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Metabolic and Hepatorenal Modulatory Effects of Coriander Seeds in an Experimental Model of Obesity in Rats

Abeer A Aljehani¹, Lobna Saad Mohammed Abd Elmeged²

¹ Department of Food and Nutrition, Faculty of Human Sciences and Design, King Abdulaziz University, Jeddah, 21589 Saudi Arabia.

aaaaljehani1@kau.edu.sa. ORCID:0000-0002-6697-1606

² Department of Nutrition, Applied College, Al-Baha University, Al-Makhwa, Saudi Arabia. Email: lobna@bu.edu.sa

² Food Quality Monitoring Administration, General Administration of Technical Inspection, Menoufia University, Shibin el Kom, Menofia Governorate 6131567, Egypt. Email: lobna_lolo_2007@yahoo.com

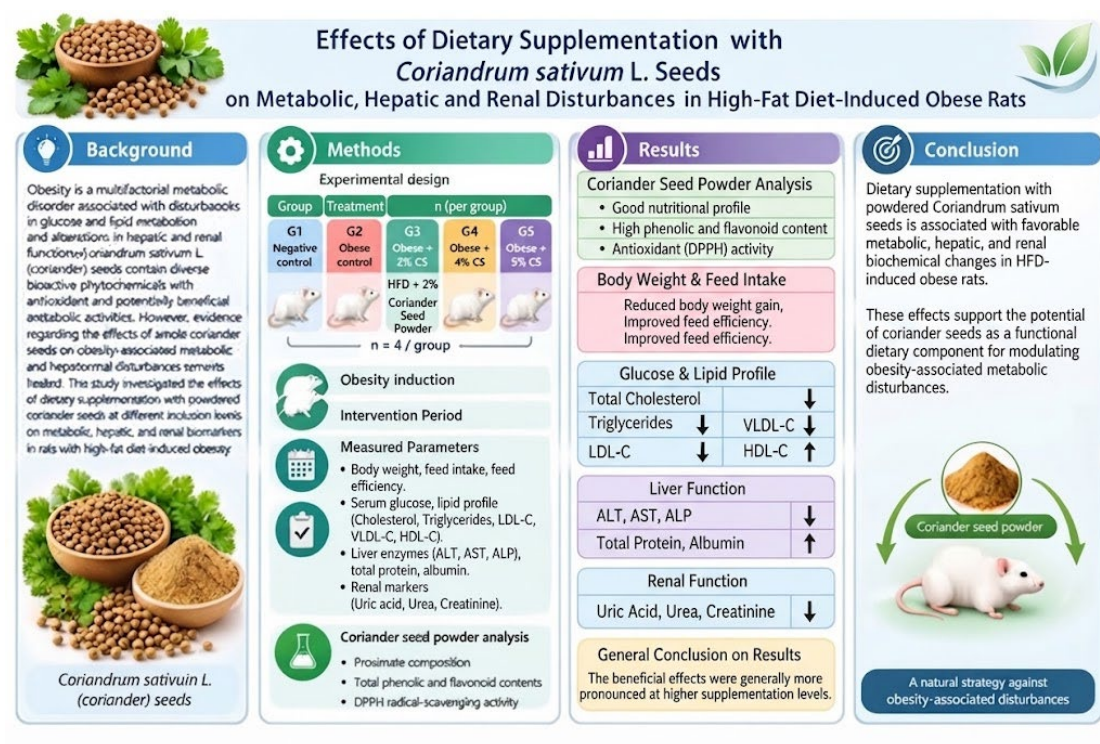
ABSTRACT:

Background: Obesity is a multifactorial metabolic disorder associated with disturbances in glucose and lipid metabolism and impaired hepatic and renal function. *Coriandrum sativum* L. (coriander) seeds contain bioactive phytochemicals with antioxidant and potentially beneficial metabolic activities. However, evidence regarding the effects of whole coriander seeds on obesity-associated metabolic and hepatorenal disturbances remains limited. This study investigated the effects of dietary supplementation with powdered coriander seeds at different inclusion levels on metabolic, hepatic, and renal biomarkers in rats with high-fat diet-induced obesity.

Methods: Twenty adult male Sprague-Dawley rats were randomly allocated into five groups (n = 4/group): negative control, obese control, and three obese groups receiving diets supplemented with 2%, 4%, or 5% powdered coriander seeds. Obesity was induced using a high-fat diet, followed by a 28-day dietary intervention. Body weight gain, feed intake, and feed efficiency were assessed. Serum glucose, lipid profile, liver enzymes, total protein, albumin, uric acid, urea, and creatinine were determined. Proximate composition, total phenolic and flavonoid contents, and DPPH radical-scavenging activity of coriander powder were also evaluated.

Results: Coriander powder exhibited measurable phenolic and flavonoid contents and antioxidant activity. Supplementation reduced body weight gain and improved serum glucose and lipid profiles, with lower total cholesterol, triglycerides, LDL-C, and VLDL-C and higher HDL-C. It also attenuated elevations in ALT, AST, and ALP and improved total protein and albumin concentrations. Furthermore, coriander supplementation reduced elevated serum uric acid, urea, and creatinine levels in obese rats. Beneficial changes were generally more pronounced at higher supplementation levels.

Conclusion: Dietary supplementation with powdered *Coriandrum sativum* seeds was associated with favorable metabolic, hepatic, and renal biochemical changes in high-fat diet-induced obese rats, supporting their potential as a functional dietary component for modulating obesity-associated metabolic disturbances.



Graphical Abstract: Protective Effects of *Coriandrum sativum* Seeds Against Obesity-Associated Metabolic, Hepatic, and Renal Disturbances.

INTRODUCTION

Obesity has emerged as one of the most important global public health challenges of the twenty-first century. It is a complex, chronic, and multifactorial disorder characterized by excessive or abnormal accumulation of adipose tissue that adversely affects metabolic health. The worldwide prevalence of obesity has increased markedly over recent decades, with approximately 890 million adults living with obesity in 2022, representing about 16% of the global adult population. The rapid increase in obesity has been attributed to complex interactions among excessive energy intake, reduced physical activity, genetic susceptibility, environmental factors, and changes in dietary patterns. Importantly, obesity is no longer regarded simply as an abnormal accumulation of body fat; rather, it is increasingly recognized as a systemic metabolic disorder associated with a broad spectrum of metabolic and organ-specific complications (World Health Organization, 2025).

The metabolic consequences of obesity include impaired glucose regulation, insulin resistance, dyslipidemia, chronic low-grade inflammation, oxidative stress, and abnormal lipid deposition in metabolically active organs. These alterations substantially increase the risk of developing type 2 diabetes mellitus, cardiovascular disease, metabolic dysfunction-associated steatotic liver disease, and chronic kidney disease (Aruwa & Sabiu, 2024). Insulin resistance represents one of the central pathological mechanisms linking obesity with these metabolic abnormalities. Persistent nutrient excess and adipose tissue dysfunction promote increased release of free fatty acids and inflammatory mediators, which interfere with insulin signaling in peripheral tissues and contribute to impaired glucose utilization and compensatory hyperinsulinemia, as shown in Figure 1. As the disease progresses, pancreatic β -cells may become unable to compensate adequately for the increased metabolic demand, resulting in deterioration of glucose homeostasis and, ultimately, hyperglycemia (Roden and Shulman, 2019).

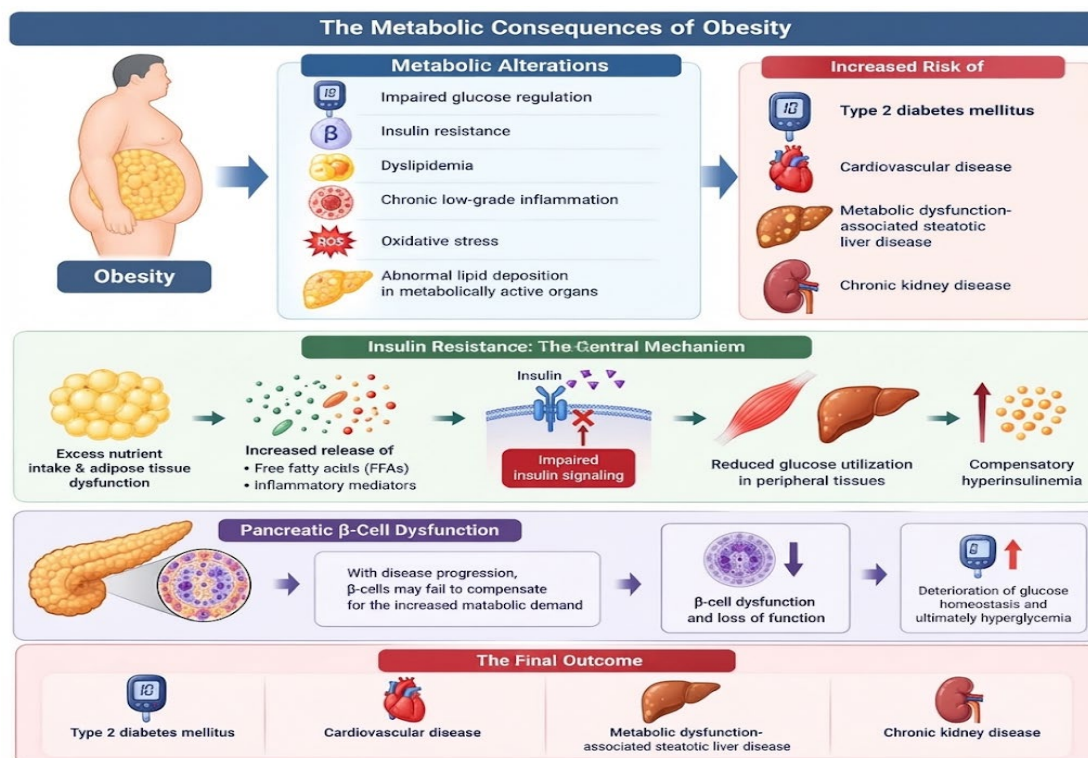


Figure 1: The Metabolic Consequences of Obesity

Dyslipidemia is another major metabolic abnormality associated with obesity. Excessive lipid availability and altered hepatic lipid metabolism may lead to increased circulating triglycerides and low-density lipoprotein cholesterol, accompanied by reduced high-density lipoprotein cholesterol. Such lipid disturbances are closely associated with insulin resistance and contribute to the development of atherogenic cardiovascular risk. Moreover, excessive lipid flux to the liver can promote hepatic triglyceride accumulation and metabolic dysfunction-associated steatotic liver disease. The close relationship between obesity, insulin resistance, dyslipidemia, and hepatic lipid accumulation highlights the interconnected nature of metabolic disturbances associated with excessive adiposity (European Association for the Study of the Liver et al., 2024). The liver plays a central role in the regulation of glucose, lipid, and energy metabolism and is therefore particularly vulnerable to the metabolic consequences of obesity. Insulin resistance promotes increased hepatic de novo lipogenesis and altered fatty acid metabolism, resulting in hepatic lipid accumulation. Persistent hepatic lipid overload may induce oxidative stress, mitochondrial dysfunction, inflammatory signaling, and hepatocellular injury. These pathological processes can be reflected biochemically by alterations in serum hepatic enzymes and, if sustained, may contribute to progressive liver injury (Yau et al., 2024). In addition, metabolic dysfunction-associated steatotic liver disease is strongly associated with type 2 diabetes and cardiovascular disease, emphasizing the importance of evaluating hepatic function when investigating potential nutritional interventions for obesity.

The kidneys are also affected by obesity-related metabolic disturbances. Obesity may contribute to renal dysfunction through several interrelated mechanisms, including insulin resistance, activation of inflammatory pathways, oxidative stress, abnormal lipid metabolism, altered hemodynamics, and increased intraglomerular pressure. Obesity-associated renal injury may initially remain clinically silent but can progress to structural and functional abnormalities of the kidney. The relationship between obesity, dyslipidemia, metabolic dysfunction, and renal disease is therefore increasingly recognized as an important component of the metabolic syndrome (Iqbal et al., 2024). Consequently, assessment of renal biomarkers, such as serum urea and creatinine, can provide useful information regarding the systemic effects and safety of dietary interventions intended to ameliorate obesity-associated metabolic abnormalities.

In addition to hepatic and renal complications, obesity may exert significant effects on pancreatic structure and function. The pancreas is particularly sensitive to chronic metabolic stress caused by excessive nutrient availability, insulin resistance, and

lipid accumulation. Experimental studies have demonstrated that high-fat feeding can produce pancreatic abnormalities, including islet alterations, adipocyte accumulation, vacuolization, and degenerative changes. Gulen et al. (2015) reported that high-fat diets induced pancreatic tissue alterations accompanied by changes in insulin resistance and islet morphology in rats. Similarly, prolonged high-fat feeding in obese rats has been associated with pancreatic fat accumulation, fibrosis, inflammatory lesions, and acinar cell injury. These observations suggest that pancreatic histopathological assessment may provide valuable complementary evidence when evaluating the effects of dietary interventions on obesity-associated metabolic dysfunction. Given the multifaceted nature of obesity and its complications, increasing attention has been directed toward dietary strategies and plant-derived functional foods as complementary approaches for improving metabolic health. Medicinal and culinary plants contain diverse bioactive compounds that may exert antioxidant, anti-inflammatory, hypoglycemic, hypolipidemic, and hepatoprotective effects. Among these plants, *Coriandrum sativum* L., commonly known as coriander, has attracted considerable scientific interest because of its nutritional and pharmacological properties. Coriander belongs to the Apiaceae family and has a long history of culinary and traditional medicinal use. Its seeds contain numerous biologically active constituents, including essential oils, terpenoids, phenolic compounds, flavonoids, fatty acids, phytosterols, and other phytochemicals (Valizadeh et al., 2024).

The biological activities of coriander have been attributed to the complex mixture of phytochemicals present in its seeds and other plant parts. Linalool is considered one of the major constituents of coriander essential oil, while the seeds also contain fatty acids, sterols, tocopherols, carotenoids, and phenolic compounds. The composition and concentration of these compounds may vary according to cultivar, geographical origin, environmental conditions, maturity, and processing methods (Wei et al., 2019). Several reviews have highlighted the antioxidant, anti-inflammatory, hypoglycemic, hypolipidemic, hepatoprotective, and other potentially beneficial biological activities of *C. sativum*, supporting its potential application as a functional food or dietary intervention (Mahleyuddin et al., 2022).

Of particular relevance to obesity-associated metabolic disturbances, experimental evidence indicates that coriander seeds may influence glucose and lipid metabolism. Demonstrated that administration of an aqueous coriander seed extract to an experimental rat model improved hyperglycemia and reduced circulating insulin, insulin resistance, total cholesterol, low-density lipoprotein cholesterol, and triglycerides. These findings suggested that coriander may exert multiple metabolic effects rather than acting exclusively on blood glucose. Similarly, (Sobhani et al. 2022) reported a significant hypolipidemic effect of coriander seeds in rats fed a high-fat, cholesterol-enriched diet. Coriander supplementation was associated with reductions in total cholesterol and triglycerides, together with favorable changes in lipoprotein fractions. The authors suggested that alterations in cholesterol metabolism, including enhanced cholesterol degradation and bile acid synthesis, may contribute to the lipid-lowering effect of coriander seeds. The potential metabolic actions of coriander are also biologically plausible based on its phytochemical composition. Phenolic compounds, flavonoids, terpenoids, phytosterols, and essential oil constituents may collectively contribute to antioxidant and metabolic effects. Oxidative stress is closely linked to obesity-induced insulin resistance, hepatic lipid accumulation, and pancreatic dysfunction; therefore, the antioxidant properties of coriander may be relevant to its potential protective effects against obesity-associated metabolic injury. Nevertheless, the biological response to whole coriander seeds may differ from that of isolated extracts or individual phytochemicals, and the dose, duration, dietary matrix, and experimental model may influence the observed effects (Aissaoui et al., 2011).

Although previous studies have investigated the hypoglycemic and hypolipidemic properties of coriander, comparatively less attention has been given to the simultaneous assessment of its effects on glucose metabolism, lipid profile, hepatic enzymes, renal function, and pancreatic histopathology within a single experimental obesity model. Such a multidimensional assessment is important because obesity is a systemic disorder in which metabolic, hepatic, renal, and pancreatic abnormalities develop concurrently. Furthermore, evaluating different dietary concentrations of coriander seed powder may provide useful information regarding the potential dose-related nature of its biological effects.

Therefore, the present study was designed to investigate the modulatory effects of dietary coriander seed powder (*Coriandrum sativum* L.) at different concentrations on selected metabolic and biochemical parameters in an experimental model of obesity in albino rats. The study specifically evaluated blood glucose and lipid profile, together with biochemical markers of hepatic and renal function. In addition, pancreatic tissue was subjected to histopathological examination to determine whether coriander seed supplementation was associated with morphological improvement in obesity-related pancreatic alterations. By integrating biochemical and histopathological assessments, this study aimed to provide a broader understanding of the

potential metabolic and hepatorenal benefits of coriander seeds and their possible protective effects on pancreatic tissue in experimental obesity

2. MATERIALS AND METHODS

2.1. Materials

2.1.1. Plant Mate

Coriander seeds (*Coriandrum sativum* L.) were obtained from a local commercial source in Al-Baha City, Saudi Arabia. The seeds were visually inspected and manually cleaned to remove foreign materials, damaged seeds, and other visible impurities. They were then washed with distilled water and air-dried at room temperature under laboratory conditions until completely dry. The dried seeds were ground into a fine, homogeneous powder using a laboratory mechanical grinder. The resulting powder was transferred to clean, airtight containers and stored under conditions that protected it from excessive moisture, heat, and light until incorporation into the experimental diets.

Coriandrum sativum L. contains a variety of bioactive phytochemicals, including phenolic compounds, flavonoids, terpenoid compounds, and other volatile constituents. Experimental evidence has also demonstrated hypoglycemic and hypolipidemic effects of an aqueous extract of coriander seeds in obese, hyperglycemic, and hyperlipidemic *Meriones shawi* rats. (Aissaoui et al., 2011)

2.1.2. Experimental Animals

Twenty adult male Sprague–Dawley albino rats, weighing 150 ± 10 g, were obtained from the animal house facility of the Institute of Nutrition, Cairo, Egypt. The animals were selected to be of comparable age and body weight and were apparently healthy at the beginning of the experiment.

2.1.3. Dietary Ingredients and Chemicals

The dietary ingredients used for the experimental diets, including casein, corn oil, cellulose, sucrose, corn starch, vitamin mixture, mineral mixture, and choline chloride, were obtained from Morgan Co., Cairo, Egypt. Analytical-grade chemicals and reagents used for biochemical and histological analyses were obtained from El-Nasr Pharmaceutical Chemicals, El-Amireia, Cairo, Egypt.

2.2. Preparation of Coriander Seed Powder

The coriander seeds were thoroughly cleaned and washed with distilled water to remove adhering dust and particulate matter. The seeds were then dried at room temperature under laboratory conditions until a constant weight was achieved. The dried seeds were subsequently ground into a fine, homogeneous powder using a laboratory mechanical grinder. The resulting powder was transferred to clean, airtight containers and stored under conditions that minimized exposure to moisture, heat, and light until incorporation into the experimental diets.

2.3. Experimental Animals and Housing Conditions

The animals were acclimatized for seven days before the initiation of the experimental procedures. During the acclimatization period and throughout the experimental period, the rats were housed under controlled laboratory conditions at a temperature of 22 ± 2 °C, relative humidity of 50–60%, and a 12-h light/12-h dark cycle. During acclimatization, the animals had free access to standard laboratory chow and drinking water.

Throughout the experimental period, the animals were monitored regularly, and appropriate measures were taken to minimize stress and unnecessary discomfort. Animal care, housing, and experimental procedures were conducted in accordance with applicable institutional guidelines for the care and use of laboratory animals (National Research Council, 2011). The study was reported in accordance with the ARRIVE 2.0 guidelines for animal research (Percie du Sert et al., 2020).

2.4. Induction of Experimental Obesity

Experimental obesity was induced by feeding the designated rats a high-fat diet (HFD) for four weeks. The HFD was formulated to contain 30% fat, provided as a 1:1 mixture of tallow and corn oil, together with casein, mineral and vitamin

mixtures, fiber, DL-methionine, choline chloride, and bile acids. Corn starch was used as the principal carbohydrate source and was added in an amount sufficient to complete the formulation to 100 g. The detailed composition of the basal and experimental diets is presented in Table 1.

The diet formulation was based on a purified rodent-diet framework and was modified to provide the experimental high-fat formulation used in the present study (Reeves et al., 1993).

Table (1): The basic and experimental diets' compositions.

Component (g)	Control (-)	Control (+)	2% powdered coriander seeds	4% powdered coriander seeds	5% f powdered coriander seeds
Test ingredients	---	---	2	4	5
Casein	20	20	20	20	20
Corn oil	4.7	4.7	4.7	4.7	4.7
Mineral mix	3.5	3.5	3.5	3.5	3.5
Vitamin mix	1	1	1	1	1
Cellulose	5	5	5	5	5
Cholin chloride	2	2	2	2	2
Sucrose	10	10	10	10	10
Corn starch	Up to 100	Up to 100	Up to 100	Up to 100	Up to 100

2.5. Induction of Experimental Obesity

To establish the experimental obesity model, the designated rats were fed a high-fat diet (HFD) for four weeks. The experimental diet was formulated as a modified purified rodent diet, with its basic formulation informed by the AIN-93 dietary framework (Reeves et al., 1993). The HFD contained 30% fat, provided as a 1:1 mixture of tallow and corn oil, 12% casein, 4% mineral mixture, 1% vitamin mixture, and 5% fiber. DL-methionine (0.3%), choline chloride (2%), and bile acids (0.2%) were incorporated into the diet, while corn starch was used as the principal carbohydrate source to complete the formulation to 100 g.

2.6. Ethical Housing and Environmental Conditions

The animal experiment was conducted at the Animal House Facility, Faculty of Home Economics, Menoufia University, Egypt. Adult male Sprague-Dawley rats, weighing 150 ± 10 g and aged 14–16 weeks, were housed in standardized polypropylene cages under controlled laboratory conditions. The ambient temperature was maintained at 22 ± 2 °C, with a relative humidity of 50–60% and a 12-h light/12-h dark cycle. Animals had free access to drinking water and the designated experimental diets throughout the study.

Before initiation of the experimental procedures, all animals were acclimatized for seven days under the same environmental conditions. During the acclimatization period, the animals were monitored for general health and allowed to adapt to the housing environment. Throughout the study, appropriate measures were taken to minimize stress, discomfort, and unnecessary suffering. Animal care and experimental procedures were conducted in accordance with the applicable institutional guidelines for the care and use of laboratory animals (National Research Council, 2011).

2.7. Experimental Design and Grouping

Twenty rats were randomly allocated to five experimental groups, with four animals per group ($n = 4$), as follows:

- Group 1 – Negative control: Healthy rats fed the basal diet.
- Group 2 – Positive control: Obese rats fed the corresponding experimental diet without coriander supplementation.
- Group 3 – 2% coriander: Obese rats fed the designated diet supplemented with 2% powdered *C. sativum* seeds.
- Group 4 – 4% coriander: Obese rats fed the designated diet supplemented with 4% powdered *C. sativum* seeds.
- Group 5 – 5% coriander: Obese rats fed the designated diet supplemented with 5% powdered *C. sativum* seeds.

The dietary intervention was continued for 28 consecutive days. Coriander seed powder was incorporated into the respective experimental diets at concentrations of 2%, 4%, and 5% (w/w). The control groups received their corresponding diets without coriander supplementation.

The experimental design, group allocation, sample size, and outcome assessment were reported in accordance with the ARRIVE 2.0 recommendations (Percie du Sert et al., 2020).

2.8. Proximate Chemical Analysis

The proximate composition of the coriander seed powder was determined by analyzing its moisture, ash, crude protein, and lipid contents using applicable official analytical methods of AOAC INTERNATIONAL. All determinations were performed in triplicate, and the results were expressed as appropriate for each component.

2.9. Determination of Phenolic Profile Using HPLC

The phenolic profile of the coriander seed powder was analyzed using a high-performance liquid chromatography (HPLC) system (Agilent Technologies 1260 Infinity II) equipped with a diode-array detector (DAD) and an autosampler. Chromatographic separation was performed using an Eclipse XDB-C18 column (150×4.6 mm, 5 μ m) coupled with a compatible guard column. The mobile phase consisted of acetonitrile and 2% aqueous acetic acid, applied using a gradient elution program. The flow rate was maintained at 0.8 mL/min, and the total analysis time was 60 min.

Before injection, the prepared samples were filtered through a 0.45- μ m membrane filter, and 50 μ L of the filtrate was injected into the HPLC system. Detection was performed at 280, 320, and 360 nm. Phenolic compounds were identified by comparing their retention times and UV-DAD spectral characteristics with those of authentic reference standards.

Note: The exact analytical reference for the stated HPLC conditions should be provided from the original laboratory protocol or source paper before the citation “Kim et al. (2006)” is retained.

2.10. Growth Performance and Biological Evaluation

Feed intake was recorded daily, and body weight was measured weekly throughout the experimental period under standardized conditions. Body weight gain percentage (BWG%) and feed efficiency ratio (FER) were calculated from the recorded body weight and feed-intake data.

At the end of the experimental period, the relevant organs were collected and weighed, and relative organ weights were calculated as the percentage of organ weight relative to final body weight.

The equations used for BWG%, FER, and relative organ weight should be explicitly reported to ensure reproducibility.

2.11. Biochemical Analyses

Serum biochemical analyses were performed to evaluate the effects of dietary coriander supplementation on glucose metabolism, lipid metabolism, hepatic function, and renal function.

The biochemical parameters were determined using commercially available diagnostic kits and the corresponding enzymatic or colorimetric procedures according to the manufacturers' instructions. The name of each kit manufacturer, assay method,

wavelength, and measurement units should be specified for each biochemical parameter to ensure methodological reproducibility.

2.11.1. Blood Glucose

Serum glucose concentration was determined using a commercial enzymatic colorimetric assay according to the manufacturer's instructions. Glucose concentration was included as a metabolic outcome because disturbances in glucose homeostasis are associated with metabolic dysfunction. Previous experimental research has reported hypoglycemic effects of an aqueous coriander seed extract in obese, hyperglycemic, and hyperlipidemic *Meriones shawi* rats (Aissaoui et al., 2011).

2.11.2. Lipid Profile

The serum lipid profile was evaluated by determining total cholesterol (TC), triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) using commercial diagnostic assays according to the manufacturers' instructions. Low-density lipoprotein cholesterol (LDL-C) and very-low-density lipoprotein cholesterol (VLDL-C) were calculated only if these parameters were actually derived from the measured lipid concentrations according to a predefined calculation method.

Because commonly used LDL-C equations such as the Friedewald equation have limitations in rats, particularly in hypercholesterolemic animals, the specific calculation method used in the present study should be explicitly reported rather than described simply as an "established method" (Sánchez-Muniz et al., 2008).

Previous experimental research has reported hypolipidemic effects of an aqueous coriander seed extract in metabolically impaired *Meriones shawi* rats (Aissaoui et al., 2011).

2.11.3. Hepatic Function

Hepatic function was assessed by determining serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities, together with any additional hepatic biomarkers actually measured in the study. All assays were performed using commercial diagnostic kits according to the manufacturers' instructions.

Previous experimental research has reported protective effects of *C. sativum* extracts against carbon tetrachloride-induced hepatotoxicity in rats (Sreelatha et al., 2009). However, because that study used a CCl₄-induced hepatotoxicity model rather than a diet-induced obesity model, its findings are considered supporting evidence for the biological activity of coriander rather than direct evidence of an effect on obesity-associated liver dysfunction.

2.11.4. Renal Function

Renal function was assessed by determining the serum renal biomarkers actually measured in the study, such as urea and creatinine, using commercial diagnostic kits according to the manufacturers' instructions.

Aissaoui et al. (2011) measured urea and creatinine in *Meriones shawi* rats treated with an aqueous coriander seed extract and reported no significant effect on these parameters. Therefore, this study may be cited as relevant background evidence for the assessment of renal biochemical parameters, but it should not be cited as evidence that coriander improves renal function.

2.12. Statistical Analysis

Data were expressed as mean $\pm$ standard deviation (SD). Differences among the five experimental groups were analyzed using one-way analysis of variance (ANOVA). When a significant overall difference was detected, a prespecified post hoc multiple-comparison test was applied to determine differences between individual groups. Statistical significance was set at $p < 0.05$.

The statistical analysis should also specify the statistical software used, the exact number of animals included in each analysis, the experimental unit, and the procedures used to assess the assumptions underlying the statistical tests, in accordance with ARRIVE 2.0 recommendations (Percie du Sert et al., 2020).

2.13. Ethical Considerations

All experimental procedures involving animals were reviewed and approved by the Institutional Research Ethics Committee of Al-Baha University, Saudi Arabia (Approval No. 47119677; dated 17 May 2026). The study was conducted in accordance

with the approved animal-care protocol and applicable principles for the humane care and use of laboratory animals. Appropriate measures were taken throughout the study to minimize animal stress, discomfort, and unnecessary suffering.

3. RESULTS

3.1 Chemical composition of dried coriander seed powder

The proximate chemical composition of the dried coriander seed powder is presented in Figure 2. The moisture content was $8.10 \pm 0.03\%$, while crude protein, crude fat, ash, and crude fiber contents were $12.50 \pm 0.05\%$, $9.50 \pm 0.04\%$, $5.80 \pm 0.03\%$, and $9.80 \pm 0.05\%$, respectively, on a wet-weight basis. The carbohydrate content, calculated by difference, was $54.30 \pm 0.14\%$. The corresponding values calculated on a dry-weight basis were $13.60 \pm 0.06\%$ for crude protein, $10.34 \pm 0.05\%$ for crude fat, $6.31 \pm 0.04\%$ for ash, $10.66 \pm 0.06\%$ for crude fiber, and $59.09 \pm 0.15\%$ for carbohydrates. The estimated energy value was 358.50 ± 0.48 kcal/100 g on a wet-weight basis and increased to 390.10 ± 0.52 kcal/100 g on a dry-weight basis.

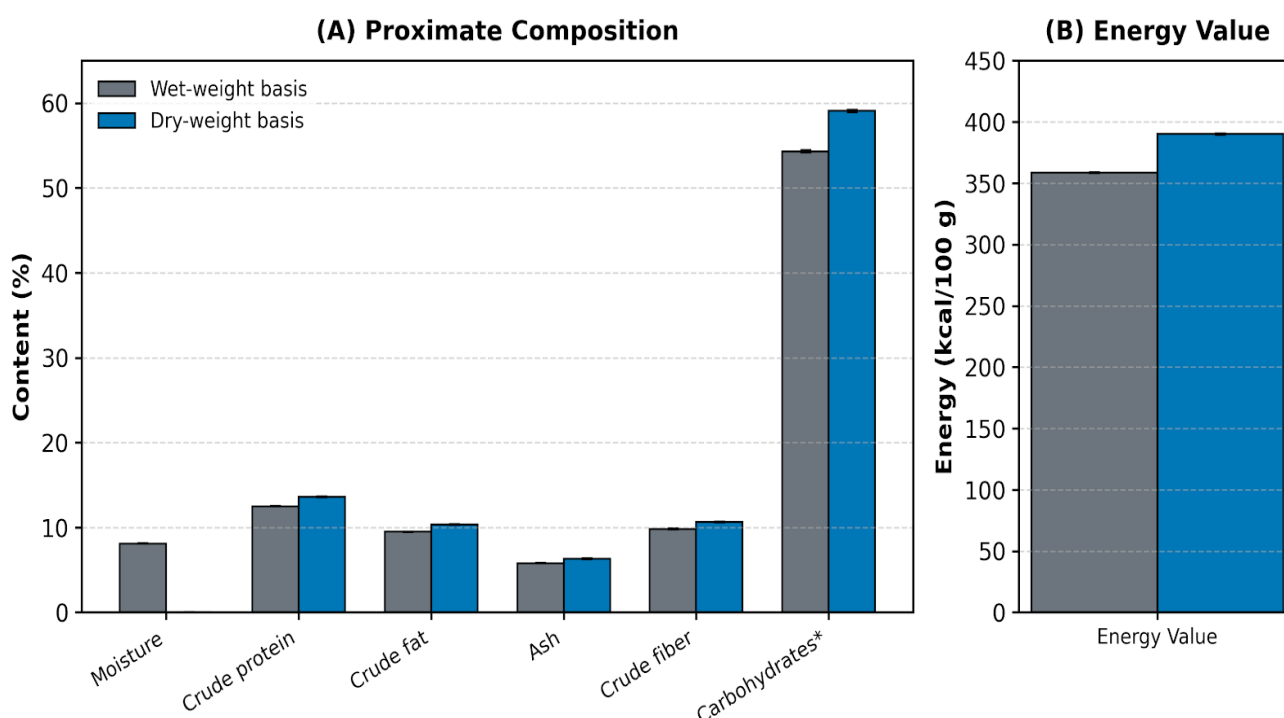


Figure 2: Proximate composition and energy value of dried coriander seed powder on wet-weight and dry-weight bases. Values are expressed as mean $\pm$ standard deviation (SD). *Carbohydrate content was calculated by difference. Dry-weight basis values were calculated after correction for moisture content.

The antioxidant-related characteristics of the coriander seed powder are presented in Figure 3. The total phenolic content was 183.45 ± 2.10 mg GAE/100 g dry matter, whereas the total flavonoid content was 62.80 ± 1.15 mg RE/100 g dry matter. The DPPH radical-scavenging capacity of the coriander seed powder was 41.25 ± 0.90 mg Trolox/100 g dry matter. These findings indicate that the ground coriander seeds contained measurable amounts of phenolic and flavonoid compounds and exhibited DPPH radical-scavenging activity under the experimental conditions.

Bioactive Compounds and Antioxidant Activity of Coriander Seed Powder

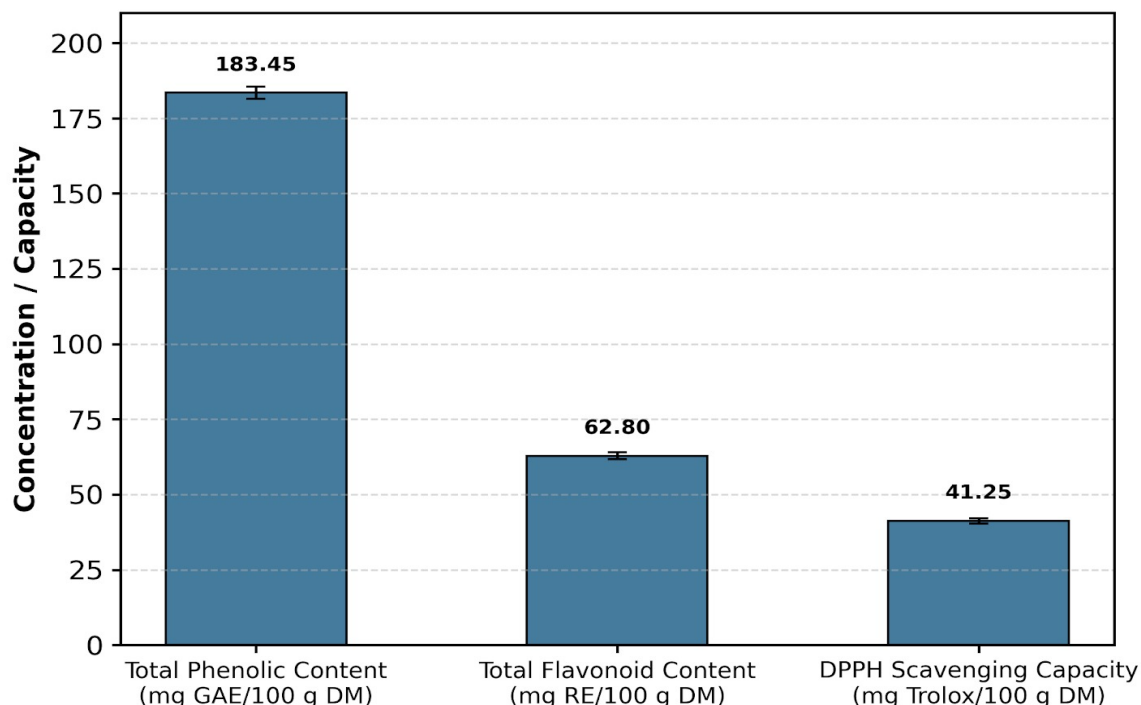


Figure 3: Total phenolic and flavonoid contents and DPPH radical-scavenging capacity of coriander seed powder. Values represent mean $\pm$ standard deviation (SD)..

3.2. Biological evaluation

The effects of dietary supplementation with ground coriander seeds on body weight gain (BWG), feed intake (FI), and feed efficiency ratio (FER) in obese rats are presented in Figure 4. The obese control group recorded the highest BWG (58.23 ± 0.32 g), which was significantly greater than that of the negative control group (20.15 ± 0.20 g; $p < 0.05$). Dietary supplementation with ground coriander seeds at levels of 2%, 4%, and 5% significantly reduced BWG to 45.62 ± 0.41 , 37.84 ± 0.35 , and 31.76 ± 0.28 g, respectively, compared with the obese control group. The reduction in BWG was more pronounced at the higher supplementation levels.

With respect to feed intake, the obese control group showed the highest value (24.45 ± 1.24 g/day), whereas the negative control group recorded 18.45 ± 1.24 g/day. Rats fed diets containing 2%, 4%, and 5% ground coriander seeds exhibited feed intake values of 22.85 ± 1.18 , 21.32 ± 1.15 , and 20.15 ± 1.10 g/day, respectively. The 4% and 5% coriander-supplemented groups showed significantly lower feed intake than the obese control group ($p < 0.05$), while the difference observed in the 2% group was not statistically significant.

The FER also differed among the experimental groups. The obese control group recorded a FER of 0.103 ± 0.003 , whereas the corresponding values for rats receiving 2%, 4%, and 5% ground coriander seeds were 0.091 ± 0.003 , 0.082 ± 0.002 , and 0.076 ± 0.002 , respectively. The 4% and 5% supplementation levels resulted in significantly lower FER values than the obese control group ($p < 0.05$). Overall, supplementation with ground coriander seeds produced a dose-dependent reduction in BWG and was accompanied by decreases in feed intake and feed efficiency, with the most pronounced effects observed at the 5% supplementation level.

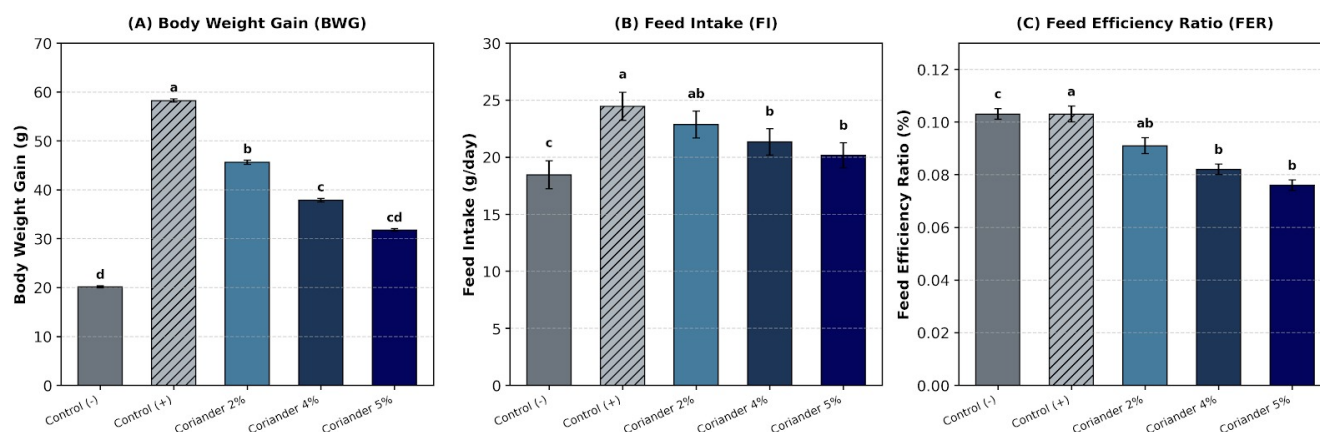


Figure 4. Effects of dietary supplementation with ground coriander seeds on body weight gain (BWG), feed intake (FI), and feed efficiency ratio (FER) in obese rats. Data are presented as mean $\pm$ standard deviation (n = 4). Within each parameter, means with different superscript letters differ significantly ($p < 0.05$).

3.3 Biochemical Analysis

• Results (Serum Lipid Profile)

The effect of dietary supplementation with ground coriander seeds on the serum lipid profile of obese rats is presented in **Figure 5**. The obese control group showed a significant elevation in serum total cholesterol (TC), triglycerides (TG), LDL-C, and VLDL-C levels, accompanied by a significant reduction in HDL-C, compared with the negative control group ($p < 0.05$). The serum TC concentration increased from 71.45 ± 4.74 mg/dL in the negative control group to 159.16 ± 5.18 mg/dL in the obese control group. Dietary supplementation with ground coriander seeds at levels of 2%, 4%, and 5% significantly reduced serum TC to 118.60 ± 4.23 , 101.35 ± 4.66 , and 86.72 ± 3.77 mg/dL, respectively. The greatest reduction was observed in rats receiving the 5% coriander seed diet.

A similar pattern was observed for serum TG levels. The obese control group recorded the highest TG concentration (129.35 ± 2.61 mg/dL), whereas the negative control group showed the lowest value (67.20 ± 3.24 mg/dL). Supplementation with 2%, 4%, and 5% ground coriander seeds reduced TG levels to 101.42 ± 4.38 , 88.43 ± 4.61 , and 74.78 ± 3.51 mg/dL, respectively. These reductions were statistically significant compared with the obese control group ($p < 0.05$), with the 5% supplementation level producing the most pronounced improvement.

Regarding HDL-C, the obese control group exhibited a significantly lower concentration (32.88 ± 2.50 mg/dL) than the negative control group (45.39 ± 2.60 mg/dL). Dietary supplementation with 2%, 4%, and 5% ground coriander seeds increased HDL-C levels to 37.85 ± 2.18 , 40.72 ± 2.26 , and 43.56 ± 2.47 mg/dL, respectively. The increase in HDL-C was more evident with increasing coriander seed supplementation, and the 5% treatment produced a value approaching that of the negative control group.

Serum LDL-C and VLDL-C concentrations followed a pattern similar to that observed for TC and TG. The obese control group recorded the highest LDL-C and VLDL-C concentrations (57.60 ± 1.77 and 25.87 ± 0.13 mg/dL, respectively), compared with 12.62 ± 1.74 and 13.44 ± 0.21 mg/dL, respectively, in the negative control group. Administration of ground coriander seeds at 2%, 4%, and 5% progressively decreased LDL-C levels to 40.46 ± 1.65 , 32.94 ± 1.75 , and 28.20 ± 1.87 mg/dL, respectively. Similarly, VLDL-C levels decreased to 20.28 ± 0.16 , 17.69 ± 0.20 , and 14.96 ± 0.70 mg/dL, respectively. Overall, the findings demonstrate that dietary inclusion of ground coriander seeds improved the serum lipid profile of obese rats, with the 5% supplementation level producing the most favorable overall lipid response.

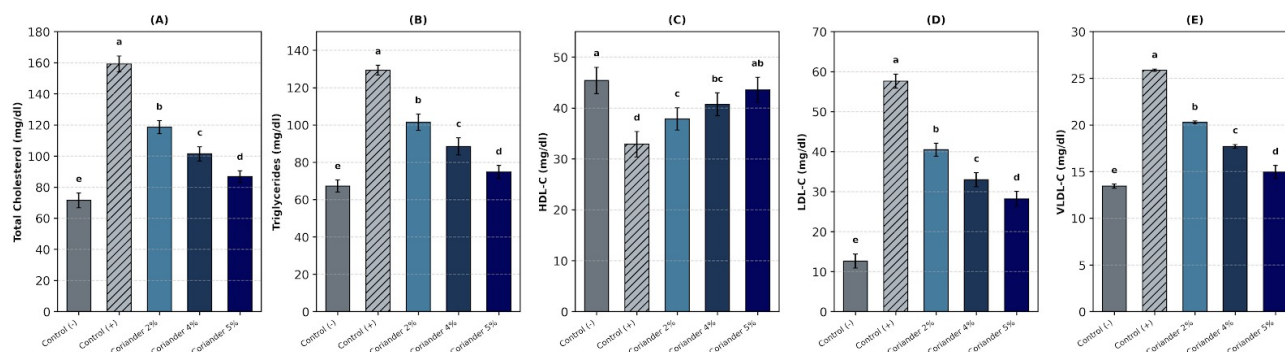


Figure 5. Serum lipid profile of obese rats following dietary supplementation with ground coriander seeds, including (A) total cholesterol (TC), (B) triglycerides (TG), (C) high-density lipoprotein cholesterol (HDL-C), (D) low-density lipoprotein cholesterol (LDL-C), and (E) very-low-density lipoprotein cholesterol (VLDL-C). Data are presented as mean $\pm$ standard deviation ($n = 4$). For each parameter, means designated by different superscript letters are significantly different ($p < 0.05$).

• Results (Liver Function)

The effects of dietary supplementation with dried ground coriander seeds on liver function biomarkers and serum protein levels in obese rats are presented in **Figure 6**. The obese control group exhibited significantly higher serum activities of ALT, AST, and ALP than the negative control group ($p < 0.05$), with values of 92.45 ± 2.05 , 83.92 ± 1.08 , and 71.50 ± 1.05 U/L, respectively, compared with 29.85 ± 1.82 , 18.52 ± 1.15 , and 30.12 ± 1.54 U/L in the negative control group. Dietary supplementation with dried ground coriander seeds resulted in a significant reduction in the activities of these liver enzymes compared with the obese control group. At supplementation levels of 2%, 4%, and 5%, ALT activity decreased to 76.80 ± 1.55 , 63.75 ± 1.14 , and 50.92 ± 1.65 U/L, respectively. Similarly, AST activity decreased to 67.90 ± 1.04 , 54.80 ± 1.18 , and 41.80 ± 1.12 U/L, whereas ALP activity declined to 59.85 ± 1.22 , 47.65 ± 1.10 , and 39.75 ± 1.05 U/L, respectively. The progressive reduction in liver enzyme activities with increasing coriander supplementation was most pronounced at the 5% level.

Regarding serum protein status, the obese control group showed significantly lower total protein and serum albumin concentrations than the negative control group ($p < 0.05$). Total protein decreased from 5.42 ± 0.10 g/dL in the negative control group to 3.68 ± 0.08 g/dL in the obese control group, while serum albumin decreased from 3.52 ± 0.19 to 2.10 ± 0.03 g/dL. Supplementation with dried ground coriander seeds partially restored these parameters. Total protein concentrations increased to 4.52 ± 0.15 , 4.81 ± 0.11 , and 4.98 ± 0.10 g/dL in the 2%, 4%, and 5% coriander-supplemented groups, respectively. Serum albumin concentrations similarly increased to 2.58 ± 0.04 , 2.89 ± 0.05 , and 3.12 ± 0.07 g/dL, respectively. The highest values of both total protein and serum albumin were observed in rats receiving the 5% coriander seed diet.

Overall, dietary supplementation with dried ground coriander seeds improved the assessed liver function biomarkers and serum protein profile in obese rats. The magnitude of improvement increased with the level of coriander supplementation, with the 5% treatment producing the most favorable response among the tested dietary levels.

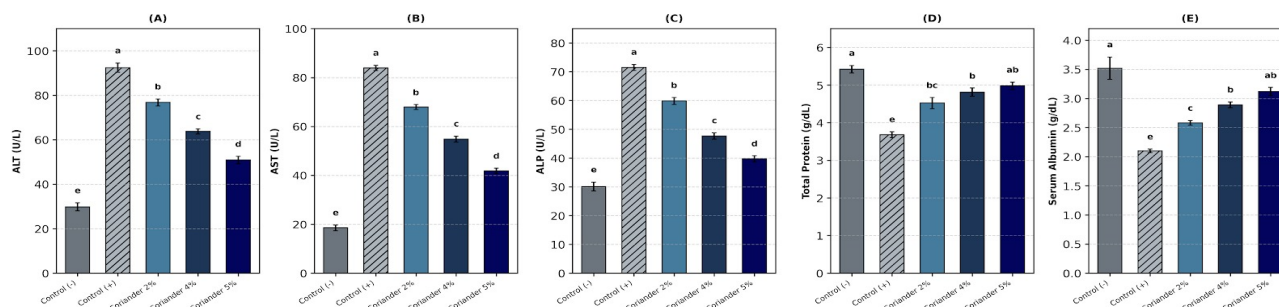


Figure 6. Effects of dietary supplementation with dried ground coriander seeds on liver function biomarkers and serum protein levels in obese rats: (A) alanine aminotransferase (ALT), (B) aspartate aminotransferase (AST), (C) alkaline phosphatase (ALP), (D) total protein, and (E) serum albumin. Data are presented as mean $\pm$ standard deviation ($n = 4$). For each parameter, means bearing different superscript letters are significantly different ($p < 0.05$).

Results (Renal Function Biomarkers)

The effects of dietary supplementation with dried ground coriander seeds on serum uric acid, urea, and creatinine levels in obese rats are presented in **Figure 7**. The obese control group exhibited significantly higher serum concentrations of uric acid, urea, and creatinine than the negative control group ($p < 0.05$). Uric acid concentration increased from 6.72 ± 0.12 mg/dL in the negative control group to 9.18 ± 0.10 mg/dL in the obese control group, while serum urea increased from 23.15 ± 0.18 to 34.38 ± 0.25 mg/dL. Similarly, creatinine concentration increased from 0.94 ± 0.02 to 1.40 ± 0.02 mg/dL.

Dietary supplementation with dried ground coriander seeds significantly attenuated these increases in a dose-related manner. In rats receiving 2%, 4%, and 5% coriander seeds, serum uric acid concentrations decreased to 7.38 ± 0.18 , 6.80 ± 0.19 , and 6.25 ± 0.22 mg/dL, respectively. Serum urea levels were also reduced to 27.85 ± 0.28 , 24.75 ± 0.12 , and 22.10 ± 0.14 mg/dL, respectively. The corresponding serum creatinine concentrations were 1.15 ± 0.04 , 1.01 ± 0.03 , and 0.88 ± 0.03 mg/dL, respectively.

The greatest reductions in serum uric acid and urea were observed in rats receiving the 5% coriander seed diet, with values approaching or slightly below those of the negative control group. Creatinine levels also declined progressively with increasing coriander supplementation, and the 4% and 5% treatments showed values statistically comparable with the negative control group based on the superscript lettering. Overall, dietary supplementation with dried ground coriander seeds improved the measured renal function-related biochemical parameters in obese rats, with the 5% supplementation level producing the most pronounced overall response.

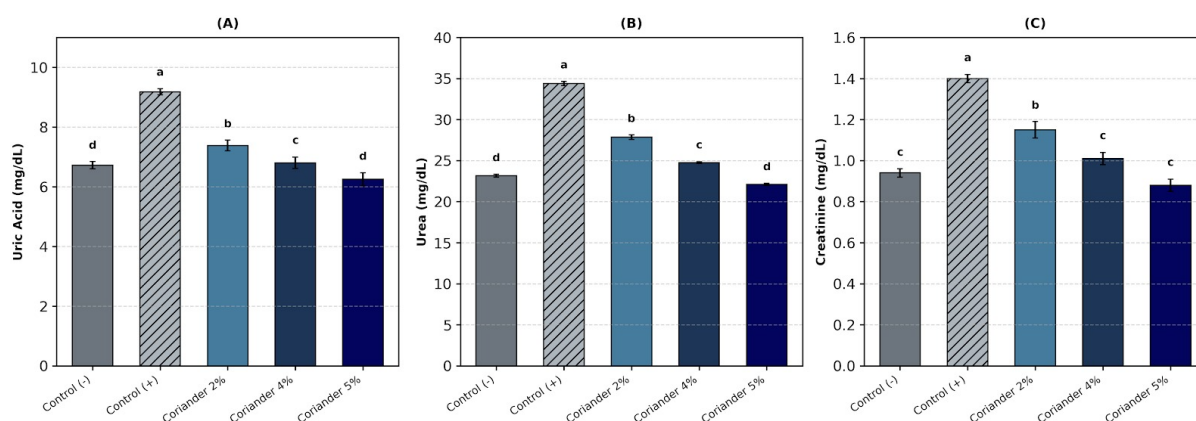


Figure 7. Effects of dietary supplementation with dried ground coriander seeds on serum uric acid, urea, and creatinine concentrations in obese rats: (A) uric acid, (B) urea, and (C) creatinine. Data are presented as mean $\pm$ standard deviation ($n = 4$). For each parameter, means bearing different superscript letters are significantly different ($p < 0.05$).

• Results (Serum Glucose Levels)

The effect of dietary supplementation with dried ground coriander seeds on serum glucose levels in obese rats is presented in **Figure 8**. The obese control group exhibited a significantly higher serum glucose concentration (222.45 ± 1.35 mg/dL) than the negative control group (91.25 ± 1.95 mg/dL; $p < 0.05$).

Dietary supplementation with dried ground coriander seeds significantly reduced serum glucose levels compared with the obese control group. Serum glucose concentrations decreased to 146.10 ± 2.15 , 120.85 ± 1.40 , and 100.80 ± 1.15 mg/dL in rats receiving diets supplemented with 2%, 4%, and 5% dried ground coriander seeds, respectively ($p < 0.05$). The reduction in serum glucose became progressively more pronounced with increasing levels of coriander supplementation.

Among the supplemented groups, the 5% coriander treatment produced the lowest serum glucose concentration, which was 100.80 ± 1.15 mg/dL and approached the value observed in the negative control group. Overall, dietary supplementation with dried ground coriander seeds improved the elevated serum glucose levels associated with obesity, with the 5% supplementation level producing the most pronounced reduction among the tested dietary treatments.

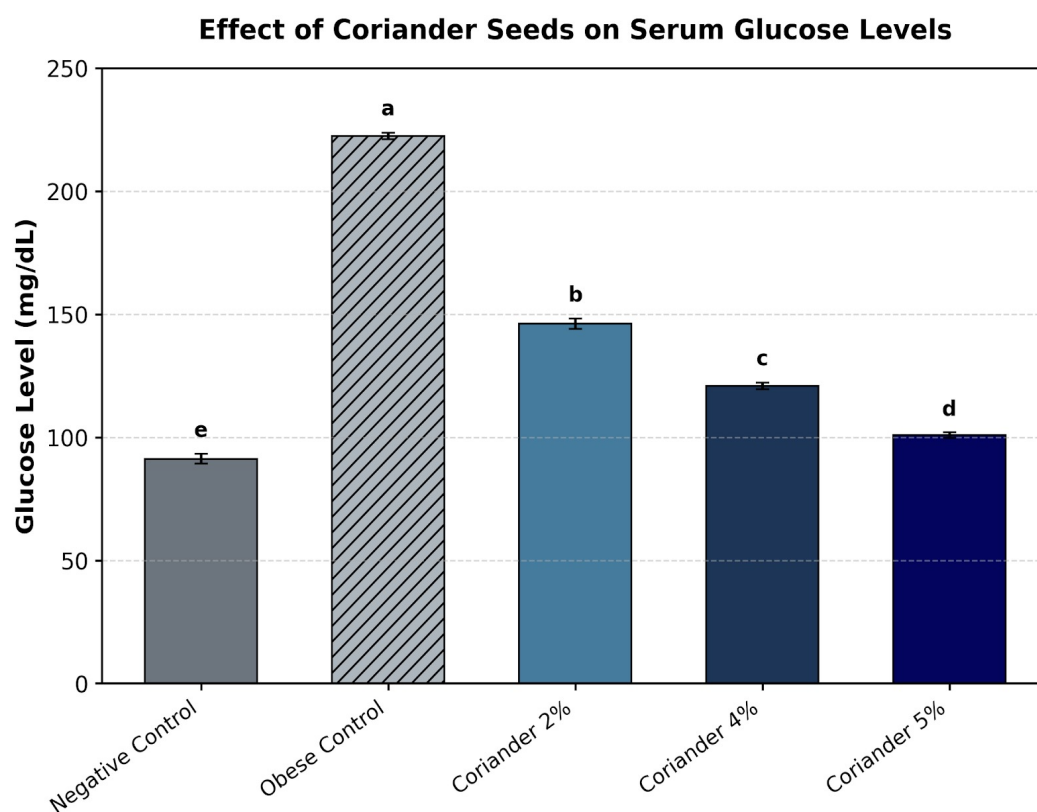


Figure 8. Effects of dietary supplementation with dried ground coriander seeds on serum glucose levels in obese rats. Data are presented as mean $\pm$ standard deviation ($n = 4$). Means bearing different superscript letters are significantly different ($p < 0.05$).

4. Discussion

The present study investigated the metabolic and biochemical effects of dietary supplementation with dried ground coriander seeds (*Coriandrum sativum* L.) in rats with experimentally induced obesity. The findings demonstrated that obesity was associated with marked alterations in body weight gain, serum glucose, lipid metabolism, hepatic biomarkers, serum protein concentrations, and renal function-related parameters. Dietary incorporation of coriander seed powder at 2%, 4%, and 5% attenuated several of these abnormalities, with the 5% supplementation level generally producing the greatest numerical improvement. These findings support the view that coriander seeds may exert multiple metabolic effects rather than acting on a single biochemical pathway.

4.1. Chemical and antioxidant characteristics of coriander seeds

The coriander seed powder used in the present study contained measurable amounts of protein, fat, fiber, ash, and carbohydrates, together with phenolic and flavonoid compounds and detectable DPPH radical-scavenging activity. The total phenolic and flavonoid contents were 183.45 ± 2.10 mg GAE/100 g dry matter and 62.80 ± 1.15 mg RE/100 g dry matter, respectively, while the DPPH radical-scavenging capacity was 41.25 ± 0.90 mg Trolox/100 g dry matter. The presence of these phytochemical constituents is relevant when interpreting the biological effects observed in the treated animals. Coriander seeds contain several classes of bioactive compounds, including volatile constituents, fatty acids, sterols, tocopherols, carotenoids, and phenolic compounds. Their concentrations can vary substantially according to genotype, geographical origin, cultivation conditions, maturity, and processing procedures. Consequently, differences in the chemical composition of coriander preparations may contribute to variation among experimental studies. (Wei et al. 2019) emphasized this compositional variability and reported antioxidant and other biological activities associated with coriander and its constituents. The antioxidant activity observed in the present coriander powder may be particularly relevant in the context of obesity. Excess nutrient availability and metabolic dysfunction can increase oxidative stress, which may contribute to disturbances in glucose and lipid metabolism and to tissue injury. However, the present study measured antioxidant capacity in the plant material rather than oxidative stress biomarkers in the experimental animals. Therefore, the antioxidant findings support a potential biological explanation for some of the observed effects but do not establish that an antioxidant mechanism was responsible for the metabolic improvements.

4.2. Effects on body weight gain and feed intake

The obese control rats exhibited substantially greater body weight gain and feed intake than the negative control animals. Supplementation with coriander seed powder reduced body weight gain at all tested concentrations, and the reduction became progressively greater at the higher supplementation levels. Feed intake was also reduced, particularly in the 4% and 5% groups, while feed efficiency was lower in these groups than in the obese control group.

The reduction in body weight gain is of particular interest because body-weight regulation represents an important component of the metabolic phenotype associated with obesity. Nevertheless, the present data do not establish whether the lower weight gain resulted primarily from reduced energy intake, altered nutrient utilization, changes in energy expenditure, or a combination of these factors. The concomitant reduction in feed intake suggests that differences in dietary consumption contributed to the lower weight gain observed in the coriander-supplemented groups (Sahib et al., 2013). The phytochemical composition of coriander provides a possible basis for biological activity, but direct mechanisms controlling appetite or energy expenditure were not examined in the present experiment. Previous investigations have nevertheless reported metabolic effects of coriander preparations in experimental animals, supporting the possibility that coriander may influence several components of metabolic homeostasis simultaneously (Wei et al., 2019). Such comparisons should be made cautiously because previous studies have used different plant parts, extracts, doses, and experimental models.

4.3. Effects on serum glucose

A marked elevation in serum glucose was observed in the obese control group, whereas dietary supplementation with coriander seed powder significantly reduced glucose concentrations. Serum glucose declined from 222.45 ± 1.35 mg/dL in the obese control group to 146.10 ± 2.15 , 120.85 ± 1.40 , and 100.80 ± 1.15 mg/dL following supplementation with 2%, 4%, and 5% coriander seed powder, respectively. The glucose concentration in the 5% group approached that of the negative control group.

The present finding is consistent with previous experimental evidence indicating that *Coriandrum sativum* may influence glucose homeostasis. Aissaoui et al. (2011) reported that an aqueous extract of coriander seeds reduced plasma glucose and improved metabolic parameters in rats exhibiting an obese-hyperglycemic-hyperlipidemic phenotype. Although that study evaluated an aqueous seed extract rather than whole ground seeds incorporated into the diet, the direction of the glucose response is consistent with the present findings. Experimental evidence involving dietary plant powders has also demonstrated improvements in glucose concentrations in experimental models. For example, Abd Elmegeed and Alzahrani (2022) reported significant reductions in serum glucose in obese rats receiving graded concentrations of *Cichorium* powder, with the lowest glucose concentration observed at the highest tested inclusion level. Similarly, Alzahrani et al. (2025) reported significant reductions in glucose following administration of graded concentrations of *Ammi visnaga* to hyperglycemic rats, together with

improvements in several biochemical parameters. These findings provide methodological support for the present observation that dietary supplementation with plant powders may influence glucose concentrations under experimental conditions, although the plant species, disease model, and experimental protocols differed among studies.

Further support can be obtained from the study of Mashnafi et al. (2025), in which graded dietary supplementation with thyme powder was investigated in high-calorie-diet-induced obese rats. The authors reported that obesity was associated with increased serum glucose and that dietary supplementation with 2%, 4%, and 6% thyme powder reduced the elevated glucose concentrations, together with reductions in body-weight gain. Likewise, Alzahrani et al. (2024) evaluated graded dietary supplementation with *Ziziphus* fruit powder in hyperglycemic Sprague-Dawley rats and assessed serum biochemical parameters after 28 days of treatment. Although the experimental model differed from the present HFD-induced obesity model, the study provides additional evidence for the use of graded plant-powder supplementation in evaluating changes in glucose and related metabolic variables.

The hypoglycemic effect observed in the present study may be related to the phytochemical composition of coriander and its potential influence on glucose metabolism. However, the present experiment did not measure insulin concentrations, glucose tolerance, or molecular markers of insulin signaling. Therefore, it would be inappropriate to attribute the observed reduction in serum glucose to a specific mechanism, such as enhanced insulin secretion or improved insulin sensitivity. The available evidence supports an association between coriander supplementation and improved glucose concentrations, but the precise biological mechanisms remain to be established.

The progressive reduction in glucose observed with increasing coriander supplementation is nevertheless noteworthy. Because the present experiment compared three dietary inclusion levels, the findings suggest a concentration-associated response under the conditions of the present study. A similar approach using graded dietary concentrations has been applied in experimental studies of other plant powders, including *Cichorium*, thyme, and *Ammi visnaga* (Abd Elmeged & Alzahrani, 2022; Alzahrani et al., 2025; Mashnafi et al., 2025). Nevertheless, formal dose-response analysis would be required before describing this relationship as a definitive dose-dependent pharmacological effect.

4.4. Effects on serum lipid profile

The obese control rats exhibited a pronounced dyslipidemic profile, characterized by increased serum total cholesterol (TC), triglycerides (TG), LDL-C, and VLDL-C, together with a reduction in HDL-C. Dietary supplementation with coriander seed powder attenuated these alterations, with the 5% supplementation group showing the greatest overall improvement in the lipid profile. The present findings are consistent with experimental and recent clinical evidence supporting the lipid-modulating potential of *Coriandrum sativum* seeds. Early experimental work demonstrated that dietary coriander seeds exerted hypolipidemic effects in rats fed a high-fat, cholesterol-enriched diet. Chithra and Leelamma (1997) reported reductions in total cholesterol and triglycerides, together with decreased LDL- plus VLDL-cholesterol and increased HDL-cholesterol in coriander-treated rats. The authors proposed that increased plasma lecithin-cholesterol acyltransferase activity, enhanced hepatic bile acid synthesis, and increased fecal elimination of cholesterol metabolites could contribute to the observed hypocholesterolemic effect. Similarly, Dhanapakiam et al. (2008) reported favorable changes in lipid metabolism following dietary incorporation of coriander seeds in rats receiving a high-fat diet supplemented with cholesterol. More recent evidence provides additional support for the lipid-modulating effects of coriander. In a randomized, double-blind, placebo-controlled clinical trial published in 2025, Zaman et al. reported that six weeks of coriander seed powder supplementation significantly reduced serum total cholesterol, triglycerides, and LDL-C in adults with type 2 diabetes mellitus, although anthropometric measures did not change substantially. Although this clinical population differs from the HFD-induced obese rat model used in the present study, the findings provide contemporary human evidence that coriander seed powder may favorably influence circulating lipid parameters. In addition, (Hardiany et al. 2026) investigated coriander seed ethanolic extract in high-fat diet-induced obese rats and reported a significant reduction in plasma triglycerides, accompanied by improvements in hepatic oxidative stress, liver function, and histological alterations. Notably, the extract did not significantly reduce body weight, Lee index, or body mass index, indicating that lipid and hepatic benefits may occur independently of a measurable reduction in overall body weight. Further recent experimental evidence has demonstrated that aqueous *C. sativum* seed extract can improve diet-induced glucolipid metabolic disturbances. In a 2026 study, coriander supplementation reduced serum total cholesterol, triglycerides, and LDL-C and improved hepatic lipid accumulation and liver injury in animals exposed to a high-sugar/high-fat diet. Collectively, these findings are compatible with the present observation that coriander supplementation was

associated with an improved serum lipid profile. The mechanisms underlying these effects, however, should be interpreted cautiously. Previous studies have proposed alterations in cholesterol metabolism, bile acid synthesis, cholesterol esterification, and fecal sterol elimination as possible contributors to the hypolipidemic activity of coriander (Chithra & Leelamma, 1997; Dhanapakiam et al., 2008). These mechanisms cannot be directly confirmed in the present study because cholesterol-metabolizing enzymes, bile acid synthesis, lecithin–cholesterol acyltransferase activity, and fecal sterol excretion were not measured. Therefore, the present results demonstrate an association between dietary coriander supplementation and improvement in circulating lipid parameters rather than establishing a specific molecular mechanism. The greater improvement observed in the 5% coriander group is also noteworthy and suggests that the magnitude of the lipid response may vary according to the dietary inclusion level under the conditions of the present experiment. Nevertheless, this pattern should be interpreted as a concentration-associated response rather than definitive evidence of a pharmacological dose-response relationship. Moreover, differences among studies in coriander preparation, including whole seed powder, aqueous extracts, and ethanolic extracts, as well as differences in dose, duration, animal model, and background diet, may contribute to variation in the magnitude and pattern of lipid responses.

4.5. Effects on hepatic biomarkers and serum proteins

The obese control group exhibited significant increases in serum ALT, AST, and ALP activities, accompanied by reductions in serum total protein and albumin concentrations. Dietary supplementation with coriander seed powder attenuated the elevations in hepatic enzyme activities and increased serum total protein and albumin levels, with the most pronounced changes observed in the 5% supplementation group. The elevation of circulating hepatic enzymes in obese animals is consistent with metabolic disturbances induced by excessive dietary fat intake and hepatic lipid accumulation. Experimental high-fat feeding has been associated with increased serum aminotransferase activities and the development of hepatic steatosis and metabolic dysfunction in rodents. Accordingly, the lower ALT, AST, and ALP activities observed following coriander supplementation may indicate an improvement in metabolic and hepatic status. However, serum enzyme activities alone cannot establish the presence, severity, or absence of specific histopathological liver lesions.

The present findings are consistent with experimental evidence supporting the hepatoprotective potential of *Coriandrum sativum*. (Sreelatha et al., 2009) reported that *C. sativum* extracts reduced serum hepatic enzyme activities and oxidative damage in rats subjected to carbon tetrachloride-induced hepatotoxicity, accompanied by improvements in hepatic antioxidant status and histopathological alterations. Although this model involved chemically induced liver injury rather than diet-induced obesity, the findings provide evidence that coriander-derived preparations can influence biochemical and structural indicators of hepatic injury.

More recent studies provide evidence that is more directly relevant to diet-induced metabolic dysfunction. Gu et al. (2024) investigated *C. sativum* leaf extract in high-fat diet-induced metabolic dysfunction-associated steatotic liver disease in C57BL/6 mice. Treatment with the extract improved serum biochemical parameters, reduced hepatic lipid accumulation, and attenuated histopathological alterations, while modulation of the AMPK-related metabolic pathway and inflammatory signaling was also observed. These findings support the possibility that coriander-derived preparations may exert hepatic benefits in the context of diet-induced metabolic dysfunction. However, this study used a leaf extract rather than coriander seed powder, and therefore its findings should not be considered directly equivalent to the present dietary intervention.

More recently, Hardiany et al. (2026) evaluated an ethanolic extract of coriander seeds in high-fat diet-induced obese rats. Coriander treatment was associated with lower plasma triglyceride concentrations, reduced hepatic oxidative stress, decreased ALT and AST activities, and improvement in liver histological scores. Importantly, the study used a diet-induced obesity model, making it particularly relevant to the present experiment. Nevertheless, the preparation used in that study was an ethanolic seed extract administered at a defined dose, whereas the present study incorporated dried, ground coriander seed powder directly into the diet. Differences in extraction procedure, phytochemical composition, dose, route of administration, and treatment duration should therefore be considered when comparing the magnitude of the hepatic response between the two studies.

Additional recent evidence was provided by Xu et al. (2026), who investigated aqueous *C. sativum* seed extract in mice exposed to a high-sugar/high-fat diet. The extract reduced serum ALT, AST, and alkaline phosphatase activities and improved hepatic lipid accumulation, oxidative stress, and histopathological alterations. The authors also reported

improvements in hepatic metabolic and inflammatory pathways. These findings are particularly relevant because they demonstrate that coriander seed preparations can improve biochemical and structural indicators of liver injury in a diet-induced metabolic dysfunction model. Nevertheless, the study involved mice and an aqueous extract administered by oral gavage, whereas the present experiment involved rats receiving whole coriander seed powder through the diet. Thus, the findings support the biological plausibility of the present results but do not establish that identical mechanisms are responsible.

The improvement in serum total protein and albumin concentrations in the coriander-treated groups may also reflect an overall improvement in metabolic and hepatic status. However, serum protein and albumin concentrations are influenced by several physiological and nutritional factors, and the present experiment did not directly evaluate hepatic protein synthesis, albumin gene expression, or protein turnover. Therefore, the observed increases should be interpreted as biochemical changes accompanying the broader metabolic response rather than as direct evidence of restored hepatic synthetic function.

Overall, the present findings, together with recent experimental evidence, support an association between coriander supplementation and improvement in biochemical indicators of hepatic status in diet-induced metabolic dysfunction. However, because the present study did not directly measure hepatic lipid content, oxidative stress, inflammatory signaling, or molecular markers of hepatic metabolism, mechanistic explanations should remain cautious. The concurrent improvement in serum hepatic enzymes, total protein, and albumin suggests an overall favorable biochemical response to coriander supplementation, particularly at the 5% dietary inclusion level, but does not by itself establish a specific hepatoprotective mechanism.

4.6. Effects on renal function-related biomarkers

Obesity was associated with increased serum uric acid, urea, and creatinine concentrations in the present experiment. Dietary supplementation with coriander seed powder reduced these parameters, with the 5% supplementation group showing the lowest uric acid and urea concentrations and a creatinine concentration approaching that of the negative control group. These findings indicate that coriander supplementation was associated with an improvement in the selected renal biochemical markers under the experimental conditions. This observation is biologically plausible because obesity and excessive dietary fat intake have been associated with metabolic, oxidative, and inflammatory alterations that may adversely affect renal function. Evidence from human studies indicates that overweight and obesity are associated with an increased risk of kidney dysfunction, although the magnitude of this association may vary according to metabolic status and the presence of other risk factors (Valizadeh et al., 2024).

Experimental evidence also supports a potential protective effect of *Coriandrum sativum* against metabolic disturbances that may involve renal function. Aissaoui et al. (2011) reported that administration of coriander seed extract to obese, hyperglycemic, and hyperlipidemic rats improved several metabolic parameters without producing adverse alterations in serum urea or creatinine. More recent experimental studies have further demonstrated beneficial effects of coriander seed preparations in diet-induced metabolic dysfunction. Hardiany et al. (2026) reported that coriander seed ethanolic extract administered to high-fat diet-induced obese rats improved several biochemical and hepatic parameters, including reductions in oxidative stress and liver enzyme activities. Similarly, Xu et al. (2026) reported that aqueous *C. sativum* seed extract improved metabolic disturbances and reduced biochemical indicators of tissue injury in animals exposed to a high-sugar/high-fat diet. Although these recent studies provide biological support for the metabolic effects of coriander, their findings should not be interpreted as direct evidence of renal protection because renal function was not the primary outcome in all of these models.

The reduction in serum uric acid observed following coriander supplementation may also be relevant to the overall metabolic improvement. Elevated uric acid concentrations have been associated with obesity and metabolic dysfunction and may accompany alterations in renal handling of urate (Valizadeh et al., 2024). Nevertheless, the mechanism responsible for the reduction in uric acid in the present study cannot be established because uric acid production, renal urate transport, and urinary uric acid excretion were not directly assessed.

The observed reductions in serum urea and creatinine may therefore suggest an improvement in the biochemical indicators associated with renal status. However, these parameters should not be considered comprehensive measures of renal function. Serum creatinine is influenced by factors such as muscle mass and creatinine production, whereas serum urea can be affected

by dietary protein intake, hydration status, and hepatic metabolism. Likewise, serum uric acid is influenced by both production and renal excretion. Therefore, the present findings should be interpreted as changes in selected renal-related biochemical markers rather than definitive evidence that coriander supplementation prevented or reversed structural renal injury.

More comprehensive assessment, including renal histopathology, urinary biomarkers, urinary protein or albumin excretion, and direct or validated estimates of glomerular filtration, would be required to confirm a renoprotective effect. Accordingly, the present results support an association between dietary coriander supplementation and improvement in serum uric acid, urea, and creatinine concentrations, particularly at the 5% supplementation level, while the underlying renal mechanisms remain to be elucidated.

4.7. Possible relationship between phytochemical composition and the observed metabolic effects

The overall pattern observed in the present study suggests that coriander seed supplementation was associated with simultaneous improvements in glucose regulation, lipid metabolism, hepatic biochemical markers, and selected renal biomarkers. Such a broad response is consistent with the complex phytochemical composition of coriander rather than with the action of a single identified compound. Coriander seeds contain volatile constituents such as linalool as well as phenolic compounds, flavonoids, sterols, fatty acids, tocopherols, and carotenoids. Oxidative stress may provide one potential link between these findings. The coriander powder demonstrated measurable phenolic and flavonoid contents and DPPH radical-scavenging activity in the present study. Previous experimental work has demonstrated antioxidant activity of coriander seed preparations and reported attenuation of oxidative stress in animal models. Nevertheless, because oxidative stress markers were not measured in the tissues or serum of the animals in the current experiment, the antioxidant capacity of the powder should be considered a plausible contributing factor rather than a demonstrated mechanism. Another possible explanation involves the interaction between lipid and glucose metabolism. The improvement in the lipid profile was accompanied by a reduction in serum glucose and body weight gain. Since disturbances in these metabolic pathways frequently coexist in obesity, improvement in one component may occur alongside improvement in others. However, the present study was not designed to establish causal relationships among these outcomes. Additional measurements of insulin, inflammatory cytokines, hepatic lipid content, oxidative stress markers, and metabolic signaling pathways would be needed to clarify the mechanisms involved.

4.8. Overall interpretation and study limitations

Taken together, the present findings indicate that dietary supplementation with dried ground coriander seeds was associated with favorable changes in several metabolic and biochemical parameters in obese rats. The reductions in body weight gain, serum glucose, total cholesterol, triglycerides, LDL-C, VLDL-C, ALT, AST, ALP, uric acid, urea, and creatinine, together with the increase in HDL-C, total protein, and albumin, suggest a broad metabolic response to coriander supplementation. The response was generally more pronounced at the 5% inclusion level. Several limitations should nevertheless be considered. First, the experiment involved a relatively small number of animals, which may limit the statistical power and generalizability of the findings. Second, the study measured biochemical outcomes but did not directly determine insulin sensitivity, inflammatory mediators, oxidative stress biomarkers in the animals, or molecular pathways involved in the response. Third, the present findings were obtained in an experimental rat model and therefore should not be extrapolated directly to human obesity. Finally, comparisons with previous studies should account for differences in coriander preparation, extraction method, dose, duration, animal model, and dietary composition. Despite these limitations, the consistency of the present findings with earlier experimental evidence on the hypoglycemic and hypolipidemic properties of coriander seeds provides supportive evidence for the metabolic activity of *C. sativum*. The results therefore suggest that coriander seed powder may have potential as a dietary component associated with improved metabolic profiles in experimental obesity. Further studies using larger sample sizes and mechanistic endpoints are warranted to determine the pathways responsible for these effects and to establish the reproducibility and safety of different dietary concentrations.

5. CONCLUSION

The present study demonstrated that dietary supplementation with powdered *Coriandrum sativum* L. seeds was associated with favorable changes in several metabolic and biochemical parameters in rats with high-fat diet-induced obesity. Coriander seed powder exhibited measurable phenolic and flavonoid contents and antioxidant activity, supporting its potential biological

activity as a plant-derived dietary component. Dietary incorporation of coriander at 2%, 4%, and 5% was associated with reductions in body weight gain, serum glucose, total cholesterol, triglycerides, LDL-C, and VLDL-C, together with an increase in HDL-C. Improvements were also observed in serum hepatic biomarkers, including ALT, AST, and ALP, as well as in total protein and albumin concentrations. In addition, coriander supplementation was associated with lower serum uric acid, urea, and creatinine concentrations, indicating favorable changes in the selected renal-related biochemical parameters.

Overall, the observed effects across multiple metabolic and organ-related biomarkers suggest that whole coriander seed powder may have potential as a functional dietary component for modulating obesity-associated metabolic disturbances. The generally greater magnitude of improvement observed at the 5% inclusion level suggests a concentration-associated response under the conditions of the present experiment. However, this observation should not be interpreted as definitive evidence of a dose-dependent pharmacological effect, particularly because formal dose-response analysis was not performed.

The findings should be interpreted within the limitations of the experimental design, including the small sample size, the use of an animal model, and the absence of direct measurements of insulin sensitivity, inflammatory mediators, tissue oxidative stress, hepatic lipid accumulation, and molecular signaling pathways. Furthermore, changes in serum urea, creatinine, and uric acid should be regarded as alterations in selected renal-related biomarkers rather than conclusive evidence of renoprotection. Similarly, improvements in serum hepatic enzymes and proteins do not by themselves establish structural recovery or restoration of hepatic function.

Taken together, the present findings provide experimental evidence that dietary coriander seed powder is associated with improvements in several biochemical manifestations of high-fat diet-induced obesity and support further investigation of *C. sativum* seeds as a potential functional food ingredient. Future studies with larger sample sizes, longer intervention periods, comprehensive histopathological evaluation, and mechanistic assessment of glucose, lipid, inflammatory, oxidative, hepatic, renal, and pancreatic pathways are warranted to clarify the biological mechanisms, optimal dietary concentrations, and long-term safety of coriander seed supplementation.

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