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Original Article

Impact of Low-Carbohydrate Diet Supplemented with Cinnamon Bark and Moringa Seed Oil Extracts on Lipid Profile in Obese Albino Rats

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Abstract: Dyslipidemia is a preventable cardiovascular risk factor, and low-carbohydrate (LC) diets with or without the addition of bioactive plant oils can modulate lipid metabolism. This study aimed to investigate the impact of an LC diet, alone or in combination with cinnamon bark oil and/or moringa seed oil, on the lipid profiles of obese albino rats. In addition, we determined whether the co-supplementation of these oils could enhance the lipid-modulating effects of an LC diet. Eighty-one obese adult rats were randomly divided into five groups: control, low-carb diet, low-carb diet with cinnamon bark oil extract (LCC), low-carb diet with moringa seed oil extract (LCM), and low-carb diet with cinnamon bark and moringa seed oil extracts (LCCM), and treated for 12 weeks. Total cholesterol (TC), triglyceride (TG), low-density lipoprotein (LDL), high- density lipoprotein (HDL), and very low-density lipoprotein (VLDL) levels were determined. Compared with the baseline, following the LC diet intervention, TC declined by 31.45%; it was even higher in groups treated with cinnamon (40.12%) or moringa (47.45%), while mixed oils caused an intermediate decrease (28.68%). Significant reductions of 54.09% were noted with the LC diet, and similar reductions were achieved with the LCC (37.07%) and LCM (48.82%) diets, however, a significantly lower reduction (20.53%) was recorded with the combined oil treatment. TG increased in the control (103.60%) and LC diet (35.80%) groups, was close to baseline in the cinnamon group (2.35%), decreased in the LCM diet group (14.93%), and was slightly lower in the combined oil group (5.34%). VLDL exhibited a similar trend. HDL levels decreased in all intervention groups. Collectively, cinnamon or moringa seemed to augment LC diet-induced amelioration of atherogenic lipids, however, the co-supplementation did not exhibit additive effects, and reductions in HDL require further attention.

1. Introduction

Dyslipidemia, defined by abnormal levels of plasma lipids—primarily total cholesterol (TC), triglyceride (TG), low-density lipoprotein (LDL), and high- density lipoprotein (HDL), is a major modifiable risk factor for atherosclerosis and cardiovascular diseases, the leading cause of mortality globally [1-3]. The increasing prevalence of dyslipidemia is associated with changes in dietary habits, sedentary lifestyle, obesity, and metabolic syndromes [4]. The global burden of dyslipidemia, particularly in low- and middle-income countries, necessitates the development of cost-effective and natural interventions to prevent and manage lipid disorders [5, 6].

Dietary modifications have long been the cornerstone of dyslipidemia management [7]. Among various dietary strategies, the low-carbohydrate (LC) diet has received considerable attention for its effects on lipid metabolism, glycemic control, and body composition [8, 9]. The LC diet often involves reducing carbohydrate intake to <130 grams/day or <45% of the total energy intake, thereby promoting adaptations in energy metabolism that favor lipolysis and ketogenesis [10, 11]. Clinical and preclinical data demonstrate that the LC diet reduces TG, increases HDL, and lowers [12-14]. However, the LDL response to LC diets is heterogeneous, depending on dietary fatty acid intake and sources [15, 16]. However, there are still some key gaps in the literature. The additive effects of a LC diet, cinnamon (*Cinnamomum verum*), and/or moringa (*Moringa oleifera*) oils on dyslipidemia are unclear. The current study fills these gaps by evaluating their additive effects on the lipid profiles of obese rats.

The incorporation of natural lipid-lowering agents, such as plant-based essential oils, has emerged as a novel strategy for improving the therapeutic efficacy of LC diets [17, 18]. Cinnamon bark oil and moringa seed oil have attracted significant scientific interest as functional compounds owing to their bioactive components and metabolic improvement effects [19-21].

Cinnamon bark essential oil is composed of components such as cinnamaldehyde, eugenol, linalool, and a range of polyphenolic compounds that are documented for their strong antioxidant, anti-inflammatory, and hypolipidemic activity [22-24]. Cinnamon extracts have been shown to decrease serum TC, LDL, and TG levels while increasing HDL levels in rodent models of hyperlipidemia and diabetes [25-28]. The hypolipidemic effects of cinnamon are thought to be mediated through modulation of hepatic lipid metabolism, increased bile acid excretion, and enhanced lipoprotein lipase activity [27]. Human studies also provide supportive evidence; [29, 30] demonstrated that daily intake of cinnamon powder significantly improved lipid and glucose parameters in patients with type 2 diabetes mellitus.

Similarly, moringa, widely known as the "miracle tree," is rich in health-promoting phytochemicals, particularly in its seeds and seed oil [31-36]. Moringa seed oil contains unsaturated fatty acids,mainly oleic acid, and sterols, flavonoids, tocopherols, and polyphenols that contribute to its pharmacological effects [37-41]. Several animal studies have reported that moringa seed extracts can significantly reduce serum cholesterol and LDL, while improving HDL [42-44]. The mechanisms of action include inhibition of 3-hydroxy-3-methylglutaryl-coenzyme A reductase, upregulation of antioxidant defense, and modulation of lipid transport and clearance [45, 46].

Animal models, particularly albino rats (*Rattus norvegicus*), are widely used to study lipid metabolism because of their well-characterized physiology and responsiveness to dietary and pharmacological interventions [47, 48]. Studies have successfully used albino rats to simulate human-like lipid disorders, evaluate the hypolipidemic potential of natural compounds, and explore dietary-gene interactions [28, 49]. In addition, the controlled laboratory setting enables uniform assessment of dietary intake and its metabolic biomarkers as well as of tissue-specific effects [50-52].

Although LC diets and plant bioactive compounds have been investigated as separate factors, but their combined effects on dyslipidemia have not yet been characterized in previous studies. This study aimed to address this gap by evaluating an integrated dietary phytochemical strategy. We hypothesize that the dual therapy would result in greater reductions in TC, TG, and LDL, and HDL cholesterol compared to the individual treatments. Thus, this study aimed to assess the impact of cinnamon bark oil and moringa seed oil (individually and in conjunction with a LC diet) on the serum lipid profile of obese albino rats. These findings provide evidence for designing functional foods or integrative dietary therapies to ameliorate lipid disorders and cardiovascular risk.

2. Materials and Methods

2.1. Experimental Design and Randomization

A total of 120 adult albino rats, aged 16 weeks, healthy, and weighing 203.25–295.38 g, were obtained from an animal house-accredited facility. Rats were kept in polypropylene cages with ad libitum access to food and water in standard laboratory conditions (23 ± 1°C, 20 ± 5% relative humidity, and 12-hour light/dark cycle). The study was conducted as a randomized, controlled, parallel-group

animal study in accordance with the guidelines for the Care and Use of Laboratory Animals in Biomedical Research [53] for the handling and use of laboratory animals.

2.2. Material Collection and Preparation

Fresh bark of true cinnamon (*Cinnamomum verum*) and moringa seeds (*Moringa oleifera*) were collected from a certified herbal vendor and taxonomically identified. Prof. A. Muhan. Voucher specimens were deposited at the Herbarium of Halabja Technical College. Kurdistan Regional, Iraq, under voucher numbers (HTC001) and (HTC002), respectively. The bark and seed were washed thoroughly with running tap water to remove dust and impurities, and shade-dried at room temperature (25 ± 2°C) to a constant weight, which took 7–10 days. The bark was air-dried and ground into pieces (1–2 cm) using a stainless-steel grinder to increase surface area for the distillation. The moringa seed was stored in a dark container at 4 °C.

2.3. Preparation of Extracted Oils

2.3.1. Cinnamon Bark Extracted Oil

Cinnamon bark was procured from a recognized source in India (Cinnamomum zeylanicum). Air-dried and pulverized bark essential oil was obtained via hydrodistillation for 3 h using a Clevenger-type apparatus. The oil was recovered, filtered, and stored in amber vials at 4°C until use [54].

2.3.2. Moringa Seed Extracted Oil

To obtain the virgin moringa seed oil, de-husked, ground, and matured moringa seeds were cold -pressed and extracted. The oil was stored in the dark at 4°C to retain bioactive compounds [55]

2.4. Diets Design and Preparation

The The basal diet of the control group was made from soybeans, local Iraqi wheat grain, salt, and limestone with 58.82% carbohydrate, 19.52% protein, and 6.15% fat, containing a balanced supply of vitamins and minerals (dissolved in drinking water), calculated to meet the maintenance requirements for adult rats according to the Nutrient Requirements of Laboratory Animals (NRC) [56]. The high-fat diet of the experimental group was prepared based on that of walnut, peanut, almond, and wheat with the addition of salt or fineness lime into this diet to include the following composition: 57.69% fat, 18.54% protein, and 14.91% carbohydrate, plus sodium from salt, and calcium from fineness lime.

2.5. Chemical Determination of Diets

Moisture content was determined using the Association of Official Agricultural Chemists (AOAC) method 930.15 [57]. Approximately 2 g of the feed sample was weighed onto a covered aluminum dish and dried in an oven at 135°C for 2 h. The sample was cooled in a desiccator and then re-weighed to determine the loss in weight due to drying, which was reported as a percentage of the original weight. The percentage of dry matter was calculated as 100% minus the determined moisture content. The ash content was analyzed using a muffle furnace [58]. Crude fiber content was determined using the Weende method [59]. The soxhlet extraction technique was used to measure fat content [60]. The crude protein content of the samples was assessed using the Kjeldahl method [61].

2.6. Fatty Acid Determination in Moringa Seed Extracted Oil

The fatty acid content was analyzed by gas chromatography with flame ionization detection (GC–FID) on an Agilent 7890A system fitted with an FID detector and Chem Station software. The capillary column (100 m × 0.25 mm i.d., film thickness 0.20 μm) used was an HP-88 (Agilent Technologies, USA) designed for fatty acid methyl ester (FAME) analysis. The injector split was 1 μL, and the FAME sample for injection was prepared according to the AOAC [62]. The oven temperature program started at 120°C for 1 min, then ramped to 175°C at 10°C/min and held for 10 min. The temperature was then increased to 210°C at 5°C/min and held for 5 min, followed by a final increase to approximately 230°C at the same rate, which was maintained for an additional 20 min. Nitrogen was used as the carrier gas at a fixed flow rate of 1 mL/min, and hydrogen and air were supplied to the flame ionization detector. The injector and detector were maintained at 250°C. Specific fatty acids were identified by comparing their retention

times with those of authentic FAME standards (Upelco 37 Component FAME Mix, Sigma). Quantification was performed by area normalization, and values are reported as a relative % of the total identified fatty acids. ChemStation (Agilent Technologies, USA) was used for data collection and analysis [63].

2.7. Bioactive Compound Identification by Gas Chromatography-Mass Spectrometry (GC-MS)

The essential oil was analyzed using a GC–MS system comprising an Agilent 7890A gas chromatograph coupled to an Agilent 5975C mass -selective detector (USA). A split/splitless injector and HP-5MS capillary column (30 m×0.25 mm id., 0.25µm film thickness; Agilent Technologies, USA) were installed on the instrument. The maximum cooking oven temperature was programmed to 80°C for 3 min, ramped at 8°C/min to 180°C, and held for another 3 min. Helium was used as the carrier gas at a flow rate of 1.0 mL/min. Electron ionization was performed at 70 eV, with the injector set in split mode at a 500:1 ratio. MS spectra were acquired in the m/z range of 40–500 m/z. The compounds were identified by matching the obtained mass spectra with retention indices stored in commercial libraries (Wiley 7n and NIST) and the Chem Station software [64].

2.8. Rat Screening

Eighty-one obese Albino rats were used in this study. These animals were purchased from the animal house of the Human Development University. Screening included anthropometric measurements (body weight and naso-anal length) to calculate body mass index and the Lee index, along with a visual examination to exclude participants showing signs of inflammation, fighting injuries, or eating abnormalities. Following these criteria, rats were randomized into five groups: control (Co), LC diet, and LC diet supplemented with various types of essential oils: cinnamon bark oil (LCC), moringa seed oil (LCM), or cinnamon combined with moringa seed oil (LCCM). A total of 81 rats will be included in the study from late October 2024 to late January 2025.

2.9. Study Groups and Ethical Approval

A total of 81 rats (203.25–295.38g) were randomly divided into five groups (n=16, except for the control group, n=17); animals in group I as the control and were fed the control rat diet. Group II (LC) rats were fed an LC diet, group III rats were fed an LC diet supplemented with 50 mg/kg body weight (BW)/d of cinnamon bark extract oil (LCC), group IV rats were fed an LC diet supplemented with 300 mg/kg BW/d of moringa seed extract oil (LCM), and group V rats were fed an LC diet supplemented with the same doses of the extracted oils (LCCM) as described above. The experiments were conducted over 12 weeks (Figure 1).

All experimental procedures were performed in accordance with the guidelines of the (Sulaimani Polytechnic University) Institutional Animal Care and Use Committee under protocol number (91/245).

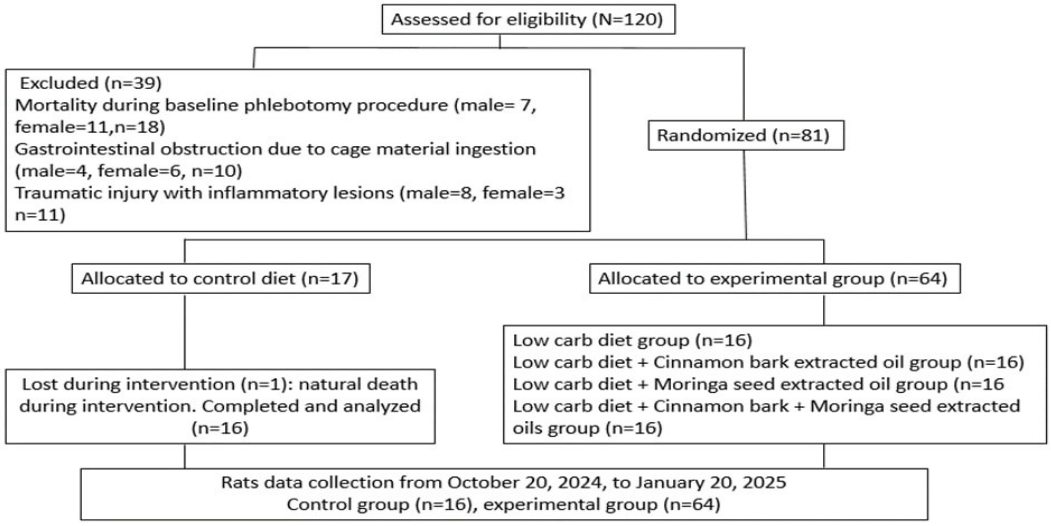

Figure 1: Clinical study design flowchart.

2.10. Administration and Monitoring

The rats were acclimatized for one week before being assigned to the experimental diet groups. The body weights and food intakes of the rats were recorded weekly. The treated oils were delivered via oral gavage once daily to the rats to ensure accurate dosing of the rats..

2.11. Blood Sample and Lipid Analysis

Rats were fasted for 12 h and anesthetized with diethyl ether prior to surgery. Blood was obtained directly from the hearts of anesthetized rats into non-heparinized (ethylenediaminetetraacetic acid tube) and gel tubes at the end of the 12-week feeding period. After centrifugation of the blood at 3000 × g for 10 min at 4°C, the plasma was used to determine the lipid profile, including TC, LDL, HDL, and TG levels, using a COBAS6000 analyzer (Roche Diagnostics, Mannheim, Germany).

2.12. Sample Size Calculation

The sample size was calculated using a power analysis for analysis of variance (ANOVA), assuming a medium effect size (f = 0.30), α = 0.05, and 80% statistical power. The required number of was 70 animals (14 per treatment group). To compensate for the expected attrition during the 12-week intervention, 15% was added, resulting in a final sample size of 80. The animals were randomly assigned to five groups (Figure 1). Thus, each of the five treatment groups contained 16 rats.

2.13. Statistical Analysis

Data were analyzed using SPSS software version. 21. Distributions' normality and homogeneity of variance were evaluated before using parametric tests. The effects of treatment and sex were analyzed using a two-way ANOVA, with sex and treatment as the main factors, and a post hoc Tukey's test was used for multiple comparisons. An unpaired T-test is used to compare groups with the control group. Statistical significance was established at the $p < 0.05$ threshold. Data were presented as mean ± standard deviation (SD).

3. Results

In a trial involving 120 animals, 81 rats were randomly allocated after exclusion criteria were applied. A total of 39 animals were excluded: Excluded (n=39), mortality during baseline phlebotomy procedure (n=25), gastrointestinal obstruction due to cage material ingestion (n=14), traumatic injury with inflammatory lesions (n=10). 81 animals were randomly allocated to four groups (LC, LCC, LCM, and LCCM), with 16 rats per group, and the control group contained 17 rats. .

3.1. Fatty Acid Profile

According to GC–FID analysis, the fatty acid profile was favorable, and due to its high oleic acid content, it is widely recognized for its cardioprotective properties and for improving blood lipid levels. Other major components were palmitic, stearic, vaccenic, and behenic acids, with minor amounts of linoleic and α-linolenic acids. The GC-FID profiling of the completefull fatty acid composition is shown in figure 2, and the chromatographic profile is presented in table 1.

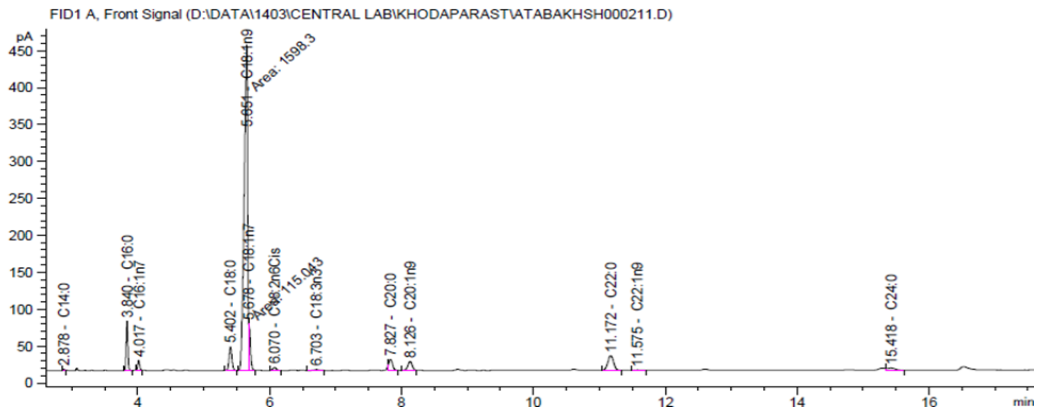

Figure 2: Chromatographic profile of fatty acid composition in moringa seed extracted oil as determined by GC–FID.

Table 1: Fatty acid composition and concentration of the moringa seed extracted oil as determined by GC–FID.

Fatty Acid	Common name	Concentration (%)
C14:0	Myristic acid [1]	0.11
C16:0	Palmitic acid [1]	5.57
C16:1n7	Palmitoleic [2]	1.29
C18:0	Stearic acid [1]	4.37
C18:1n9	Oleic acid [2]	71.14
C18:1n7	Vaccenic [2]	5.12
C18:2n6	Linoleic [3]	0.64
C18:3n3	α-Linolenic [3]	0.25
C20:0	Arachidic [1]	2.47
C20:1n9	Gondoic [2]	2.54
C22:0	Behenic acid [1]	5.39
C22:1n9	Erucic acid [2]	0.10
C24:0	Lignoceric [1]	1.01

* **Note:** 1, 2, and 3 are abbreviations for saturated (SFA), monounsaturated (MUFA), and polyunsaturated fatty acids (PUFA), respectively.

3.2. Bioactive Compounds Profile in Cinnamon Bark Extracted Oil

GC–MS analysis identified several bioactive molecules in cinnamon bark extracted oil, such as terpenoids, phenolics, and aromatic analysis identified several bioactive molecules, including terpenoids, phenolics, and aromatic aldehydes (Figure 3). The chromatographic profile revealed major and minor compounds widely known for their antioxidant, antimicrobial, and anti-inflammatory properties. The main components were cinnamaldehyde (63.22%), trans-caryophyllene (5.40%), 1, 8-cineole (5.98%), linalool (4.10%), and α-pinene (3.41%). Additional synergistic compounds include p-cymene, α-phellandrene, eugenol, cinnamyl acetate, and α-copaene at lower concentrations.

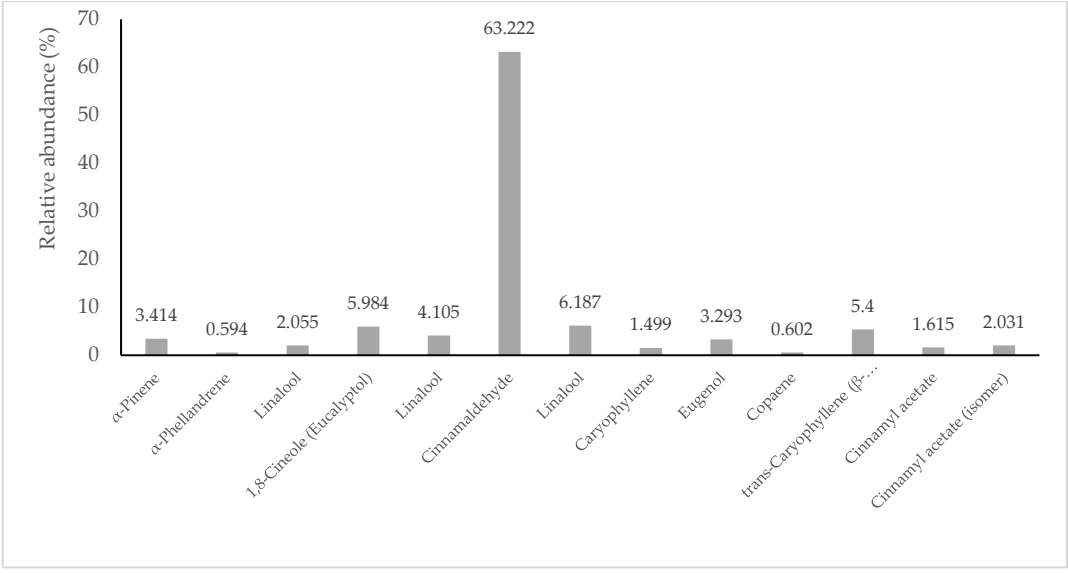

Figure 3: Cinnamon bioactive compounds identified by GC–MS analysis in cinnamon bark extracted oil.

3.3. Effect of Different Dietary Treatments on Body Weights

Figure 4 shows the weekly body weight changes in a group of obese rats during the 12-week intervention and treatment periods. In the control group, in which the rats were fed a standard diet, body weight increased over 12 weeks. Group II, when fed a LC diet as a single intervention to reduce obesity, showed a consistent reduction in body weight over 12 weeks. The LCC group showed a sharper reduction in body weight than the control group. After six weeks, the rate of weight loss increased significantly, with lower and more stable weights in subsequent weeks. Animals fed in LCM group showed no significant changes in overall body weight compared to other groups. A slight reduction in

body weight over 12 weeks indicates relatively continuous consumption despite the gradual and shallow regulation methods. Feeding the combination of two plants as a single treatment, moringa and cinnamon (LCCM group), showed a significant reduction in body weight after week 5 and also showed a progressive and consistent reduction in weight until week 12.

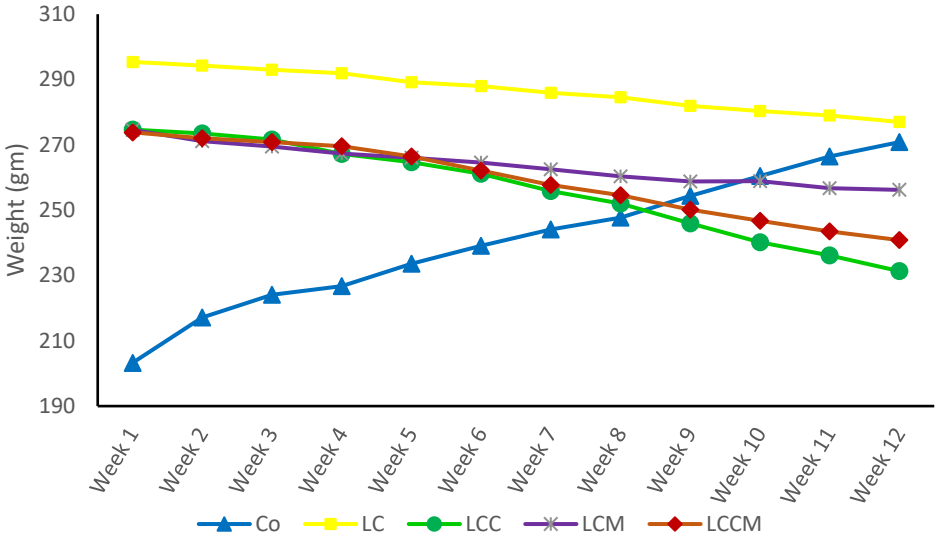

Figure 4: Weekly body weight changes in groups of obese rats after 12 weeks of treatment.

3.4. Lee Index

The change in the Lee Index of obese rats before and after 12 weeks of dietary intervention is shown in figure 5. At the end of the experimental period, the Lee Index of the control group decreased slightly. However, at the end of the 12-week experiment, the LC, LCC, LCM, and LCCM groups, showed a clear decline in the Lee Index measurements. The decline was most significant in LCCM group. The LCC group followed by LCM group displayed a significant improvement in obesity indicators owing to a decrease in adiposity and body fat mass in the treated rats. The general decline in the Lee Index across treatments confirmed that the LC dietary treatment is anti-obesity, and its effectiveness is further increased when combined with moringa and cinnamon, as both have a synergistic effect that reduces body fat mass and helps maintain homeostasis.

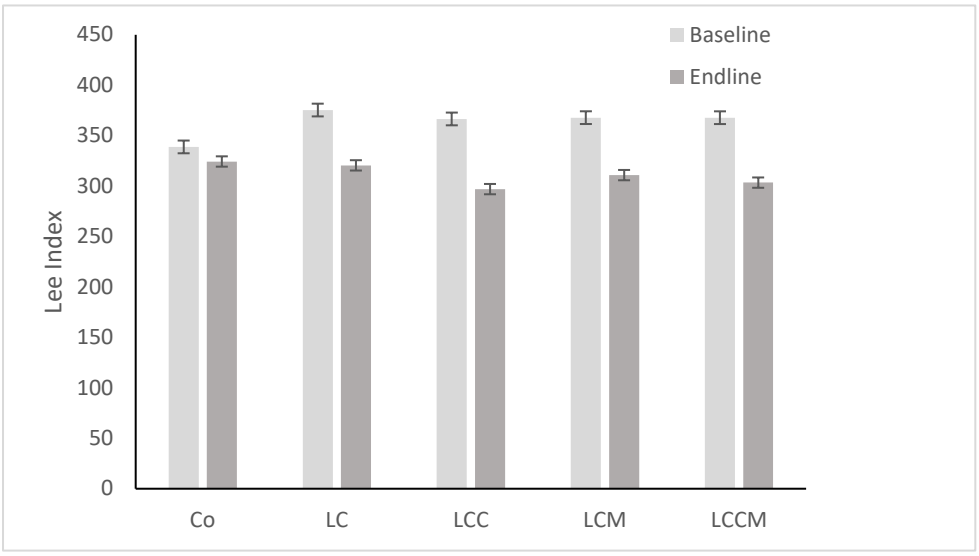

Figure 5: Comparison of baseline and endline of Lee index among groups.

3.5. Lipid Profile

Table 2 shows the TC levels (mg/dL) of rats fed different diets for 12 weeks. In the control group, there was no significant change in TC levels, with a slight 2.5% decrease (p = 0.724). In contrast, rats fed a LC diet showed a significant 31.45% decrease in TC (p = 0.002). For those in LCC group, TC levels decreased even more steeply by 40.12% (p = 0.001). However, at the TC level, rats in LCM group showed a significant (p = 0.008) 47.45% reduction in body weight. Notably, the combined treatment in LCCM group resulted in a 28.68% reduction in TC levels (p = 0.003), which, although significant, was less than the effect of either supplement alone.

Table 2: Comparison of baseline and endline of total cholesterol level among groups.

Study groups	Baseline (Mean ± SD) (mg/dL)	Endline (Mean ± SD) (mg/dL)	Change (%)	p-value
Control	59.42 ± 8.75	57.93 ± 11.53	-2.50	0.724 ns
LC	64.77 ± 7.32	44.40 ± 4.68	-31.45	0.002 **
LCC	62.60 ± 8.35	37.48 ± 2.81	-40.12	0.001 **
LCM	63.40 ± 11.39	33.32 ± 10.87	-47.45	0.008 **
LCCM	59.68 ± 4.63	42.57 ± 6.54	-28.68	0.003 **

Significance between-group values were calculated using ANOVA test. Data are expressed as mean ± SD. Significance levels: *: $p < 0.05$, **: $p \leq 0.01$, ns: non-significant difference.

In table 3, all dietary treatments effectively lowered TC compared with the control group, with the greatest decrease observed in LCM group.

Table 3: Comparison of baseline and endline of total cholesterol level among groups.

Study groups	Mean ± SD (mg/dL)	P-value (vs Co)	Significance
Control	58.16 ± 11.53	—	—
LC	44.16 ± 4.68	0.034	*
LCC	37.43 ± 2.81	0.006	**
LCM	33.19 ± 10.87	0.003	**
LCCM	42.77 ± 6.54	0.022	*

Priori comparisons of treatments with the control group were conducted using independent-samples t-tests. Data are expressed as mean ± SD. Significance levels: *: $p < 0.05$, **: $p \leq 0.01$.

As shown in table 4, the control group showed a significant increase in TG levels. TG increased by 103.6% from 32.83 ±13.41 mg/dL at baseline to 66.85 ± 38.30 mg/dL at the end of our study. Rats fed thean LC diet had a nonsignificant increase in TG levels of 35.8%, from 33.75 ± 17.31 mg/dL to 45.83 ± 12.24 mg/dL. In the LCC group, TG levels did not change significantly, increasing only slightly (from 39.05 ± 15.60 mg/dL to 39.97 ± 15.24 mg/dL), with a p- value of 0.934. Rats in LCM group experienced a nonsignificant decrease in TG levels of 14.93%, falling from 45.88 ± 16.07 mg/dL at baseline to 39.03 ± 12.16 mg/dL at the end of our study. In animals of LCCM group, TG levels increased slightly by 5.34% (from 31.52 ± 10.95 mg/dL to 33.20 ± 4.61 mg/dL).

Table 4: Comparison of baseline and endline triglyceride levels among groups.

Study groups	Baseline (Mean ± SD) (mg/dL)	Endline (Mean ± SD) (mg/dL)	Change (%)	p-value
Control	32.83 ± 13.41	66.85 ± 38.30	103.60	0.039 *
LC	33.75 ± 17.31	45.83 ± 12.24	35.80	0.082 ns
LCC	39.05 ± 15.60	39.97 ± 15.24	2.35	0.934 ns
LCM	45.88 ± 16.07	39.03 ± 12.16	-14.93	0.526 ns
LCCM	31.52 ± 10.95	33.20 ± 4.61	5.34	0.723 ns

Significance between-group values were calculated using ANOVA test. Data are expressed as mean ± SD. Significance levels: *: $p < 0.05$, ns: non-significant difference.

Overall, these results indicate that moringa and cinnamon, when combined with an LC diet (LCCM group), exert a synergistic triglyceride-lowering effect (Table 5).

Table 5: Effect of dietary treatments on triglyceride levels in the groups.

Study groups	Mean ± SD (mg/dL)	P-value (vs Co)	Significance
Control	66.85 ± 38.30	-	-
LC	46.67 ± 12.24	0.0781	ns
LCC	39.25 ± 15.24	0.0217	*
LCM	36.33 ± 12.16	0.0151	*
LCCM	34.68 ± 4.61	0.0083	**

Pairwise comparisons with the control group were conducted using independent-sample t-tests. Data are expressed as mean ± SD. Significance levels: *: $p < 0.05$, **: $p \leq 0.01$, ns: non-significant difference.

Table 6 shows the LDL levels (mg/dL) of obese rats subjected to different dietary interventions for 12 weeks. In the control group, LDL levels showed a small, nonsignificant reduction of 14.55%, from 17.75 ± 3.89 mg/dL at baseline to 15.17 ± 5.75 mg/dL at the end of the study. On an LC diet, rats had a marked reduction in LDL level by 54.09%, falling from 15.77 ± 6.18 mg/dL to 7.24 ± 2.35 mg/dL. Similarly, rats supplemented with the LCC diet had a significant 37.07% decrease in LDL levels, from 14.63 ± 5.53 mg/dL to 9.21 ± 6.32 mg/dL. In the LCM group, LDL levels decreased by 48.82%, from 13.30 ± 4.53 mg/dL at the start to 6.81 ± 1.26 mg/dL after 12 weeks. The LCCM diet resulted in a non-significant 20.53% decrease. From 14.72 ± 4.50 mg/dL, it reduced to 11.70 ± 7.33 mg/dL.

Table 6: Comparison of baseline and endline of low-density lipoprotein levels among groups.

Study groups	Baseline (Mean ± SD) (mg/dL)	Endline (Mean ± SD) (mg/dL)	Change (%)	p-value
Control	17.75 ± 3.89	15.17 ± 5.75	-14.55	0.066 ns
LC	15.77 ± 6.18	7.24 ± 2.35	-54.09	0.005 **
LCC	14.63 ± 5.53	9.21 ± 6.32	-37.07	0.044 *
LCM	13.30 ± 4.53	6.81 ± 1.26	-48.82	0.014 *
LCCM	14.72 ± 4.50	11.70 ± 7.33	-20.53	0.395 ns

Significance between-group values were calculated using ANOVA test. Data are expressed as mean ± SD. *: $p < 0.05$, **: $p \leq 0.01$, ns: nonsignificant difference.

A LC diet alone or in combination with moringa (LCM group) effectively reduced LDL compared to the control diet (Table 7).

Table 7: Effect of dietary treatments on low-density lipoprotein levels.

Study groups	Mean ± SD (mg/dL)	P-value (vs Co)	Significance
Control	13.90 ± 5.75	-	-
LC	6.97 ± 2.35	0.0165	*
LCC	9.51 ± 6.32	0.1205	ns
LCM	7.78 ± 1.26	0.0381	*
LCCM	11.96 ± 7.33	0.4822	ns

Pairwise comparisons with the control group were conducted using independent-sample t-tests. Data are expressed as mean ± SD. Significance levels: * $p < 0.05$, ns: non-significant difference.

Table 8 shows the HDL levels in obese rats fed various dietary interventions for 12 weeks. During the study period, the control group showed no significant change in HDL concentration. The value remained almost constant at 33.37 ± 6.63 mg/dL after 1 month of observation and 33.40 ± 5.71 mg/dL at the end of the study. A minimal increase of 0.10% was calculated from these findings. In contrast, rats fed the LC diet showed a significant decrease in HDL concentration, from 41.57 ± 8.24 mg/dL to 29.45 ± 6.78 mg/dL, a 29.16% decrease. Likewise, in the LCC group, HDL concentration also showed a highly significant decline of 47.29% (from 41.40 ± 7.66 mg/dL to 21.82 ± 5.47 mg/dL). Rats on an LCM diet also grew well. The HDL level fell by 45.26% from 40.57 ± 8.59 mg/dl at the start to 22.21 ± 9.76 mg/dL at the

end of this study period. Similarly, for the LCCM diet group, HDL level also showed a highly significant drop of 37.90%. It decreased from 40.07 ± 7.33 mg/dL at baseline to 24.88 ± 4.73 mg/dL.

Table 8: Comparison of baseline and endline high-density lipoprotein level among groups.

Study groups	Baseline (Mean ± SD) (mg/dL)	Endline (Mean ± SD) (mg/dL)	Change (%)	p-value
Control	33.37 ± 6.63	33.40 ± 5.71	0.10	0.991 ns
LC	41.57 ± 8.24	29.45 ± 6.78	-29.16	0.009 **
LCC	41.40 ± 7.66	21.82 ± 5.47	-47.29	0.001 ***
LCM	40.57 ± 8.59	22.21 ± 9.76	-45.26	0.007 **
LCCM	40.07 ± 7.33	24.88 ± 4.73	-37.90	0.005 **

Significance between-group values was calculated using ANOVA test. Data are expressed as mean ± SD. Significance levels: *: $p < 0.05$, **: $p \leq 0.01$, ***: $p \leq 0.001$, ns: non-significant difference.

The results indicate that while the control group maintained relatively higher HDL levels (Table 9), all dietary treatments—particularly those involving moringa and cinnamon—led to a significant reduction in HDL cholesterol.

Table 9: Effect of dietary treatments on high -density lipoprotein among groups.

Study groups	Mean ± SD (mg/dL)	P-value (vs Co)	Significance
Control	35.54 ± 5.71	-	-
LC	28.67 ± 6.78	0.0872	ns
LCC	21.11 ± 5.47	0.0010	***
LCM	21.79 ± 9.76	0.0014	**
LCCM	24.64 ± 4.73	0.0081	**

Pairwise comparisons with the control group were conducted using independent-sample t-tests. Data are expressed as mean ± SD. Significance levels: **: $p \leq 0.01$, ***: $p \leq 0.001$, ns: non-significant difference.

Table 10 presents the VLDL concentrations in obese rats across the different dietary drug-treated groups after 12 weeks of the experiment. The control diet exhibited a significant elevation in VLDL, this was raised by 103.38% (6.67 ± 2.66 mg/dL at baseline increased by 103.38% (6.67 ± 2.66 mg/dL at baseline to 13.56 ± 7.87 mg/dL after the diet), showing a nonsignificant increase in VLDL by 33.63% (6.83 ± 3.43 to 9.13 ± 2.53 mg/dL). The VLDL in individuals with cinnamon (LCC group) did not exhibit any changes either, leveling up by 0.96% (7.83±3.25 to 7.91 ±2.96 mg/dL); this group following on the LC cohort in terms of declining VLDL levels did show a small raising during therapy period was similar but it were insignificant increased hyperlipidemias decline as remained unchanged however; what occurred is high-lpa) decreasing by only −39%- 33% (95% CI:- 45%- 19%;- 31%- 40%) and TG at ratch (cinnamon)/TG decreased also when turned, doubling expanded hyperlipidemias, showing decreased methods, lesser or less specificity.

Similarly, VLDL was reduced non-significantly in LCM-fed rats by 14.80%, from 9.17±2.93 mg/dL to 7.81±2.36 mg/dL. In addition, there was a 7.65% increase in the plasma cholesterol levels from 6.17±2.14 mg/dL to 6.64±0.92 mg/dL in LCCM group.

Table 10: Comparison of baseline and endline very low-density lipoprotein levels among the groups.

Study groups	Baseline (Mean ± SD) (mg/dL)	Endline (Mean ± SD) (mg/dL)	Change (%)	p-value
Control	6.67 ± 2.66	13.56 ± 7.87	103.38	0.042 *
LC	6.83 ± 3.43	9.13 ± 2.53	33.63	0.104 ns
LCC	7.83 ± 3.25	7.91 ± 2.96	0.96	0.973 ns
LCM	9.17 ± 2.93	7.81 ± 2.36	-14.80	0.501 ns
LCCM	6.17 ± 2.14	6.64 ± 0.92	7.65	0.626 ns

Significance between-group values was calculated using ANOVA test. Data are expressed as mean ± SD. Significance levels: * $p < 0.05$, ns: non-significant difference.

Overall, these findings indicate that LC diets, particularly when combined with cinnamon supplementation (LCC group), effectively reduced VLDL concentrations in obese rats (Table 11).

Table 11: Effect of dietary treatments on very low -density lipoprotein among groups.

Study groups	Mean ± SD (mg/dL)	P-value (vs Co)	Significance
Control	35.54 ± 5.71	-	-
LC	28.67 ± 6.78	0.0872	ns
LCC	21.11 ± 5.47	0.0010	***
LCM	21.79 ± 9.76	0.0014	**
LCCM	24.64 ± 4.73	0.0081	**

Pairwise comparisons with the control group were conducted using independent-sample t-tests. Data are expressed as mean ± SD. Significance levels: **: $p \leq 0.01$, ***: $p \leq 0.001$, ns: non-significant difference.

4. Discussion

4.1. Fatty Acid

The fatty acid composition achieved by GC–FID revealed an unequivocal ratio between SFA: MUFA: PUFA (18.92:80.19:0.89), showing the predominance of a lipid source in monounsaturated fatty acids (MUFAs), mainly oleic acid (C18:1n9). This compositional profile is similar to that of high-oleic plant oils, such as olive oil, which are known for their cardiometabolic protection, oxidative stability, and functional nutritional properties. The enrichment in oleic acid (~71%) highlighted the monounsaturated-rich nature of the sample, which is similar to dietary lipids that are known to be beneficial for reducing LDL cholesterol levels and increasing those of HDL and anti-inflammatory responses, as well as decreasing visceral fat accumulation and central obesity via the AMP-activated protein kinase signaling pathway and oleoyl ethanolamide synthesis [65-67]. Among the different SFAs, palmitic acid (C 16:0, 5.57%) and stearic acid (C18:0, 4.37%) were the predominant fatty acids. High palmitic acid intake has been associated with dyslipidemia and insulin resistance; however, this amount in the profile may not be harmful to health. In contrast, stearic acid is neutral in metabolism. It may be beneficial for lipid profiles in some instances through reduced low-density lipoprotein cholesterol levels [68, 69]. Other SFA types also appeared among the top ten, specifically behenic acid (C22:0, 5.39%), arachidic acid (C20:0, 2.47%), and lignoceric acid (C24:0, 1.01%), in lower amounts but hypothesized to function in membrane fluidity regulation or energy metabolism. The predominant fatty acid classes were jointly made up by MUFAs, with oleic acid being the most abundant (compositional characteristic of MUFA, 9.95%), followed by substantial proportions of vaccenic acid (C18:1n7, 5.12%) and less palmitoleic acid (C16:1n7) (1.29%). Increasingly appreciated for their insulin-sensitizing and anti-inflammatory properties, even low doses of these fatty acids may enhance the performance parameters of this fat exploiter [70]. Conversely, PUFA levels were low (trace), comprising linoleic acid (C18:2n6; 0.64%) and α-linolenic acid (C18:3n3; 0.25%). Although PUFAs comprise only a small proportion of the total lipid pool in cells, their importance is pronounced because they contribute to membrane fluidity and serve as precursors for bioactive lipid mediators [70, 71]. However, their extensive oxidative stability and low rancidity during storage and processing are attributed to their low lipid contents. Such compositional attributes may suggest possible applications as food and nutraceutical constituents, which require durability and high levels of lipid peroxidase resistance [71]. However, low omega-3 FA levels also indicate that additional dietary supplementation with a source rich in PUFA (e.g., canola and camelina seeds or soybean oil) is needed to meet the essential fatty acid requirements [72, 73]. The grouping of both high MUFA and moderate SFA with PUFA stands out as proprietary, since it provides an antagonistic synergism or co-benefit (health-oriented [cardio-protective, per nutrition but], industrially stability-oriented). Moreover, this combination can be useful for other healthy dietary fats, favoring the promotion of functional foods or supplements, as oxidative stability is a key hindrance to developing such properties. Other investigations addressing in vivo bioavailability and the effects of such an FA profile, focusing on the potential effects on inflammatory cytokines, oxidative biomarkers, or lipid metabolism pathways, should also be conducted.

4.2. Bioactive Compounds

GC–MS analysis of the sample identified a series of volatile bioactive compounds (terpenes, phenolics, and aromatic aldehydes) known to exhibit antioxidant, antimicrobial, and anti-inflammatory activities. Cinnamaldehyde, a phenylpropanoid aldehyde agent, has been reported to exhibit antimicrobial, antioxidant, and anti-inflammatory activities among the biogenic compounds determined in this study [74]. Cinnamaldehyde also enhanced glucose metabolism, blood vessel function, and immune modulation, which are important for metabolic homeostasis and disease protection [75-77]. One of the most abundant compounds was 1,8-cineole (eucalyptol), a cyclic ether monoterpenoid with bronchodilator and anti-inflammatory properties. The content is interesting because 1,8-cineole has been linked to enhanced respiratory performance and pulmonary function, greater oxygen transfer, and less oxidative stress (reducing factors that increase physiological reserve [78-80]. Linalool was also classified as an important acid-based behavior in the profile behavior section by Patch Array analysis. Linalool is a terpene alcohol with high antioxidant potential, analgesic, and neuroprotective effects. In addition, the gene is involved in stress tolerance, immune response, and a pathway linking tissue damage by inflammatory mediators to adaptive mechanisms, which is under investigation [81-83]. Trans-caryophyllene (β-caryophyllene) is a sesquiterpene that acts as a selective CB2 cannabinoid receptor agonist, conferring widespread anti-inflammatory and analgesic properties. Accordingly, this molecule can be classified as a potential regulator of the immune response and a preventive agent against chronic inflammatory diseases. Analysis of the clinical material identified competent eugenol, an anti-inflammatory and antimicrobial agent. It reduces inflammation and lipid peroxidation and increases cellular resistance to oxidative stress [84, 85]. Cinnamyl acetate was also present in moderate proportions. The values of this cinnamyl alcohol ester, which are appreciated, include its odor and biological activities, such as antimicrobial and antioxidant effects, which may help improve the overall functional characteristics of the diet [86-88]. Overall, the chemical constituents suggest that the prepared sample contains several terpenoids and phenylpropanoids, including cinnamaldehyde, linalool, 1,8-cineole, eugenol, and caryophyllene. They are thought to play major roles in the formulation of antioxidant, antimicrobial, and anti-inflammatory activities and have potential applications in health, wellness, and functional product development.

4.3. Effect of Different Dietary Treatments on Body Weights

In this study, the LCCM group showed the greatest relative reduction in body weight among all the treatment groups. The LCC and LCM groups showed the second- and third-greatest reductions, respectively. These results suggest a synergistic therapeutic effect between moringa and cinnamon oils, with combined administration resulting in a higher metabolic rate and energy expenditure than either extract alone. The weight loss trends observed in the LCCM group were consistent with those of previous studies [81]. Kilany *et al* reported a weight reduction effect of moringa seed oil on body weight and fat deposition in obese rats due to the modulation of lipid metabolism and hepatic enzyme activity [89]. Adiga [90] reported that *Cinnamomum zeylanicum* significantly promoted body weight gain and improved insulin sensitivity in rats fed a high diet. Collectively, these results suggest that oleic acid from moringa and flavonoids, cinnamaldehyde, and eugenol from cinnamon promote lipid oxidation, thermogenesis, and metabolic control. The trend of bodyweight loss observed in the LC group, although less pronounced than that in the supplemented groups, also seems to corroborate the published evidence. These studies have shown that ketone body production and fat metabolism are significantly enhanced when carbohydrate intake is insufficient. While the metabolic impact of LC enhances body fat metabolism, supplementation with moringa and cinnamon oligomers appears to increase the rate of fat metabolism relative to the former group [91]. In this study, the synergistic effect of the two extracts likely had a greater impact on insulin signaling, antioxidant enzyme activity, and lipid expression. Therefore, it is clear that the body reductions observed in these groups of animals can be attributed to high rates of upregulation. The key difference between this study and existing studies is the best key experience, which occurred in the seventh week. These results concur with those of Ezzat *et al.* [92], suggesting that the lipid-lowering and anti-obesity effects of moringa and cinnamon are delayed and take time to become apparent. These findings suggest that bioactive oils remain at

appropriate levels for an extended period in the lipid metabolism pathway. In conclusion, these results show that each of the coma die groups has significant anti-obesity effects and holds great potential for clinical application.

4.4. Lee Index

Body weight changes varied among the treatment groups, with the LC diet supplemented with Moringa seed and cinnamon bark oil producing the highest reduction. The LC diet with cinnamon bark oil and the LC diet with moringa seed oil groups also showed considerable declines relative to baseline. A plausible explanation for these results is that the effects of moringa seed and cinnamon bark oils are synergistic; therefore, combined administration results in greater fat metabolism and energy expenditure than when either oil is administered alone. Various studies support the findings of the LCCM group's reduced weights. Kilany *et al.* [89] revealed that moringa seed oil reduced fat and body weight in obese rats by regulating lipid metabolism and hepatic activity.

Adiga [90] reported that *Cinnamomum zeylanicum* bark oil considerably inhibited weight gain and increased insulin sensitivity in rats fed a high-fat diet. Thus, their results agree that the bioactive contents in these plant extracts, eugenol, cinnamaldehyde, flavonoids, and other phytochemicals, play a critical role in lipid oxidation thermogenesis and the regulation of feeding behavior. The results of the LC diet are consistent with those of previous studies, as carbohydrate restriction enhances ketone production and fat oxidation. Hence, in agreement with Volek *et al.* [91], LC diets induce a metabolic switch that enhances fatty acid oxidation, leading to greater fat loss and weight loss. In the present study, moringa and cinnamon oils considerably strengthened these effects. Discrepancies observed after half the experimental duration in the intervention results might be due to the multiple effects of phytol on lipid turnover regulation, antioxidant defense system, and insulin signaling. The difference from the other groups is due to the influence of their supplements; therefore, the reduced effect of cinnamon and moringa can be interpreted as a lack of influence.

The immersion loss in the current study is also consistent with the results of Ezzat *et al*. [92], who found that the effects of moringa on lipids and anti-obesity become stronger with increased administration time. Generally, the results indicate that LC intake, especially when supplemented with moringa seed and cinnamon bark-extracted oils, is associated with reduced obesity, increased lipid utilization, reduced storage, and improved metabolic activity in rats. Hence, the joint use of these natural extracts with a carbohydrate-limited diet appears promising as a nutritional approach to obesity and related metabolic problems.

4.5. Effects of Different Dietary Treatments on Lipid Profile

The 12-week LC diet in obese albino rats produced significant changes in lipid regulation, including TC (–20.4 ± 8.6 mg/dL) and LDL cholesterol (LDL-C) (–8.5 ± 4.4 mg/dL) levels. The hypolipidemic effects are also in accordance with the findings in the literature, indicating that LC diets lower both LDL-C and TC levels in individuals, particularly those from the obesity/type 2 diabetes population groups [93, 94]. In line with this, systematic reviews have also shown better outcomes of those on local coverage determinations (LCDs) on serum lipid markers than low-fat dietary interventions. However, the extent of these effects may vary according to the dietary composition and baseline metabolic profile [94]. Therefore, the cholesterol reduction noted here is consistent with the literature on improved lipid profiles upon carbohydrate deprivation.

However, an important distinction lies in the response of HDL cholesterol (HDL-C) and triglyceride levels to exercise. While this study did find a substantial decrease in HDL cholesterol (−12.1 ± 7.1 mg/dL) but an increase in triglycerides (+12.1 ± 13.6 mg/dL) and VLDL (+2.3 ± 2.8 mg/dL), the preponderance of clinical evidence is still weighted toward higher HDL levels and lower triglyceride levels with LC diets [94-96]. Despite the higher prevalence of type 2 diabetes and obesity, LCDs increased HDL-C levels while decreasing TG levels, even among patients with diabetes and obesity patients. In contrast, our results indicate that these lipid parameters tend to exhibit dyslipidemic profiles. Studies in animal models have similarly indicated that LC and high-fat diets increase dyslipidemia but improve it with the addition of fish oil [97]. These differences could be attributable to species-specific variations in lipid metabolism, the animals' obesogenic nature, and/or the

macronutrient composition of their diet, which may have reduced the HDL-raising and TG-lowering effects of the test ingredients.

Taken together, these findings provide evidence that carbohydrate-restricted diets may be potent inhibitors of serum TC and LDL-C levels in obese albino rats, but have a deleterious impact on HDL-C and TG levels. The response to carbohydrate restriction is more heterogeneous than previously reported. This indicates that dietary context and physiological state are both important, modifiable factors that determine whether a response to an intervention that modifies carbohydrate consumption is beneficial or dyslipidemic.

Treatment with the herb cinnamon for the rats fed on an LC diet (–25.1±9.2 mg/dL) led to a significant suppression of the serum total cholesterol level during this study; conversely, a comparable lowering effect on blood lipid levels has been reported in other animal model studies subjected to the administration of herbs containing high polyphenol content, including cinnamon [35]. Cinnamon extracts have also been reported to reduce serum cholesterol levels in dyslipidemic or high-fat diet-fed rats [25, 98-100]. Furthermore, the antihyperlipidemic potential of cinnamon has been substantiated by meta-analyses of randomized controlled trials that show small yet significant reductions in total cholesterol levels across populations [101]. This agreement supports the potent cholesterol-modulatory effect of cinnamon, irrespective of diet.

Regarding triglycerides and LDL-C, the results were not confirmatory, displaying minor or conflicting effects of cinnamon in humans [102, 103]. Reduction of triglycerides and LDL. Some studies have reported declines in triglycerides and LDL levels [25, 102], whereaswhile others, including systematic reviews, have found no significant effects [101, 102]. Hence, the neutral findings in our analyses are indeed more consistent with recent interpretations of the effects of cinnamon on these lipids, which are dependent on dose, treatment duration, and endogenous metabolic states.

A notable difference is the reaction of HDL cholesterol. Although we found a significant decrease in serum HDL levels (–19.6 ± 6.2 mg/dL), most other studies reported either increased or unchanged HDL levels after cinnamon intervention. Several studies have reported that HDL levels increase with a concomitant decrease in LDL and triglyceride levels in dyslipidemia and high-fat diet-fed animal models [25, 99, 100, 104]. Similarly, clinical studies have mostly observed neutral or beneficial effects of cinnamon on HDL levels [101, 104]. The different dietary backgrounds of our study, in which cinnamon was combined with carbohydrate restriction, may explain this discrepancy in results. The LC diet alters lipid metabolism and may have masked or overcome the normal HDL-promoting effects of cinnamon's bioactive substances. In addition, a discrepancy in baseline health, as measured using healthy albino rats versus dyslipidemic or diabetic models used in other studies, could be another reason for the differences in the HDL response.

The current study results are consistent with the overall literature, in which total cholesterol is reduced, and TG and LDL show some sensitivity to the response. In contrast, the reduction in HDL cholesterol levels was robust. These differences emphasize the importance of considering the context of diet, metabolism, and species when evaluating the lipid -regulatory effects of cinnamon. This interaction needs to be studied in conjunction with the macronutrient profile and cinnamon supplementation to explain this discrepancy.

In this study, , the seed oil of moringa had the highest lipid-lowering effect as a supplement to an LC diet in rats. Rats supplemented with moringa seed oil showed significantly lower total cholesterol (30.1 ± 17.4 mg/dL), triglycerides (6.8 ± 24.6 mg/dL), and VLDL cholesterol (−1.4 ± 4.6 mg/dL) than the control group rats, which is consistent with several animal and clinical trials that demonstrated the strong hypolipidemic effects of moringa seed oil and its extracts. Rodent and zebrafish models show a substantial decrease in serum TGs and TC levels after supplementation [104], whereas human studies that supplement functional foods with moringa seed show lower levels of TC, LDL-C, and TGs [105]. In addition, studies in hyperlipidemic and diabetic rats have shown the potential of moringa to improve cholesterol metabolism and reduce tissue lipid deposition, with efficacy comparable to that of atorvastatin [106]. These findings highlight the remarkable hypocholesterolemic potential of moringa, owing to its high concentrations of bioactive components, including oleic acid, phytosterols, and

polyphenols, which improve cholesterol homeostasis and inhibit intestinal cholesterol absorption [107, 108].

An important difference between the current and previously published results lies in the impact ofn HDL cholesterol. In the present study, HDL levels were lower (–18.4 ± 10.4 mg/dL) after the consumption of moringa seed oil. However, most earlier studies reported that HDL levels in participants treated with moringa were higher than those in the control group. For example, Kareem *et al.* [109] observed increased HDL levels in male rats. In addition, HDL elevation in ovariectomized rats has been reported by Kusolrat and Kupittayanant [110]. Simultaneously, moringa supplementation in the diets of hyperlipidemic patients increased HDL and reduced atherogenic lipid levels [105]. This clear contrast may be due to differences in the nutritional backgrounds. This study employed moringa, which, when ingested with a carbohydrate-restricted diet, individually modifies lipid metabolism and can therefore mask or attenuate HDL upregulation. Additionally, it used obese albino rats instead of probes such as hyperlipidemic or diabetic animals, which are frequently used in other studies and may have caused the contrasting effect on HDL levels.

The simultaneous administration of the LC diet with cinnamon bark and moringa seed oil in obese albino rats from the present study induced modest changes in lipid metabolism, represented by a significant decrease in total cholesterol (−17.1 ± 7.5 mg/dL) coupled with reduced amounts of HDL cholesterol (−15.2 ± 7.9 mg/dL). These results partly match those reported in the literature, as it is well documented that the two substances alone (cinnamon and moringa) show hypolipidemic activity, that is, they decrease total cholesterol, LDL, and triglyceride levels [105]. In fact, human dietary interventions with moringa supplementation have shown significant reductions in TC, LDL, and TG levels. In contrast, cinnamon bark oil has been reported to reduce serum cholesterol and triglyceride levels in both preclinical and clinical studies [107, 111]. The cholesterol-lowering effects observed in the present study are, in that case, fully consistent with previous evidence on their regulation of lipid metabolism.

This study reported a significant decrease in HDL levels, which contrasts with most of other studies that reported either maintenance or elevation of serum HDL levels following moringa supplementation in experimental animals and human patients [105, 109]. Similarly, polyherbal formulations containing cinnamon and other plant materials mainly increased HDL levels alongside reductions in LDL, VLDL, and triglyceride levels [112, 113]. This This difference can be explained, at least in part, by the dietary background employed in our study: carbohydrate restriction, as a component of the diet, was used to induce obesity in rats, which may have interacted with phytochemicals to negate or reverse thean anticipated elevation in HDL. Although some studies have highlighted the potential synergistic effects of moringa and cinnamon in combination [113], our results did not show any additive or synergistic effects beyond those observed for each extract alone. Rather, the results support common mechanisms of action, including antioxidant effects and modulation of lipid absorption [108], which may have left little room for increased lipid-lowering ability when the two were combined.

The findings of this study largely agree with those of published studies and confirm the lipid-lowering effects of cinnamon and moringa extracts; however, they differ in two key aspects: HDL cholesterol was reduced rather than increased, and no synergistic benefit was observed when both extracts were combined. These findings underscore the role of dietary and metabolic contexts in mediating the effects of phytochemicals on lipid metabolism and identify an area for further research into how macronutrient background/obesity status alters interactions among herbs.

5. Conclusions

The current study was conducted to evaluate the impact of moringa and cinnamon bark oils, when supplemented with an LC diet, on the lipid profiles of obese albino rats. Although the diet alone modestly improved lipid markers, the addition of these bioactive oils resulted in significant increases in total cholesterol, LDL-C, VLDL-C, and triglyceride levels. Cholesterol levels decreased more with LCM than with LCC treatment. In contrast, the LC diet resulted in a larger decrease in LDL-C with the LC diet. HDL-C levels decreased after the LC diet with cinnamon; however, they remained higher than

those in the other intervention groups. The general trend is that adding plant oils to LC diets shifts the lipid profile toward a less atherogenic pattern.

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