

Role of Aqueous *Coriandrum Sativum* Seed Extract in Diet Induced Glucolipid Metabolic Disorder Through Gut-Liver Axis Regulation

Jawahar Vel M V M¹ Dr.V. Sathiya²

¹Ph.D. Research Scholar (Full-Time), Department of Nutrition and Dietetics, PSG College of Arts & Science

²Assistant Professor, Department of Nutrition and Dietetics College of Arts & Science

Abstract

Background: Glucolipid metabolic disorders—encompassing central obesity, non-alcoholic fatty liver disease (NAFLD), insulin resistance, and dyslipidemia—present a critical global health burden driven predominantly by energy-dense, high-sugar high-fat diets (HSFD). Conventional therapeutic agents are frequently accompanied by long-term adverse events, prompting interest in natural dietary interventions. This study investigated the pro-efficacy and molecular mechanisms of aqueous *Coriandrum sativum* seed extract (YS) in an HSFD-induced mouse model.

Methods: Phytochemical profiling of YS was conducted via gas chromatography-mass spectrometry (GC-MS). Male Kunming mice received an HSFD along with oral gavage of YS at 1.0 g/kg/day (YS-L) or 2.0 g/kg/day (YS-H) for eight consecutive weeks. Physiological parameters, glucose/insulin dynamics (HOMA-IR), serum and hepatic lipid profiles (TC, TG, LDL-C), liver enzymes (ALT, AST, AKP), and redox status (SOD, MDA) were quantified. Histopathological changes in hepatic, pancreatic, and colonic tissues were evaluated alongside hepatic gene and protein expression (*FAS*, *NF-κB*, *IL-6*, *PPARα*, *LDLR*). Gut microbiota dynamics were profiled using full-length 16S rRNA gene sequencing paired with PICRUST2 functional predictions.

Results: GC-MS identified nine major constituents in YS, led by 4-ethenyl-2-methoxyphenol (11.62%) and hexadecanoic acid (6.13%). YS treatment dose-dependently mitigated body weight gain, visceral adiposity, hyperglycemia, hyperinsulinemia, and HOMA-IR while rescuing hepatic glycogen storage. YS normalized lipid profiles, lowered ALT, AST, and AKP levels, reduced MDA, and elevated SOD activity. Histopathology confirmed structural proion across hepatic, pancreatic, and colonic tissues. At the molecular level, YS suppressed hepatic expression of lipogenic (*FAS*) and pro-inflammatory (*NF-κB*, *IL-6*) targets while upregulating *PPARα* and *LDLR* protein expression. Full-length 16S rRNA sequencing showed that YS restored microbial diversity, enriching beneficial taxa (*Lactobacillus murinus*, *Lachnospiraceae_UCG-006*, *Streptococcus danieliae*) and suppressing pro-inflammatory *Desulfovibrio*, alongside enriching carbohydrate and energy metabolic pathways.

Conclusion: Aqueous *Coriandrum sativum* seed extract serves as a potent, multi-targeted natural agent capable of reversing diet-induced glucolipid metabolic disorders through coordinated regulation of lipid metabolism, inflammation, and gut microbial ecosystem dynamics along the gut-liver axis.

Keywords: *Coriandrum sativum*; glucolipid metabolic disorder; NAFLD; gut-liver axis; 16S rRNA sequencing; lipid metabolism

1. Introduction

Metabolic syndrome (MetS), type 2 diabetes mellitus (T2DM), dyslipidemia, and non-alcoholic fatty liver disease (NAFLD/MASLD) represent interconnected metabolic crises across the modern world. The widespread adoption of diets high in refined sugars and saturated fats (HSFD) disrupts systemic energy balance, triggering lipid accumulation in visceral adipose depots and liver parenchymal tissue. Visceral adiposity acts as an active endocrine organ, releasing excess free fatty acids and pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). This chronic low-grade inflammatory state blunts insulin signaling pathways, inducing compensatory hyperinsulinemia, enhanced hepatic *de novo* lipogenesis, and elevated very-low-density lipoprotein (VLDL) secretion. Simultaneously, chronic nutrient overload overwhelms mitochondrial respiratory capacity, leading to the excessive generation of reactive oxygen species (ROS). Oxidative stress impairs cellular macromolecules and activates nuclear factor-kappa B (NF-κB) transcription factors, creating a self-perpetuating feedback loop of tissue inflammation and metabolic distress. Beyond direct tissue injury, recent paradigm shifts highlight the **gut-liver axis** as a central regulator of metabolic homeostasis. Diet-induced gut dysbiosis compromises intestinal epithelial barrier integrity, permitting bacterial endotoxins (such as lipopolysaccharides, LPS) to translocate into the portal circulation, directly exacerbating hepatic inflammation and lipid metabolic derangements.

Although pharmacological therapies—including statins, metformin, and GLP-1 receptor agonists—are effective, their long-term clinical utility can be constrained by gastrointestinal intolerance, hepatotoxicity, or myopathy. Consequently, naturally derived botanical products and "medicinal food homology" interventions have attracted attention for their multi-component, multi-target properties. *Coriandrum sativum* L. (coriander), an annual herb belonging to the Apiaceae family, has been used historically across culinary and traditional medicine systems. While leaves and essential oils have been widely studied, the therapeutic efficacy and precise molecular mechanisms of aqueous extracts derived from *Coriandrum sativum* seeds—particularly regarding gut microbial modulation and hepatic lipid gene regulation—remain insufficiently defined.

This study evaluates the therapeutic effects of aqueous *Coriandrum sativum* seed extract in HSFD-fed mice, detailing its impact on glycemic regulation, lipid handling, organ histopathology, hepatic transcriptional networks, and gut microbial ecosystem dynamics.

2. Methodology

2.1 Extraction and GC-MS Phytochemical Profiling

Coarsely ground *Coriandrum sativum* seeds (TongRenTang, Beijing, China) underwent dual aqueous extraction at 100°C (1000 mL for 1 h, followed by 800 mL for 45 min). Concentrated aqueous extracts were partitioned with ethyl acetate, dried under nitrogen stream, and reconstituted for Gas Chromatography-Mass Spectrometry (GC-MS) analysis on a Shimadzu GCMS-QP2010SE system utilizing a DB-5MS capillary column (30 m times 0.25 mm, 0.25 μm). Separation proceeded with helium carrier gas (1.0 mL/min) over a temperature ramp (70°C to 230°C). Mass spectra were recorded in full-scan mode (m/z 40--350) at 70 eV electron ionization and matched against the NIST 17 library (similarity index ≥ 90%).

2.2 Animal Model and Experimental Design

Male Kunming (KM) mice (5 weeks old, 32--34 g, n=32) were housed in specific-pathogen-free (SPF) conditions (24 pm 1°C, 12 h light/dark cycle) at Tianjin University of Traditional Chinese Medicine (Approval No. TCM-LAEC2024162z1030). Mice were randomized into four experimental groups (n=7--8 per group):

1. **ND Group:** Standard chow and drinking water.
2. **HSFD Group:** High-sugar high-fat diet (67% standard chow, 10% lard, 20% sucrose, 2.5% cholesterol, 0.5% sodium cholate) plus 20% fructose drinking solution *ad libitum*.
3. **YS-L Group:** HSFD diet + oral gavage of low-dose aqueous extract (1.0 g/kg/day).
4. **YS-H Group:** HSFD diet + oral gavage of high-dose aqueous extract (2.0 g/kg/day).

Interventions were administered daily for 56 consecutive days (8 weeks). ND and HSFD controls received equivalent volumes of normal saline.

2.3 Biochemical and Histological Assays

Glycemic & Serum Lipid Parameters: Fasting blood glucose (FBG) was monitored using tail-vein blood.

Serum insulin was quantified via enzyme-linked immunosorbent assay (ELISA), and the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) was calculated as:

$$\text{HOMA-IR} = \frac{\text{FBG (mmol/L)} \times \text{FINS (mIU/L)}}{22.5}$$

Biochemical Markers: Serum and hepatic total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (AKP), superoxide dismutase (SOD), malondialdehyde (MDA), and hepatic glycogen were assayed using enzymatic colorimetric commercial kits. **Histopathology:** Paraffin-embedded liver, pancreas, and colon sections (4 μm) were stained with Hematoxylin and Eosin (H&E).

Cryosectioned liver samples (8 μm) were subjected to Oil Red O staining to visualize neutral lipid accumulation.

2.4 Gene and Protein Expression (RT-qPCR and Western Blot)

Total RNA was extracted from hepatic tissue using TransZol Up Plus reagent and reverse-transcribed into cDNA. Quantitative real-time PCR (RT-qPCR) was performed using SYBR Green chemistry targeting *FAS*, *NF-κB*, *PPARα*, *LDLR*, and *IL-6*, normalized to beta-actin using the $2^{-\Delta\Delta CT}$ method. Protein expression levels of *PPARα*, *IL-*

6, NF- κ B, and LDLR were measured by SDS-PAGE and Western blotting with chemiluminescent deion.

2.5 Full-Length 16S rRNA Gene Sequencing and Bioinformatics

Fecal/cecal microbial genomic DNA was isolated, and full-length 16S rRNA genes (V1–V9 regions) were amplified with universal primers 27F and 1492R. Libraries were sequenced on a Pacific Biosciences (PacBio) SMRT platform. Sequence processing and amplicon sequence variant (ASV) dereplication were executed via QIIME2 and DADA2 against the SILVA database (v138.1). Alpha/beta diversity indices, LEfSe biomarker discovery, Spearman correlation analysis, and PICRUST2 functional predictions were performed in R.

3. Results

3.1 Phytochemical Identification of Extract Constituents

GC-MS analysis identified nine primary volatile and lipophilic constituents in the aqueous seed extract:

Peak	Compound Name	Molecular Formula	Retention Time (min)	Relative Concentration (%)
1	2,6-dimethylocta-3,7-diene-2,6-diol	C ₁₀ H ₁₆ O ₂	14.837	1.35%
2	4-ethenyl-2-methoxyphenol	C ₉ H ₁₀ O ₂	21.522	11.62%
3	3-methylhepta-1,6-dien-3-ol	C ₈ H ₁₄ O	23.453	1.08%
4	(2E)-2,6-dimethylocta-2,7-diene-1,6-diol	C ₁₀ H ₁₈ O ₂	24.923	2.58%
5	Dodecanal	C ₁₂ H ₂₄ O	27.528	0.51%
6	Hexadecane	C ₁₆ H ₃₄	31.952	1.10%
7	2,6,10,14-tetramethylhexadecane	C ₂₀ H ₄₂	49.709	1.47%
8	Hexadecanoic acid	C ₁₆ H ₃₂ O ₂	58.223	6.13%
9	Octadecanoic acid	C ₁₈ H ₃₆ O ₂	66.304	4.28%

4-ethenyl-2-methoxyphenol (a vinyl-substituted guaiacol derivative) represented the predominant component (11.62%), supplemented by monoterpene alcohols and long-chain fatty acids.

3.2 Mitigation of Obesity, Visceral Adiposity, and Organ Indices

HSFD-fed mice showed progressive body weight gain over 8 weeks, significantly exceeding ND controls ($p < 0.01$). Administration of YS, particularly YS-H (2.0 g/kg), suppressed weight accumulation starting at week 3 ($p < 0.01$ at week 8). YS-H significantly reduced the liver index ($p < 0.001$ vs. HSFD) and epididymal visceral fat mass ($p < 0.05$),

while partially restoring elevated renal mass indices.

3.3 Improvement of Glycemic Homeostasis and Pancreatic Preservation

HSFD induced severe hyperglycemia, hyperinsulinemia, and insulin resistance:

Fasting Blood Glucose: Significantly elevated in HSFD mice ($p < 0.001$), but restored toward normoglycemia in the YS-H group ($p < 0.001$). **Insulin Resistance:** Elevated HOMA-IR in HSFD mice ($p < 0.001$) was significantly lowered by YS-H ($p < 0.05$). **Hepatic Glycogen:** HSFD-depleted hepatic glycogen stores ($p < 0.001$) were restored following YS treatment ($p < 0.001$ for YS-H). **Pancreatic Histology:** H&E staining of HSFD pancreases revealed distorted islet architecture, irregular borders, and vacuolar degeneration. YS-H preserved islet structural integrity, maintained smooth boundaries, and restored acinar cell alignment.

3.4 Attenuation of Dyslipidemia, Hepatic Steatosis, and Oxidative Stress

YS treatment demonstrated clear lipid-lowering and hepatoprotective actions: **Lipid Profiles:** HSFD elevated serum TC, TG, and LDL-C ($p < 0.001$), all of which were reduced by YS-H ($p < 0.001$). Hepatic TC and TG concentrations were similarly lowered ($p < 0.01$). **Liver Function Markers:** Serum ALT, AST, and AKP levels were elevated in HSFD-fed animals ($p < 0.001$). YS-H treatment reduced ALT ($p < 0.05$), AST ($p < 0.01$), and AKP ($p < 0.001$), indicating attenuation of hepatocellular damage. **Oxidative Status:** Hepatic MDA levels were suppressed, while antioxidant SOD enzymatic activity was increased by YS-H ($p < 0.05$). **Liver Histology:** Oil Red O staining confirmed extensive macrovesicular steatosis in HSFD controls, which was reduced in YS-H-treated mice. H&E staining confirmed diminished hepatocyte ballooning and restored sinusoidal architecture.

3.5 Regulation of Hepatic Lipid Metabolic and Inflammatory Signaling

Transcriptional and translational profiling revealed that YS targets central metabolic pathways: **Lipogenesis:** mRNA expression of *FAS* (Fatty Acid Synthase) was upregulated by HSFD ($p < 0.01$) and suppressed by YS-H ($p < 0.05$). **Inflammatory Cascade:** *NF- κ B* and *IL-6* transcript levels were elevated in HSFD livers ($p < 0.01$) and downregulated by YS-H ($p < 0.01$). Protein expression verified suppression of NF- κ B and IL-6. **Lipid Oxidation & Clearance:** HSFD disrupted *PPAR α* and *LDLR* expression. YS-H significantly increased protein levels of PPAR α and LDLR, supporting hepatic fatty acid oxidation and receptor-mediated low-density lipoprotein clearance.

3.6 Preservation of Colonic Mucosal Integrity

H&E staining of colonic tissue in HSFD mice showed mucosal injury, including crypt distortion, epithelial thinning, submucosal spacing, and inflammatory cell infiltration. YS-H treatment preserved mucosal structure, maintaining tight crypt layout, continuous epithelial coverage, and normal goblet cell architecture.

3.7 Restructuring of Gut Microbiota Dynamics

Full-length 16S rRNA gene sequencing revealed distinct microbial shifts:

- Diversity Metrics:** YS-H intervention reversed HSFD-induced drops in alpha-diversity indices (Chao1, ACE, Shannon). Beta-diversity ordinations (PCoA, NMDS, PLS-DA) confirmed separation between the microbial community structures of HSFD and YS-H groups.
- Taxonomic Realignment:** HSFD decreased *Bacteroidota* abundance while promoting *Proteobacteria* and *Desulfobacterota*. YS-H restored *Bacteroidota* levels, re-introduced *Verrucomicrobiota*, and expanded *Lactobacillus* populations.
- Discriminative Taxa (LEfSe):**
 - HSFD Enriched:** *Helicobacter ganmani*, *Ileibacterium valens*, *Corynebacterium stationis*, and *Desulfovibrio*.
 - YS-H Enriched:** *Lactobacillus murinus* (LDA = 4.51, $p = 0.004$), *Lachnospiraceae_UCG-006*, *Streptococcus danieliae*, and *Adlercreutzia caecicola*.
- Phenotypic Correlations:** Spearman analysis demonstrated that *L. murinus* and *Lachnospiraceae_UCG-006* correlated negatively with FBG, LDL-C, IL-6, and NF- κ B, while correlating positively with hepatic SOD activity and glycogen content. Conversely, *Desulfovibrio* correlated positively with ALT, hepatic TG, and pro-inflammatory signaling.

5. **Functional Metagenomics (PICRUSt2):** Predicted functional profiles indicated that YS-H enriched microbial gene modules corresponding to "Carbohydrate Metabolism", "Amino Acid Metabolism", "Energy Metabolism", and "ABC Transporters".

4. Discussion

Dietary management of metabolic syndrome requires interventions capable of targeting interconnected systemic pathways. This study demonstrates that an aqueous extract of *Coriandrum sativum* seeds acts through multiple targets along the gut-liver axis to reverse diet-induced metabolic dysfunction.

The gut-liver axis serves as a key communication highway between the gastrointestinal tract and the liver. High-fat, high-sugar diets disrupt this connection by degrading the pro-ve mucosal barrier and shifting the microbiota toward an endotoxin-producing profile. Translocated LPS and bacterial byproducts enter the portal vein, activating Kupffer cell TLR4/NF- κ B pathways and triggering hepatic inflammation, oxidative stress, and lipid accumulation.

Aqueous coriander extract addresses these disruptions at both ends of the axis. Within the colon, YS treatment preserves crypt depth and mucosal structure. Microbiome profiling revealed that YS enriches short-chain fatty acid (SCFA)-producing bacteria, particularly members of *Lachnospiraceae* and *Lactobacillus murinus*. SCFAs (such as acetate, propionate, and butyrate) serve as critical energy substrates for colonocytes, strengthen tight junction proteins, stimulate GLP-1/GLP-2 secretion from intestinal L-cells, and suppress systemic inflammation. Concurrent reduction of sulfate-reducing bacteria like *Desulfovibrio* minimizes mucosal barrier damage and endotoxin burden.

In the liver, YS coordinates a metabolic shift in gene and protein expression. Downregulation of *FAS* suppresses *de novo* lipogenesis, curbing the synthesis of new triglycerides. Simultaneously, upregulation of PPAR α protein promotes mitochondrial beta-oxidation of fatty acids, while restored LDLR expression enhances receptor-mediated clearance of circulating LDL particles. The suppression of NF- κ B and IL-6 downstream signaling mitigates local hepatic inflammation, preventing the progression of simple steatosis to non-alcoholic steatohepatitis (NASH). These molecular actions are reinforced by antioxidant effects, as evidenced by reduced MDA levels and elevated SOD activity.

The active phytoconstituents identified by GC-MS—such as 4-ethenyl-2-methoxyphenol, phenolic acids, and monoterpenoids—likely drive these observed bioactivities. Phenolics and flavonoids are known to activate Nrf2 antioxidant cascades and AMPK signaling pathways, providing a plausibly synergistic basis for the multi-targeted actions of the crude aqueous extract.

5. Conclusion

This study demonstrates that aqueous *Coriandrum sativum* seed extract effectively ameliorates high-fat, high-sugar diet-induced glucolipid metabolic disorders in mice. Through multi-target actions along the gut-liver axis, YS intervention:

Improves glycemic control, lowers systemic insulin resistance, and preserves pancreatic islet architecture.

Reduces serum and hepatic lipid accumulation while pro-ing against liver injury and oxidative stress.

Modulates key hepatic targets by suppressing lipogenic (*FAS*) and inflammatory (NF- κ B, IL-6) pathways while promoting lipid oxidation and clearance (PPAR α , LDLR).

Restores intestinal mucosal integrity and reshapes the gut microbiota by enriching beneficial taxa (*Lactobacillus murinus*, *Lachnospiraceae*_UCG-006) and suppressing pro-inflammatory microbes (*Desulfovibrio*). Overall, these findings support the potential of *Coriandrum sativum* seed extract as a functional food component or natural therapeutic agent for the management of metabolic syndrome, NAFLD, and type 2 diabetes.

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Submitting a manuscript must be accompanied by a statement from the authors disclosing all financial and personnel relationships that might bias their work, if any. Moreover, they are requested to provide evidence of the presence or absence of upcoming conflicts. A separate page should cover this proclamation and if there are further details, they could be mentioned in the covering letter. If an organization encompasses any financial interest in the outcome of the study, the authors are appealed to provide a declaration that has full access to all data in the study and take complete responsibility for the integrity of data and the accuracy of data analysis.

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Data Availability Statement:

All data analysed during this study are included in this published article and its supplementary information files. Additional details or extracted datasets are available from the corresponding author upon reasonable request.

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