar rats (180-200 g) were randomly divided into nine groups (n=6). Hyperlipidemia was induced by single intraperitoneal injection of Triton X-100 (60 mg/kg)²³. After 72 hours, confirmed hyperlipidemic rats received daily oral treatment for seven days: normal control (saline), hyperlipidemic control (saline), atorvastatin (10 mg/kg), and graded doses of extracts (50, 100, 200 mg/kg). Blood samples were collected for lipid profile analysis including total cholesterol (TC), triglycerides (TG), HDL, LDL, and VLDL. Hepatic and fecal lipid contents were determined. Cardiovascular risk indices were calculated using established formulae²⁴.

Polyherbal Tablet Development

Polyherbal formulation (PHF) was prepared by mixing equal proportions (1:1 w/w) of *B. laciniosa* and *F. racemosa* extracts. Four tablet formulations (PHT1-PHT4) were developed using direct compression method with varying concentrations of disintegrating and binding agents. Precompression parameters including angle of repose, bulk density, tapped density, Carr's index, and Hausner's ratio were evaluated²⁵. Drug-excipient compatibility was assessed using FTIR spectroscopy.

Post-compression Tablet Evaluation

Tablets were evaluated for physical parameters including thickness, diameter, hardness, friability, weight variation, and disintegration time following pharmacopoeial standards²⁶. Stability studies were conducted under accelerated conditions (40±2°C/75±5% RH) for 30 days following ICH Q1A guidelines.

Anti-obesity Activity Study

Twenty-four male Sprague Dawley rats (150-200 g) were randomly assigned to four groups (n=6): normal control (standard diet), disease control (high-fat diet), standard treatment (HFD + orlistat 30

mg/kg), and test group (HFD + PHT4 200 mg/kg). High-fat diet was administered for eight weeks with treatment intervention during the final four weeks²⁷. Body weight, food intake, and anthropometric parameters were monitored weekly. Blood samples were analyzed for lipid profile, liver enzymes, glucose, leptin, and adiponectin levels. Histopathological examination of liver and adipose tissues was performed using hematoxylin and eosin staining.

Statistical Analysis

Data were analyzed using GraphPad Prism 5.0 software. Results are expressed as mean $\pm$ standard deviation. Statistical significance was determined using one-way ANOVA followed by Dunnett's multiple comparison test. P-values < 0.05 were considered statistically significant.

RESULTS AND DISCUSSION

Phytochemical Screening

Qualitative phytochemical analysis revealed the presence of alkaloids, glycosides, triterpenoids, carbohydrates, steroids, saponins, flavonoids, and phenolic compounds in both *B. laciniosa* and *F. racemosa* extracts. Tannins and amino acids were absent in both extracts.

Table 1: Preliminary Phytochemical Screening of Ethanolic Extracts of *Bryonia laciniosa* and *Ficus racemosa*

Phytochemical Constituents	<i>Bryonia laciniosa</i>	<i>Ficus racemosa</i>
Alkaloids	+	+
Glycosides	+	+
Triterpenoids	+	+
Carbohydrates	+	+
Steroids	+	+
Saponins	+	+
Flavonoids	+	+
Phenols	+	+
Tannins	—	—
Amino acids	—	—

In Vitro Antioxidant Activity

Both extracts demonstrated significant antioxidant activity in DPPH and FRAP assays. *B. laciniola* extract showed superior DPPH radical scavenging activity (IC_{50} : 96.85 ± 0.04 $\mu\text{g/mL}$) compared to *F. racemosa* (IC_{50} : 115.82 ± 0.06 $\mu\text{g/mL}$), though both were less potent than ascorbic acid (IC_{50} : 47.47 ± 0.04 $\mu\text{g/mL}$). Similarly, in FRAP assay, *B. laciniola* (IC_{50} : 99.43 ± 0.06 $\mu\text{g/mL}$) exhibited stronger reducing power than *F. racemosa* (IC_{50} : 102.81 ± 0.05 $\mu\text{g/mL}$) compared to ascorbic acid standard (IC_{50} : 53.90 ± 0.06 $\mu\text{g/mL}$).

Table 2: Antioxidant activity of extracts by DPPH assay

Extracts	$IC_{50} \pm SD$ (DPPH) (mg/mL)
Ethanol extract of <i>Bryonia Laciniola</i>	96.85 ± 0.04
Ethanol extract of <i>Ficus Racemosa</i>	115.82 ± 0.06
Ascorbic acid	47.47 ± 0.04

Pancreatic Lipase Inhibitory Activity

Both extracts exhibited dose-dependent pancreatic lipase inhibition. *B. laciniola* demonstrated superior anti-lipase activity (IC_{50} : 27.70 ± 0.15 $\mu\text{g/mL}$) compared to *F. racemosa* (IC_{50} : 36.88 ± 0.14 $\mu\text{g/mL}$), though both were less potent than orlistat (IC_{50} : 18.15 ± 0.34 $\mu\text{g/mL}$).

Table 3: Pancreatic Lipase Inhibitory Potential (IC_{50} values) of Different Extracts of Selected Plants and Orlistat

Extract / Standard	$IC_{50} \pm SD$ ($\mu\text{g/mL}$)
Ethanol (EBL)	27.70 ± 0.15
Ethanol (EFR)	36.88 ± 0.14
Orlistat	18.15 ± 0.34

Acute Toxicity Studies

No mortality or clinical signs of toxicity were observed in rats administered extract doses up to 5000 mg/kg body weight. Gross necropsy revealed no pathological abnormalities, classifying both extracts as GHS Category 5 ($LD_{50} > 2000$ -5000 mg/kg), indicating low acute toxicity potential.

Anti-dyslipidemic Activity

Triton X-100 administration significantly elevated serum TC, TG, LDL, and VLDL levels while reducing HDL compared to normal controls ($p < 0.01$). Treatment with both extracts produced dosedependent improvements in lipid profiles. *F. racemosa* at 200 mg/kg (EFRH) demonstrated the most pronounced effects, reducing TC from 113.93 ± 3.19 to 82.64 ± 1.94 mmol/L, comparable to atorvastatin efficacy (82.01 ± 4.20 mmol/L). Similar patterns were observed for TG, LDL, and VLDL reduction, with concomitant HDL elevation.

Hepatic lipid analysis revealed significant reduction in cholesterol and triglyceride accumulation following extract treatment. EFRH showed superior hepatoprotective effects (hepatic TC: 20.52 ± 1.82 mmol/L) compared to hyperlipidemic controls (35.08 ± 2.12 mmol/L). Fecal lipid excretion increased significantly in treatment groups, indicating enhanced lipid elimination. Cardiovascular risk indices including atherogenic index, cardiac index, and coronary artery index were markedly reduced following treatment, with EFRH showing the most favorable profile.

Table 4: Effect of Extract EBL and EFR on Serum Lipid Profile in Triton X-100-Induced Hyperlipidemic Rats

Groups	TC (mmol/L)	TG (mmol/L)	LDL (mmol/L)	VLDL (mmol/L)	HDL (mmol/L)
Normal control	79.54 ± 1.80^c	87.83 ± 2.72^c	27.16 ± 1.13^c	17.57 ± 0.54^c	33.68 ± 1.29^c
Hyperlipidemic control	113.93 ± 3.19^b	188.70 ± 2.89^b	50.46 ± 1.77^b	37.80 ± 0.62^b	26.83 ± 1.17^b
Atorvastatin (10 mg/kg)	82.01 ± 4.20^c	95.86 ± 3.42^{bc}	31.67 ± 1.46^{bc}	19.17 ± 0.68^{bc}	28.84 ± 0.84^{bc}
EBLL (50 mg/kg)	90.84 ± 1.79^{bc}	117.47 ± 4.47^{bc}	39.09 ± 1.37^{bc}	23.49 ± 0.89^{bc}	29.18 ± 0.50^{bc}
EBLI (100 mg/kg)	88.04 ± 2.32^{bc}	114.74 ± 5.65^{bc}	37.37 ± 1.17^{bc}	22.95 ± 1.13^{bc}	30.09 ± 1.01^{bc}
EBLH (200 mg/kg)	84.68 ± 2.76^{ac}	103.04 ± 3.09^{bc}	33.14 ± 1.25^{bc}	20.61 ± 0.62^{bc}	32.14 ± 1.10^c
EFRL (50 mg/kg)	94.59 ± 2.66^{bc}	122.22 ± 3.03^b	38.22 ± 1.34^{bc}	24.44 ± 0.61^{bc}	29.42 ± 1.01^{bc}

EFRI mg/kg)	(100	83.12 ± 2.50 ^c	112.61 5.90 ^{bc}	±	35.79 ± 1.31 ^{bc}	23.32 ± 1.18 ^{bc}	31.60 ± 1.26 ^{bc}
EFRH mg/kg)	(200	82.64 ± 1.94 ^c	105.24 3.15 ^{bc}	±	31.26 ± 1.37 ^{bc}	19.96 ± 0.63 ^{bc}	34.87 ± 0.77 ^c

Values are presented as mean ± SD (N=6), values with different superscript letters indicates significant differences compared with control group (a = P#<0.05; b = P##<0.01; ns = P> 0.05), and hyperlipidemic group (c = P**<0.01) by ANOVA followed by Dunnett test.

Table 5: Hepatic cholesterol and triglyceride concentration of hyperlipidemic rats following oral administration of EBL and EFR

Groups	Hepatic TC (mmol/l)	Hepatic TG (mmol/l)
Normal control	20.95 ± 0.84 ^c	0.33 ± 0.02 ^c
Hyperlipidemic	35.08 ± 2.12 ^b	0.73 ± 0.02 ^b
Atorvastatin (10 mg/kg)	21.19 ± 1.41 ^c	0.40 ± 0.02 ^{bc}
EMCL (50 mg/kg)	28.97 ± 1.67 ^{bc}	0.48 ± 0.04 ^{bc}
EMCI (100 mg/kg)	24.23 ± 2.01 ^{bc}	0.47 ± 0.03 ^{bc}
EMCH (200 mg/kg)	21.08 ± 1.67 ^c	0.46 ± 0.02 ^{bc}
EPCL (50 mg/kg)	28.49 ± 1.20 ^{bc}	0.51 ± 0.04 ^{bc}
EPCI (100 mg/kg)	23.39 ± 1.55 ^{bc}	0.50 ± 0.03 ^{bc}
EPCH (200 mg/kg)	20.52 ± 1.82 ^c	0.45 ± 0.03 ^{bc}

Values are presented as mean±SD (N=6), values with different superscript letters indicates significant differences compared with control group (a = P#<0.05; b = P##<0.01; ns = P>0.05), and hyperlipidemic group (c = P**<0.01) by ANOVA followed by Dunnett test.

Table 6: Faecal cholesterol and triglyceride of hyperlipidemic rats after treatment of EBL and EFR (± SD, N=6)

Groups	Fecal Cholesterol (mmol/l)		Fecal Triglycerides (mmol/l)	
	First 3 days	Last 3 days	First 3 days	Last 3 days
Normal control	50.13 ± 1.92	50.88 ± 1.82	0.83 ± 0.03	0.84 ± 0.04
Hyperlipidemic	36.43 ± 1.35	33.74 ± 1.56	1.16 ± 0.04	1.17 ± 0.03
Atorvastatin (10 mg/kg)	36.61 ± 2.15	54.36 ± 4.02	1.15 ± 0.04	1.86 ± 0.04
EBLL (50 mg/kg)	37.72 ± 3.24	46.33 ± 2.51	1.19 ± 0.02	1.65 ± 0.03

EBLI (100 mg/kg)	38.02 ± 2.21	46.60 ± 2.63	1.16 ± 0.04	1.71 ± 0.03
EBLH (200 mg/kg)	36.88 ± 3.30	48.55 ± 4.25	1.17 ± 0.04	1.81 ± 0.03
EFRL (50 mg/kg)	37.42 ± 2.53	47.68 ± 1.89	1.16 ± 0.02	1.62 ± 0.03
EFRI (100 mg/kg)	37.91 ± 2.61	46.87 ± 3.50	1.18 ± 0.02	1.72 ± 0.03
EFRH (200 mg/kg)	37.96 ± 1.62	50.19 ± 2.34	1.14 ± 0.02	1.82 ± 0.04

Tablet Formulation and Evaluation

Pre-formulation studies revealed acceptable micromeritic properties for all formulations. PHT4 demonstrated optimal characteristics with angle of repose ($20.91 \pm 0.06^\circ$), Carr's index ($12.43 \pm 5.01\%$), and Hausner ratio (1.13 ± 0.06), indicating excellent flowability. FTIR analysis confirmed drug-excipient compatibility without chemical interactions.

Post-compression evaluation showed PHT4 had superior physical properties: hardness (4.55 ± 0.08 kg/cm²), friability ($0.50 \pm 0.02\%$), and disintegration time (27.38 ± 0.81 minutes). Stability studies under accelerated conditions revealed no significant changes in evaluated parameters over 30 days.

Table 7: Post-compression evaluation of polyherbal tablets

Parameters	PHT 1	PHT 2	PHT 3	PHT 4
Diameter (mm)	10.01 ± 0.01	10.01 ± 0.01	10.04 ± 0.05	10.00 ± 0.01
Thickness (mm)	5.00 ± 0.01	5.01 ± 0.01	5.01 ± 0.01	5.00 ± 0.01
Hardness (kg/cm ²)	2.50 ± 0.06	2.83 ± 0.09	3.21 ± 0.09	4.55 ± 0.08
Friability (%)	0.64 ± 0.03	0.59 ± 0.02	0.56 ± 0.03	0.50 ± 0.02
Weight Variation (%)	449.08 ± 0.51	450.04 ± 0.57	449.13 ± 0.50	450.08 ± 0.06
Disintegration (min)	44.53 ± 1.91	36.56 ± 1.36	30.31 ± 0.79	27.38 ± 0.81

Anti-obesity Activity

PHT4 demonstrated significant anti-obesity effects in high-fat diet-induced obese rats. Treatment for four weeks resulted in progressive body weight reduction from 226.17 ± 7.96 g to 184.33 ± 6.12 g,

showing superior efficacy compared to orlistat (193.67 ± 7.47 g) and significantly different from disease controls (328.67 ± 5.47 g) ($p < 0.01$).

Serum lipid analysis revealed significant improvements in all parameters. Total cholesterol, triglycerides, LDL, and VLDL were markedly reduced, while HDL levels increased compared to disease controls. Liver enzyme levels (SGOT, SGPT) normalized, indicating hepatoprotective effects. Hormonal analysis showed reduced leptin and increased adiponectin levels, suggesting improved metabolic homeostasis.

Histopathological examination revealed preserved hepatic architecture in PHT4-treated animals with minimal lipid accumulation, contrasting with extensive hepatic steatosis observed in disease controls. Adipose tissue analysis showed reduced adipocyte size and improved tissue morphology following treatment.

Table 8: Effect of oral administration of PHT4 on body weight of HFD-induced obese rats for four weeks

Treatment Groups	Day 1	Day 7	Day 14	Day 21	Day 28
NC	212.83 ± 14.39	218.83 ± 14.92	225.50 ± 15.48	231.17 ± 15.88	237.33 ± 16.08
DC (Disease Control)	232.33 ± 11.22	258.00 ± 10.86	267.67 ± 5.85	291.17 ± 6.27	328.67 ± 5.47
Orlistat	224.50 ± 14.80	219.50 ± 15.55	215.67 ± 8.64	209.83 ± 7.19	193.67 ± 7.47
PHT4	226.17 ± 7.96	216.67 ± 5.57	209.83 ± 7.47	193.17 ± 5.56	184.33 ± 6.12

The present investigation demonstrates the significant anti-obesity and anti-dyslipidemic potential of a novel polyherbal formulation combining *B. laciniosa* and *F. racemosa* extracts. The observed therapeutic effects can be attributed to the synergistic actions of diverse bioactive compounds present in both plants, including flavonoids, phenolic acids, saponins, and terpenoids^{28, 29}.

The substantial pancreatic lipase inhibitory activity exhibited by both extracts represents a primary mechanism underlying their anti-obesity effects. Pancreatic lipase catalyzes the hydrolysis of dietary triglycerides, and its inhibition reduces fat absorption, leading to decreased caloric intake and weight reduction³⁰. *B. laciniosa* demonstrated superior anti-lipase activity, which may be attributed to its rich content of bioactive compounds such as bryonin and flavonoid derivatives³¹.

The significant antioxidant activity observed in both extracts contributes to their therapeutic efficacy through multiple pathways. Obesity-associated oxidative stress promotes adipogenesis, inflammation, and metabolic dysfunction³². The demonstrated free radical scavenging and reducing properties of the extracts may counteract these pathological processes, supporting weight management and metabolic homeostasis³³.

The comprehensive in vivo anti-dyslipidemic studies revealed dose-dependent improvements in lipid profiles, with *F. racemosa* showing particularly pronounced effects on HDL elevation and cardiovascular risk index reduction. These findings align with previous reports of *Ficus* species' hypolipidemic properties, attributed to compounds such as leucocyanidin and β -sitosterol^{34, 35}.

The successful development of a stable polyherbal tablet formulation (PHT4) with optimal pharmaceutical properties enabled effective delivery of the active compounds. The superior antiobesity effects observed with the combination formulation compared to individual extracts suggest synergistic interactions between the plant constituents, supporting the traditional polyherbal approach in Ayurvedic medicine³⁶.

The histopathological evidence of hepatoprotection and reduced adipose tissue hypertrophy further validates the multi-target therapeutic approach of the polyherbal formulation. The normalization of liver enzymes and improvement in metabolic biomarkers including leptin and adiponectin indicate comprehensive metabolic benefits beyond simple weight reduction³⁷.

Limitations of this study include the relatively short treatment duration and the need for long-term safety evaluation. Future clinical studies are warranted to establish human efficacy and safety profiles. Additionally, detailed mechanistic studies investigating specific molecular pathways and biomarker changes would enhance understanding of the therapeutic mechanisms.

CONCLUSIONS

The polyherbal formulation combining *B. laciniosa* and *F. racemosa* extracts demonstrates significant anti-obesity and anti-dyslipidemic properties through multiple complementary mechanisms including pancreatic lipase inhibition, antioxidant activity, and metabolic regulation. The optimized tablet formulation (PHT4) showed superior therapeutic efficacy with favorable pharmaceutical properties and stability profile. These findings support the potential clinical application of this polyherbal formulation as a safe and effective alternative for obesity management, warranting further clinical investigation.

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