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NUTRITIONAL ASSESSMENT AND LIPID PROFILE IN RATS FED A DIET SUPPLEMENTED WITH SUNFLOWER OIL AND COCONUT OIL

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Background. Coconut oil is traditionally recognized for its potential health benefits, particularly in metabolic regulation. This study aimed to evaluate the effects of a high-fat diet supplemented with coconut oil (CO) compared with sunflower oil (SO) on body weight, food intake, and plasma biochemical parameters in Wistar rats.

Materials and Methods. Fifteen male Wistar rats (≈80 g) were divided into three groups: D1 (16% casein + 8% SO), D2 (16% casein + 32% SO), and D3 (16% casein + 28% SO + 4% CO). After two months, plasma glucose, total protein, albumin, triglycerides, total cholesterol, HDL, and LDL were analyzed using enzymatic assay kits.

Results. Rats fed the coconut oil-supplemented diet (D3) showed significantly lower blood glucose, triglyceride, and total cholesterol levels, along with a notable increase in HDL cholesterol, compared with the control groups. Moreover, body weight gain and food intake were markedly reduced in the coconut oil group.

Conclusion. These findings demonstrate that coconut oil supplementation exerts beneficial effects on lipid metabolism and weight regulation, suggesting its potential role as a functional dietary fat for managing obesity and metabolic disorders.

Keywords: obesity, coconut oil, lipid metabolism, vegetable oils, sunflower oil



INTRODUCTION

The increasing prevalence of obesity has become a major public health concern worldwide. Overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health. According to the World Health Organization (WHO), a body mass index (BMI) above 25 is classified as overweight, whereas a BMI of 30 or higher is considered obese (Furukawa *et al.*, 2004; Wei *et al.*, 2009).

In 2022, the WHO reported that 2.5 billion adults globally were overweight, including 890 million who were obese (BMI ≥ 30 kg/m²). Approximately 16% of adults aged 18 years and older were obese, while over 390 million children and adolescents aged 5–19 years were overweight. In 2024, an estimated 35 million children under five years old were classified as overweight (WHO, 2025).

Excessive fat accumulation results from multiple factors, including high-calorie intake (Pérez-Escamilla *et al.*, 2012), sedentary lifestyles, reduced physical activity, hormonal imbalances (Coutinho *et al.*, 2013), and various environmental, genetic, economic, and cultural influences (Winter *et al.*, 2013; Drewnowski, 2009). Obesity is closely linked to several chronic diseases, such as hypertension, cardiovascular disease, metabolic syndrome, and dyslipidemia. Other complications include hepatic steatosis, cerebrovascular disease, respiratory dysfunction (e.g., asthma), osteoarthritis (Woolf & Pfleger, 2003; Song *et al.*, 2004), gastrointestinal disorders, gout, proteinuria, elevated hemoglobin levels, and potential immune impairment. Furthermore, obesity has been associated with reproductive disorders, an increased risk of certain cancers, and psychosocial issues (Guerra *et al.*, 2004; Hampel *et al.*, 2005; Ehrmann, 2005). The severity of these complications underscores the urgent need for effective obesity prevention and treatment strategies.

Current preventive approaches primarily emphasize dietary and lifestyle modifications, including calorie restriction and regular physical activity (WHO, 2007). Natural products have gained increasing attention for their potential anti-obesity effects (Santos *et al.*, 2010). Many bioactive compounds from natural sources can target multiple metabolic pathways, providing potential advantages over synthetic drugs (Rayalam *et al.*, 2008; Liu, 2003). The anti-obesity mechanisms of these compounds include modulation of lipid absorption, regulation of energy balance, enhancement of lipolysis, and inhibition of lipogenesis, adipogenesis, and preadipocyte differentiation (Rayalam *et al.*, 2008).

Nutritional guidelines generally recommend reducing the intake of saturated fats – commonly found in meat and dairy products – and increasing the consumption of polyunsaturated fatty acids (PUFAs) from vegetable oils (U.S. Department of Agriculture, 2024; Kritchevsky *et al.*, 1998). However, some evidence suggests that certain saturated fats, particularly those derived from coconut oil and rich in medium-chain triglycerides (MCTs), may provide beneficial effects against the development and progression of metabolic syndrome (Xue *et al.*, 2009; Nagao & Yanagita, 2010).

Virgin coconut oil (VCO) is a functional food of growing scientific interest, traditionally used as both a medicinal remedy and dietary supplement in South India and other tropical regions. VCO is a natural, unrefined oil extracted from fresh coconut kernels, notable for its high content of medium-chain fatty acids and polyphenolic antioxidants. Studies have shown that VCO intake reduces lipogenesis and stimulates fatty acid catabolism in rats (Illam *et al.*, 2017). Additionally, it exerts chemoprotective, anti-inflammatory, antipyretic and thermoregulatory activities (Vysakh *et al.*, 2014; Famurewa *et al.*, 2017). Lauric acid, which accounts for approximately 52% of VCO, enhances oxidative metabolism and helps reduce lipid accumulation (Lekshmi Sheela *et al.*, 2016).

Accumulating evidence has challenged long-standing misconceptions regarding VCO's high saturated fat content. Recent studies have demonstrated its potential functional properties, including hypocholesterolemic, anti-obesity, anti-hepatosteatotic, antioxidant, anti-inflammatory, antimicrobial (including HIV-inhibitory), and cardioprotective effects (Gopalakrishna *et al.*, 2010; Oyi *et al.*, 2010). VCO may also improve glycemic control by regulating both insulin secretion and sensitivity (Marten *et al.*, 2006) and has been investigated for its potential roles in cancer prevention and as a protective agent during chemotherapy (Boemeke *et al.*, 2015). Moreover, its high vitamin E content supports skin and hair health (Nevin & Rajamohan, 2010).

Given this background, the present study was designed to provide new insights into the metabolic effects of virgin coconut oil (VCO) when incorporated as a partial replacement for sunflower oil in a high-fat diet. Although the anti-obesity and hypolipidemic effects of VCO have been previously documented, most studies have focused on its use as a pure supplement or in isolation from other dietary lipids. In contrast, our experimental design simulates a more realistic nutritional context by evaluating the impact of moderate inclusion of VCO alongside sunflower oil within a high-fat dietary regimen.

Furthermore, our investigation extends beyond conventional lipid analysis by simultaneously examining biochemical parameters, offering a more comprehensive understanding of the systemic effects of dietary VCO. Through this integrative approach, the study aims to determine whether partial substitution of sunflower oil with coconut oil can improve lipid metabolism, reduce obesity-related biomarkers, and promote better metabolic balance in Wistar albino rats.

MATERIALS AND METHODS

Vegetable oils and chemical reagents. In this study, commercial sunflower oil (Fleurial Plus, Cevital, Algeria) was employed. Virgin coconut oil (VCO) (*Pure Natural Sri Lankan*, Hemani International KEPZ, Karachi, Pakistan) was used, commercially available in Algeria. Virgin coconut oil was obtained from fresh, mature, organically grown coconuts by the cold-press extraction method. Hemani coconut oil appears white in its solid form and clear in its liquid form. All other chemicals and reagents used in the present study were purchased from Sigma-Aldrich (USA).

Animals and diets. The experimental protocol was approved by the Local Ethics Committee for Animal Experiments in Tlemcen, Algeria; tests on Wistar rats were approved by the Ethical Committee of the University of Tlemcen, Algeria, No 165–2019.

Fifteen 4-week-old male Wistar albinos rats (average body weight: 80 ± 5 g; Pasteur Institute, Algiers) were housed in stainless-steel cages under controlled environmental conditions (22 ± 2 °C, 12-h light/dark cycle, $55 \pm 5\%$ relative humidity) with ad libitum access to food and water.

The animals were randomly assigned into three groups ($n = 5$ per group) and a specific diet was administered to each group:

Group 1 (Control): Diet containing 4% sunflower oil (SO)

Group 2: Diet containing 32% SO

Group 3: Diet containing 28% SO + 4% virgin coconut oil (CO)

The lipid percentages in the diets were selected based on established high-fat diet models for rodents. All diets were formulated to be isocaloric to isolate the effect of fat type on metabolic outcomes, where lipid content typically ranges from 8% (normal-

fat control) to 30–35% (high-fat diet) of the total feed weight (Hariri & Thibault, 2010; Buettner *et al.*, 2007). Body weight gain was registered daily over the two-month experimental period. All diets were prepared in the laboratory.

The ingredient composition of the diets is presented in **Table 1**, and their fatty acid profiles are shown in **Table 2**. The fatty acid and ingredient composition of the oils were based on certified data provided by the supplier, as both VCO and SO were obtained from commercial food-grade sources.

Table 1. Diet ingredient composition

Constituents (g/100g diet)	Diet 1 4% SO	Diet 2 32% SO	Diet 3 4% CO + 28% SO
Casein	16	16	16
Methionine	0.3	0.3	0.3
Starch	60.35	32.35	32.35
Sucrose	05	05	05
Cellulose	05	05	05
Mineral mix	07.35	07.35	7.35
Vitamin mix	02	02	02
Oil	04	32	32
Total	100	100	100
Energetics values (Kcal/100g)	371.88	517.48	517.48

Table 2. Fatty acid composition of the diets

Fatty acids %	Diet 1 4% SO	Diet 2 32% SO	Diet 3 4% CO + 28% SO
Myristic acid	0	0	0.03
Palmitic acid	0.62	4.96	4.66
Stearic acid	0.2	1.6	1.64
Oleic acid	0.51	4.09	3.89
Linolenic acid	0.07	0.57	0.52
Linoleic acid	2.52	20.1	20.66
Arachdnic acid	0.07	0.60	0.05

The vitamin and mineral mixtures used in all experimental diets were prepared according to the AIN-93G formulation (Reeves *et al.*, 1993). Each 100 g of diet contained 7.35 g of mineral mix and 2 g of vitamin mix. The mineral mix included calcium carbonate, potassium phosphate monobasic, sodium chloride, ferric citrate, zinc carbonate, manganese carbonate, copper carbonate, potassium iodate, and sodium selenite. The vitamin mix contained retinol acetate, cholecalciferol, α -tocopheryl acetate, menadione, thiamin HCl, riboflavin, pyridoxine HCl, niacin, calcium pantothenate, folic acid, biotin, and cyanocobalamin.

Sample preparation. At the end of the experimental period, the animals were fasted overnight and anesthetized with a light dose of chloral hydrate to minimize stress. Blood samples were collected either from the abdominal aorta puncture using sterile syringes, and the rats were subsequently euthanized by cervical dislocation in accordance with the institutional animal ethics guidelines.

Furthermore, a statement has been inserted to confirm ethical compliance: “All animal procedures were conducted following the guidelines for the care and use of laboratory animals and were approved by the Ethical Committee of the University of Tlemcen, Algeria, No 165–2019”. These additions ensure that the experimental procedures meet international ethical standards and clarify how blood sampling and euthanasia were performed.

Blood samples were collected in heparinized tubes for biochemical analysis. Plasma was separated by centrifugation at $1600 \times g$ for 10 min and stored in aliquots at $-20\text{ }^{\circ}\text{C}$ until analysis.

Plasma biochemical analysis. Triglycerides, plasma cholesterol, high-density lipoprotein cholesterol (HDL-C), total protein, albumin, and glucose levels were determined using commercially available enzymatic assay kits (Spinreact, Spain), according to the manufacturer’s guidelines.

Statistical analysis. Data are expressed as mean \pm standard deviation (SD). Statistical analyses were executed using analysis of variance (ANOVA) followed by Tukey’s post hoc test. Differences were considered statistically significant at $P < 0.05$.

RESULTS AND DISCUSSION

The means (\pm standard error) of body weight are presented in **Table 3**. The statistical analysis showed that weight gain in Diet 2 was significantly higher than in Diet 1 ($p = 0.01$).

Supplementation with coconut oil (Diet 3) attenuated weight gain compared to Diet 2, but no significant difference was observed ($p = 0.15$) between Diet 3 and Diet 1 ($p = 0.10$).

These findings indicate that Diet 2 induces a greater weight gain compared to Diet 1, while Diet 3 shows intermediate values that are not significantly different.

Table 3. Body weight gains

Parameters	Diet 1	Diet 2	Diet 3
Initial B weight (g)	86.80 \pm 3.15 ^b	88 \pm 2.54	90 \pm 2.45
Final B Weight (g)	279.20 \pm 4.49 ^{ab}	319.80 \pm 5.73 ^c	294.80 \pm 4.96
Body Weight Gains (g) (w0–w8)	192.40 ^{ab}	231.80 ^c	204.80

Note: Values represent the mean \pm standard error of the mean (SEM) of rats per group. The letters (a, b, c) indicate significant differences between groups (post-hoc test).
a = Diet 1 vs. Diet 2; b = Diet 1 vs. Diet 3; c = Diet 2 vs. Diet 3

Weight gain results from a positive energy balance, which is influenced by multiple factors, including the consumption of energy-dense diets (Mohamed *et al.*, 2009).

In the present study, supplementation with coconut oil led to a reduction in body weight in rats. This finding aligns with several reports suggesting that the addition of coconut oil to high-fat or cafeteria diets significantly decreases body weight, even in the presence of high dietary energy content. A likely mechanism underlying this effect is an increase in energy consumption induced by coconut oil consumption (Fatma *et al.*, 2009).

The weight-reducing effect of coconut oil has been widely documented (Binnert *et al.*, 1998; St-Onge *et al.*, 2003) and is largely attributed to its high medium-chain triglyceride (MCT) content. MCTs are quickly digested and utilized for energy generation, similarly to carbohydrates, and consequently are not present in systemic circulation like long-chain fatty acids. Consequently, they are less likely to be stored in adipose tissue or contribute to weight gain (Crozier *et al.*, 1987). Several studies have shown that MCTs enhance fatty acid oxidation and boost metabolic rate (St-Onge *et al.*, 2003; Tsuji *et al.*, 2001), thereby contributing to reduced body weight.

Nutritional evaluation. As shown in **Table 4**, rats on Diet 2 exhibited lower food intake but higher energy intake compared to those on Diet 1, due to the higher fat content of the diet. Supplementation with coconut oil in Diet 3 reduced food intake and consequently decreased energy intake, contributing to the observed attenuation in weight gain.

Table 4. Nutritional evaluation of rats during the obesity induction period

Food intake g/rat/day			
Weeks of experimentation	Diet 1	Diet 2	Diet 3
Week 1	21.42±1.34 ^{ab}	13.57±1.70	10.78±1.98
Week 4	21±1.25 ^{ab}	17.77±0.77	15.71±1.11
Week 5	26.14±1.75 ^a	19.52±0.42	20.51±0.53 ^a
Week 8	29.57±0.85 ^{ab}	17.37±0.10	18.50±0.37
Caloric intake Kcal/rat/day			
Weeks of experimentation	Diet 1	Diet 2	Diet 3
Week 1	25.70±4.98 ^a	50.48±8.79	40.08±7.36
Week 4	25.20±4.64 ^{ab}	66.10±3.98	58.42±4.12
Week 5	31.36±6.50 ^{ab}	72.57±2.17	76.27±1.97
Week 8	35.48±3.16 ^{ab}	64.61±2.17	68.79±1.37

Note: Values represent the mean ± standard error of the mean (SEM) of rats per group. The letters (a, b, c) indicate significant differences between groups (post-hoc test).
a = Diet 1 vs. Diet 2; b = Diet 1 vs. Diet 3; c = Diet 2 vs. Diet 3

During the initial phase of the present experiment, a decrease in food intake was observed. This early reduction could be explained by many factors, including the high energy density of the diets (Guyton *et al.*, 2006) and their richness in fat, which has been demonstrated to stimulate the release of cholecystokinin. This gut hormone reduces food intake, mainly through activation of the melanocortin pathway in the hypothalamus (Guyton *et al.*, 2006).

Biochemical parameters. Although total protein and albumin levels were slightly higher in the coconut oil group (Diet 3), differences between groups were not statistically significant. However, glucose levels were significantly lower in Diet 3 compared to Diet 2 (**Table 5**).

Table 5. Biochemical analysis

Biochemical parameters	Diet 1	Diet 2	Diet 3
Glucose (g/L)	0.80±0.03 ^{ab}	2.04 ±0.09 ^c	1.39±0.05
Total protein (g/dL)	4.89±0.06	4.90±0.24	4.96±0.36
Albumin (g/dL)	3.38±0.13	3.17±0.23	2.97±0.15

Note: Values represent the mean ± standard error of the mean (SEM) of rats per group. The letters (a, b, c) indicate significant differences between groups (post-hoc test).
a = Diet 1 vs. Diet 2; b = Diet 1 vs. Diet 3; c = Diet 2 vs. Diet 3

The beneficial effects of virgin coconut oil (VCO) on glucose metabolism have been well established in experimental models (Newell-Fugate *et al.*, 2017). Studies by A. E. Newell-Fugate *et al.* (2017) and A. Narayanankutty *et al.* (2016) reported that diets containing coconut oil were associated with the absence of hyperglycemia and reduced insulin levels, suggesting a potential role of coconut oil in glucose homeostasis. VCO supplementation has also been shown to prevent insulin resistance and hyperglycemia in high-fructose-fed rats (Wein *et al.*, 2009). These effects may be attributed to the metabolic properties of medium-chain fatty acids (MCFAs), which appear to improve glycaemic control, in contrast to long-chain saturated fatty acids such as palmitate (C16:0) and stearate (C18:0), which have been implicated in the pathogenesis of type 2 diabetes mellitus (Mandal, 2017).

Lipid profile. The results shown in **Table 6**, indicate that a high fat diet (Diet 2) is associated with an unfavorable lipid profile, characterized by significant increases in total cholesterol, triglycerides, and particularly LDL-cholesterol, accompanied by a reduction in HDL-cholesterol. Supplementation with coconut oil (Diet 3) induced a favorable effect on the lipid profile, producing a significant decrease in triglyceride and LDL-cholesterol levels.

Table 6. Lipid profile

Lipid parameters	Diet 1	Diet 2	Diet 3
Total cholesterol (mg/dL)	54.04±4.35 ^a	62.23 ±3.76 ^b	57.09±4.32
Triglycerides (mg/dL)	41.22±3.22 ^a	57.78±5.46 ^c	46.77±3.62
HDL cholesterol level (mg/dL)	40.01±2.78 ^{a^b}	29.73±2.05	32.51±4.25
LDL cholesterol level (mg/dL)	5.78±0.11 ^{ab}	20.94±0.43 ^c	12.32±1.20

Note: Values represent the mean ± standard error of the mean (SEM) of rats per group. The letters (a, b, c) indicate significant differences between groups (post-hoc test).
a = Diet 1 vs. Diet 2; b = Diet 1 vs. Diet 3; c = Diet 2 vs. Diet 3

Several studies have demonstrated that coconut oil consumption exerts beneficial effects on the serum lipid profile, including reductions in total cholesterol and triglycerides (Liu et al., 2009 ; Nevin et al., 2004). In the present study, we observed a decrease in HDL-cholesterol in rats fed 32% sunflower oil; in contrast, coconut oil supplementation was associated with an increase in HDL-cholesterol levels. HDL-cholesterol plays a key role in reverse cholesterol transport, carrying cholesterol from peripheral tissues back to the liver for excretion or reuse. Increased consumption of saturated fatty acids has been associated with greater HDL-cholesterol synthesis, which in turn supports reverse cholesterol transport (Harris *et al.*, 2017).

These findings are in agreement with previous research. For instance, D. A. Cardoso *et al.* (2015) reported an increase in HDL-cholesterol among obese women after 28 days of coconut oil supplementation. Similarly, a study in which participants consumed 13 mL of coconut oil daily alongside a balanced diet for six months also reported increased HDL-cholesterol levels (Elshemy, 2018).

The observed reduction in body weight and plasma triglycerides in rats fed VCO-supplemented diets can be attributed to the metabolic properties of medium-chain fatty acids, which are rapidly oxidized for energy rather than stored as fat. Additionally, the elevated HDL levels suggest enhanced reverse cholesterol transport, while decreased LDL concentrations reflect reduced hepatic lipid accumulation. These findings support the hypothesis that partial substitution of sunflower oil with VCO improves lipid metabolism through modulation of energy utilization pathways. While previous studies have established the general hypolipidemic and thermogenic effects of medium-chain triglycerides (MCTs), the present results extend this understanding by demonstrating that partial replacement of polyunsaturated sunflower oil with VCO within a high-fat diet confers comparable metabolic improvements. This finding suggests that the beneficial effects of MCTs can be achieved without complete substitution, reflecting a more feasible dietary strategy. Furthermore, our data indicate concurrent improvements in plasma proteins and HDL/LDL balance, providing additional biochemical evidence for the systemic benefits of moderate VCO incorporation.

CONCLUSION

The present study demonstrates that partial substitution of sunflower oil with virgin coconut oil (VCO) in a high-fat diet significantly improves lipid metabolism and reduces obesity-related parameters in Wistar rats. VCO supplementation led to decreased plasma glucose, cholesterol, and triglyceride levels, alongside increased HDL concentrations and moderated weight gain. These effects are likely associated with the metabolic activity of medium-chain fatty acids that enhance lipid oxidation and energy utilization. Collectively, these findings suggest that moderate inclusion of VCO in dietary fats may serve as a beneficial strategy for managing dyslipidemia and obesity.

This study provides new evidence that partial substitution of sunflower oil with virgin coconut oil (VCO) in a high-fat diet can improve lipid metabolism and mitigate obesity-related disturbances in Wistar rats. Unlike previous studies that examined VCO as an isolated supplement, our findings demonstrate that even moderate inclusion of VCO (4% of total diet) exerts measurable benefits on lipid profile and weight regulation. These results suggest that small, practical dietary modifications involving VCO could offer an accessible nutritional strategy for managing metabolic disorders associated with high-fat consumption.

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COMPLIANCE WITH ETHICAL STANDARDS

Conflict of interest: the authors declare no conflict of interest.

Funding: no funding was received for conducting this study.

Animal rights: the rats were treated ethically, following the international guidelines for treating laboratory animals. In vivo manipulations tests on Wistar rats were approved by the Ethical Committee of the University of Tlemcen, Algeria, No 165–2019. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. All efforts were made to minimize pain and distress.

AUTHOR CONTRIBUTIONS

Investigation, [Y.S-M.; S-M.Z.; C-S.D.; S.H.]; visualization, [S-M.Z.; M.R-K.]; validation, [Y.S-M.]; writing original draft, [Y.S-M.; S-M.Z.], conceptualization, [Y.S-M.]; data curation, [Y.S-M.; S-M.Z.]; writing – writing – review & editing, [Y.S-M.; S-M.Z.].

All authors have read and agreed to the published version of the paper.

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ОЦІНКА ХАРЧОВОГО СТАТУСУ І ЛІПІДНОГО ПРОФІЛЮ У ЩУРІВ, ЯКИХ ГОДУВАЛИ РАЦІОНОМ ІЗ ДОДАВАННЯМ СОНЯШНИКОВОЇ ТА КОКОСОВОЇ ОЛІЇ

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Вступ. Кокосову олію традиційно вважають продуктом із потенційними корисними властивостями для здоров'я, зокрема, щодо регуляції метаболізму. Метою цього дослідження було оцінити вплив високожирової дієти з додаванням кокосової

олії (КО), порівняно зі соняшниковою олією (СО), на масу тіла, споживання корму та біохімічні показники плазми крові у щурів лінії Wistar.

Матеріали і методи. П'ятнадцять самців щурів Wistar (≈ 80 г) були розподілені на три групи: D1 (16% казеїну + 8% СО), D2 (16% казеїну + 32% СО) та D3 (16% казеїну + 28% СО + 4% КО). Через два місяці визначали рівні глюкози, загального білка, альбуміну, тригліцеридів, загального холестерину, HDL і LDL у плазмі крові з використанням наборів для ферментативного аналізу.

Результати. У щурів, які отримували дієту з додаванням кокосової олії (D3), спостерігали достовірно нижчі рівні глюкози крові, тригліцеридів і загального холестерину, а також помітне підвищення рівня холестерину HDL порівняно з контрольними групами. Крім того, у групі з кокосовою олією було значно знижено приріст маси тіла та споживання корму.

Висновки. Отримані результати свідчать, що додавання кокосової олії чинить сприятливий вплив на ліпідний обмін і регуляцію маси тіла. Це вказує на потенційну роль кокосової олії як функціонального харчового жиру для контролю ожиріння та метаболічних порушень.

Ключові слова: ожиріння, кокосова олія, обмін ліпідів, рослинні олії, соняшникова олія