Research article

Hypolipidemic effect and modulation of hepatic enzymes by different edible oils in obese Wistar rats

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ABSTRACT

The current study assessed the hypolipidemic effect and modulation of hepatic enzymes by different edible oils in obese Wistar rats. In order to conduct this study, 36 Wistar rats that were collected at 5 weeks of age and weighed an average of 70 g were split into two groups: 28 of them were fed a high-fat diet (HFD) and 8 of them were fed a control diet. After 5 weeks of feeding, rats from the HFD (obese, n = 4) and the control diet group (n = 4) were sacrificed. Subsequently, the rest of obese rats (n = 24) were separated into six groups, including the continuing high-fat (CHF) diet group, rice bran oil (RBO) diet group, olive oil (OO) diet group, soybean oil (SO) diet group, cod liver oil (CLO) diet group, and sunflower oil (SFO) diet group, and the continuing control diet group (n = 4). Rats from each group were sacrificed following an additional 5 weeks, and all analytical tests were carried out. The results found that the interventions of RBO, CLO, and SFO in obese rats reduced their body weight non-significantly when compared with CHF. It was also observed that a non-significant reduction in weight of the heart, AAT, and EAT occurred by RBO,

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OO, SO, and CLO, while SFO reduced the AAT level significantly ($p < 0.05$). Besides, RBO, OO, SO, CLO, and SFO decreased IBAT and liver fat significantly compared to CHF. Similarly, the administration of RBO, OO, SO, and CLO reduced ALT significantly. RBO reduced GGT ($p < 0.05$) significantly, but other oils did not. The given oil has the efficiency to reduce TC, TAG, and LDL-C but increase HDL-C significantly. These findings suggest that different edible oils can ameliorate obesity, regulate lipid profiles, and modulate hepatic enzymes.

1. Introduction

Around the world, obesity is one of the key reasons of death and the most dangerous public health problem nowadays [1,2]. It is a substantial risk issue for cardiovascular disease (CVD), several types of cancer, diabetes, especially type 2 diabetes, and mortality in general [3–6]. In America, over 80% of predicted obesity-related deaths are attributed to those with a body mass index (BMI) of more than 30 kg/m^2. In contrast, obesity rates in Europe fluctuated between 4.0% and 28.3% for men as well as 6.2%–36.5% for women (BMI $>$30 kg/m^2) [7,8]. Around 17% of adult population in Bangladesh suffer from overweight and obesity problems. In contrast, in 1980, 7% of adults and 3% of children were overweight or obese [9,10]. A BMI of 30–35 kg/m^2 often results in a reduction of 2–4 years in longevity, but serious obesity (BMI $>$40 kg/m^2) causes a reduction of 8–10 years in life span [11]. The World Health Organization foresees that undernourishment and infectious diseases may be replaced by concerns about overweight and obesity, which affect 34.3% of Americans over 20 and cost \$503.2 billion in treatment and productivity in 2010 [12,13].

Obesity intensifications the danger of raised hepatic enzymes by two to three times, contrasting with the three-fold increase in steatosis risk at ultrasonography [14]. Omega-3 and omega-6 fatty acids are considered healthier due to their potential to protect against cancer, heart inflammatory diseases, and reduce belly fat size [15,16]. Food administration is the keystone of a remedy for people who are obese because it improves or even normalizes adiposity and related diseases [17]. The finest food menu or diet plan prevents gaining of weight, overweightness, metabolic disorder, and type 2 diabetes [18]. Substituting saturated fatty acids with
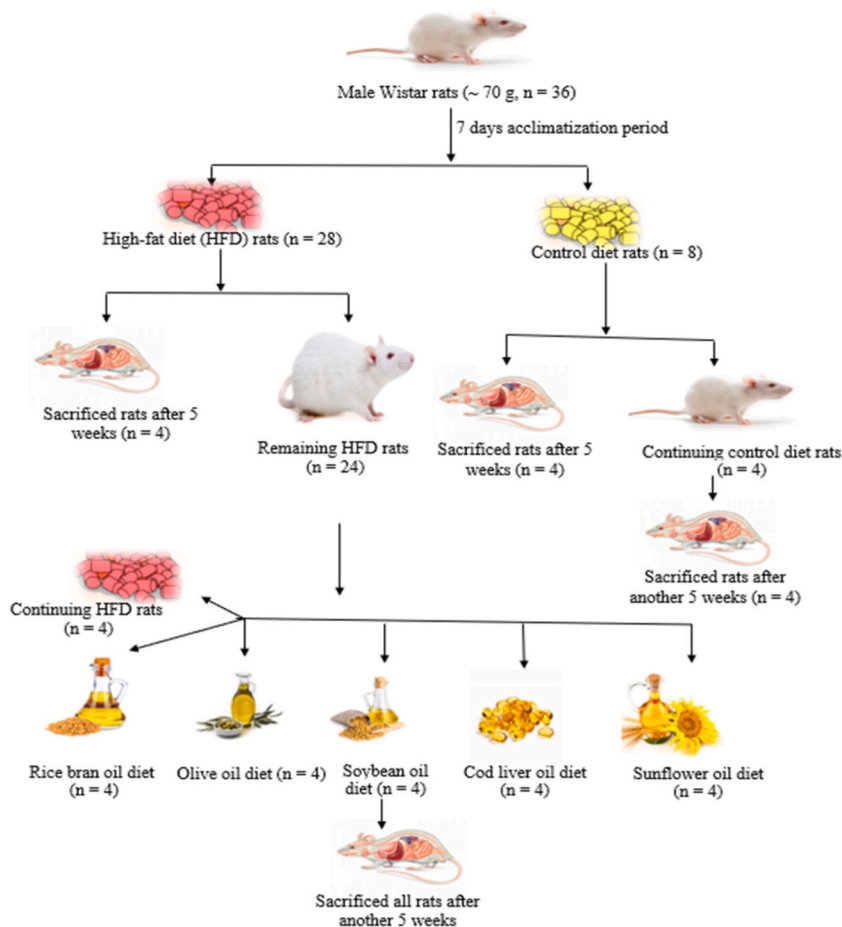

Fig. 1. Schedule of the research protocol.

unsaturated fatty acids has proven beneficial for controlling lipid profiles and obesity-related diseases [19]. Polyunsaturated fatty acids (PUFA) benefit liver fat reduction, mainly by changing the liver marker [20]. Currently, weight loss by food and workout therapy is the foremost dealing for non-alcoholic fatty liver disease (NAFLD), and it has been demonstrated to progress hepatic enzymes [21–23] and diminish plasma triglycerides [22,24].

Continuous weight management, managing the promising lipid profile for the long term, and plummeting fat storage from different fat depots are positive outcomes in preventive measures against obesity and in lowering its complications [25]. Dietary oil, which comprises a range of oils such coconut, mustard, palm, sesame, soybean, rapeseed, sunflower, olive, and cod liver oil, is one of the most significant sources of fat in the human diet. The fatty acid profile of these oils varies extensively, considering the PUFA content. PUFA aid in the decrease of liver fat primarily by altering the liver marker, decreasing fat depots, and enhancing high-density lipoprotein cholesterol (HDL-C) in the body by lowering low-density lipoprotein cholesterol (LDL-C) [20,26]. The unsaturated fatty acid profiles of rice bran oil (RBO), olive oil (OO), soybean oil (SO), cod liver oil (CLO), and sunflower oil (SFO) may be promising in lowering aberrant fat deposition and NAFLD. Furthermore, Virgin olive oil and other oils can reduce obesity and other CVD complications due to their high content of PUFA [27]. Still, liver enzymes show a vital part in signaling the possibility of developing liver diseases in extreme cases, like diabetes, liver cirrhosis, and so on. Different types of oils are capable of reducing obesity and liver enzymes, but the extensive study of the possessions of diverse edible oils on hyperlipidemia and NAFLD is still limited. Many investigations have been done on the impacts of various formulated foods on obesity and NAFLD, but the comparisons of different edibles are still low. So, a significant research field is to attempt to prevent obesity and its complications through dietary correction of fat sources. In this perspective, the authors wanted to elucidate the hypolipidemic effect and modulation of hepatic enzymes by different edible oils in obese Wistar rats.

2. Materials and methods

2.1. Trail animals and laboratory setting

36 male Wistar rats, weighted at an average of 70 g, were procured from the Animal House, Department of Pharmacy, Jahangirnagar University, Bangladesh, at the age of 4–5 weeks. The animals were given a week to get used to lab settings before being used in the trial. Rats were kept in temperature-controlled clinical research setting at 23 ± 5 °C with a 12:12 h light-dark cycle. The Animal Care Committee allowed all of the investigations conducted by this research, and all rats were housed in the accommodations in compliance with animal handling and management guidelines.

2.2. Research design

The study was finished in two stages (Fig. 1). The rats were split into two groups at random for the first period: the control group (n = 8) taken a control diet, while the HFD group (n = 28) received HFD. 4 rats from the control group and 4 from the HFD had been sacrificed after a period of 5 weeks of feeding. Subsequently, the 6 groups comprised of the stayed obese rats (n = 24) were the high-fat diet (CHF), RBO, OO, SO, CLO, and SFO meals. The rats were sacrificed after a further 5 weeks of feeding, and the organs, serum, and fat tissue were all isolated as in the initial stage. After sacrificing, essential analytical tests were done. Rats were given water and diet *ad libitum*.

2.3. Development of diets

Table 1 includes all of the items used in the first and second stages of the experiment and control diet preparations. The diet was

Table 1
Ingredients of control and experimental diet (g/100 g) for first phase and second phase.

Ingredients	First Phase diet (g)		Second Phase diet (g)				
	Control	HFD	RBO	OO	SO	CLO	SFO
Wheat	42	42	42	42	42	42	42
Wheat bran	20	20	20	20	20	20	20
Rice polish	5	0	1	1	1	1	1
Ghee	0	10	0	0	0	0	0
Egg yolk	0	5	0	0	0	0	0
Respective oil	0	0	15	15	15	15	15
Fish meal	10	5	5	5	5	5	5
Till cake	10	5	5	5	5	5	5
Vitamin GS	1	1	1	1	1	1	1
Oil	1	1	0	0	0	0	0
Salt	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Molasses	0.5	0.5	0.5	0.5	0.5	0.5	0.5
SMP	4	4	4	4	4	4	4
MCD diet	6	6	6	6	6	6	6

SMP = Skimmed Milk Powder, MCD = Methionine- and Choline-Deficient.

created using a conventional procedure, and the components were bought at a nearby market [28]. Therefore, proximate composition of control diet, HFD, and oil-based formulated diet are shown in Table 2.

2.4. Fatty acid composition of selected oils

This standard technique [29] was used to assess the fatty acid profile of the selected oils (RBO, OO, SO, CLO, and SFO) (Table 3). The oil samples were collected from the retail shop of Tangail and Dhaka in Bangladesh.

2.5. Measurement of body weight and serum separation

From the first day of the trial to the last, the weight of the rats used for the experiment was measured in triplicate. Body weight growth was determined using the average weight.

Mean weight gain = Final weight (gm) – Primary weight (gm) (1).

Rats were starved for 12 h and then put into the metabolic cages at the ending of each treatment session. Next, an intraperitoneal dose of ketamine K (5 mg/100 g body weight; Abbott, IL, USA) was used to induce unconsciousness the rats. A sterilized syringe was used to draw blood from the abdominal aorta, and the sample was then put in a test tube to clot for 20–30 min. The serum was then refrigerated at −40 °C until examination, and it was centrifuged for 10 min at 3000 rpm at 4 °C.

2.6. Separation of tissue

Wistar rats' fat tissue was thoroughly separated into three distinct regions. In this clinical study, brown adipose tissue (BAT) was separated from interscapular brown adipose tissue (IBAT), abdominal adipose tissue (AAT) was separated from dorsolumber, inguinal, and gluteal posterior subcutaneous depots, and epididymal adipose tissue (EAT) was separated from mediastinal, retroperitoneal, gonadal, and perirenal visceral depots.

2.6.1. Isolation of interscapular brown adipose tissue (IBAT)
After euthanasia, the rats were placed on their abdomens, their heads facing the investigator. In order to wet the coat and prevent hair from getting on the samples, the shoulder area is heavily rinsed with 70% ethanol. With tongs, the skin immediately behindhand the head is elevated and then cut with scissors. From this point to the center of the back, the skin has been heavily incised, and the field has been cleared. IBAT was revealed to be shaped like a butterfly. Then, removed the white portion of the fat pad just above the IBAT by wiping it with a paper towel, carefully dissecting the fat pad, and then carefully dissecting the brown fat butterfly. In every situation, extra care is taken to avoid muscles closely related to brown fat. When the sample was ready, the pad's components were divided as needed [30].

2.6.2. Separation of abdominal adipose tissue (AAT)
With its tail pointed in the direction of the viewer, the rat was lying on its back. The skin of the abdomen was deeply incised after being rinsed with ethanol. Dissect the lymph nodes among the fat after removing the pad, then discard them [30].

2.6.3. Separation of epididymal adipose tissue (EAT)
After removing the abdominal adipose tissue, the abdominal wall was opened. With one hand, dissect the fat tissue or handled the gonadal tract to remove the genitalia from the abdominal cavity. On the other hand, gently pulled the fat away from the different tissues to reveal the genitalia. Warm saline was used to collect the tissue, which was then blotted and weighed to the closest milligram [30].

2.7. Separation of liver and heart

The abdominal wall was opened, the AAT, EAT, and BAT were removed, and the liver and heart were removed. Removed carefully by dissecting the liver and heart with one hand, and separated from the body. Heated saline was used for obtaining the liver and heart, which were then blotted and weighed to the closest milligram [30].

Table 2
Proximate composition of control, HFD, and oil-based formulated diet per 100 g.

Parameter	Control diet	HFD	Oil-based formulated diet
Moisture (g)	8.26 ± 0.44^a	7.98 ± 0.39^b	9.41 ± 0.45^c
Ash (g)	7.29 ± 0.39^d	4.12 ± 0.55^e	5.73 ± 0.38^f
Carbohydrate (g)	66.49 ± 1.68^g	62.21 ± 0.43^h	61.09 ± 1.11^i
Protein (g)	10.27 ± 0.23^j	6.59 ± 0.46^k	8.91 ± 0.38^l
Fat (g)	6.28 ± 1.09^m	17.97 ± 0.59^n	12.88 ± 0.49^o
Fiber (g)	1.41 ± 0.31^p	1.13 ± 0.29^q	1.98 ± 0.42^r

The data is shown as Mean ± SD. According to the Duncan Multiple Range Test (DMRT) of One-Way ANOVA, values in each bar in a row with distinct superscripts are statistically significant from one another ($p < 0.05$) across various diets.

Table 3

Fatty acid composition of selected oils.

Fatty acid composition	Results				
	RBO	OO	SO	CLO	SFO
A) Saturated fatty acids %	22.6010	9.8067	15.5368	16.7858	9.6953
a) Myristic acid (C14:0)	0.1930	–	0.0546	5.0937	0.0403
b) Pentadecyclic acid (C15:0)	–	–	–	0.2332	–
c) Palmitic acid (C16:0)	19.4083	8.3715	11.3950	9.1802	6.1967
d) Margaric acid (C17:0)	–	–	–	0.3497	–
e) Stearic acid (C18:0)	2.3733	1.1469	3.7900	1.9290	2.8113
f) Arachidic acid (C20:0)	0.4994	0.2883	0.1649	–	0.1400
g) Behenic acid (C22:0)	0.1270	–	0.1323	–	0.5070
B) Unsaturated fatty acids %	77.3990	90.1933	84.4632	83.2142	90.3047
i) Monounsaturated fatty acids (MUFA) %	43.0626	86.3620	22.1584	46.2061	27.5735
a) Myristoleic acid (C14:1)	–	–	–	0.0692	–
b) Palmitoleic acid (C16:1)	0.0412	0.5061	–	7.4242	0.0455
c) Oleic acid (C18:1)	42.6623	85.6769	22.0759	13.6534	27.4385
d) Vaccenic acid (C18:1)	–	–	–	2.3365	–
e) Eicosenoic acid (C20:1)	0.3591	0.1790	0.0825	12.1603	0.0895
f) Erucic acid (C22:1)	–	–	–	10.5625	–
ii) Polyunsaturated fatty acids (PUFA) %	34.3364	3.8313	62.3048	37.0081	62.7312
a) Tetradecadienoic acid (C14:2)	–	–	–	0.1439	–
b) Hexadecadienoic acid (C16:2)	–	–	–	0.7299	–
c) Hexadecatrienoic acid (HTA) (C16:3)	–	–	–	0.1581	–
d) Hexadecatetraenoic acid (C16:4)	–	–	–	0.2676	–
e) Linoleic acid (C18:2)	33.4521	3.2177	55.5454	1.5492	62.6910
f) Linolenic acid (C18:3)	0.8843	0.6136	6.7594	4.1662	0.0402
g) Eicosadienoic acid (C20:2)	–	–	–	2.0004	–
h) Eicosatrienoic acid (C20:3)	–	–	–	0.2846	–
i) Arachidonic acid (C20:4)	–	–	–	0.8903	–
j) Eicosapentaenoic acid (EPA) (C20:5)	–	–	–	10.2287	–
k) Docosadienoic acid (C22:2)	–	–	–	0.5955	–
l) Docosatrienoic acid (C22:3)	–	–	–	0.2059	–
m) Adrenic acid (C22:4)	–	–	–	0.1095	–
n) Docosapentanoic acid (DPA) (C22:5)	–	–	–	0.0711	–
o) Docosahexanoic acid (DHA) (C22:6)	–	–	–	15.6072	–

2.8. Determination of liver fat

The percentages of each animal's total liver fat were calculated after the animals were slaughtered. To prevent undigested debris from interfering, the carcass's stomach and intestines were removed. From each rat, around 9 g of liver were taken. The livers were then weighed again, and the difference between the first and last weights was used to determine the proportion of water in the liver. The dehydrated livers were placed into cellulose thimbles that had been previously weighed. The amount of water in the liver sample was determined after it was dried for 24 h at 105 °C in an air oven. *n*-hexane was used as the solvent in a Soxhlet system to extract the lipids from dried livers for 4 h. A computation was made of the recovered lipid content. Each animal underwent this procedure three times [31].

2.9. Analysis of biochemical parameters

Standardized diagnostic techniques (enzyme colorimetric & enzyme kinetic techniques) were used for the measurement of triglycerides (TAG), serum total cholesterol (TC), HDL-C, aspartate aminotransferase (AST), plasma alanine aminotransferase (ALT), alkaline phosphatase (ALP), and gamma glutamic transpeptidase (GGT) with a biochemical instrument Siemens dimension RxL Clinical Chemistry Autoanalyzer, USA using commercial kits (Siemens kits; AHDL GA6104; Siemens Healthcare Diagnostic Inc. Newark DE, 19,714, USA).

LDL-C and VLDL-C were measured by using the Friedwald equations [32]:

$$LDL\text{-}C(mg/dL) = TC(mg/dL) \text{-} HDL\text{-}C(mg/dL) \text{-} TAG\,(mg/dL)/5 \tag{2}$$

$$VLDL\text{-}C = triglycerides/5 \tag{3}$$

2.10. Statistical analysis

For all variables, descriptive statistics were computed using the Statistical Package for Social Sciences (SPSS) software (version 25.0) and the data is shown as Mean ± SEM. The statistical significance of the variations in group means was evaluated using the independent sample student's t-test. Differences were significant at $**p < 0.01$, and $*p < 0.05$.

3. Results and discussion

3.1. Obesity development phase

3.1.1. Consequence of HFD on body weight and BMI at 5th weeks

Following a five-week HFD, the rats' weight rose considerably ($p < 0.01$) than to the control group (Table 4). Feeding with HFD made them obese (0.78 g/cm^2) according to the Lee index [33,34], where the normal range of BMI is 0.45–0.68 g/cm^2. In line with other studies showing increased body weight growth in rats, the study proposes that HFD plays a major role in obesity and weight gain [35–37]. Likewise, additional research has shown that HFD leads to mice becoming fatter and developing insulin resistance [38,39]. HFD diets are one of the crucial factors in increasing the weight of rats, which triggers obesity and other metabolic disorders [40,41].

3.1.2. Consequence of HFD on the organ weight, cholesterol level, and hepatic enzymes

During the first phase, the weight of rats in the liver, heart, AAT, EAT, and liver fat rose considerably ($p < 0.01$) due to the HFD, and there was also a substantial ($p < 0.05$) rise in the weight of IBAT (Table 5). The weight of liver and heart found in this research is reliable with previous investigation that linked HFD to a rise in liver and heart weight [35,37]. This rise in liver weight might be brought on by the buildup of triglycerides and other lipids in the liver, which is a hallmark of nonalcoholic fatty liver disease (NAFLD) [36]. The increase in AAT and EAT weights seen in this study aligns with other research that linked HFD to an increase in adipose tissue weight [42].

Various metabolic diseases like insulin resistance, dyslipidemia, and inflammation are linked to this increase in adipose tissue weight, which may be caused by the buildup of triglycerides and other lipids in the body fat [43,44]. The current research found a significant rise in IBAT weight, which is interesting because it has been suggested that HFD can activate IBAT, which is essential for maintaining homeostasis by releasing energy as heat through thermogenesis. It has been that activation of IBAT is a probable healing goal for managing overweightness and associated metabolic problems in response to HFD [45,46].

HFD instigated a substantial elevation ($p < 0.05$) of serum TAG, TC, and VLDL-C, but in the case of LDL-C, it was reduced non-significantly compared to control diet group rats (Table 5). This study's observation of the hyperlipidemic effect of HFD is consistent with other research that has shown a favorable relationship between HFD and serum lipid levels [35–37]. The liver produces and secretes more VLDL-C particles due to HFD, which can raise TAG and TC levels [37].

HDL-C levels were shown to decrease in this study, along with an increase in VLDL-C levels [42,47]. Increased triglyceride production and hepatic steatosis result from fatty acids entering the liver more readily while eating an HFD [45]. Increased lipogenesis, decreased lipolysis, and adipocyte hypertrophy are responsible for the increased adiposity in rats fed an HFD [37].

It also shows HFD significantly reduced HDL-C ($p < 0.05$). Contrary, HFD caused a noteworthy elevation ($p < 0.05$) of ALT, but ALP, AST, and GGT increased non-significantly compared to control diet group rats (Table 5). HFD significantly increased ($p < 0.05$) ALT serum levels, but not AST or GGT. Elevated levels of ALT and ALP indicate liver injury, which may be brought on by a buildup of fat in the liver. Additionally, increased ALP levels have been linked to cholestasis, a condition marked by the buildup of bile acids and cholesterol in the liver [35]. Therefore, the rise in ALP levels seen in this study might result from cholestasis brought on by the HFD.

Interestingly, there was no discernible increase in the AST or GGT levels in the HFD group. Elevated AST and GGT levels have been connected to liver impairment and are commonly employed as markers of liver function [48]. The study's findings on the non-significant increase in AST and GGT levels might be attributed to the short period of HFD. A prolonged HFD regimen could likely outcome in a more noticeable upsurge in AST and GGT values.

3.2. Experimental phase

3.2.1. Consequence of various edible oils on weight of body and BMI of obese rats

Table 6 shows that as compared to the rats in the CC diet group, the growth in the CHF group rose considerably ($p < 0.05$). Table 6 also illustrate that the body weight gain rate diminished non-significantly in RBO, OO, SO, CLO, and SFO compared to the CHF group. These results align with earlier research that demonstrated that different oils have various impacts on body weight gain non-significantly [49].

Although several theories have been proposed, the mechanisms behind the anti-obesity actions of oils remain incompletely understood. It has been shown that the bioactive and antioxidant compounds included in oil enhance lipid metabolism and inhibit rat body weight gain. Although the present study found non-significant weight gain in the rats [50–52].

The BMI of rats was measured according to the Lee index [33,34], and the table shows that feeding with different oils except OO showed the obese rats turn to the normal range of BMI, which indicates the positive outcome of oils on obesity but it was occurred

Table 4
Consequence of diet on the increment of body weight at 5th weeks of rats.

Group	Primary weight (g)	Final weight (g)	Weight gain (g)	Weight gain (g/rat/day)	BMI (g/cm^2)
Control	62 ± 3.05	127 ± 9.68	65	1.85	0.47 (normal)
HFD	71 ± 0.66	203 ± 2.40	132**	3.77	0.78 (obese)

The data is shown as Mean ± SEM. The statistical significance of the variations in group means was assessed using the independent sample student's t-test. **$p < 0.01$ when compared initial body weight to final body weight.

Table 5
Effect of HFD on the weight of various organ weight, fat profile, and hepatic enzymes at 5th weeks.

Parameters	Control group	HFD group	P value
Liver (g)	4.99 ± 0.41	8.85 ± 0.36	**
Heart (g)	0.50 ± 0.05	0.75 ± 0.06	**
AAT (g)	0.45 ± 0.08	2.36 ± 0.19	**
EAT (g)	0.93 ± 0.09	2.08 ± 0.44	**
IBAT (g)	0.18 ± 0.06	0.70 ± 0.09	*
Liver Fat (mg)	21.47 ± 0.03	39.79 ± 0.01	**
TC (mg/dL)	55 ± 4.42	94 ± 3.67	*
TAG (mg/dL)	57 ± 3.17	80 ± 6.64	*
HDL-C (mg/dL)	59 ± 3.17	11 ± 6.64	*
LDL-C (mg/dL)	40 ± 3.46	39 ± 2.65	–
VLDL-C (mg/dL)	11 ± 0.57	21 ± 3.33	*
ALT (IU/L)	23 ± 11.26	44 ± 3.53	*
AST (IU/L)	79 ± 10.60	82 ± 7.62	–
ALP (IU/L)	79 ± 5.50	160 ± 6.50	–
GGT (IU/L)	1.67 ± 0.67	3.67 ± 0.67	–

The data is shown as Mean ± SEM. The statistical significance of the variations in group means was assessed using the independent sample student's t-test. **$p < 0.01$, and *$p < 0.05$ when contrast control diet to HFD.

Table 6
Effect of different oil diet on growth and BMI at 11th of obese rats.

Group	Primary weight (g)	Final weight (g)	Weight gain (g)	Weight gain (g/rat/day)	BMI (g/cm^2)
CC	184 ± 8.17	242 ± 14.06	58	1.65	0.68
CHF	200 ± 3.16	286 ± 10.77	86*	2.45	0.80
RBO	206 ± 7.82	273 ± 8.61	67	1.91	0.68
OO	202 ± 4.56	272 ± 8.23	70	2.00	0.69
SO	208 ± 4.57	278 ± 6.24	70	2.00	0.68
CLO	184 ± 8.01	228 ± 11.08	44	1.25	0.65
SFO	193 ± 6.10	239 ± 8.18	46	1.31	0.59

The data is shown as Mean ± SEM. The statistical significance of the variations in group means was assessed using the independent sample student's t-test. *$p < 0.05$ when comparing CC to CHF and CHF to respected oil groups. CC = Continuing Control.

non-significantly ($p > 0.05$). The BMI of the control diet cluster was discovered to be within the normal range. However, the BMI of the rats given CHF significantly increased ($p < 0.05$), indicating that these rats were obese. This study's findings align with other research that demonstrated the practical benefits of various oils on obesity [49].

3.2.2. Consequence of various oils on weight of organ, cholesterol level, and hepatic enzymes at 11th weeks of obese rats
  The weight of the liver, heart, AAT, EAT, IBAT, and liver fat were all decreased over a period of five weeks (6–11 weeks) by feeding

Table 7
Effect of different edible oils on weight of organ, cholesterol level, and hepatic enzymes at 11th weeks of obese rats.

Parameters	Treatment Phase						
	CC	CHF	RBO	OO	SO	CLO	SFO
Liver (g)	6.97 ± 0.45	10.1 ± 0.56*	9.02 ± 0.76	9.05 ± 0.89	8.86 ± 0.69	7.31 ± 0.54*	7.2 ± 0.71
Heart (g)	0.72 ± 0.08	0.82 ± 0.09	0.77 ± 0.02	0.74 ± 0.04	0.81 ± 0.05	0.67 ± 0.08	0.65 ± 0.10
AAT (g)	2.8 ± 0.73	6.95 ± 0.75*	4.99 ± 0.49	5.29 ± 0.54	5.07 ± 0.18	4.03 ± 0.86*	3.22 ± 0.58
EAT (g)	2.93 ± 0.67	6.24 ± 0.69	3.90 ± 0.41	4.04 ± 0.43	4.32 ± 0.39	2.96 ± 0.61	2.99 ± 0.06
IBAT (g)	0.26 ± 0.07	1 ± 0.07**	0.59 ± 0.04*	0.47 ± 0.04**	0.56 ± 0.06*	0.58 ± 0.06*	0.49 ± 0.09*
Liver Fat (mg)	25.19 ± 0.03	48.53 ± 0.03**	32.38 ± 0.026**	40.24 ± 0.02*	36.14 ± 0.01**	34.95 ± 0.04**	34.82 ± 0.01**
TC (mg/dL)	46 ± 4.45	138 ± 8.89**	48 ± 3.31**	78 ± 5.49*	54 ± 6.78*	49 ± 1.34**	50 ± 4.31**
TAG (mg/dL)	72 ± 1.20	99 ± 6.36**	54 ± 3.93**	58 ± 3.41*	60 ± 4.48*	30 ± 4.48**	57 ± 2.08**
HDL-C (mg/dL)	47 ± 0.578	14 ± 4.67**	39 ± 4.16**	32 ± 6.5*	33 ± 3.89*	40 ± 3.89**	34 ± 1.15*
LDL-C (mg/dL)	16 ± 1.53	40 ± 5.36*	5 ± 1.73**	20 ± 5.76	16 ± 2.33*	18 ± 5.36*	13 ± 4.48*
VLDL-C (mg/dL)	9 ± 0.33	27 ± 3.17**	10 ± 0.33**	16 ± 2.33*	11 ± 0.88**	10 ± 0.00**	10 ± 0.00**
ALT (IU/L)	54 ± 9.07	83 ± 6.17	49 ± 2.00**	58 ± 5.46*	40 ± 0.00**	63 ± 6.98	70 ± 0.00
AST (IU/L)	72 ± 1.76	88 ± 7.31	70 ± 7.02	79 ± 1.15	83 ± 9.40	77 ± 4.33	85 ± 0.58
ALP (IU/L)	62 ± 8.61	236 ± 10.50*	126 ± 7.00	161 ± 11.00	148 ± 12.00	197 ± 2.02	177 ± 9.52
GGT (IU/L)	1.67 ± 0.67	4.34 ± 0.33	1.67 ± 0.33	2.67 ± 0.77	3.33 ± 0.67	2 ± 0.59	2.67 ± 0.33

The data is shown as Mean ± SEM. The statistical significance of the variations in group means was assessed using the independent sample student's t-test. **$p < 0.01$, and *$p < 0.05$, when compared CC to CHF and CHF to RBO, OO, SO, CLO, and SFO groups.

different dietary oils to obese rats that had been given high fat levels. These reductions were observed at both significant and non-significant levels (Table 7). The different fatty acid compositions of the oils utilized in the study can account for the observed differences in organ weights and liver fat content. In the current study, the high-fat-induced obese rat's liver weight was considerably greater than that of the control group. On the other hand, weight of liver dropped after five weeks of RBO, OO, SO, CLO, and SFO feeding. The RBO, SO, CLO, and SFO groups showed the most substantial reduction in liver fat (p < 0.01), followed by OO (p < 0.05). These outcomes are reliable with the prior investigation, which demonstrated that various oils have distinct impacts on hepatic lipid metabolism [53]. The adding of omega-3 fatty acids in these oils, which have been demonstrated to have anti-obesity possessions, may be the source of the decline in body fat weight seen in the CLO and SFO groups [54]. The increased concentration of monounsaturated fatty acids (MUFA) in the SO group may cause a decrease in liver fat content [55]. Additionally, the current study revealed no apparent variations in the weight of the heart between the various oil-fed rats. A high-fat diet, however, has been linked to heart lipid buildup and malfunction [56]. Therefore, the short study duration may be partially responsible for the observed lack of noticeable changes in heart weight.

On the contrary, the study also examined the hypolipidemic effects of several oil groups, such as RBO, OO, SO, CLO, and SFO, on high-fat-induced obese rats. After consuming the various oil groups for five weeks, the results revealed significantly lower TC and TAG (p < 0.01 for RBO, CLO, SFO, and p < 0.05 for OO and SO); HDL-C (p < 0.01 for RBO, CLO, and p < 0.05 for OO, SO, and SFO); LDL-C (p < 0.01 for RBO and p < 0.05 for SO, CLO, and SFO); and VLDL-C (p < 0.01 for RBO, SO, CLO, and SFO except OO (p < 0.05) among the rats (Table 7). Unsaturated fatty acids, abundant in RBO, OO, SO, CLO, and SFO, have been shown to positively affect lipid metabolism [57]. For instance, oleic acid, which is abundant in RBO and has been found to drop LDL-C and raise HDL-C levels [58]. In both animal and human research, OO has been shown to have hypolipidemic effects because it is also high in oleic acid [59]. Linoleic acid, which has been demonstrated to reduce TC, LDL-C, and VLDL-C levels, is a good source of fat in SO [60]. On the other hand, CLO is abundant in EPA and DHA, which have been revealed to lower plasma TAG levels. Last but not least, SFO has a significant amount of unsaturated fatty acids, especially linoleic acid, which has been shown to lower points of TC and LDL-C [61].

The effects of the various oil groups on TC, TAG, LDL-C, HDL-C, and VLDL-C levels were used to evaluate the hypolipidemic effect of the groups in the current investigation. The outcomes presented that all of the oil groups considerably lowered TC levels, with the exception of OO. RBO, SO, and CLO considerably lowered TAG levels whereas SFO markedly increased HDL-C levels. Furthermore, RBO, CLO, and SFO dramatically decreased (p < 0.01) LDL-C levels whereas SO considerably decreased (p < 0.01) VLDL-C levels. The different oil groups' fatty acid composition might be the cause of the observed hypolipidemic effect. The unsaturated fatty acids in the oils may reduce hepatic TC formation, accelerate bile acid synthesis, enhance LDL receptor function, and promote fatty acid oxidation to lower blood cholesterol levels [62]. The presence of bioactive substances like tocopherols and phenolic compounds in oils may be a factor in the oils' hypolipidemic impact [61].

On high-fat-induced obese rats, ALT, AST, ALP, and GGT were reduced over the course of five weeks at varying significant and non-significant levels by feeding RBO, OO, SO, CLO, and SFO. ALT, AST, ALP, and GGT levels significantly decreased in rats fed RBO, SO, and CLO, but non-significantly decreased in rats given OO and SFO. It's possible that the oils protect the liver because of the reported drop in liver enzymes.

Oleic acid and linoleic acid, abundant in RBO and SO, have been demonstrated to diminish liver fat buildup and enhance liver function [63]. Due to RBO's capacity to increase fatty acid oxidation and decrease hepatic lipid production, this may have a hypolipidemic impact [64]. While SO's capacity to boost LDL receptor activity, improve fatty acid oxidation, and encourage bile acid synthesis may be responsible for its hypolipidemic effect [65]. EPA and DHA, in particular, are abundant in CLO and have been shown to minimize liver lipid buildup and enhance liver function [66]. The potential of CLO to decrease hepatic lipogenesis, increase fatty acid oxidation, and decrease inflammation may be responsible for its hypolipidemic effects [67,68]. The findings of this research are in line with earlier research that revealed the hypolipidemic impact of various oils on liver function [69].

3.3. Hypolipidemic effect of different edible oils diet in obese rats over HFD to improve lipid profile

The effectiveness of various edible oils over HFD in enhancing cholesterol levels is shown in Table 8. The CHF had significantly higher TC, TAG, and LDL-C. Contrary to the rats fed the CHF group, those fed the RBO, OO, SO, CLO, and SFO groups had significantly lower points of TC, TAG, and LDL-C and higher levels of HDL-C. These findings suggest that consuming these oils may protect against the onset of dyslipidemia and may even enhance the lipid profile in obese rats. The profile of lipids may be impacted by the MUFA and PUFA present in the regarded oils. Edible oils have been recommended as a dietary approach to reduce the risk of metabolic disorders

Table 8
Efficiency of different edible oils over HFD (11th weeks) on improving lipid profile.

Treatment groups	TC (%)	TAG (%)	LDL-C (%)	HDL-C (%)
CHF	46.80↑	23.75↑	2.56↑	27.27↑
RBO	48.94↓	32.50↓	87.18↓	254.54↑
OO	17.02↓	27.50↓	48.72↓	190.90↑
SO	42.55↓	25.00↓	58.97↓	200.00↑
CLO	47.87↓	62.50↓	53.85↓	263.64↑
SFO	46.81↓	28.75↓	66.67↓	209.00↑

↑ indicates increase.
↓ indicates decrease.

[70].

4. Conclusion

In a nutshell, the current research suggests that feeding different edible oils to obese Wistar rats was beneficial in reducing hyperlipidemic status, reducing fat reserves from various fat depots, and improving blood serum fat profiles (improved HDL-C and lowered TC, TAG, LDL-C, and VLDL-C). All the oils showed positive results for the reduction of hyperlipidemia. Administration of different edible oils to obese rats also reduced all the hepatic enzymes compared to the CHF group, and the oils opted to reduce the risk of cardiovascular complications. No oils were found capable of producing a significant change in AST level, regardless of whether RBO showed the effect of significantly reducing all the other hepatic enzymes. Other oils could reduce ALT levels significantly, whereas SFO could not make any significant changes to hepatic enzymes. The various edible oils had hypolipidemic effects that improved liver function, controlled lipid profiles, and reduced cholesterol levels in people. Further research is needed to determine how this oil affects human lipid metabolism and liver function.

Ethics statement

The requirements for animal handling and usage were followed in keeping all the rats in the cages. The Department of Food Technology and Nutritional Science at Mawlana Bhashani Science and Technology University's "Ethical Review Committee (ERC)" assessed and granted permission for all of the study's investigations into protecting animal issues. The panel's ethical authorization number is "MBSTU/FTNS/42/2023/10."

Data availability statement

Data will be made available on request.

CRediT authorship contribution statement

Md Abdul Alim: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation. **Tarana Jannat Mumu:** Methodology, Investigation. **Ummay Salma Tamanna:** Methodology, Investigation. **Md Moin Khan:** Methodology, Investigation. **Md Imran Miah:** Methodology, Investigation. **Md Shahikul Islam:** Methodology, Investigation. **Zannat Ara Jesmin:** Methodology, Investigation. **Tayeba Khan:** Writing – review & editing, Visualization. **Md Rakibul Hasan:** Writing – review & editing, Visualization. **Md Jahangir Alam:** Writing – review & editing, Visualization, Methodology, Formal analysis. **Khan Md Murtaja Reza Linkon:** Writing – review & editing, Visualization, Validation, Resources. **Md Nannur Rahman:** Writing – review & editing, Visualization, Validation, Resources. **Rokeya Begum:** Writing – review & editing, Visualization. **Utpal Kumar Prodhan:** Writing – review & editing, Visualization, Validation, Supervision, Project administration, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] L.A. Barness, J.M. Opitz, E. Gilbert-Barness, Obesity: genetic, molecular, and environmental aspects, Am. J. Med. Genet. 143 (24) (2007) 3016–3034, https://doi.org/10.1002/ajmg.a.32035.

[2] R.T. Hurt, C. Kulisek, L.A. Buchanan, S.A. McClave, The obesity epidemic: challenges, health initiatives, and implications for gastroenterologists, Gastroenterol. Hepatol. 6 (12) (2010) 780–792.

[3] L. Akil, H.A. Ahmad, Relationships between obesity and cardiovascular diseases in four southern states and Colorado, J. Health Care Poor Underserved 22 (4 Suppl) (2011) 61–72, https://doi.org/10.1353/hpu.2011.0166.

[4] N. Gray, G. Picone, F. Sloan, A. Yashkin, Relation between BMI and diabetes mellitus and its complications among US older adults, South. Med. J. 108 (1) (2015) 29–36, https://doi.org/10.14423/SMJ.0000000000000214.

[5] M. Abdelaal, C.W. le Roux, N.G. Docherty, Morbidity and mortality associated with obesity, Ann. Transl. Med. 5 (7) (2017) 161, https://doi.org/10.21037/atm.2017.03.107.

[6] S.S. Doerstling, C.H. O'Flanagan, S.D. Hursting, Obesity and Cancer Metabolism: a Perspective on interacting tumor-intrinsic and extrinsic factors, Front. Oncol. 7 (2017) 216, https://doi.org/10.3389/fonc.2017.00216.

[7] A. Berghöfer, T. Pischon, T. Reinhold, C.M. Apovian, A.M. Sharma, S.N. Willich, Obesity prevalence from a European perspective: a systematic review, BMC Publ. Health 8 (2008) 1–10. https://doi:10.1186/1471-2458-8-20.

[8] Prospective Studies Collaboration, G. Whitlock, S. Lewington, P. Sherliker, R. Clarke, J. Emberson, J. Halsey, N. Qizilbash, R. Collins, R. Peto, Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies, Lancet (London, England) 373 (9669) (2009) 1083–1096, https://doi.org/10.1016/S0140-6736(09)60318-4.

[9] Adult Rates of Overweight and Obesity Rise in Bangladesh. Bangladesh Obesity Release. International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR, B), 2020. Retrieved from, https://www.icddrb.org/dmdocuments/Bangladesh%20. obesity%20release.pdf.

[10] S. Banik, M. Rahman, Prevalence of overweight and obesity in Bangladesh: a systematic review of the literature, Curr. Obes. Rep. 7 (2018) 247–253, https://doi.org/10.1007/s13679-018-0323-x.

[11] G. Whitlock, S. Lewington, P. Sherliker, R. Clarke, J. Emberson, J. Halsey, N. Qizilbash, R. Collins, R. Peto, Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies, Lancet (London, England) 373 (9669) (2009) 1083–1096, https://doi.org/10.1016/S0140-6736(09)60318-4.

[12] J. Loscalzo, A. Fauci, E. Braunwald, K.L. Dennis, S.L. Hauser, D.L. Longo, Chapter 17, Fever versus Hyperthermia. Harrison's Principles of Internal Medicine, McGraw-Hill Medical, 2008. ISBN 0-07-146633-9.

[13] L. Akil, H.A. Ahmad, Relationships between obesity and cardiovascular diseases in four southern states and Colorado, J. Health Care Poor Underserved 22 (4 Suppl) (2011) 61–72, https://doi.org/10.1353/hpu.2011.0166.

[14] G. Marchesini, S. Moscatiello, S. Di Domizio, G. Forlani, Obesity-associated liver disease, J. Clin. Endocrinol. Metabol. 93 (11 Suppl 1) (2008) S74–S80, https://doi.org/10.1210/jc.2008-1399.

[15] M. Garaulet, J.J. Hernandez-Morante, J. Lujan, F.J. Tebar, S. Zamora, Relationship between fat cell size and number and fatty acid composition in adipose tissue from different fat depots in overweight/obese humans, Int. J. Obes. 30 (6) (2006) 899–905, https://doi.org/10.1038/sj.ijo.0803219, 2005.

[16] A.P. Simopoulos, An increase in the omega-6/omega-3 fatty acid ratio increases the risk for obesity, Nutrients 8 (3) (2016) 128, https://doi.org/10.3390/nu8030128.

[17] V.R. Johnson, T.B. Washington, S. Chhabria, E.H. Wang, K. Czepiel, K.J.C. Reyes, F.C. Stanford, Food as medicine for obesity treatment and management, Clin. Therapeut. 44 (5) (2022) 671–681, https://doi.org/10.1016/j.clinthera.2022.05.001.

[18] A. Astrup, Dietary management of obesity, JPEN - J. Parenter. Enter. Nutr. 32 (5) (2008) 575–577, https://doi.org/10.1177/0148607108321707.

[19] B.A. Hannon, S.V. Thompson, R. An, M. Teran-Garcia, Clinical outcomes of dietary replacement of saturated fatty acids with unsaturated fat sources in adults with overweight and obesity: a Systematic Review and Meta-Analysis of Randomized Control Trials, Ann. Nutr. Metabol. 71 (1–2) (2017) 107–117, https://doi.org/10.1159/000477216.

[20] H.M. Parker, N.A. Johnson, C.A. Burdon, J.S. Cohn, H.T. O'Connor, J. George, Omega-3 supplementation and non-alcoholic fatty liver disease: a systematic review and meta-analysis, J. Hepatol. 56 (4) (2012) 944–951, https://doi.org/10.1016/j.jhep.2011.08.018.

[21] J.B. Dixon, P.S. Bhathal, N.R. Hughes, P.E. O'Brien, Nonalcoholic fatty liver disease: improvement in liver histological analysis with weight loss, Hepatology (Baltimore, Md 39 (6) (2004) 1647–1654, https://doi.org/10.1002/hep.20251.

[22] J.B. Dixon, P.S. Bhathal, P.E. O'Brien, Weight loss and non-alcoholic fatty liver disease: falls in gamma-glutamyl transferase concentrations are associated with histologic improvement, Obes. Surg. 16 (10) (2006) 1278–1286, https://doi.org/10.1381/096089206778663805.

[23] A. St George, A. Bauman, A. Johnston, G. Farrell, T. Chey, J. George, Effect of a lifestyle intervention in patients with abnormal liver enzymes and metabolic risk factors, J. Gastroenterol. Hepatol. 24 (3) (2009) 399–407, https://doi.org/10.1111/j.1440-1746.2008.05694.x.

[24] S.G. Mattar, L.M. Velcu, M. Rabinovitz, A.J. Demetris, A.M. Krasinskas, E. Barinas-Mitchell, G.M. Eid, R. Ramanathan, D.S. Taylor, P.R. Schauer, Surgically-induced weight loss significantly improves nonalcoholic fatty liver disease and the metabolic syndrome, Ann. Surg. 242 (4) (2005) 610–620, https://doi.org/10.1097/01.sla.0000179652.07502.3f.

[25] K.M. Gadde, C.K. Martin, H.R. Berthoud, S.B. Heymsfield, Obesity: pathophysiology and management, J. Am. Coll. Cardiol. 71 (1) (2018) 69–84, https://doi.org/10.1016/j.jacc.2017.11.011.

[26] L. Boateng, R. Ansong, W.B. Owusu, M. Steiner-Asiedu, Coconut oil and palm oil's role in nutrition, health and national development: a review, Ghana Med. J. 50 (3) (2016) 189–196.

[27] C. Sayon-Orea, S. Carlos, M.A. Martínez-Gonzalez, Does cooking with vegetable oils increase the risk of chronic diseases?: a systematic review, Br. J. Nutr. 113 (Suppl 2) (2015), https://doi.org/10.1017/S0007114514002931. S36–S48.

[28] M.O.F. Mridha, P. Noor, R. Khaton, D. Islam, M. Hossain, Effect of *Spirulina platensis* on lipid profile of long evans rats, Bangladesh J. Sci. Ind. Res. 45 (3) (2010) 249–254, https://doi.org/10.3329/bjsir.v45i3.6533.

[29] A. Barison, C.W. da Silva, F.R. Campos, F. Simonelli, C.A. Lenz, A.G. Ferreira, A simple methodology for the determination of fatty acid composition in edible oils through 1H NMR spectroscopy, Magn. Reson. Chem.: MRC 48 (8) (2010) 642–650, https://doi.org/10.1002/mrc.2629.

[30] L. Casteilla, L. Pénicaud, B. Cousin, D. Calise, Choosing an adipose tissue depot for sampling: factors in selection and depot specificity, Methods Mol. Biol. 456 (2008) 23–38, https://doi.org/10.1007/978-1-59745-245-8_2.

[31] J.A. Domínguez-Avila, E. Alvarez-Parrilla, J.A. López-Díaz, I.E. Maldonado-Mendoza, M. delC. Gómez-García, L.A. de la Rosa, The pecan nut (Carya illinoinensis) and its oil and polyphenolic fractions differentially modulate lipid metabolism and the antioxidant enzyme activities in rats fed high-fat diets, Food Chem. 168 (2015) 529–537, https://doi.org/10.1016/j.foodchem.2014.07.092.

[32] N. Rifai, G.R. Warnick, Measurement of lipids, lipoproteins, and apolipoproteins, in: C.A. Burtis, E.R. Ashwood, D.E. Bruns (Eds.), Tietz Textbook of Clinical Chemistry and Molecular Diagnosis, fourth ed., Elsevier Saunders, St. Louis, Missouri, 2006, pp. 938–952.

[33] E.L. Novelli, Y.S. Diniz, C.M. Galhardi, G.M. Ebaid, H.G. Rodrigues, F. Mani, A.A. Fernandes, A.C. Cicogna, J.L. Novelli Filho, Anthropometrical parameters and markers of obesity in rats, Lab. Anim 41 (1) (2007) 111–119, https://doi.org/10.1258/002367707779399518.

[34] Y. Prashar, N.J. Patel, Methanolic fruit extract of Myrica nagi protects the hypothalamus and attenuates inflammation associated with gold thioglucose- and high-fat diet-induced obesity via various adipokines, J. Ayurveda Integr. Med. 14 (2) (2023) 100582, https://doi.org/10.1016/j.jaim.2022.100582.

[35] J. Makkonen, K.H. Pietiläinen, A. Rissanen, J. Kaprio, H. Yki-Järvinen, Genetic factors contribute to variation in serum alanine aminotransferase activity independent of obesity and alcohol: a study in monozygotic and dizygotic twins, J. Hepatol. 50 (5) (2009) 1035–1042, https://doi.org/10.1016/j.jhep.2008.12.025.

[36] H. Tilg, A.R. Moschen, Adipocytokines: mediators linking adipose tissue, inflammation and immunity, Nat. Rev. Immunol. 6 (10) (2006) 772–783, https://doi.org/10.1038/nri1937.

[37] H. Poudyal, S.K. Panchal, L.C. Ward, J. Waanders, L. Brown, Chronic high-carbohydrate, high-fat feeding in rats induces reversible metabolic, cardiovascular, and liver changes, Am. J. Physiol. Endocrinol. Metabol. 302 (12) (2012) E1472–E1482, https://doi.org/10.1152/ajpendo.00102.2012.

[38] M.H. Oosterveer, A. Grefhorst, T.H. van Dijk, R. Havinga, B. Staels, F. Kuipers, A.K. Groen, D.J. Reijngoud, Fenofibrate simultaneously induces hepatic fatty acid oxidation, synthesis, and elongation in mice, J. Biol. Chem. 284 (49) (2009) 34036–34044, https://doi.org/10.1074/jbc.M109.051052.

[39] A. Dittmann, N.J. Kennedy, N.L. Soltero, N. Morshed, M.D. Mana, Ö.H. Yilmaz, R.J. Davis, F.M. White, High-fat diet in a mouse insulin-resistant model induces widespread rewiring of the phosphotyrosine signaling network, Mol. Syst. Biol. 15 (8) (2019) e8849, https://doi.org/10.15252/msb.20198849.

[40] M. Bastías-Pérez, D. Serra, L. Herrero, Dietary options for rodents in the study of obesity, Nutrients 12 (11) (2020) 3234, https://doi.org/10.3390/nu12113234.

[41] E. Rodríguez-Correa, I. González-Pérez, P.I. Clavel-Pérez, Y. Contreras-Vargas, K. Carvajal, Biochemical and nutritional overview of diet-induced metabolic syndrome models in rats: what is the best choice? Nutr. Diabetes 10 (1) (2020) 24, https://doi.org/10.1038/s41387-020-0127-4.

[42] P. Dandona, A. Aljada, A. Chaudhuri, P. Mohanty, R. Garg, Metabolic syndrome: a comprehensive perspective based on interactions between obesity, diabetes, and inflammation, Circulation 111 (11) (2005) 1448–1454, https://doi.org/10.1161/01.CIR.0000158483.13093.9D.

[43] E.E. Kershaw, J.S. Flier, Adipose tissue as an endocrine organ, J. Clin. Endocrinol. Metabol. 89 (6) (2004) 2548–2556, https://doi.org/10.1210/jc.2004-0395.

[44] U.J. Jung, M.S. Choi, Obesity and its metabolic complications: the role of adipokines and the relationship between obesity, inflammation, insulin resistance, dyslipidemia and nonalcoholic fatty liver disease, Int. J. Mol. Sci. 15 (4) (2014) 6184–6223, https://doi.org/10.3390/ijms15046184.

[45] H. Cao, K. Gerhold, J.R. Mayers, M.M. Wiest, S.M. Watkins, G.S. Hotamisligil, Identification of a lipokine, a lipid hormone linking adipose tissue to systemic metabolism, Cell 134 (6) (2008) 933–944, https://doi.org/10.1016/j.cell.2008.07.048.

[46] X. Liu, Z. Zhang, Y. Song, H. Xie, M. Dong, An update on brown adipose tissue and obesity intervention: function, regulation and therapeutic implications, Front. Endocrinol. 13 (2023) 1065263, https://doi.org/10.3389/fendo.2022.1065263.

[47] J.O. Hill, K.L. Olson, Obesity and hyperlipidemia, in: The Metabolic Syndrome, Humana Press, 2008, pp. 69–84, https://doi.org/10.1007/978-1-59745-361-5_5.

[48] J.H. van Beek, M.H. de Moor, E.J. de Geus, G.H. Lubke, J.M. Vink, G. Willemsen, D.I. Boomsma, The genetic architecture of liver enzyme levels: GGT, ALT and AST, Behav. Genet. 43 (4) (2013) 329–339, https://doi.org/10.1007/s10519-013-9593-y.

[49] Y. Chen, Z. Li, H. Wang, Z. Wang, Y. Li, H. Liu, Association of obesity-related genetic variants with metabolic syndrome in 8162 subjects from rural China, J. Diabetes Complicat. 31 (4) (2017) 657–661, https://doi.org/10.1016/j.jdiacomp.2016.12.010.

[50] D.F. Coelho, L.O. Pereira-Lancha, D.S. Chaves, D. Diwan, R. Ferraz, P.L. Campos-Ferraz, J.R. Poortmans, A.H. Lancha Junior, Effect of high-fat diets on body composition, lipid metabolism and insulin sensitivity, and the role of exercise on these parameters, Brazil. J. Med. Biol. Res. = Rev. Brasil. Pesqui. Med. Biol. 44 (10) (2011) 966–972, https://doi.org/10.1590/s0100-879x2011007500107.

[51] J. Choo, H. Ueshima, Y. Jang, K. Sutton-Tyrrell, A. El-Saed, T. Kadowaki, T. Takamiya, T. Okamura, Y. Ueno, Y. Nakamura, A. Sekikawa, J.D. Curb, L.H. Kuller, C. Shin, Difference in carotid intima-media thickness between Korean and Japanese men, Ann. Epidemiol. 18 (4) (2008) 310–315, https://doi.org/10.1016/j.annepidem.2007.08.002.

[52] C.L. Kien, J.Y. Bunn, C.L. Tompkins, J.A. Dumas, K.I. Crain, D.B. Ebenstein, T.R. Koves, D.M. Muoio, Substituting dietary monounsaturated fat for saturated fat is associated with increased daily physical activity and resting energy expenditure and with changes in mood, Am. J. Clin. Nutr. 97 (4) (2013) 689–697, https://doi.org/10.3945/ajcn.112.051730.

[53] J. Wang, S. Yan, H. Xiao, H. Zhou, S. Liu, Y. Zeng, B. Liu, R. Li, Z. Yuan, J. Wu, J. Yi, Y.B.S. Razack, L. Wen, Anti-obesity effect of a traditional Chinese dietary habit-blending lard with vegetable oil while cooking, Sci. Rep. 7 (1) (2017) 14689, https://doi.org/10.1038/s41598-017-14704-2.

[54] A. Safaeiyan, A. Ostadrahimi, M. Sanayei, Y. Rasmi, P. Taraghijou, Effect of omega-3 fatty acids on appetite, energy and macronutrient intake and body weight in obese adults: a randomized clinical trial, Prog. Nutr. 20 (2) (2018) 203–209, https://doi.org/10.23751/pn.v20i2-S.5481.

[55] T. Sarabhai, S. Kahl, J. Szendroedi, D.F. Markgraf, O.P. Zaharia, C. Barosa, C. Herder, F. Wickrath, P. Bobrov, J.H. Hwang, J.G. Jones, M. Roden, Monounsaturated fat rapidly induces hepatic gluconeogenesis and whole-body insulin resistance, JCI Insight 5 (10) (2020) e134520, https://doi.org/10.1172/jci.insight.134520.

[56] J.A. Wali, N. Jarzebska, D. Raubenheimer, S.J. Simpson, R.N. Rodionov, J.F. O'Sullivan, Cardio-Metabolic effects of high-fat diets and their underlying mechanisms-A Narrative Review, Nutrients 12 (5) (2020) 1505, https://doi.org/10.3390/nu12051505.

[57] L. Schwingshackl, M. Christoph, G. Hoffmann, Effects of olive oil on markers of inflammation and endothelial function-A Systematic Review and Meta-Analysis, Nutrients 7 (9) (2015) 7651–7675, https://doi.org/10.3390/nu7095356.

[58] K.M.C. Nogoy, H.J. Kim, Y. Lee, Y. Zhang, J. Yu, D.H. Lee, S.H. Choi, High dietary oleic acid in olive oil-supplemented diet enhanced omega-3 fatty acid in blood plasma of rats, Food Sci. Nutr. 8 (7) (2020) 3617–3625, https://doi.org/10.1002/fsn3.1644.

[59] M.I. Covas, K. Nyyssönen, H.E. Poulsen, J. Kaikkonen, H.J. Zunft, H. Kiesewetter, A. Gaddi, R. de la Torre, J. Mursu, H. Bäumler, S. Nascetti, J.T. Salonen, M. Fitó, J. Virtanen, J. Marrugat, EUROLIVE Study Group, The effect of polyphenols in olive oil on heart disease risk factors: a randomized trial, Ann. Intern. Med. 145 (5) (2006) 333–341, https://doi.org/10.7326/0003-4819-145-5-200609050-00006.

[60] N.A. Azemi, A.K. Azemi, L. Abu-Bakar, V. Sevakumaran, T.S.T. Muhammad, N. Ismail, Effect of linoleic acid on cholesterol levels in a high-fat diet-induced hypercholesterolemia rat model, Metabolites 13 (1) (2022) 53, https://doi.org/10.3390/metabo13010053.

[61] J.K. Bird, P.C. Calder, M. Eggersdorfer, The role of *n*-3 long chain polyunsaturated fatty acids in cardiovascular disease prevention, and interactions with statins, Nutrients 10 (6) (2018) 775, https://doi.org/10.3390/nu10060775.

[62] P. Silva Figueiredo, A. Carla Inada, G. Marcelino, C. Maiara Lopes Cardozo, K. de Cássia Freitas, R. de Cássia Avellaneda Guimarães, A. Pereira de Castro, V. Aragão do Nascimento, P. Aiko Hiane, Fatty acids consumption: the role metabolic aspects involved in obesity and its associated disorders, Nutrients 9 (10) (2017) 1158, https://doi.org/10.3390/nu9101158.

[63] E. Juárez-Hernández, N.C. Chávez-Tapia, M. Uribe, V.J. Barbero-Becerra, Role of bioactive fatty acids in nonalcoholic fatty liver disease, Nutr. J. 15 (1) (2016) 72, https://doi.org/10.1186/s12937-016-0191-8.

[64] N.K. Maurya, P. Arya, N.S. Sengar, Hypolipidemic effect of rice bran oil on chronic renal failure (undergoing hemodialysis) patients, Plant Arch. 20 (2020) 3285–3289.

[65] B.M. Rasmussen, B. Vessby, M. Uusitupa, L. Berglund, E. Pedersen, G. Riccardi, A.A. Rivellese, L. Tapsell, K. Hermansen, KANWU Study Group, Effects of dietary saturated, monounsaturated, and n-3 fatty acids on blood pressure in healthy subjects, Am. J. Clin. Nutr. 83 (2) (2006) 221–226, https://doi.org/10.1093/ajcn/83.2.221.

[66] M.Y. Wei, T.A. Jacobson, Effects of eicosapentaenoic acid versus docosahexaenoic acid on serum lipids: a systematic review and meta-analysis, Curr. Atherosclerosis Rep. 13 (6) (2011) 474–483, https://doi.org/10.1007/s11883-011-0210-3.

[67] V. Saraswathi, J.D. Morrow, A.H. Hasty, Dietary fish oil exerts hypolipidemic effects in lean and insulin sensitizing effects in obese LDLR-/- mice, J. Nutr. 139 (12) (2009) 2380–2386, https://doi.org/10.3945/jn.109.111567.

[68] G. Murali, G.L. Milne, C.D. Webb, A.B. Stewart, R.P. McMillan, B.C. Lyle, M.W. Hulver, V. Saraswathi, Fish oil and indomethacin in combination potently reduce dyslipidemia and hepatic steatosis in LDLR (-/-) mice, J. Lipid Res. 53 (10) (2012) 2186–2197, https://doi.org/10.1194/jlr.M029843.

[69] M.H. Lai, Y.T. Chen, Y.Y. Chen, J.H. Chang, H.H. Cheng, Effects of rice bran oil on the blood lipids profiles and insulin resistance in type 2 diabetes patients, J. Clin. Biochem. Nutr. 51 (1) (2012) 15–18, https://doi.org/10.3164/jcbn.11-87.

[70] C.deO. Rosa, C.A. Dos Santos, J.I. Leite, A.P. Caldas, J. Bressan, Impact of nutrients and food components on dyslipidemias: what is the evidence? Adv. Nutr. (Bethesda, Md 6 (6) (2015) 703–711, https://doi.org/10.3945/an.115.009480.