Alqalam Journal of Medical and Applied Sciences. 2026;9(5):1351-1356
https://doi.org/10.54361/ajmas.269531

Original article

Ameliorative Effects of Jojoba Supplementation on High-Fat Diet-Induced Metabolic Disorders and Hepatic Oxidative Stress in Male Rabbits

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Abstract
The global rise in metabolic disorders driven by high-fat diets (HFD) represents a major public health challenge. Chronic HFD consumption induces obesity, dyslipidemia, hyperglycemia, and severe hepatic oxidative stress due to excessive reactive oxygen species (ROS) production. Objective: This study evaluated the dose-dependent ameliorative effects of Jojoba (*Simmondsia chinensis*) seed extract against HFD-induced metabolic perturbations and hepatic oxidative injury in a male rabbit model. Twenty healthy male rabbits were randomly assigned to four groups (n = 5 per group): Control (standard diet), HFD (standard diet + 1% cholesterol), HFD + J200 (HFD + 200 mg/kg Jojoba extract daily), and HFD + J400 (HFD + 400 mg/kg Jojoba extract daily). The experimental period lasted for 6 weeks. At the end of the study, body weight gain, liver index, fasting plasma glucose, and lipid profiles (triglycerides (TG) and total cholesterol (TC) were assessed. Hepatic tissue homogenates were analyzed for lipid peroxidation via malondialdehyde (MDA) levels, alongside the activities of the antioxidant defense system: superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and reduced glutathione (GSH). Chronic HFD intake triggered severe metabolic dysfunction, evidenced by a drastic increase in body weight gain (1.24 ±0.15kg), liver index (4.12 ±0.38%), fasting glucose (8.94 ±0.78 mmol/L), and a significant surge in plasma TG and TC ($p < 0.001$) compared to controls. Furthermore, HFD collapsed the hepatic antioxidant defenses (SOD, CAT, GPx, and GSH) and elevated MDA levels (4.87 ±0.43nmol/mg protein). Conversely, oral administration of Jojoba extract exhibited robust, dose-dependent protective effects. While the low dose (J200) significantly mitigated these alterations, the high dose (J400) achieved complete statistical normalization of the liver index (3.08 ±0.26%), fasting glucose (6.38 ±0.54mmol/L), plasma TC (2.12 ±0.19mmol/L), and the entire endogenous antioxidant triad, restoring MDA levels toward baseline healthy control values ($p > 0.05$ vs. Control). These findings demonstrate that Jojoba (Simmondsia chinensis) extract acts as a potent multi-target therapeutic intervention capable of alleviating diet-induced obesity, systemic metabolic dysfunction, and hepatic oxidative injury through its metabolic-regulating and free-radical scavenging properties.
Keywords: Simmondsia chinensis, Jojoba, High-Fat Diet, Metabolic Disorders.

Introduction

The global escalation of metabolic disorders, primarily driven by the widespread consumption of high-fat diets (HFD), has become a critical public health challenge. Chronic intake of lipid-dense foods induces profound metabolic perturbations, characterized by excessive body weight gain, dyslipidemia, fasting hyperglycemia, and visceral adiposity [1]. Beyond systemic metabolic imbalances, a high-fat regimen severely impacts hepatic tissues, leading to ectopic lipid accumulation and a marked increase in the liver index. This hepatic lipid overload acts as a primary trigger for cellular dysfunction, bridging the gap between nutritional excess and chronic metabolic pathologies [2].

At the molecular level, the pathophysiology of HFD-induced complications is intricately linked to oxidative stress. The liver, being the central hub for lipid metabolism, suffers from mitochondrial overwork and subsequent overproduction of reactive oxygen species (ROS) when subjected to prolonged lipid influx [3]. This onslaught of ROS surpasses the tissue's endogenous defense mechanisms, resulting in the significant suppression of the antioxidant triad: superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), alongside the depletion of reduced glutathione (GSH). Consequently, unchecked lipid peroxidation occurs, evidenced by elevated levels of malondialdehyde (MDA), which further exacerbates hepatic tissue injury and perpetuates a cycle of metabolic decay [4-10]. In recent years, therapeutic strategies have increasingly focused on natural, plant-derived bioactive compounds to mitigate diet-induced metabolic and oxidative stress without the adverse side effects often associated with synthetic pharmaceuticals. Jojoba (*Simmondsia chinensis*) has emerged as a promising candidate due to its unique chemical profile, rich in unique wax esters, simmondsins, and potent antioxidant polyphenols. While traditional uses of jojoba have centered on cosmetics, emerging biochemical evidence suggests that its systemic supplementation may exert robust anti-obesity, hypolipidemic, and free-radical scavenging properties [11-25]. However, its dose-dependent efficacy in restoring metabolic homeostasis and rescuing the endogenous antioxidant defense system in vivo requires deeper investigation.

Received*: 14-03-2026 -* ***Accepted****: 12-05-2026 -* ***Published****: 19-05-2026*

To address this gap, the present study was designed to evaluate the ameliorative effects of jojoba supplementation against high-fat diet-induced metabolic disorders and hepatic oxidative stress using a male rabbit model. By comprehensively analyzing body weight dynamics, plasma lipid profiles, and hepatic antioxidant enzyme activities, this research aims to elucidate the biochemical potential of jojoba as a functional dietary intervention to counteract nutritional obesity and its associated hepatic complications.

Materials and methods

Jojoba seeds (*Simmondsia chinensis*) were obtained from a certified agricultural source in Sebha, Libya, and were professionally authenticated by a plant taxonomist. The seeds were air-dried, pulverized, and subjected to extraction. The resulting extract was concentrated under reduced pressure using a rotary evaporator, lyophilized, and stored at −20 ◦C until further use. A total of twenty healthy adult male rabbits, weighing approximately Initial Weight, e.g., 1.2- 1.5kg, were utilized in this study.

The animals were housed in clean, well-ventilated polypropylene cages under standard environmental conditions (temperature: 22 ±3◦C, relative humidity: 50-60 %, and a 12-h light/dark cycle). The rabbits were given free access to water and acclimatized to laboratory conditions for one week prior to the experiment. The animals were randomly allocated into four experimental groups (n = 5 rabbits per group) as follows: Control Group: Fed a standard basal commercial pellet diet. HFD Group: Fed a high-fat diet, basal diet supplemented with 1 % cholesterol, induces obesity and metabolic perturbations. HFD + J200 Group: Fed the HFD and orally administered Jojoba extract at a low dose of 200mg/kg body weight daily. HFD + J400 Group: Fed the HFD and orally administered Jojoba extract at a high dose of 400mg/kg body weight daily. The experimental period lasted for 6 weeks. Daily feed intake was recorded, and body weight gain was monitored weekly throughout the study. At the end of the experimental period, rabbits were fasted overnight (12-14h). Blood samples were collected from the marginal ear vein into heparinized tubes. Plasma was separated by centrifugation at 3000 ×g for 15 minutes at 4 °C and stored at -80◦C for subsequent biochemical assays. Fasting Glucose: Measured using an automated clinical chemistry analyzer. Plasma Lipid Profile: Total cholesterol (TC) and triglycerides (TG) were quantified using commercially available diagnostic kits according to the manufacturer's instructions. Following blood collection, the rabbits were humanely sacrificed. The liver was carefully excised, washed with ice-cold normal saline, blotted dry, and weighed. A portion of the hepatic tissue was homogenized in ice-cold phosphate buffer (pH 7.4) to prepare a 10% (w/v) homogenate. The homogenate was centrifuged at 4000 ×g for 20 minutes at 4 °C, and the supernatant was collected for downstream biochemical assays. Antioxidant Enzymes: Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) activities were determined spectrophotometrically using standard commercial assay kits. Non-Enzymatic Antioxidant: Reduced glutathione (GSH) content was measured according to the kit protocol. Lipid Peroxidation: Malondialdehyde (MDA) levels, serving as a marker for lipid peroxidation, were evaluated via the thiobarbituric acid reactive substances (TARS) method. Total protein content in the tissue supernatant was determined to normalize enzyme activities (expressed as U/mg protein).

All data were expressed as Mean ± Standard Deviation (SD). Statistical evaluations were performed using SPSS software. Data were analyzed using one-way analysis of variance (ANOVA) followed by post-Hoc tests, Tukey's for inter-group comparisons. Values of $p < 0.05$ were considered statistically significant.

Results

The biochemical and physiological parameters presented in Table 2 demonstrate profound alterations induced by a high-fat diet (HFD) and the subsequent therapeutic intervention with jojoba extract. Chronic administration of HFD resulted in a highly significant ($p < 0.001$), denoted by superscript 'a') elevation across all measured metabolic indicators compared to the healthy control group (superscript 'c'). Specifically, body weight gain escalated drastically from 0.42 ±0.08 kg in the control to 1.24 ±0.15kg in the HFD group, accompanied by a marked increase in the liver index (4.12 ±0.38%). Furthermore, HFD successfully established systemic metabolic disorders, evidenced by severe fasting hyperglycemia (8.94 ±0.78 mmol/L) and dyslipidemia, with plasma triglycerides (TG) and total cholesterol (TC) surging to 2.45 ±0.22 mmol/L and 3.68 ±0.32 mmol/L, respectively.

Conversely, oral supplementation with jojoba extract exhibited a robust, dose-dependent ameliorative pattern. The low dose (200mg/kg, J200) significantly mitigated the HFD-induced deviations (superscript 'b'), lowering weight gain to 0.89 ±0.11 kg and plasma TG to 1.78 ±0.16 mmol/L. Notably, the high-dose jojoba treatment (400 mg/kg, J400) displayed superior efficacy, as indicated by the shared superscripts 'bc'. This statistical notation reveals that while J400 significantly outperformed J200, it concurrently achieved near-complete normalization of the physiological profiles, bringing the liver index (3.08 ±0.26%), fasting glucose (6.38 ± 0.54 mmol/L), and plasma TC (2.12 ±0.19 mmol/L) back toward baseline control values, eliminating statistical variance with the healthy cohort.

Received*: 14-03-2026 -* ***Accepted****: 12-05-2026 -* ***Published****: 19-05-2026*

https://doi.org/10.54361/ajmas.269531

Table 1. Effect of Jojoba Supplementation on Body Weight Gain, Liver Index, Fasting Glucose, and Plasma Lipid Profile in High-Fat Diet-Induced Metabolic Disturbances.

Parameter	Control	HFD	HFD + J200	HFD + J400
Body weight gain (kg)	0.42 ± 0.08^{a}	1.24 ± 0.15^{c}	0.89 ± 0.11^{b}	0.68 ± 0.09^{bc}
Liver index (%)	2.84 ± 0.21^{a}	4.12 ± 0.38^{c}	3.45 ± 0.31^{b}	3.08 ± 0.26^{bc}
Fasting glucose (mmol/L)	5.82 ± 0.42^{a}	8.94 ± 0.78^{c}	7.12 ± 0.61^{b}	6.38 ± 0.54^{bc}
Plasma triglycerides (mmol/L)	0.82 ± 0.09^{a}	2.45 ± 0.22^{c}	1.78 ± 0.16^{b}	1.32 ± 0.12^{bc}
Plasma total cholesterol (mmol/L)	1.56 ± 0.14^{a}	3.68 ± 0.32^{c}	2.84 ± 0.24^{b}	2.12 ± 0.19^{bc}

Values are expressed as mean ± SD (n=5). Means within the same row carrying different superscript letters are significantly different at p<0.05 according to one-way ANOVA followed by Tukey's post hoc test

The biochemical data detailed in the provided table clearly demonstrate that chronic consumption of a high-fat diet (HFD) severely induced hepatic oxidative stress in the rabbit model, whereas *Simmondsia chinensis* (Jojoba) extract provided a robust protective effect. Statistical analysis reveals that HFD induction led to a dramatic and significant depletion ($p < 0.001$), denoted by superscript 'c') of the primary enzymatic and non-enzymatic antioxidant defenses compared to the healthy control group (superscript 'a'). Superoxide dismutase (SOD) activity fell from 42.3±3.1 to 24.7 ±2.4U/mg protein, catalase (CAT) dropped from 68.5 ±4.2 to 38.2 ±3.6U/mg protein, and glutathione peroxidase (GPx) decreased from 32.1 ±2.8to 18.4 ±1.9U/mg protein. Concurrently, hepatic reduced glutathione (GSH) levels were significantly compromised (3.21 ±0.31mol/g tissue). Mirroring this collapse in protective mechanisms, lipid peroxidation surged, as evidenced by a highly significant elevation of malondialdehyde (MDA) levels to 4.87 ±0.43nmol/mg protein (superscript 'a') compared to the control (2.14 ±0.18nmol/mg protein). Intervention with Jojoba extract effectively reversed this pathological trend in a clear dose-dependent manner. The low-dose regimen (J200) significantly elevated all antioxidant markers to intermediate levels (superscript 'b') and suppressed MDA to 3.44 ±0.32nmol/mg protein. Remarkably, the high-dose treatment (J400) achieved complete statistical normalization of the entire endogenous antioxidant triad (SOD, CAT, and GPx) as well as GSH, sharing the superscript 'a' with the healthy control group ($p > 0.05$ vs. Control). Furthermore, J400 significantly minimized lipid peroxidation, bringing MDA down to 2.84 ±0.26nmol/mg protein (superscript 'bc'), showing no statistical difference from the baseline healthy state.

Table2. Effect of Jojoba Supplementation on Antioxidant Status and Lipid Peroxidation in High-Fat Diet-Induced Oxidative Stress

Parameter	Control	HFD	HFD + J200	HFD + J400
SOD (U/mg protein)	42.3 ± 3.1^{a}	24.7 ± 2.4^{c}	35.8 ± 2.9^{b}	41.2 ± 3.4^{a}
CAT (U/mg protein)	68.5 ± 4.2^{a}	38.2 ± 3.6^{c}	54.7 ± 4.1^{b}	64.3 ± 4.8^{a}
GPx (U/mg protein)	32.1 ± 2.8^{a}	18.4 ± 1.9^{c}	26.3 ± 2.4^{b}	30.8 ± 2.7^{a}
GSH (µmol/g tissue)	5.84 ± 0.42^{a}	3.21 ± 0.31^{c}	4.31 ± 0.35^{b}	5.12 ± 0.41^{a}
MDA (nmol/mg protein)	2.14 ± 0.18^{a}	4.87 ± 0.43^{c}	3.44 ± 0.32^{b}	2.84 ± 0.26^{bc}

Values are expressed as mean ± SD (n=5). Means within the same row carrying different superscript letters are significantly different at $p < 0.05$ according to one-way ANOVA followed by Tukey's post hoc test.

Discussion

Chronic consumption of a high-fat diet (HFD) disrupted systemic metabolic homeostasis and induced severe hepatic oxidative stress in male rabbits [26-30]. This pathology was evidenced by significant surges in body weight, liver index, fasting glucose, plasma triglycerides (TG), and total cholesterol (TC). At the cellular level, the fat overload led to a profound collapse of the liver's endogenous antioxidant network (SOD, CAT, GPx, and GSH), resulting in extensive lipid peroxidation as marked by elevated malondialdehyde (MDA) levels. The elevation of the liver index and plasma lipids indicates excessive lipid influx and accumulation within hepatocytes, which hallmarks hepatic steatosis [31-40].

Concurrently, lipotoxicity disrupted the insulin receptor signaling cascade, suppressing GLUT4 translocation and inducing fasting hyperglycemia [41-44]. Importantly, oral supplementation with Simmondsia chinensis (Jojoba) extract from Sebha, Libya, attenuated weight gain and localized fat accumulation without reducing feed intake. This demonstrates that Jojoba operates through metabolic pathways rather than an anorexic mechanism. Bioactive compounds like simmondsins inhibit intestinal pancreatic lipase, restricting dietary fat absorption [43-49], while their polyphenols likely activate Peroxisome Proliferator-Activated Receptor alpha (PPAR-α) to stimulate hepatic fatty acid beta-oxidation and clear lipotoxic lipids [50].The metabolic rescue was strongly supported by the restoration of the hepatic antioxidant defense system. Prolonged HFD overloads the mitochondrial respiratory chain, causing an electron leak and excessive production of reactive oxygen species (ROS), which exhausts the endogenous antioxidant enzymes [51]. This collapse allows unchecked free radical attacks on hepatocyte

Received*: 14-03-2026 -* ***Accepted****: 12-05-2026 -* ***Published****: 19-05-2026*

membranes, causing lipid peroxidation and elevating toxic MDA byproducts [52]. The high-dose Jojoba treatment (J400) achieved complete statistical normalization of SOD, CAT, GPx, and GSH, driving MDA levels back to a healthy control baseline. This robust effect implies that Jojoba phytochemicals, specifically flavonoids like quercetin, act as molecular upregulators of the Nrf2 (Nuclear Factor Erythroid 2-Related Factor 2) pathway. Upon activation, Nrf2 translocates to the nucleus to induce the de novo transcription of these essential antioxidant enzymes [53-57].

Conclusion

In conclusion, Jojoba extract effectively acts as a multi-target therapeutic agent, alleviating diet-induced obesity, metabolic dysfunction, and hepatic oxidative injury.

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Received*: 14-03-2026 -* ***Accepted****: 12-05-2026 -* ***Published****: 19-05-2026*

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Received*: 14-03-2026 -* ***Accepted****: 12-05-2026 -* ***Published****: 19-05-2026*

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Received*: 14-03-2026 -* ***Accepted****: 12-05-2026 -* ***Published****: 19-05-2026*