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PHYTOCHEMICAL INVESTIGATION AND PHARMACOLOGICAL EVALUATION OF *BETULA UTILIS* ON CAFETERIA INDUCED OBESITY IN RATS

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ABSTRACT

Obesity, resulting from an imbalance between caloric intake and energy expenditure, leads to excessive fat accumulation and increases the risk of chronic diseases like diabetes and coronary artery disease. A 10-week cafeteria diet in rats induces obesity by promoting fat deposition. Betula utilis, a medicinal plant rich in phytochemicals such as betulinic acid, exhibits numerous pharmacological effects, including anti-obesity properties. Betulinic acid and other compounds in Betula utilis inhibit enzymes like pancreatic lipase and α-amylase, crucial for fat and carbohydrate metabolism. This study aimed to assess the impact of Betula utilis extract on cafeteria diet-induced obesity in Wistar rats. The extract was administered orally in doses of 100, 200, and 400 mg/kg from the fourth to the tenth week. Various parameters, including body weight, BMI, Lee's index, feed intake, and biochemical markers (serum cholesterol, LDL, VLDL, triglycerides, and glucose levels), were evaluated. Results showed that Betula utilis extract significantly reduced obesity markers and improved biochemical profiles in a dose-dependent manner, with the highest dose being the most effective. The extract also reduced adipose tissue weight in different fat depots. These findings suggest that *Betula utilis* extract effectively mitigates obesity, providing a pharmacological basis for its potential use in treating obesity in humans.

Keywords: Obesity, *Betula utilis*, phytochemicals, etc.

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INTRODUCTION

Obesity is characterized by an excess of body fat, while being overweight indicates a weight that exceeds what is considered healthy for a person's height. Though related, these terms describe different conditions. Weight is influenced by various factors, including an individual's genetic makeup, caloric intake, consumption of high-fat foods, and level of physical activity. The equilibrium between calories consumed and expended is unique to each person. Overweight and obesity rank as the fifth highest risk factor for global mortality. Each year, at least 2.8 million adults lose their lives due to these conditions [1,2] Additionally, 44% of diabetes cases, 23% of ischemic heart disease cases, and between 7% and 41% of certain cancers can be attributed to excess weight. According to WHO's 2008 global estimates, 1.5 billion individuals were overweight, including over 200 million men and nearly 300 million women classified as obese. In total, more than 10% of the world's adult population was obese [3]. Currently, only two medications, sibutramine and orlistat, are approved for long-term obesity treatment. Both drugs can result in a 5 to 10% reduction in body weight but come with their own set of limitations and potential side effects. Traditional medicines derived from medicinal plants are utilized by approximately 60% of the global population. Ayurveda claims that the bark of the Betula utilis plant can help control obesity. However, no scientific data currently exist to support this claim. Traditional medicines derived from medicinal plants are utilized by approximately 60% of the global population. Ayurveda claims that the bark of the Betula utilis plant can help control obesity [4]. However, no scientific data currently exist to support this claim. In the present study, the ethanolic extract of Betula utilis bark has been selected for its potential anti-obesity activity. This extract is reported to possess various properties, including anti-inflammatory , antioxidant , anti-HIV , antibacterial , and mild antihyperglycemic activities. The objective of this study is to examine the impact of the ethanolic extract of Betula utilis bark on obesity induced in rats by a cafeteria diet. Additionally, the study aims to explore the potential mechanisms underlying the extract's anti-obesity effects by assessing body weight, serum lipid profile, and glucose levels. Orlistat was used as the reference standard drug in this investigation.

MATERIALS AND METHODS

Collection Plant Material

The fresh *Betula utilis* bark used for the present studies was collected from outskirt of Jaipur Rajasthan, in January 2024.

Extraction

The bark of *Betula utilis* (Bhojpatra) was dried in the shade. The dried bark was cut into small pieces and then powdered using a mixer grinder. A total of 45 grams of powdered Betula utilis bark was defatted with 1.25 liters of petroleum ether. Subsequently, the defatted bark powder was extracted with 1.25 liters of 95% ethanol using a Soxhlet apparatus at 40°C for approximately 72 hours. After completing the extraction, the extract was concentrated using a vacuum rotary evaporator to obtain a yellowish-brown residue. This residue was then placed in a vacuum desiccator for 4-5 days to dry, after which it was used for subsequent experiments.

Phytochemical study

The ethanolic bark extract of Betula utilis was subjected to standard phytochemical screening tests for various phytoconstituents [5].

Experimental Animals

Healthy Wistar albino rats (150–200g) of either sex were used for the experiment were procured from the animal house of Idma Lab, Panchkula. They were maintained under standard conditions (temperature 22 ± 2^0C, Relative humidity 60 ± 5% and 12 h light/dark cycle). The animals were housed in sanitized polypropylene cages containing sterile paddy husk as bedding. They had free access to standard pellet diet and water ad libitum. The Institutional Animal Ethics Committee approved the experimental protocol (IAEC 0424-05).

Assessment of anthropometric parameters

The raise in the body weight as compared to age matched normal rats is regard as obesity. Body Mass Index (BMI) i.e. weight (g)/ height (cm) 2, Lee index i.e. (Body Wt)1/3 / ano-nasallength (cm) x 1000 was calculated after the treatment as an index of obesity and compared with normal rats. Body weight was measured weekly. Food intake measurements for individual rats were recorded biweekly. To evaluate the effect of high calorie diet and drug interventions, fat depots (Epididymal, retroperitoneal and mesenteric fat depots) was isolated, remove from nearby tissues, and weigh individually and after that total weight was calculated [6].

Assessment of Biochemical Parameters

The hyperlipidemia was assessed by estimating the levels of Total cholesterol, High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL), Very Low Density Lipoprotein (VLDL) and Triglycerides in blood serum using commercially available kits. Values were expressed in mg/dl. Additionally glucose level in serum was also estimated by using commercially available kits. Values are expressed in mg/dl.

Estimation of serum total cholesterol

The total cholesterol was estimated by cholesterol oxidase peroxidase CHOD-POD method using commercially available kit (ADI Diagnostics [P] Ltd., Hyderabad, India). 1000 µl of cholesterol reagent was added to 10 µl of serum, 10 µl of standard cholesterol (200 mg/dl) and 10 µl of purified water to prepare test, standard and blank, respectively. All the test tubes were incubated at room temperature for 20 mins. The absorbances of test and standard samples were noted against blank at 520 nm spectophotometrically [7].

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{CHE}} \text{Cholesterol} + \text{Free Fatty acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{CHOD}} \text{Cholest} - 4\text{ene } 3 - \text{one} + H_2O_2$$

$$H_2O_2 + \text{Phenol} + 4 - \text{Aminoantip yrine} \xrightarrow{\text{POD}} \text{Red quinoneimi ne Complex} + H_2O_2$$

Cholesterol is determined after enzymatic hydrolysis and oxidation. Cholesterol esters are hydrolysed by the enzyme cholesterol esterase (CHE) to give free cholesterol and free fatty molecules. This free cholesterol gets oxidized in the presence of cholesterol oxidase (CHOD) to liberate Cholest-4ene 3-one and H_2O_2. The indicator quinoneimine is formed from hydrogen peroxide and 4-Aminoantipyrine in the presence of phenol and peroxides (POD). The intensity of the colour complex is directly proportional to the cholesterol concentration present in sample.

The serum total cholesterol was calculated using the following formula:

$$\text{Total cholesterol (mg/dl)} = \frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \times 200$$

Estimation of serum triglycerides

The serum triglyceride was estimated by glycerol phosphate oxidase peroxidase GPO-PAP method (Fossati *et al.,* 1982) using commercially available kit (ADI Diagnostics [P] Ltd., Hyderabad, India). 1 ml of Triglyceride reagent was added to 10 µl of serum and 10 µl of standard (200 mg/dl) and 10 µl of purified water to prepared test, standard and blank respectively. All the test tubes were incubated at room temperature for 20 min. The absorbances of test and standard samples were noted against blank at 520 nm spectrophotometrically.

$$\text{Triglycerides} + H_2O \xrightarrow{\text{Lipase}} \text{Glycerol} + \text{Fatty acids}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{GK}} \text{Glycerol} - 1 - \text{Phosphate (G} - 3 - \text{P)} + \text{ADP}$$

$$G - 1 - P + O_2 \xrightarrow{\text{GPO}} H_2O_2 + \text{Dihydroxyacetone phosphate}$$

$$H_2O_2 + 4\ AAP + 4-Chlorophenol \xrightarrow{POD} Purplish\ brown\ quinoneimine\ complex + H_2O + HCl$$

Serum triglycerides are hydrolysed to glycerol and free fatty acids by lipase. In the presence of ATP and glycerol kinase (GK), glycerol is converted to glycerol-1-phosphate which is then oxidized by glycerol-1-phosphate oxidase (GPO) to yield H_2O_2. The H_2O_2, thus formed reacts with 4-aminoantipyrine (4-AAP) and 4-Chlorophenol in presence of enzyme peroxidase (POD) to form purplish brown quinoneimine complex, which is measured spectophotometrically.

The serum triglyceride was calculated using the following formula:

$$\text{Triglycerides (mg/dl)} = \frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \text{ X } 200$$

Estimation of high density lipoprotein (HDL)

The HDL was estimated by cholesterol oxidase peroxidase CHOD-POD method (Allain *et al.*, 1974) using commercially available kit (ADI Diagnostics [P] Ltd., Hyderabad, Indi Step1. 200 µl of serum and 200 µl of precipitating reagent were taken into the centrifuge tube, mixed well and were incubated at room temperature for 5 min and then centrifuged at 3000 rpm for 10 min to get clear supernatant. Step 2- 1000 µl of cholesterol reagent was added to 100 µl of supernatant (from step1), 100 µl of HDL cholesterol standard (50 mg/dl) and 100 µl of purified water to prepare test, standard and blank, respectively. All the test tubes were incubated at room temperature for 10 min. The absorbances of test and standard samples were noted against blank at 505 nm spectophotometrically. On addition of the precipitating reagent to the serum, followed by centrifugation, HDL fraction remains in the supernatant while the lipoprotein precipitate out.

$$Cholesterol\ ester + H_2O \xrightarrow{CHE} Cholesterol + Free\ Fatty\ acids$$

$$Cholesterol + O_2 \xrightarrow{CHOD} Cholest-4ene\ 3-one + H_2O_2$$

$$H_2O_2 + Phenol + 4-Aminoantipyrine \xrightarrow{POD} Red\ quinoneimine\ Complex + H_2O_2$$

Cholesterol is determined after enzymatic hydrolysis and oxidation. Cholesterol esters are hydrolysed by the enzyme cholesterol esterase (CHE) to give free cholesterol and free fatty molecules. This free cholesterol gets oxidized in the presence of cholesterol oxidase (CHOD) to liberate Cholest-4ene 3-one and H_2O_2. The indicator quinoneimine is formed from hydrogen peroxide and 4-Aminoantipyrine in the presence of phenol and peroxides (POD). The intensity of the colour complex is directly proportional to the cholesterol concentration

present in sample. The serum High density lipo protein cholesterol was calculated using the following formula:

$$\text{HDL cholesterol (mg/dl)} = \frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \text{ X } 50$$

Estimation of Very Low Density Lipoprotein (VLDL) and Low Density Lipoprotein (LDL) Level

VLDL and LDL concentrations were calculated from the Friedewald equation (Friedewald *et al.,* 1972):

VLDL Level

Serum VLDL levels (mg/dl) = Triglyceride level/ 5, and

LDL Level

Serum LDL levels (mg/dl) = Total cholesterol-(HDL level + VLDL level)

Estimation of serum glucose

At the end of the experimental protocol, the blood samples were collected and serum was separated. The serum samples were frozen until analyzing the biochemical parameters. The glucose concentration was estimated by glucose oxidase peroxidase GOD-POD method using commercially available kit (Reckon Diagnostics [P] Ltd., Vadodara, India). 1000 µl of working glucose reagent was added to 10 µl of serum, 10 µl of standard glucose (100 mg/dl) and 10 µl of purified water to prepare test, standard and blank, respectively. All the test tubes were incubated at room temperature for 20 min. The absorbances of test and standard samples were noted against blank at 520 nm spectophotometrically [8].

$$\text{Glucose} + O_2 + H_2O \longrightarrow \text{Gluconate} + H_2O_2$$

$$H_2O_2 + \text{4 Aminoantipyrine} + \text{Phenol} \xrightarrow{\text{Peroxidase}} \text{Red Quinoneimine dye} + H_2O$$

Glucose is oxidized to gluconic acid and hydrogen peroxide in presence of glucose oxidase. Hydrogen peroxide further reacts with phenol and 4 aminoantipyrine by the catalytic action of peroxidase to form a red colour quinoneimine dye complex. The intensity of the colour formed is directly proportional to the amount of glucose present in the sample. The serum glucose was calculated using the following formula:

$$\text{The concentration of glucose (mg/dl)} = \frac{\text{Absorbance of Test}}{\text{Absorbance of Standard}} \text{ X } 100$$

Histopathological study

The Cafeteria diet induced change in size of body fat depots were assessed histologically for fat depots. During dissections, standard necropsy procedure was maintained between animals to minimize any variance in tissue collection. A wet-tissue weight is taken immediately after removal using an analytical balance. Special care was taken to remove any non adipose-associated tissues from the depot including glands and lymph nodes. Once weighed, 100–500 mg of the adipose tissue of interest was placed in a sealable tube and covered by a ratio of >10 mL of 10% formalin per gram of WAT. Covered tissue is placed at 4 ℃ for 72 h, then switched to 70% ethanol (equal volume as was used for formalin) for 48 h. A standard procedure was strictly followed for WAT fixing and dehydration [9].

RESULTS AND DISCUSSION

Preliminary Phytochemical Screening: The ethanolic extract of BU showed the presence of alkaloids, carbohydrates, saponins, triterpenoids and steroids (Table 1).

Table 1. Preliminary phytochemical analysis of BU

Phytoconstitudents	Ethanolic extract of BU
Triterpenoids	+
Alkaloids	+
Carbohydrates	+
Steroids	+
Saponins	+
Glycosides	-
Tannins	-
Protein	-

+ present, -Absent,

Effect of various pharmacological interventions on body weight and feed intake.

Obesity was induced in normal rats by feeding a Cafeteria diet for 10 weeks. The mean body weight of the experimental groups were almost similar at the start of the experiment. At the end of the study, there is substantial ($p<0.05$) increase in body weight in Cafeteria fed rats as compared to the normal control group (Table 2). The feed intake (gm) decreased in Cafeteria

diet treated group, while the feed consumption (kcal) increased as compared to the normal control ($p<0.05$). However oral supplementation of *Betula utilis* extract in high, medium and low dose (400, 200 and 100 mg/kg) produced significant ($p<0.05$) dose dependent reduction in body weight and feed intake (gm) as compared to the Cafeteria fed group. There was no significant *per se* effect of *Betula utilis* extract and orlistat.

Effect of various pharmacological interventions on Lee's index and body mass index (BMI).

Cafeteria diet caused a significant rise in Lee's index and BMI in Cafeteria diet group (Table 3) as compared to the normal age matched control group fed on normal chow diet. *Betula utilis* extract in high, medium and low dose (400, 200 and 100 mg/kg) produced significant ($p<0.05$) dose dependent reduction in lee's index and BMI when compared to the Cafeteria diet fed group. There was no significant *per se* effect of *Betula utilis* extract and orlistat.

Effect of various pharmacological interventions on different fat depots

Administration of Caafeteria diet for 10 weeks caused a significant ($p<0.05$) increase in body fat depots: epididymal, retroperitoneal, mesenteric fat depots and total fat depot (Table 3). Treatment with *Betula utilis* extract administration produced significant ($p<0.05$) dose dependent decrease in body fat depots: epididymal, retroperitoneal, mesenteric fat and total fat in comparison to Cafeteria control (Table 6.

Effect of various pharmacological interventions on serum biochemical parameters

Cafeteria diet induces significant elevation of serum total cholesterol (TC), triglycerides (TG), LDL, VLDL and decrease in HDL in Cafeteria diet group as compared to age matched normal animals on standard diet. (Table 3). Oral treatment with graded dose of *Betula utilis* extract (100, 200 and 400 mg/kg) caused a significant ($p<0.05$) reduction in serum total cholesterol (TC), triglycerides (TG), LDL, VLDL of HFD fed rats and significant increase in the level of HDL as compared to Cafeteria control group (Table 6). There was no significant *perse* effect of *Betula utilis* extract and orlistat.

Table 2. Effect of normal diet & Cafeteria diet on the body weight, body mass index, Lee index and Feed intake in gram and feed intake in Kcal:

Parameter	Normal control	Cafeteria diet
Initial Body weight (gm)	209.33 ± 14.7	214.67 ± 19.4
Final Body weight (gm)	263.16 ± 7.61	363.5 ± 22.5[a]
Body mass index (g/cm^2)	0.78 ± 0.04	1.17 ± 0.04[a]
Lee index ($gm^{1/3}/cm*1000$)	351.0 ± 8.56	408.86 ± 11.05[a]
Feed intake (gm)	24.32 ± 3.53	22.9 ± 2.02
Feed intake (Kcal)	95.25 ± 13.26	115.71 ± 11.88[a]

All values are expressed as Mean ± S.D; [a] = $p< 0.05$ vs normal diet control

Table 3. Effect of Cafeteria diet on the serum glucose, serum lipid profile:

Parameter	**Normal diet control**	**Cafeteria diet**
GLU (mg/dl)	93.32 ± 3.08	159.87 ± 2.56[a]
TC (mg/dl)	98.63 ± 0.82	158.42 ± 4.03[a]
TG (mg/dl)	69.35 ± 3.57	149.29 ± 1.85[a]
HDL (mg/dl)	32.27±1.66	21.29 ± 0.60[a]
VLDL (mg/dl)	13.64 ± 0.73	29.85 ± 0.37[a]
LDL (mg/dl)	53.70 ± 1.90	107.27 ± 3.66[a]

All values are expressed as Mean ± S.D; [a] = $p< 0.05$ vs normal diet control

Table 4. Effect of normal diet and Cafeteria diet on the various fat pads:

Parameter	Normal diet control	Cafeteria diet
MES (gm)	2.71 ± 0.28	7.05 ± 0.32[a]
RET (gm)	1.54 ± 0.30	7.61 ± 0.42[a]
EPI(gm)	1.90 ± 0.10	4.9 ± 0.54[a]
TF (gm)	6.15 ± 0.47	19.56 ± 1.30[a]

All values are expressed as Mean ± S.D; [a] = $p < 0.05$ vs normal diet control

Table 5. Effect of various pharmacological interventions on the body weight, body mass index, lee index, feed intake in gram and feed intake in Kcal:

Parameters	Initial Body weight (gm)	Final Body Weight (gm)	Body mass Index (gm/cm^2)	Lee index ($gm^{1/3}/cm$ *1000)	Feed intake (gm)	Feed Intake (Kcal)
Normal diet treatment						
Normal diet control	209.33 ± 14.7	263.16 ± 7.61	0.78 ± 0.04	351.05 ± 8.56	24.32 ± 3.53	95.25 ± 13.26
Betula utilis extract – *perse*	209 ± 10.01	246 ± 12.05	0.85 ± 0.08	342.63 ± 8.02	22.07 ± 1.16	83.34 ± 0.92
Orlistat per se	204±9.1	253 ± 6.5	0.81 ± 0.07	340.52 ± 7.5	22.03 ± 1.10	84.34 ± 0.90
Cafeteria diet treatment						
Cafeteria diet	214.67 ± 19.4	363.5 ± 22.5[a]	1.17 ± 0.04[a]	408.86 ± 11.05[a]	22.9 ± 2.02	115.71 ± 11.88[a]
Cafeteria diet + *Betula utilis* extract (low)	209.82 ± 18.65	306 ± 12.03	0.96 ± 0.05	378.71 ± 7.91	16.03 ± 1.18	91.75 ± 6.22
Cafeteria diet + *Betula utilis* extract (medium)	205.66 ± 10.2	259.31 ± 8.15	0.86 ± 0.05	365.20 ± 8.82	15.03 ± 0.71	86.41 ± 3.84
Cafeteria diet + *Betula utilis* extract (High)	215.5 ± 6.11	250.33 ± 11.5	0.84 ± 0.08	368.21 ± 12.44	12.86 ± 0.30	80.26 ± 1.66

All values are expressed as Mean ± S.D; [a] = $p < 0.05$ vs. normal control,[b] = $p < 0.05$ vs. Cafeteria Diet.

Table 6. Effect of various pharmacological interventions on the serum glucose and lipid profile:

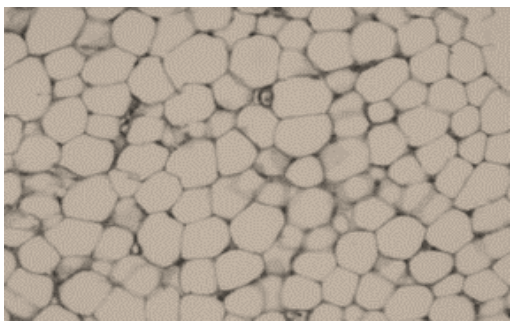
Parameters	Serum glucose (mg/dl)	Serum total cholesterol (mg/dl)	Serum triglyceride (mg/dl)	Serum HDL (mg/dl)	Serum VLDL (mg/dl)	Serum LDL (mg/dl)
Normal diet treatment						
Normal diet control	93.32 ± 3.08	98.63 ± 0.82	69.35 ± 3.57	32.27 ± 1.66	13.64 ± 0.73	53.7 ± 1.90
Betula utilis extract –*per se*	101.54 ± 1.06	98.30± 2.15	76.55 ± 1.22	33.29 ± 0.82	15.31 ± 0.24	59.69 ± 2.40
Orlistat Per se	101.16 ± 1.28	97.16± 0.53	61.37 ± 1.50	30.70 ± 0.91	12.27 ± 0.30	54.18 ± 0.84
Cafeteria diet treatment						
Cafeteria diet	159.87 ± 2.56[a]	158.42 ± 4.03[a]	149.29 ± 1.85[a]	21.29 ± 0.60[a]	29.85 ± 0.37[a]	107.27 ± 3.66[a]
Cafeteria diet + *Betula utilis* extract (low)	133.45 ± 1.54[b]	134.37 ± 1.72[b]	128.98 ± 1.41[b]	22.08 ± 0.81[b]	25.98 ± 0.21[b]	84.56 ± 2.11[b]
Cafeteria diet + *Betula utilis* extract (medium)	116.31 ± 2.20[b]	111.68 ± 2.08[b]	104.06 ± 1.02[b]	28.07 ± 0.98[b]	22.30 ± 0.31[b]	68.29 ± 2.31[b]
Cafeteria + *Betula utilis* extract (high)	101.20 ± 1.31[b]	102.92 ± 0.88[b]	88.81 ± 1.59[b]	32.29 ± 1.21[b]	18.91 ± 0.32[b]	52.60 ± 1.68[b]

All values are expressed as Mean ± S.D; [a] = $p < 0.05$ vs. normal control, [b] =$p < 0.05$ vs.

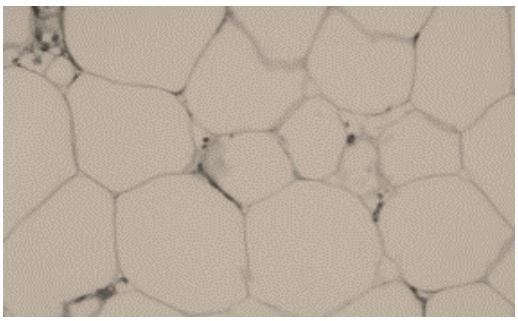
Table 7. Effect of various pharmacological interventions on the various fat pads:

Parameters	MES	RET	EPI	TF
Normal diet treatment				
Normal diet control	2.71 ± 0.28	1.54 ± 0.30	1.90 ± 0.10	6.15 ± 0.4
Betula utilis extract *per se*	2.55 ± 0.21	2.32 ± 0.08	2.18 ± 0.01	7.02 ± 0.16
Orlistat Per se	2.51 ± 0.22	2.45 ± 0.09	2.19 ± 0.10	7.15 ± 0.34
Cafeteria diet treatment				
Cafeteria diet	7.05 ± 0.32[a]	7.61 ± 0.42[a]	4.9 ± 0.54[a]	14.56 ± 1.3[a]
Cafeteria diet + *Betula utilis* extract (low)	4.05 ± 0.21[b]	4.2 ± 0.23[b]	3.98 ± 0.28[b]	12.23 ± 0.04[b]
Cafeteria diet + *Betula utilis* extract (medium)	3.58 ± 0.13[b]	3.29 ± 0.77[b]	2.22 ± 0.48[b]	8.98 ± 0.21[b]
Cafeteria diet + *Betula utilis* extract (high)	2.06 ± 0.41[b]	2.98 ± 0.41[b]	1.99 ± 0.24[b]	6.03 ± 0.44[b]

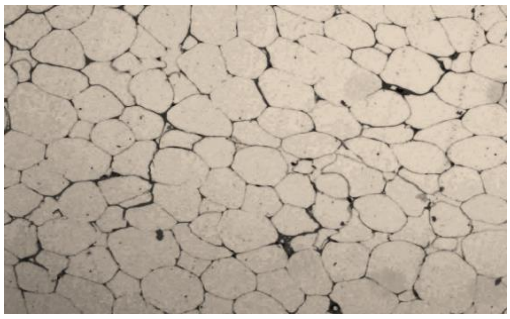
All values are expressed as Mean ± S.D; [a] = $p < 0.05$ vs. normal control,[b] = $p < 0.05$ vs. Cafeteria Diet.



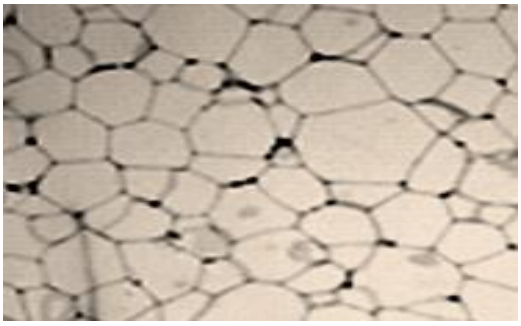
Normal Control



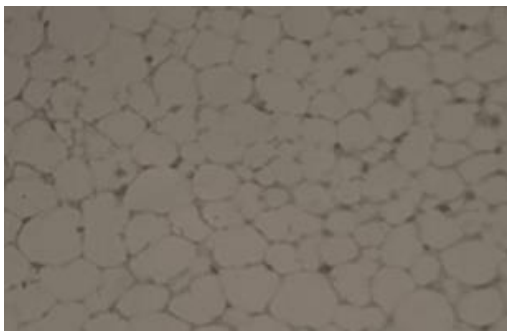
Cafeteria Diet



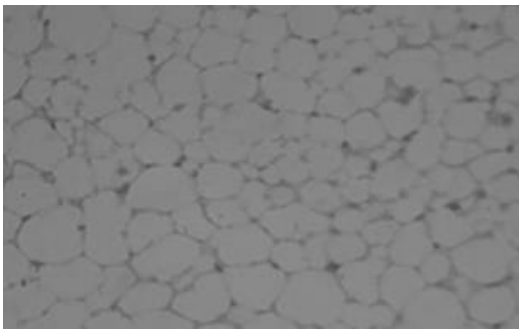
Orlistat Per se



BU Per se



Cafateria Diet + BU Low



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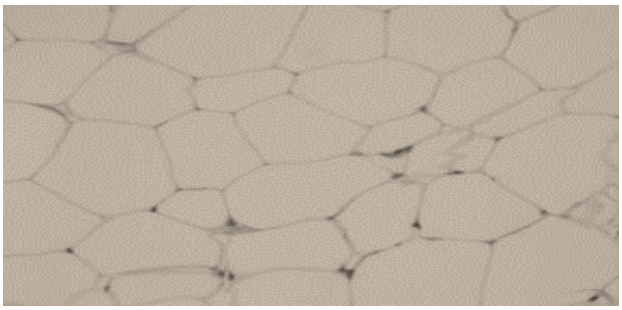

Cafeteria diet + BU High

CONCLUSION

Obesity, caused by an imbalance between caloric intake and energy expenditure, leads to fat accumulation and various chronic diseases. Betula utilis, a medicinal plant, contains compounds like betulinic acid, which exhibit anti-obesity effects by inhibiting enzymes involved in fat and carbohydrate metabolism. This study investigates the effects of Betula utilis extract on cafeteria diet-induced obesity in rats. The extract, administered in doses of 100, 200, and 400 mg/kg for six weeks, significantly reduced obesity markers and improved biochemical parameters. The highest dose was most effective, suggesting Betula utilis extract's potential in preventing and treating obesity.

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