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Assessing of The Protective Effect of Gum Arabic And L-Carnitine on Diet Induced Obesity in Rats

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Abstract This experimental study set out to investigate the protective effects of L-carnitine and gum Arabic supplementations, either alone or in combination, against High Fat Diet (HFD) obesity-related complications in male Wistar rats. A total of 48 three-month-old rats weighing 160–180g were randomly assigned to six groups. The control group was given standard diet and water, while the other groups (T1– T°) were fed with HFD with 30% animal fat for 60 days to induce hyperlipidemia and T5 maintained on a standard diet. After the lipid elevation was confirmed, cure regimens were applied for 30 days: T2=f 600 mg/kg GA, T3= f 250 mg/kg L- Carnitine, T4=Both agents, and T5=Both agents were administered alongside a standard diet.The rats of the untreated HFD group (T1) showed statistically significant higher body weight, serum lipids (total cholesterol (TC), TGs, LDL, VLDL), liver and renal function tests (AST, ALT, urea, creatinine) and atherogenic index, with reduction in HDL, in comparison with that in the control group. On the other hand, the treatment groups, especially T4 and T5 showed obvious ameliorations of lipid metabolism, organ function and body weight regulating. These findings indicate promising role for L-carnitine and gum Arabic against the metabolic disorders associated with obesity. Combined treatment provided better results, which suggested a synergic effect in treatment of diet-induced obesity.

Keywords: hyperlipidemia, obesity, gum Arabic, L-carnitine

Introduction

Obesity has emerged as a major global health concern, recognized as a non-communicable epidemic with widespread prevalence across diverse populations (1). It represents a significant burden on both public health systems and economic structures, prompting its prioritization in international health agendas (2,3). Numerous metabolic processes, alongside a complex interplay of environmental and genetic components, have been implicated in the development of obesity. Research has identified over a thousand genetic markers associated with increased susceptibility to obesity; however, lifestyle factors, particularly physical activity, may play a role in offsetting the influence of these genetic predispositions (4,5).The condition poses a considerable risk for the onset of various comorbidities, including type 2 diabetes, metabolic-associated fatty liver disease (formerly nonalcoholic fatty liver disease NAFLD), cardiovascular complications, and other disorders such as obstructive sleep apnea and osteoporosis (6).L-carnitine is an endogenous molecule synthesized from the amino acids lysine and methionine, with liver and kidney being the major synthesis sites, it is involved in lipid metabolism as it permits the entry of long-chain fatty acids into the mitochondria for degradation by β-oxidation and the generation of ATP, while the body can produce L-carnitine endogenously, dietary intake accounts for the majority of its availability (9,10). L-carnitine has antioxidant properties and can function as a scavenger of reactive oxidation products that could explain some of the beneficial cardiovascular effects (7,8). Even though most investigators consider it safe to be used as a beverage, the influence of coffee consumption on cardiovascular risk is still a matter of debate, with some studies suggesting its potential cardioprotective effect (11–13). In addition, L-carnitine supplementation has proven to beneficial for weight management and metabolic health when used either alone or in combination with exercise, resulting in decreased blood pressure and body fat percentage in obese individuals (14,15).Gum Arabic (GA) is a natural polysaccharide exudate extracted primarily from Acacia

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senegal and Acacia seyal and it acts as a protector in nature against adverse environmental effects including drought, insects and microbial threats (16). It is a water-soluble dietary fiber made up of non-viscous carbohydrates, such as arabinose and ribose, and is mainly produced in the acacia tree and is mainly grown in African countries, including Sudan, Chad, and Nigeria. GA is commonly used in food industry as a stabilizer, in pharmaceutical industry as an emulsifier and therapeutic**ally** in chronic renal failure and digestive system dysfunctions, its wide pharmacological profile including anti-obesity, antihypertensive, antihyperlipidemic, anticoagulant, antimicrobial, antidiabetic and anti-inflammatory activities has been recently highlighted in the literature (17). Additionally, the physiological roles of GA include its antioxidant capabilities, immune modulation, hypoglycemic action, and anti-gastric ulcer. Regular consumption was also found to significantly decrease plasma lipid concentrations (18–21).The present research explores the separate and synergistic roles of L-carnitine and gum Arabic in modulating obesity-related physiological outcomes in a rat model induced by a high-fat dietary regimen.

Materials and Methods

Ethical Approval

All experiment in the animals was performed in accordance with the College of Education, University of Al-Qadisiyah Institutional animal ethics Committee guidelines. The research protocol was approved under Ref: 24-19/1/2025.

Experimental Animals:

Male albino rats weighing160-180 g and aged three months were employed in the present investigation. The Animals **rats** were purchased from the animal farm of **the** College of Veterinary Medicine/University of AL-Qadisiyah. All experimental protocols were performed in the animal house of the Biology Department, College of Education of the same university.

3. Experimental Design:

The experimental protocol was divided into two major phases:

First stage: Animals **Rats** were randomized to two experimental groups. The control (Group 1) received a standard chow diet and drinking water throughout the study period. The second group received hyperlipidemic diet with 30% animal fat for 60 days so as to produce obesity related metabolic disturbances.

Second stage: This phase commenced after the 60-day hyperlipidemia induction period. Treatment with gum Arabic and L-carnitine continued for an additional 30 days across the respective groups (T2, T3, T4, and T5). The high-fat feeding was maintained during this treatment phase for the relevant groups. The animals were then redistributed into six groups (n = 8 rats per group) as follows:

The animals were allocated into six groups as follows:

- Control Group (C): Maintained on a standard diet with free access to water throughout the 90-day experimental period.
- Group T1 (Positive Control): Received a high-fat diet continuously for 90 days without any form of therapeutic treatment.
- Group T2: Subjected to a high-fat diet for 90 days and treated orally with gum Arabic at a dose of 600 mg/kg body weight daily during the final 30 days.
- Group T3: Fed a high-fat diet for the entire duration, with L-carnitine administered intraperitoneally (250 mg/kg body weight) during the last 30 days.
- Group T4: Received both gum Arabic (oral, 600 mg/kg) and L-carnitine (intraperitoneal, 250 mg/kg) during the final 30 days of a 90-day high-fat diet regimen.
- Group T5: Maintained on a standard diet throughout the study and co-treated with gum Arabic and L-carnitine at the aforementioned doses during the final 30 days.

This experimental setup facilitated the assessment of both individual and combined therapeutic strategies under standard and high-fat dietary conditions, allowing for a clearer understanding of the protective roles and potential synergistic interactions between gum Arabic and L-carnitine in counteracting obesity-related metabolic impairments.

L-Carnitine Source and Dosage Calculation:

L-carnitine was obtained from pharmacies in Al-Diwaniyah province, supplied by Premier Health Products Ltd (UK) in capsule form, with each package containing 30 capsules of 1000 mg each. Gum Arabic was purchased from the local market and ground into a fine powder using an electric grinder. Once both materials were prepared, their dosages were calculated and administered to the experimental animals accordingly.

The dosage was determined using the following formula:Dose per animal = (Animal weight (g) × Substance concentration (mg)) / 1000 (g)

Each rat was injected with ½ ml of L-carnitine and orally administered 1 ml of gum Arabic daily for one month.

Measured Parameters:

The studied parameters included body weight gain, total cholesterol, triglycerides, high-density lipoproteins (HDL), low-density lipoproteins (LDL), very low-density lipoproteins (VLDL), atherogenic index, liver enzymes (AST and ALT), and kidney function indicators (creatinine and urea).

Body Weight Gain:

Weight gain (g) = Final weight (g) − Initial weight (g)

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Relative Organ Weights:
Relative organ weight (%) = (Organ weight (g) / Body weight (g)) × 100

Lipid Profile Estimation:

Total Cholesterol Determination (22):
Measured using enzymatic kits from BioMérieux (France, 69280 IE toile) based on enzymatic transformation into quinoneimine.
- Reagents: phosphate buffer (90 mmol/L, pH 6.9), phenol (26 mmol/L), cholesterol esterase (300 U/L), cholesterol oxidase (200 U/L), peroxidase (1250 U/L), and 4-aminoantipyrine (0.4 mmol/L).
- Standard: 200 mg/100 ml.
- Procedure: Incubation at 37°C for 5 minutes, absorbance at 546 nm.
- Calculation:
Cholesterol (mg/dL) = ((A_test - A_blank) / (A_standard - A_blank)) × 200
Cholesterol (mmol/L) = Cholesterol (mg/dL) × 0.025

Triglyceride Determination (23):
Measured enzymatically using Biomerieux kits with colorimetric detection.
- Key enzymes: Lipase, glycerokinase, glycerol-3-phosphate oxidase, peroxidase, 4-aminoantipyrine.
- Calculation:
Triglycerides (mg/dL) = ((A_test - A_blank) / (A_standard - A_blank)) × 200
Triglycerides (mmol/L) = Triglycerides (mg/dL) × 0.0133

HDL-C Measurement (24):
Using Randox kits and precipitation with phosphotungstic acid and magnesium chloride.
- Calculation:
HDL-C (mg/dL) = (A_test - A_blank) × 280
HDL-C (mmol/L) = HDL-C (mg/dL) / 38.7

LDL-C Estimation (25):
LDL-C (mg/dL) = Total Cholesterol − (HDL-C + VLDL-C)

VLDL-C Estimation (22):
VLDL-C (mg/dL) = Triglycerides / 5

Atherogenic Index (26):
Atherogenic Index = Total Cholesterol / HDL-C

Liver Enzymes AST, ALT (27): Determined using colorimetric methods with Biosystem kits.
- AST (IU/L) = ((A_test − A_control) × 133) / (A_standard − A_blank)
- ALT (IU/L) = ((A_test − A_control) × 67) / (A_standard − A_blank)

Kidney Function Tests:

Urea Concentration (28):
Urea (mg/dL) = (Sample Absorbance / Standard Absorbance) × Standard Concentration

Creatinine Concentration (29):
Creatinine (mg/dL) = (Test Absorbance / Standard Absorbance) × 2

Results

Body Weight

As shown in Table (1), rats in the T1 group, which received a 30% fat diet for 45 days, exhibited a statistically significant increase in body weight gain compared to the control group (P<0.05). There was no significant variation between T1 and T2 (P>0.05). In contrast, group T3 showed a marked reduction in weight gain relative to both T1 and T2 (P<0.05). Additionally, animals in groups T4 and T5 experienced significantly less weight gain than those in T1, T2, and T3 (P<0.05), with no significant difference between T4 and T5 themselves (P>0.05). Despite this, all treated groups recorded significantly greater weight gain compared to the control group (P<0.05).

For liver weight expressed as a percentage of body weight, significant decreases (P<0.05) were recorded in groups T1, T2, and T3 when compared to the control. Conversely, group T5 exhibited a notable increase in liver weight percentage compared to T1, T2, and T3 (P<0.05), although its values did not differ significantly (P>0.05) from the control or T4. As for kidney weight percentage, all treated groups had significantly reduced values in comparison with the control group (P<0.05), yet no meaningful differences were observed among the treatment groups themselves (P>0.05), indicating a consistent renal response across treatments.

Table 1. Impact of Gum Arabic and L-Carnitine on Weight Gain and Organ-to-Body Weight Ratios in Hyperlipidemic Male Albino Rats

Group	Weight Gain (g)	Liver Weight (%)	Kidney Weight (%)
Control	2.8 ± 0.56^{d}	10.55 ± 0.39^{a}	1.30 ± 0.03^{a}
T1	52.5 ± 2.45^{a}	8.37 ± 0.67^{c}	0.975 ± 0.06^{b}
T2	48.7 ± 2.76^{a}	8.69 ± 0.59^{bc}	0.959 ± 0.10^{b}
T3	36.3 ± 1.78^{b}	8.67 ± 0.49^{bc}	0.888 ± 0.04^{b}
T4	27.5 ± 1.45^{c}	10.11 ± 0.06^{ab}	0.844 ± 0.01^{b}
T5	3.72 ± 0.68^{d}	10.47 ± 0.43^{a}	0.935 ± 0.09^{b}
LSD	5.91	1.48	0.205

Results are shown as mean ± SE. Letters in the same column indicate significant differences where P<0.05.

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Blood Lipid Profile

Table (2) presents the effects of dietary interventions on serum lipid parameters. Rats in group T1, which were maintained on a high-fat diet containing 30% animal fat for 60 days, exhibited significantly elevated levels of total cholesterol, triglycerides, LDL, and VLDL compared to the control group (P<0.05). Conversely, HDL levels were significantly reduced in this group (P<0.05).A comparison between groups T1 and T2 revealed no significant differences in total cholesterol, triglycerides, LDL, or VLDL concentrations (P>0.05), indicating that gum Arabic alone had minimal effect in mitigating lipid dysregulation under the study conditions. However, group T3, treated with L-carnitine, showed a notable decline in these lipid markers compared to both T1 and T2 (P<0.05).The most pronounced improvement was observed in groups T4 and T5, where combined administration of gum Arabic and L-carnitine resulted in significant reductions (P<0.05) in cholesterol, triglycerides, LDL, and VLDL levels, approaching values seen in the control group.As for HDL, levels were significantly increased (P<0.05) in T3, T4, and T5 compared to T1 and T2. There were no statistical differences (P>0.05) between T4, T5, and the control group, suggesting that HDL concentrations were effectively restored to near-normal levels following the combined treatment.

Table 2. Impact of Gum Arabic and L-Carnitine Supplementation on Serum Lipid Parameters in Hyperlipidemic Male Rats

Group	Cholesterol (mg/dL)	Triglycerides (mg/dL)	HDL (mg/dL)	LDL (mg/dL)	VLDL (mg/dL)
Control	58.16 ± 2.94^{d}	63.5 ± 2.77^{d}	33.3 ± 2.16^{a}	16.26 ± 1.80^{d}	12.7 ± 0.52^{c}
T1	171.0 ± 7.90^{a}	182.1 ± 9.88^{a}	12.7 ± 0.88^{c}	124.6 ± 9.43^{a}	36.4 ± 1.97^{a}
T2	160.2 ± 8.15^{a}	176.7 ± 6.72^{a}	13.9 ± 1.07^{c}	111.5 ± 5.33^{a}	35.3 ± 1.34^{a}
T3	116.5 ± 6.32^{b}	131.7 ± 5.96^{b}	23.3 ± 1.41^{b}	72.0 ± 3.33^{b}	26.3 ± 1.19^{b}
T4	80.3 ± 3.57^{c}	86.7 ± 2.95^{c}	30.6 ± 1.98^{a}	33.1 ± 2.58^{c}	17.3 ± 0.59^{c}
T5	62.4 ± 2.83^{d}	67.7 ± 3.36^{d}	32.3 ± 1.55^{a}	20.4 ± 1.71^{d}	13.5 ± 0.67^{c}
LSD	16.01	19.46	5.73	19.28	3.97

Results are shown as mean ± SE. Letters in the same column indicate significant differences where P<0.05.

Atherogenic Index

The findings displayed in Table (3) reveal a clear elevation in the atherogenic index among rats in group T1, which had been maintained on a high-fat diet (30%) for 60 days. This increase was statistically significant (P<0.05) when compared with the control group and is indicative of an increased tendency toward atherosclerotic development, likely driven by disruptions in lipid metabolism. Comparison between groups T1 and T2 showed no meaningful difference (P>0.05), suggesting that gum Arabic alone had minimal effect on moderating this cardiovascular risk indicator. In contrast, administration of L-carnitine in group T3 led to a marked reduction (P<0.05) in the atherogenic index relative to both T1 and T2.Groups T4 and T5, which received the combined intervention of L-carnitine and gum Arabic, exhibited the most substantial improvements. Their atherogenic index values were significantly lower (P<0.05) than those in all other groups and were comparable to the control group.These observations imply that the dual application of L-carnitine and gum Arabic may act synergistically to counteract lipid-related cardiovascular risks in the context of diet-induced hyperlipidemia.

Table 3. Influence of Gum Arabic and L-Carnitine on the Atherogenic Index in Male Rats Subjected to Diet-Induced Hyperlipidemia

Group	Atherogenic Index (Cholesterol / HDL)
Control	1.77 ± 0.06^{d}
T1	13.47 ± 0.92^{a}
T2	11.63 ± 0.84^{a}
T3	5.01 ± 0.46^{b}
T4	2.63 ± 0.21^{c}
T5	1.94 ± 0.08^{d}
LSD	1.33

Results are shown as mean ± SE. Letters in the same column indicate significant differences where P<0.05.

Liver Enzymes (AST and ALT)

Table (4) showed a significant increase in the hepatic enzymes (AST&ALT) in the group T down to 30% high fat diet for 60 days. This was an abnormal and statistically significant elevation **(P<0.05)**, similar to indicate that gum Arabic alone had no significant differential modulating effect on liver enzyme pattern under the present experimental condition. However, the L-Carnitine treated group (T3) had significant decrease (P<0.05) in both enzymes when compared with T1 and T2.The largest decreases in AST and ALT were recorded in groups T4 and T5 (P<0.05), where the supplementation with a mixture of L-carnitine and gum Arabic reduced the

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AST and ALT levels significantly. The enzyme activities in these groups were close to those in the control groups, which suggested that the damage of liver could be partially reduced by HA.These data suggest that gum arabic and L-carnitine may have a potential synergistic protective effect on hyperlidemia-induced liver function in high fat-fed rats.

Table 4. Impact of Gum Arabic and L-Carnitine on AST and ALT Activities in Hyperlipidemic Male Rats

Group	AST (IU/L)	ALT (IU/L)
Control	47.4 ± 2.36^{d}	31.3 ± 2.02^{d}
T1	123.5 ± 5.44^{a}	99.2 ± 3.75^{a}
T2	119.4 ± 6.89^{a}	93.4 ± 4.17^{a}
T3	90.5 ± 3.57^{b}	64.2 ± 2.48^{b}
T4	58.1 ± 2.42^{c}	39.6 ± 1.96^{c}
T5	49.5 ± 2.63^{cd}	35.1 ± 1.37^{cd}
LSD	12.12	9.06

Results are shown as mean ± SE. Letters in the same column indicate significant differences where P<0.05.

Kidney Function (Creatinine and Urea)

The results of Table (5) indicated that serum creatinine and urea were significantly increased in the T1 group after 60 days of consumption of the high fat diet. These increases were significantly different (P<0.05) were recorded between T1 and T2 for both biomarkers, this might indicate that gum Arabic alone was insufficient to ameliorate the renal effect of hyperlipidemia. However, creatinine and urea concentrations were significantly less in rats of group T3, which received L-carnitine injection, in comparison with those of T1 and T2 (P<0.05).

Group T4 and T5 showed the most significant changes and concurrent treatment with L-carnitine and gum Arabic (250 mg/kg plus 2 % w/v) resulted in significant decrease in the markers (P<0.05); nearly to normal control levels.

The results of this work emphasize the significant renoprotective effects of L-carnitine as well as gum Arabic, especially when given in combination.

Table 5. Impact of Gum Arabic and L-Carnitine on Serum Creatinine and Urea Levels in Hyperlipidemic Male Rats

Group	Creatinine (mg/dL)	Urea (mg/dL)
Control	0.654 ± 0.031^{cd}	24.1 ± 1.57^{cd}
T1	1.63 ± 0.12^{a}	55.6 ± 2.94^{a}
T2	1.59 ± 0.09^{a}	53.2 ± 2.22^{a}
T3	1.17 ± 0.08^{b}	43.4 ± 1.69^{b}
T4	0.843 ± 0.06^{c}	29.8 ± 1.28^{c}
T5	0.679 ± 0.04^{cd}	26.4 ± 1.15^{cd}
LSD	0.305	6.63

Results are shown as mean ± SE. Letters in the same column indicate significant differences where P<0.05.

Discussion

Body Weight

The present study revealed a notable rise in body weight among rats in group T1 that were maintained on a high-fat diet, in comparison with the control group. This observation aligns with earlier research reporting that increased dietary fat intake is associated with progressive body weight gain over time (30). The weight increase observed in the current experiment may be linked to the elevated fat content (30%), which likely stimulated the animals' preference for the energy-dense feed, thereby promoting greater consumption and consequent weight gain (31,32). Another study suggested that this weight increase could be due to enhanced bile secretion caused by the high-fat content, which improved digestive efficiency and nutrient absorption (33). No significant difference was observed in group T2, which received a high-fat diet (30%) along with oral administration of gum Arabic. This aligns with findings from previous studies (34,35). A study also confirmed that L-carnitine supplements did not significantly affect triglyceride levels, showing no significant differences (19).Conversely, rats in group T3, which were administered L-carnitine intraperitoneally while receiving a high-fat diet, exhibited a marked reduction in body weight gain relative to the hyperlipidemic group T1. This outcome aligns with prior research reporting significant reductions in body weight and Body Mass Index BMI among subjects supplemented with L-carnitine, reinforcing its role in weight management (36). The underlying mechanism is believed to involve L-carnitine's essential function in transporting long-chain fatty acids into mitochondria, where they undergo β-oxidation within skeletal and cardiac muscle tissues (37,38). Additional hypotheses suggest that L-carnitine contributes to weight control by enhancing insulin sensitivity, modulating appetite, and influencing hypothalamic regulation (39–41).Group T4, which received a combination of gum Arabic and L-carnitine, also demonstrated a significant decline in weight gain compared to group T1. The co-administration of both agents appears to produce a synergistic effect, surpassing the outcomes observed in groups treated with either compound individually (T2 and T3). Remarkably, group T5 - maintained on a standard diet yet given the combined treatment - exhibited the greatest reduction in body weight among all experimental groups (T1–T4).Regarding organ weights, earlier studies have indicated that the reduction in kidney mass observed in animals subjected to high-fat diets may stem from lipid-induced renal injury. Elevated lipid concentrations are known to compromise glomerular integrity by interacting

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directly with renal cells and structures, ultimately leading to nephron loss and renal atrophy (42). As for the liver, the decline in its weight may be attributed to hepatic lipid accumulation, which promotes oxidative stress through excessive free radical generation, this oxidative burden damages hepatocytes over time, resulting in cellular loss and diminished liver mass. Moreover, such oxidative stress is closely linked to the onset of liver diseases that can further contribute to reduced liver size and function (43–46).Conversely, group T4, which received both gum Arabic and L-carnitine along with a high-fat diet, showed increased relative liver weight. The synergistic effect of both compounds appears to have reduced hepatic fat, preserving liver health and size. In group T5, which received the same treatments alongside a normal diet, the increase in liver weight was even more pronounced than in T4, confirming that the combination treatment preserved liver mass in the absence of dietary fat stress.

Lipid Profile

In the current study, rats exposed to a high-fat diet for eight weeks exhibited elevated levels of total cholesterol, triglycerides, LDL, VLDL, and atherogenic index (AI) compared to the control group. Conversely, a significant reduction was observed in HDL concentrations. These alterations are consistent with previous reports that describe high-fat dietary regimens as potent inducers of dyslipidemia in animal models (47–49).The rise in circulating lipids may be partially attributed to oxidative stress triggered by excessive fat consumption, which promotes increased generation of reactive oxygen species (ROS) and disrupts lipid metabolism (50). Supporting this, other experimental studies have documented that high-fat feeding leads to elevated lipid markers alongside diminished HDL levels, correlating with enhanced lipid peroxidation and fat deposition within intestinal tissues. These changes have been shown to impair gastric motility and weaken the body's antioxidant defenses, thereby intensifying oxidative damage (51).Decreased plasma HDL levels seen in Regulatory T cells specific CXCR-4-Cre fa/fa animals might be due to blunted L-CAT-activity as LCAT is key enzyme responsible for HDL **elevation** and mediating cholesterol efflux from cells to HDL particles (52). Moreover, an increase in the Atherogenic Index of Plasma (AIP) indicates an impaired lipid metabolism which is characterized by an excess of atherogenic lipids such as TG and a paucity of beneficial HDL. This dysbalanced is generally associated with insulin resistant state and high intake of cholesterol diets (53).Interestingly, lipid profile parameters such as triglycerides, LDL, VLDL, and total cholesterol were found significantly reduced while HDL increased significantly after supplementation with gum Arabic (T2). Thus, the atherogenic index also decreased significantly. These results are supported by earlier reports that aqueous gum Arabic intake in the experimental rats decreases total and LDL **and** cholesterol profiles **demonstrably**. This hypocholesterolemic effect is believed to occur through the downregulation of hepatic HMGR mRNA (3-hydroxy-3-methyl-glutaryl-CoA reductase mRNA), reducing cholesterol biosynthesis (19). Another mechanism involves gum Arabic binding to bile acids in the intestine, reducing their absorption. During fermentation in the colon, acidic conditions are created, rendering bile acids insoluble and promoting their excretion in feces, which in turn reduces fat digestion and absorption (34).Group T3, treated with L-carnitine, also showed reductions in cholesterol, triglycerides, and LDL levels, resulting in a lower atherogenic index. These findings are supported by studies (54,55) indicating that L-carnitine reduces triglycerides**, and** VLDL **and** cholesterol by redirecting hepatic metabolism toward acetylcarnitine production rather than fat synthesis, enhancing mitochondrial fatty acid oxidation (56). Additionally, L-carnitine may exert protective effects by reducing oxidative and nitrosative stress and regulating nitric oxide production and cellular respiration (57,58). The group also exhibited elevated HDL levels, consistent with findings from studies (55,59,60). This may be explained by L-carnitine's role in reducing HDL degradation and increasing the activity of HDL lipase, which helps remodel HDL particles and improves reverse cholesterol transport, thus contributing to cardiovascular protection (61).Group T4, treated with both gum Arabic and L-carnitine, demonstrated substantial reductions in total cholesterol, triglycerides, LDL, VLDL, and atherogenic index compared to groups T1–T3. The combined treatment produced stronger effects than either agent alone. Group T5, which received both treatments on a normal diet, showed the greatest reduction in lipid markers. Both T4 and T5 also experienced increases in HDL levels, with T5 achieving the highest HDL concentration among all groups.

Liver Function Markers (AST and ALT)

Significant elevations in serum AST and ALT levels were detected in rats receiving a high-fat diet, reflecting hepatic stress-a finding that mirrors result reported in prior investigations of diet-induced metabolic disturbance (62). The administration of high-fat diets led to elevated liver enzyme activity, as also reported in other studies (49,63). One study explained that feeding mice a fat-rich diet raises AST and ALT levels in serum, indicating liver cell damage. These enzymes leak into the bloodstream when hepatocytes are damaged. The mice in that study also showed increased triglycerides and cholesterol, leading to hepatic fat accumulation and

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lipotoxicity (64).These effects have also been associated with oxidative stress, as enhanced ROS generation can trigger lipid peroxidation, ultimately compromising cellular integrity. This occurs through activation of liver enzymes such as cytochrome P450, which produces the highly reactive trichloromethyl radical. This radical interacts with mitochondrial oxygen, causing destruction of cell membrane lipids and resulting in hepatic tissue injury. Consequently, hepatocyte membrane integrity is compromised, and serum AST and ALT levels rise (65,66).In group T2, which was administered gum Arabic, a reduction in AST and ALT levels was observed, confirming gum Arabic**'s** hepatoprotective effect. These findings are consistent with earlier studies (67–69). One study explained that the observed reduction in liver enzymes is attributed to gum Arabic's antioxidant potential, derived from its active phytochemicals including phenolics, flavonoids, and carotenoids. These substances function as endogenous antioxidants by neutralizing free radicals and preserving hepatic cell membranes against oxidative damage. The study also evaluated gum Arabic**'s** hepatoprotective potential in comparison with that of vitamin C in the context of oxidative liver injury (70).Similarly, group T3, treated with L-carnitine, exhibited reductions in ALT and AST levels, which aligns with several studies (71–73). L-carnitine contributes to hepatic lipid regulation by enabling the transfer of fatty acids into mitochondria for β-oxidation, reducing steatosis and associated inflammatory responses. This effect is also supported by its role in enhancing insulin responsiveness and upregulating energy metabolism enzymes, collectively aiding in the maintenance of liver integrity (74).A significant decrease in serum ALT and AST levels was recorded for group T4 (animals given gum Arabic and L-carnitine with a highfat diet) compared with the untreated hyperlipidemic group T1. . The combined treatment produced greater reductions than gum Arabic (T2) or L-carnitine (T3) alone. Notably, group T5, which received both treatments with a normal diet, exhibited the greatest reduction in liver enzyme levels among all groups (T1–T4).

Kidney Function (Urea and Creatinine)

In this study, rats subjected to a high-fat diet exhibited elevated serum levels of urea and creatinine, mirroring observations reported in earlier research (75,76). This increase may be linked to intensified lipid peroxidation within renal tissues, which impairs the activity of key antioxidant enzymes such as superoxide dismutase (SOD) and catalase (CAT), thereby contributing to oxidative stress in the kidneys induced by HFD exposure (77). Supporting this, additional studies have demonstrated that high-fat dietary intake can compromise renal function in rats, leading to raised urea and creatinine levels, along with histopathological changes such as glomerular and tubular injury, breakdown of the filtration barrier, and apoptosis of tubular epithelial cells (78).Conversely, group T2, which received gum Arabic, exhibited reduced urea and creatinine levels. This outcome aligns with several studies (79–82). One study noted that any substance providing renal protection must first be absorbed to exert systemic effects. Gum Arabic, a water-soluble fiber that is not digested in the intestine, likely reduced serum urea and creatinine by binding these compounds in the gut and promoting their excretion in feces (83,84). Additionally, gum Arabic contains adsorbent resins that may assist in uremic toxin removal, enhancing the efficacy of natural renal detoxification processes (85).In addition, the T3 group, which was treated with L-carnitine through intraperitoneal injection, had a lower level of serum urea and creatinine, which was consistent with results from many previous studies (86–88). L-carnitine was found to have antioxidant nature with anti-inflammatory potential, which could be beneficial for renal health by removing the metabolic waste products, such as urea and creatinine, from the circulation (89). As well as providing a role as a waste disposal system, L-carnitine has been demonstrated to maintain endothelial integrity under stress (27) as a result of increased intracellular PCr which preserves cellular energy status (effects) of cellular energy supply. It also regulates inflammatory responses by reducing the expression of adhesion molecules ICAM-1 and E-selectin, and by suppressing pro-inflammatory signaling via activation of the adenosine A2A receptor pathway, all of which contribute to enhanced renal filtration and excretion (90).Significantly, the T4 group exhibited significantly low serum urea and creatinine levels as compared with the T1 hyperlipidemic group. The synergistic action of both agents produced a greater effect than either alone (T2 or T3). Group T5, which received the combined treatment along with a normal diet, exhibited the greatest reduction in urea and creatinine levels among all groups (T1–T4), confirming the protective and restorative effects of the dual therapy under non-lipidemic conditions.

Conclusion

These observations provide insights into the metabolic status following high fat diet which is characterized by increased in body mass, modified lipid profile, surge in hepatic and renal biomarkers and a decline in normal functioning of the major organs in male albino rats. Supplementation with both gum Arabic and L-carnitine separately reversed a number of these effects. Of note, the therapeutic effects of the drug combination were most impressive, indicating that a synergistic way

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existed for them to regulate lipids and protect both hepatic and renal tissues in addition to lowering the oxidative stress load. These results highlight the potential of gum Arabic and L-carnitine alone or in combination as potential natural agents against obesity-related metabolic perturbations

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Conflict of interest

No conflict of interest is found for the present study.

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