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RESEARCH ARTICLE

Anti-obesity and Antihyperlipidemic Activity of *Ougeinia oojeinensis* (Roxb.) in High Fat Diet-Induced Obesity in Experimental Animals

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Abstract

The present study was planned to assess the anti-obesity efficacy of petroleum ether, ethyl acetate, and methanolic extracts of leaves of *Ougeinia oojeinensis* (Roxb.) belonging to the family *Fabaceae*. The soxhlation method was employed to produce the extracts. Wistar rats were subjected to a high-fat diet for 40 days to induce obesity. Along with the high-fat diet, a standard drug Orlistat 50 mg/kg and various extracts of *O. oojeinensis* at 100 mg/kg and 200 mg/kg were administered for 40 days. The key markers like lipid profiles, SGOT, SGPT, glucose, body weight, food intake, body temperature, atherogenic index, coronary index, and weight of the organs were assessed. The anti-antioxidant properties like TBARS, GSH, GR, Gpx, SOD, and CAT were also estimated. The results revealed that *O. oojeinensis* with the doses 100 and 200 mg/kg showed significant Anti obese and hypolipidemic effects in rats fed with a high-fat diet.

Keywords: Antihyperlipidemic Anti-Obesity, Antioxidant, Lipid Profile, *O. oojeinensis*

1. Introduction

Obesity is one of the leading health concerns in the world today and has been linked to increased morbidity, a higher death rate, and a shorter life expectancy[1]. The lifestyle and eating habits of humans have made it an inevitable result after a certain age. The number of persons who are obese and of a hefty weight is rapidly increasing. Numerous disorders, including hyperlipidemia, diabetes, atherosclerosis, liver damage, and malignancies, are made more likely by obesity[2]. Additionally, obesity raises the financial burden placed on the person and ultimately the government[3]. Due to an unfair distribution of obesity and/or increased energy consumption, a Westernized diet presents the main risk problem for high fat. The main issue with the Western diet is that it either contains excessive amounts of energizing substances or high-fat compounds[4]. This encourages the production of free radicals and raises the risk of cardiac-related problems[5].

The lipid-lowering medications that are now on the market have a variety of negative effects. As a result, diet experts and nutritionists are continuing to look into other anti-obesity treatments that include beneficial ingredients or dietary recommendations. Despite the serious requirement for effective and safer therapies and the expected magnitude of the market for anti-obesity

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drugs, efforts are currently being made to develop these drugs[5]. The usage of natural substances for the treatment of obesity is currently underutilized but has the potential to be a fantastic alternative method for creating secure and efficient anti-obesity medications. Many investigations have demonstrated that bioactive chemicals, including steroids, alkaloids, flavonoids, tannins, and saponins have promising effects in treating stoutness by a few components.

The *Fabaceae* plant *Ougeinia oojeinensis* (Roxb.) is a well-known deciduous tree found in the Northern and Deccan parts of India. It's been used as part of Ayurvedic formulation or alone for various clinical conditions like obesity, urinary disorders, skin conditions, and conditions involving inflammation and spasms[6]. One such notable Ayurvedic formulation is Tinisha and its clinical applications were not scientifically documented[7]. The bioactive components isolated from *O. oojeinensis* are ougenin, ferreirin, genistein, kaempferol, dalbergioidin, lupeol, orobol, neophellamuretin, homoferririn, wedelolactone, isoflavanone, botulin, etc[8]. This study, thus, aimed at determining whether Petroleum Ether (PE), Ethyl Acetate (EA), and Methanolic (MA) leaf extracts of *O. oojeinensis* possess anti-obesity effects.

2. Materials and Methods

2.1 Animals

Wistar strain rats (150-200 gm) of either sex were picked after obtaining Institutional Animal Ethics Committee approvals (CPCSEA/IAEC/JIPS/19/1/1) and quarantined for one week for this study. The animals were maintained at 25±1 °C temperature, 55±10 % relative humidity and 12 hr light and dark cycle and provided with water *ad libitum* and a standard pellet diet.

2.2 Chemicals

Orlistat was purchased from Surety Healthcare, Gujarat, India. Standard diagnostic kits were employed for measuring the Triglycerides (TG), Total Cholesterol (TC), High-Density Lipoprotein (HDL), Serum Glutamate Pyruvate Transaminase (SGPT), and Serum Glutamate Oxaloacetate Transaminase (SGOT) done using standard kits and other chemicals are analytical grade.

2.3 Herbal Extract Preparation

The *O. oojeinensis* leaves were collected from Nanded district of Maharashtra. Freshly collected leaves of *O. oojeinensis* were dehydrated under shade and grinded to a coarse powder. The resultant powder was kept in an airtight amber-colored container in a cool and dry place. The soxhlation process was employed for extracting the phytoconstituents from the leaf powder. 25 gm of leaf powder was extracted using 250 ml of PE, EA, and MA for 72 hrs. These extracts of leaves of *O. oojeinensis* were used for this study and sodium carboxyl methyl cellulose (Sod. CMC) (2%) was used as a vehicle.

2.4 Selection of Dose

For the existing study, multiple doses of *O. oojeinensis* leaf extracts were taken (100 and 200 mg/kg *p.o.*), and the dose selection was done based on the earlier studies[9].

2.5 Composition of High-Fat Fat Diet (HFD)

The high-fat diet contains 20% casein, 15% corn starch, 0.3% D, L methionine, 27.5% sucrose, 5% cellulose powder, 3.5% minerals, 1% vitamin complex, 0.2% choline derivatives, 9.9% corn oil, 17.6% lard oil. The HFD was ready, dehydrated, crushed and given throughout the treatment period[10].

2.6 Experimental Design

The Wistar rats were arbitrarily distributed into nine groups — six rats in each group with 40 days of treatment[11]. The respective treatment groups and treatment protocols were stated in Table 1.

2.7 Estimation of Lipid Profile

To estimate the serum TC, HDL (CHOD-PAP), and TG (GPO-PAP), the animals are imposed to fast overnight

Table 1 Animal experimental design protocol

Treatment groups	Treatment
Group-I	0.1% Na.CMC
Group-II	High-fat diet (HFD)
Group-III	HFD+Orlistat (50 mg/kg)
Group-IV	HFD+PEOO (100mg/kg)
Group-V	HFD+ PEOO (200mg/kg)
Group-VI	HFD+EAOO (100mg/kg)
Group-VII	HFD+EAOO (200mg/kg)
Group-VIII	HFD+MAOO (100mg/kg)
Group-IX	HFD+MAOO (200mg/kg)

Note: Group-1-Normal control; Group-II-Negative control; Group-III- Positive control

and sacrificed by cervical decapitation. 2 mL of blood was withdrawn by cardiac puncture on the 40th day[12]. Friedewald formula was used to calculate LDL-C and VLDL-C levels.

2.8 Evaluation of Antioxidant Properties

2.8.1 Determination of Lipid Peroxidation (LPO)

The Thio-Barbituric Acid Reactive Substance (TBARS) quantities were measured as lipid peroxidation markers by using the standard procedure. The solution consists of 0.4 mL serum sample or liver tissue homogenate, 1.5 mL each of sodium dodecyl sulphate (SDS, 8.1%), acetate buffer (20%, pH 3.5) and TBA (0.8%) solution, heated at 95°C for about an hour. Then allowed to cool to room temperature, later 5.0 mL of n-butanol and pyridine (15:1) were added and subjected to centrifugation for 15 min at 3000 rpm. The absorbance of the separated organic layer was read at 532 nm. Malondialdehyde (MDA) equivalents were used to generate the standard curve. The standard unit to express was nM/mL in serum samples[13].

2.8.2 Determination of Glutathione (GSH) Reduction

The reduction of GSH molecule was evaluated by half mL of the serum sample added with 125 µL of TCA (25%) and the resulting solution was cooled in the ice bath for 5 minutes. Later the sample solution was treated with 5% TCA (0.6 mL) and centrifuged at 5000 rpm/10 min. From this solution, the supernatant layer (0.3 mL) was isolated and added to phosphate buffer (0.7 mL) and Ellman's reagent (2 mL). The developed yellow mixture was measured for absorbance in a colorimeter at 412 nm. The quantity of GSH was measured in µg/mL for the serum sample[14].

2.8.3 Determination of Glutathione Peroxidase (GPx)

The estimation of GPx was performed by using 0.2 mL of serum samples to Tris-HCl buffer (0.2 mL). Then 0.2 ml of EDTA, and 0.1 ml of sodium azide were added and vortexed. 0.2 mL GSH and 0.1 mL H_2O_2 were added to the resulting mixture. At 37°C, this mixture was incubated along with the control sample for 10 minutes. TCA (10%, 0.5 mL) was used to stop the reaction after 10 min. After centrifuging, the supernatant layer was collected and examined for GSH inhibition at 340 nm by colorimetric method. The serum sample GPx levels were expressed in U/m[14].

2.8.4 Glutathione Reductase (GR)

The liver collected from the experimental animals was sliced into tiny pieces and subjected to homogenization in the ice-cold solution of sucrose (0.25M, 9 mL/g) for 45 min at 14000 rpm. The pH of the supernatant layer was set to 5.5 with 0.2 M ethanoic acid and subjected to centrifugation for another 45 min at 14000 rpm. Later, the oxidation rate of glutathione disulfide (GSSG) by NADPH at 30°C is employed as standard. A 1 mL of the sample contains 1.0 mM of each GSSG, NADPH, 0.5 mM of EDTA, 0.10 M of pH 7.6 phosphate buffer, and the *glutathione reductase* was added, and the absorbance measured spectrophotometrically[15-17].

2.8.5 Determination of Superoxide Dismutase (SOD) Activity

The SOD activity was performed by using serum samples that were suspended in chloroform and ethanol and centrifuged at 1800 rpm/6 min, later, the separated layer was adjusted to pH 7.8 with phosphate buffer. By adding the EDTA, Nitro Blue Tetrazolium (NBT), and riboflavin to the above mixture, the absorbance was measured at 560 nm. After the measurement of the initial readings, the test samples were kept in a dark chamber for 15 min. After that the absorbance was read at 560 nm, and the difference was recorded between the initial and final absorbance. The enzyme-specific activity was measured and mentioned in U/mL for the serum sample[18].

2.8.6 Assay of Catalase (CAT)

The *catalase* activity was measured by taking a 0.1 mL serum sample and liver tissue homogenate in 0.9 mL phosphate buffer and H_2O_2 (0.4 mL) was added. Later, dichromate acetic-acid mixture (1:4, 2.0 mL) was added to the reaction to arrest the process after every 15, 30, 45, and 60 sec for 10 min. The sample tubes were boiled in a water bath, and cooled down to room temperature and the developed color was examined at 590 nm. 20-100 µM H_2O_2 (0.2 mM) solutions were used as standard. The enzyme was measured as U/mL for the serum sample[19].

2.9 Effect of Liver Enzymes

In a test tube, 1mL of the test extract was added and kept at 40°C temperature in a water bath for 10 min. Later 0.2 mL

of serum was added and the entire contents were vortexed. The resultant solution was incubated for 60 min for GOT and 30 min for GPT. 1mL of the 2, 4-dinitrophenyl hydrazine reagent was added immediately to stop the reaction. 10 mL of NaOH (0.4 N) was added to the above mixture at room temperature and the contents were mixed by inversion. The optical density of the solution was examined at 505 nm at the end of the 30 min by using water as the blank[17].

2.10 Effect on Blood Glucose Levels

The retro-orbital plexus puncture was used to collect samples of the anaesthetized animal's blood. Basal reading was taken at the time of grouping of animals. Blood was collected after the treatment and centrifuged at 3500 rpm/20 min. The serum from the blood was separated and the glucose concentration was determined by the GOD-POD method[20].

2.11 Improvement in Weight and Consumption of Food

The improvement of the weight was noted from the first day and then, it was measured weekly. Weighing apparatus was used to measure them. The everyday nourishment eating on behalf of all sets was assessed daily for 40 days[21].

2.12 Atherogenic and Coronary Risk Index (ACRI) Assessment

The formulae used to measure the ACRI remained as the ratios of LDL-C to HDL-C and TC to HDL-C respectively[20].

2.13 Body Temperature

The basal rectal temperature was measured using a digital clinical thermometer. The thermometer was inserted 2 cm depth into the rectum and the difference in rectal temperature was measured. The animal's rectal temperature was noted at regular intervals and respective treatments were followed[20].

2.14 Organ and Fat Pad Weights

The kidney, liver, and fat pads (retroperitoneal, epididymal, and mesenteric fat pads) were separated, followed by washing them in frozen saltwater and then, they were weighed[20].

2.15 Statistical Analysis

Statistical analysis was done by one-way ANOVA and found significant results ($P<0.05$, $P<0.01$, $P<0.001$) after Tukey's post hoc test by SPSS software.

3. Results

3.1 Phytochemical Investigation of *O. oojeinensis* Leaf Extracts

The occurrence of various phytochemical constituents in PE, EE, and ME extracts were stated in Table 2.

3.2 Effect of *O. oojeinensis* Leaf Extracts on Lipid Profile in HFD-Induced Obese Rats

The effects of PEOO, EAOO, and MAOO leaf extracts on TC, TG, HDL-C, LDL-C, and VLDL-C concentrations in obese rats induced by HFD (Table 3). The rats that received the PEOO at 100 and 200 mg/kg doses have shown a non-significant reduction in lipid profiles compared to the negative control. But rats that received the EAOO at 200 mg/kg dose have shown an insignificant decline in lipid marker profile and increased HDL-C levels when compared to the rats in the negative control. Conversely, rats given the MAOO at 100 and 200 mg/kg dose were observed a substantial decrease in lipid profiles and a rise in HDL-C levels when the comparison made with negative control respectively.

Table 2. Phytochemical profile in PE, EA, and MA extracts of *O. oojeinensis* leaves

Phytochemical	PE	EE	ME
Alkaloids	-	+	+
Glycosides	-	-	+
Saponins	-	+	+
Flavonoids	-	+	+
Tannins	+	+	+
Steroids	-	+	+
Triterpenoids	+	-	-
Protein	-	-	-
Carbohydrates	-	-	+
Fixed oils and fats	-	-	-

Table 3. Effect of *O. oojeinensis* leaf extracts on lipid profile

Group	Dose (mg/kg)	Lipid profile (mg/dl)				
		TG	TC	HDL-C	LDL-C	VLDL-C
Normal	0.1% Na.CMC	71.81±1.92	94.18 ±2.70	26.91±1.33	54.2±1.4	13.66±1.27
HFD	--	135.22±3.70	157.59 ±4.12	16.17±2.56	123.41±3.02	26.19±1.93
STD	50	71.34±1.85***	91.04±3.24***	25.52±1.46***	43.99±2.84***	13.66±1.40***
PEOO	100	130.41±3.32	156.29 ±3.42	15.11±1.66	118.37±3.13	24.11±1.43
	200	132.67±2.94	155.35 ±2.19	14.87±1.47	119.71±2.96	25.09±1.21
EAOO	100	132.36±2.58	155.36 ±2.90	13.27±1.59	118.50±2.83	24.49±1.39
	200	129.44±2.82*	150.36 ±2.92*	20.83±1.45*	117.38±2.61*	23.23±1.40*
MAOO	100	128.54±3.24**	149.48±3.60**	22.99±1.54**	116.85±3.72**	22.98±1.02**
	200	127.19±2.75***	148.11±3.93***	24.5±1.56***	115.26±2.78***	22.39±1.01***

*Statistically significant at *P<0.05, **P<0.01 and *** P<0.001*

Table 4. Effect of *O. oojeinensis* leaf extracts on liver enzymes and glucose levels

Group	Dose (mg/kg)	SGPT (IU/L)	SGOT (IU/L)	GLUCOSE
Normal	0.1% Na.CMC	12.51±1.02	29.04±1.08	53.28±4.23
HFD	---	25.04±1.13	45.89±1.34	110.34±5.82
STD	50	20.75±1.41***	42.18±1.53***	99.96±1.23***
PEOO	100	26.78±1.98	43.78±1.32	108.07±3.35
	200	25.43±1.54	45.59±1.42	108.79±3.81
EAOO	100	24.62±1.88	44.33±1.10	109.72±3.84
	200	21.89±1.22*	42.56±1.19**	101.51±3.06**
MAOO	100	21.92±1.08*	43.26±1.12*	103.08±3.54*
	200	20.97±1.27***	42.17±1.10***	100.47±3.82**

*Statistically significant at *P<0.05, **P<0.01 and *** P<0.001*

3.3 Effect of *O. oojeinensis* Leaf Extracts on Liver Enzymes and Glucose Concentrations in HFD-Induced Obese Rats

The rats which received the PEOO extract at 100 and 200 mg/kg doses have shown non-significant increments in SGPT, SGOT, and glucose levels (Table 4). Therefore, rats that received EAOO extract at 200 mg/kg dose showed a significant reduction in SGPT, SGOT, and glucose levels (compared to negative control respectively). The MAOO extract at 100 and 200 mg/kg doses had a significant decline in SGPT, SGOT, and glucose levels compared to negative control respectively.

3.4 Effect of *O. oojeinensis* Leaf Extracts on Food Intake and Weight Gain

The results have shown that negative control obese rats had high food intake and weight gain when compared to the normal control group and the rats received the phyto-extracts (100 mg/kg and 200 mg/kg), orlistat received rats (Table 5).

3.5 Effect of *O. oojeinensis* Leaf Extracts on Atherogenic and Coronary Index (ACRI) Assessment in HFD-Induced Obese Rats

The rats that received 100 and 200 mg/kg dose of PEOO extract displayed non-significant decreases in the ACRI

Table 5. Effect of *O. oojeinensis* leaf extracts on food intake, weight gain, atherogenic index, coronary index, and temperature

Group	Dose (mg/kg)	Body weight gain (gm)	Food intake (gm)	Atherogenic index	Coronary risk index	Temperature	
						0 min	120 min
Normal	0.1% Na.CMC	25.32±1.22	8.16±0.24	0.32±0.02	1.13±0.21	38.39±1.13	38.24±1.29
HFD	--	43.65±1.80	9.79±0.48	2.25±0.04	3.75±0.82	36.52±1.56	35.99±1.62
STD	50	39.10±1.33***	6.30±0.72***	2.08±0.06***	2.42±0.19***	35.95±1.03	32.15±1.38***
PEOO	100	45.02±1.62	8.69±0.48	2.21±0.04	3.66 ±0.63	36.42±1.42	34.80±1.15
	200	45.06±1.84	7.48±0.52	2.25±0.05	3.60 ±0.26	36.96±1.18	36.53±1.10
EAOO	100	45.49±1.45	7.69±0.48	2.22±0.03	3.50 ±0.30	36.06±1.32	34.91±1.27
	200	40.60±1.32*	6.66±0.41*	2.12±0.04*	2.70±0.42*	36.95±1.44	32.97±1.82*
MAOO	100	40.50±1.74*	6.69±0.45*	2.11±0.05*	2.69 ±0.56*	36.98±1.02	32.93±1.16*
	200	39.90±1.21**	6.59±0.83**	2.09±0.03**	2.55 ±0.50**	36.05±1.46	32.05±1.46***

*Statistically significant at *P<0.05, **P<0.01 and *** P<0.001*

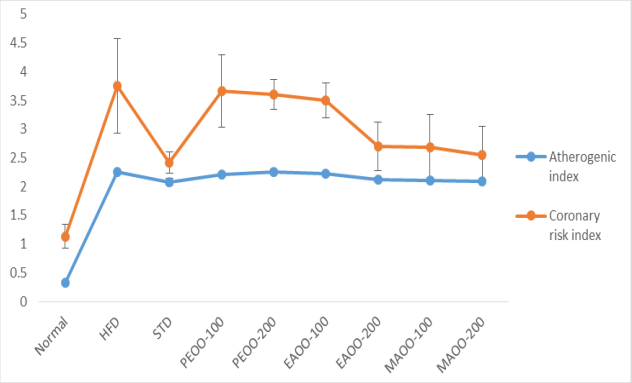

Figure 1. Effect of *O. oojeinensis* leaf extracts on atherogenic and coronary risk index.

compared to the negative control. However, the treatment of 200 mg/kg dose of EAOO extract have shown an insignificant decline in ACRI compared to the rats that are in the negative control, but MAOO extract at a dose of 100 and 200 mg/kg had a significant reduction in ACRI compared to the negative control (Figure 1, Table 5).

3.6 Effect of *O. oojeinensis* Leaf Extracts on Body Temperature in HFD-Induced Obese Rats

Typically, the negative control HFD obese rats had a high temperature when compared with the rats that received different extracts (100 mg/kg and 200 mg/kg), and orlistat-treated rats (Table 5).

Table 6. Effect of *O. oojeinensis* leaf extracts on organ weights

Group	Dose (mg/kg)	Liver	Right kidney	Left kidney
Normal	0.1%Na CMC	3.5±0.03	0.83±0.03	0.53±0.05
HFD	---	6.65 ±0.43	1.15±0.02	1.10±0.04
STD	50	5.01±0.95***	1.05±0.03***	0.99±0.02***
PEOO	100	6.78±0.51	1.21±0.03	1.04±0.06
	200	5.90±0.45	1.18±0.02	1.16±0.04
EAOO	100	6.67±0.80	1.11±0.06	1.09±0.01
	200	5.54±0.66*	1.07±0.03*	1.01±0.05*
MAOO	100	5.49±0.42*	1.08±0.04*	1.02±0.04*
	200	4.99±0.35***	1.06±0.02***	0.98±0.03***

*Statistically significant at *P<0.05, **P<0.01 and *** P<0.001*

Table 7. Effect of *O. oojeinensis* leaf extract on fat pad weights

Group	Dose (mg/kg)	Retroperitoneal fat (mg)	Epididymal fat (mg)	Mesenteric fat (mg)
Normal	0.1%Na.CMC	1.83±0.32	1.13±0.41	1.56±0.36
HFD	--	5.92±1.54	3.02±0.50	0.72±0.41
STD	50	3.48±0.59***	1.66±0.40***	2.34±0.35***
PEOO	100	4.57±0.93	2.92±0.38	1.34±0.53
	200	4.43±0.83	2.64±0.52	1.26±0.43
EAOO	100	4.65±0.75	2.87±0.74	0.97±0.69
	200	4.30±0.45**	1.72±0.62**	1.94±0.55**
MAOO	100	4.37±0.78*	2.01±0.49*	1.82±0.57*
	200	3.92±0.56**	1.89±0.38**	2.32±0.74**

*Statistically significant at *P<0.05, **P<0.01 and *** P<0.001*

Table 8. Effect of *O. oojeinensis* leaf extracts on *in vivo* antioxidant capacity in HFD-induced obese rats

Group	Dose (mg/kg)	TBARS (nmol of MDA formed/ g tissue)	GSH (mg/g tissue)	Gpx (mg of GSH consumed/ min/mg of protein)	GR (mg of GSH consumed /min/mg protein)	SOD (unit min/ mg/ protein)	CAT (µmoles H_20_2 consumed min/mg/ protein)
Normal	0.1% Na.CMC	18.45±2.12	3.63±0.43	8.34±2.03	1.96±0.13	4.93±1.43	28.53±2.31
HFD	-----	41.24±2.04	1.45±0.25	4.20±1.62	0.84±0.10	1.40±1.05	18.81±1.43
STD	50	36.32±1.17***	2.56±0.52***	7.95±1.87**	1.32±0.15***	4.89±1.23**	23.84±1.34***
PEOO	100	38.41±1.32	1.30±0.28	4.25±1.72	0.56±0.16	1.78±1.01	16.62±1.36
	200	39.60±1.78	1.44±0.46	4.75±1.48	0.61±0.18	1.99±1.24	18.51±1.58
EAOO	100	38.45±1.54	1.55±0.24	4.92±1.36	0.73±0.14	2.10±1.40	19.63±1.88
	200	36.92±1.72**	2.36±0.38*	7.42±1.41*	1.22±0.19**	4.23±1.89*	22.69±1.92*
MAOO	100	37.63±1.42*	2.24±0.29*	7.37±1.36*	1.19±0.17*	4.12±1.08*	22.35±1.84*
	200	36.33±1.20***	2.44±0.31***	7.95±2.03**	1.26±0.20**	4.68±1.65**	23.56±1.93***

*Statistically significant at *P<0.05, **P<0.01 and *** P<0.001*

3.7 Effect of *O. oojeinensis* Leaf Extracts on Organ and Fat Pad Weights in HFD-Induced Obese Rats

The PEOO-treated rats with 100 and 200 mg/kg doses revealed non-significant decreases in the organs (liver, right kidney, and left kidney) and fat pad weights (retroperitoneal fat, epididymal fat, and mesenteric fat) when compared to the negative control (Table 6). Similarly, EAOO extracts at 200 mg/kg dose has shown an insignificant decrease in all organ and fat pad weights when compared to the negative control rats (Table 7). However, MAOO extract at 100 and 200 mg/kg dose had a significant decrease in organ and fat pad weights compared to the negative control.

3.8 Effect of *O. oojeinensis* Leaf Extracts on *In Vivo* Antioxidant Capacity in HFD-Induced Obese Rats

The rats that received the PEOO extract at 100 and 200 mg/kg doses have shown a non-significant increase in antioxidants compared to the negative control. With the EAOO plant extract at 200 mg/kg dose has shown an insignificant rise in GSH, GR, GPX, SOD, and CAT levels and a decrease in TBARS levels compared to the negative

control (Table 8). On the other hand, the rats that received the MAOO extract at 100 and 200 mg/kg dose had a significant increment in GSH, GPX, GR, SOD, and CAT levels and a decrease in TBARS levels when compared to negative control respectively.

4. Discussion

Hyperlipidemia and obesity ensure remained important care for community health officials in emerging Nations as they can affect a surprising rise in the risk features of diabetes, hepatic adipose infiltration, and cardiovascular diseases. Obesity-associated illnesses are the chief source of mortality Worldwide, prominent to the expansion of numerous treatments for obesity to stop its difficulties[21]. For the treatment of obesity conventional drugs ensure limitations, outcomes show numerous adverse events and are individually used for patients with a BMI over 30 or 27-29.9 kg/m^2 through multiple medical conditions who are incapable to attain loss of weight. In this background, it would be applicable to assess the herbal extracts, especially, phytochemicals with hypolipidemic and antioxidant properties.

High fat-diet results in the deposition of fat in the liver and to rise in mass, intuitive adipose tissue, TG, and TC[22]. HFD Persuaded Fat Method is a perfect tool to know the relationship between HFD and the progress of obesity. The present work assessed the lipid-lowering properties of *O. oojeinensis* in HFD-produced fat. In outcome, management through *O. oojeinensis* led to lipid-lowering effects on weight, the inhibited lipid and sugar intensities.

Total body weight decrease is the main area in considering obesity, as investigation consumes with decreased weight in substantial or obese is connected through lessening death level[23]. In the present study, treatment with *O. oojeinensis* extracts was accompanied by a substantial decrease in complete body weight and food intake. In lipid production, acetyl-CoA is changed toward TG intended to load in fat materials, creating together free fatty acids and triglycerides[24,25]. TG remnants in the fluid connective tissue, enfolded in a hydrophobic chylomicron through the enzyme. In HDL-C elevation intensities consistently display an opposite link with coronary actions. But excessive amounts of TC and LDL-C raise the risk of atherosclerotic coronary diseases[26]. In these studies, the levels of free fatty acids, TG, TC, and LDL-C remained abolished in animals treated with *O. oojeinensis* extracts contrasted with the HFD group.

The *O. oojeinensis* extracts treated groups presented an enhanced HDL-C likened to the HFD group. Our results specify that *O. oojeinensis* hypothetically beneficial and reduces elevated lipid levels and associated complications. An extreme HFD diet can lead to Nonalcoholic Fatty Liver Disease (NAFLD) which can induce liver problems such as Non-Alcoholic Steatohepatitis (NASH) and cirrhosis of the liver[27]. In these assessments, the HFD group existing augmented SGOT and SGPT amounts and a greater increase of fats in liver tissues than the normal group[28]. Extended continued dyslipidemia has been considered a chief issue intended for circulatory intimidations similar to atherosclerosis[29,30].

The atherogenic and coronary index is measured as an indicator on the way of cardiovascular diseases. In this study, the animals fed with an HFD caused increased atherogenic and coronary index in the atherogenic diet group whereas treatment with *O. oojeinensis* extracts displayed significant lessening, so given that cardio protection.

Particularly, the HFD group's mesenteric fat, epididymal fat, and retroperitoneal fat were all higher than those of the normal group[31]. In the current assessment, the anti-obesity evaluation of *O. oojeinensis* extracts was observed in HFD animals by measuring the markers of obesity. The retroperitoneal fat, epididymal fat and mesenteric fat were found to be significantly lesser in the *O. oojeinensis* extracts treatment. In the present study, the weight of the liver was significantly higher in the HFD animals whereas the weight of the kidney was not altered. These results may suggest that PD extracts have no adverse effects on the liver. HFD-fed animals are found to possess increased oxidative stress. In the same way, the present study shows that in high lipid peroxidation and lesser levels of CAT, GSH, Gpx, GR and SOD, indicating the induction of oxidative stress in HFD induced obese group. But management with *O. oojeinensis* improved the levels of CAT, GSH, Gpx, GR, SOD and decreased MDA by obstructing the lipid accumulation in the liver.

However previous studies stated on *O. oojeinensis* for its polyphenolic, flavonoid contents and coumarins, illustrate the auspicious source of antioxidants with various pharmacological activities. Additionally, a polyherbal anti-obese preparation with *O. oojeinensis* extract as the primary ingredient as well as other cutting-edge weight-reducing and hypolipidemic herbal drugs is needed. This preparation will need to be developed to

pinpoint the precise phytoconstituents responsible for the activity at the brain level.

5. Conclusion

The *O. oojeinensis* extracts decrease the body weight and organ weight in HFD encouraged obese animals by changeable obesity parameters like amended sugar levels and lipid profiles in the serum. Lastly, we recommend that *O. oojeinensis* extracts can be used as a great beneficial substitute for the management of obesity due to its antioxidant activity might be permitted by its high phenolic contents. According to the results of the study, *O. oojeinensis* extract significantly reduced the cholesterol and glucose levels in rats given a high-fat diet, which indicated that it had an anti-obesity effect. *O. oojeinensis* extensive history of use may have therapeutic and preventative benefits in the management of certain illnesses. To determine the precise mechanism of *O. oojeinensis* anti-obesity activity and to determine its therapeutic potential in the treatment of obesity, more research involving measurements of hormones and enzymes in lipid pathways are required.

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