



Evaluation of the Role of *Chlorella vulgaris* as a Natural Environmental Resource in Improving Metabolic Disorders in Hyperlipidemic Rats.

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KEYWORDS

Chlorella vulgaris, Hyperlipidemia, Metabolic disorders, Lipid metabolism, Natural therapeutic agents, Nutraceuticals, Oxidative stress, Rat model

ABSTRACT:

Background: Hyperlipidemia, characterized by elevated blood lipid levels, is a major risk factor for metabolic disorders, and natural resources such as *Chlorella vulgaris* have shown potential in modulating lipid metabolism and improving overall metabolic health.

Objective: This study aims to investigate the effectiveness of *Chlorella vulgaris* as a natural environmental resource in mitigating metabolic disturbances associated with hyperlipidemia in rats.

Material and Methods: Twenty-four male Wistar rats were divided into four groups: negative control, positive control (high-fat diet; HFD), and two HFD groups supplemented with *Chlorella vulgaris* (4.5% and 8.5%) for 28 days. Phytochemical screening, growth performance, lipid profiles, glycemic status, liver/renal functions, and immunoglobulins (IgA/IgE) were assessed, alongside myocardial histopathology.

Results: *Chlorella vulgaris* exhibited high bioactive content, with total phenolics and flavonoids at 21.8 and 38.1 mg/100g, respectively. *Chlorella vulgaris* supplementation, particularly at 8.5%, significantly reduced body weight gain and reversed dyslipidemia by lowering total cholesterol, LDL-C, VLDL-C, and triglycerides, while elevating HDL-C ($p < 0.05$). Additionally, *Chlorella vulgaris* significantly decreased serum glucose, uric acid, and liver enzymes (ALT, AST, ALP), indicating potent hepatoprotective and metabolic regulatory effects. A notable immunomodulatory response was observed through the reduction of elevated IgA and IgE. Histopathologically, *Chlorella vulgaris* especially the 8.5% dose restored myocardial architecture, neutralizing inflammatory infiltration and myofibrillar degeneration induced by hyperlipidemic stress.

Conclusion: *Chlorella vulgaris* acts as a multi-targeted nutraceutical, reversing metabolic disturbances and protecting cardiac tissues through its rich antioxidant and anti-inflammatory phytochemical profile. These findings support its application as a functional food strategy for managing cardiovascular and metabolic syndromes.

1. Introduction

Hyperlipidemia, defined by persistently elevated blood triglycerides and cholesterol levels, represents a critical metabolic imbalance that contributes to the progression of insulin resistance, obesity, and cardiovascular disease due to its disruption of lipid homeostasis and promotion of chronic oxidative stress and inflammation. Conventional pharmacological therapies remain effective but are often limited by side effects and economic burdens, driving a growing scientific interest in natural bioactive resources as complementary or

preventive strategies in metabolic disorder management [1]. Among such resources, *Chlorella vulgaris* a unicellular green microalga has gained attention for its rich profile of proteins, essential fatty acids, dietary fibers, pigments, vitamins, and other metabolites with potential biological activity. Studies in animal models have shown that diets containing *Chlorella vulgaris* can lower serum triglycerides, total cholesterol, and liver lipids while increasing fecal excretion of lipids, suggesting modulation of lipid absorption and metabolism [2]. Additionally, meta-analytical evidence



from human randomized controlled trials indicates that supplementation with *Chlorella vulgaris* may reduce total cholesterol and low-density lipoprotein cholesterol levels, underscoring its translational relevance to dyslipidemia [3]. The mechanisms underlying the beneficial effects of *Chlorella vulgaris* on lipid metabolism are multifaceted. Dietary fibers and bioactive lipids in *Chlorella* may enhance fecal lipid excretion and reduce intestinal lipid absorption, while omega-3 fatty acids and carotenoids modulate hepatic lipid catabolism and strengthen antioxidant defenses, protecting lipoproteins from oxidative modification [4]. Additionally, polysaccharides present in *Chlorella* appear to influence gut microbiota composition, promoting the production of short-chain fatty acids that convey metabolic signals through the gut-liver axis [5, 6]. Collectively, these interactions support improved lipid turnover and broader metabolic regulation beyond the simple reduction of blood lipid levels. From an ecological and biotechnological perspective, *Chlorella vulgaris* can be cultivated using renewable agricultural residues such as sweet sorghum bagasse, producing higher biomass and lipid yields with reduced production costs, and enabling more sustainable cultivation strategies [7, 8]. Additionally, supplementation of anaerobic wastewater with carbon sources has been shown to enhance nutrient removal and increase both biomass and lipid productivity, demonstrating its potential as a sustainable feedstock for high-value products [9, 10]. Given the combined evidence of hypolipidemic effects in animal models, potential benefits on human lipid profiles, and the sustainable and eco-friendly nature of its production, *Chlorella vulgaris* emerges as a promising multifactorial natural agent for managing hyperlipidemia and associated metabolic disorders. Its cultivation can simultaneously contribute to CO capture, wastewater treatment, and biomass production for high-value applications, highlighting its potential as a sustainable component of circular bioeconomy strategies [11]. Therefore, this study aims to systematically evaluate the efficacy of *Chlorella vulgaris* in improving metabolic disturbances. The proposed multifaceted cellular mechanisms through which *C. vulgaris* exerts its hypolipidemic and anti-inflammatory effects are illustrated in Figure 2.

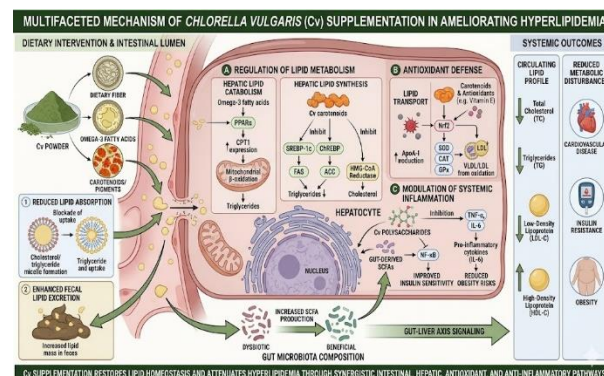


Figure 2. Proposed cellular mechanisms of *Chlorella vulgaris* in mitigating hyperlipidemia. The diagram illustrates the multifaceted pathways through which *Cv* bioactive compounds (fibers, omega-3, and carotenoids) modulate lipid homeostasis. Key mechanisms include: (1) inhibition of intestinal lipid absorption and enhancement of fecal excretion; (2) regulation of hepatic lipid catabolism and antioxidant defense; and (3) modulation of the gut-liver axis via microbiota-derived short-chain fatty acids (SCFAs), collectively reducing systemic inflammation and metabolic disorders.

2. Aim of Study

This study aims to investigate the effectiveness of *Chlorella vulgaris* as a natural environmental resource in mitigating metabolic disturbances associated with hyperlipidemia in rats.

3. Materials and Methods

3.1. Materials

3.1.1. Source and Preparation of *Chlorella vulgaris*

Source of *Chlorella vulgaris*: Dried *Chlorella vulgaris* powder was obtained from commercially available products in local markets in Jeddah, Saudi Arabia. The powder was verified for purity and quality to ensure suitability for experimental use.

Preparation of *Chlorella*-supplemented Diets: The dried powder was accurately weighed and incorporated into the standard laboratory diet to prepare two experimental concentrations: Low dose: 4.5% *Chlorella vulgaris* powder by weight of total diet. High dose: 8.5% *Chlorella vulgaris* powder by weight of total diet. The diets were thoroughly homogenized to ensure uniform distribution of the algae powder. Diets were freshly prepared weekly and stored at 4°C in airtight containers to maintain stability and prevent nutrient degradation. Rats in the experimental groups received the *Chlorella*-



supplemented diets alongside their normal feed for the duration of the 28-day experiment.

3.1.2. Lamb fat was purchased from a certified local butcher in Shibin El-Kom, Menoufia, Egypt, and utilized as part of the high-fat diet to induce hyperlipidemia in rats.

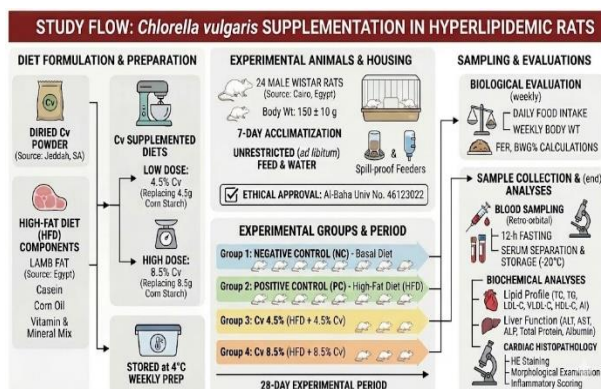


Figure 3. Comprehensive schematic of the experimental design, diet formulation, and analytical workflow. The diagram details the preparation of *Chlorella vulgaris* (Cv) supplemented diets (4.5% and 8.5%) and the induction of hyperlipidemia using a high-fat diet. It outlines the 28-day experimental timeline for the four cohorts ($n=24$ male Wistar rats), illustrating the transition from housing and acclimatization to biological sampling (blood and cardiac tissue) and subsequent biochemical and histopathological evaluations.

3.2. Biological experiment

3.2.1. Diet experiment

The basal and experimental diets were formulated to be nutritionally balanced and isocaloric. The basal diet contained casein (20 g), corn oil (4.7 g), mineral mixture (3.5 g), vitamin mixture (1 g), cellulose (5 g), choline chloride (2 g), and sucrose (10 g), with corn starch added to complete the formulation to 100 g. For the experimental groups, *Chlorella vulgaris* powder was incorporated into the diet at levels of 4.5% and 8.5%, replacing an equivalent proportion of corn starch. Specifically, 4.5 g and 8.5 g of *Chlorella vulgaris* were added per 100 g of diet in the 4.5% and 8.5% groups, respectively, while maintaining the same amounts of casein, corn oil, mineral mixture, vitamin mixture, cellulose, choline chloride, and sucrose as in the basal diet. Corn starch was adjusted accordingly to maintain a total diet weight of 100 g. This formulation allowed a controlled comparison between groups while isolating the effect of graded *Chlorella vulgaris* supplementation [12].

3.2.2. Rats

Experimental Animals and Ethical Oversight: Twenty-four adult male Wistar albino rats, weighing (150 ± 10 g) and confirmed to be clinically healthy, were procured from the Animal House of the National Research Centre (Giza, Egypt). The sample size was strategically selected based on power analysis from analogous hyperlipidemia models to ensure statistical significance while maintaining ethical parsimony in animal usage. All experimental protocols were executed in strict compliance with international guidelines for the care and use of laboratory animals. The study was formally reviewed and authorized by the Research Ethics Committee at Al-Baha University (Approval No. 46123022; April 17, 2025).

Housing and Environmental Conditioning: Upon arrival, the cohorts were transferred to a controlled facility and housed individually in sanitized, well-ventilated polycarbonate cages. Environmental parameters were maintained at a standardized 12-hour light/12-hour dark photoperiod. To ensure physiological stability, animals underwent a seven-day acclimation phase with unrestricted (*ad libitum*) access to a balanced basal diet and purified water. To maintain hygiene and nutritional accuracy, fodder was dispensed via spill-proof feeders, and hydration was provided through stainless-steel nipple-tip bottles to prevent leakage and external contamination [13].

Group Allocation and Clinical Monitoring: Following the stabilization period, the rats were partitioned into experimental groups using a simple randomization sequence. Systematic clinical observation was maintained throughout the study duration; notably, no fatalities or adverse clinical manifestations were recorded. Consequently, the initial sample size remained intact, with no exclusions required for the final analysis.

3.2.3. Animal Groups and Experimental Design:

Following a one-week acclimatization period, baseline body weights were recorded for all animals. No statistically significant differences in initial body weight were observed among the groups ($P > 0.05$). Subsequently, the rats were randomly assigned into four experimental cohorts ($n = 6$ per group) and maintained on their respective dietary regimens for 28 days. The detailed experimental sequence, including group



allocation and the integrated study flow, is illustrated in Figure 3. The groups were defined as follows:

Group 1 (Negative Control): Healthy rats fed the basal diet only and receiving no treatment.

Group 2 (Positive Control): Hyperlipidemic rats fed a high-fat diet without any additional treatment.

Group 3 (Hyperlipidemic + 4.5% *Chlorella vulgaris*): Hyperlipidemic rats fed a high-fat diet supplemented with 4.5% *Chlorella vulgaris* powder.

Group 4 (Hyperlipidemic + 8.5% *Chlorella vulgaris*): Hyperlipidemic rats fed a high-fat diet supplemented with 8.5% *Chlorella vulgaris* powder.

Hyperlipidemia was induced by feeding the animals a high-fat diet throughout the experimental period. All rats had ad libitum access to food and tap water under controlled environmental conditions [41].

3.2.4. Determination of Total Phenolic and Flavonoid Contents in Sesame Seeds

The total phenolic content (TPC) of sesame seeds (*Sesamum indicum*) was measured using the Folin–Ciocalteu colorimetric method, while the total flavonoid content (TFC) was determined by the aluminum chloride (AlCl_3) assay. Both methods are widely applied to quantify bioactive compounds in plant materials and provide reliable estimates of antioxidant potential. All measurements were performed in triplicate and expressed as mg GAE/100 g dry weight (DW) for TPC and mg CE/100 g DW for TFC, following the procedures described by [14].

3.2.5. Blood sampling:

At the end of the study, blood samples were collected following a 12-hour fasting period. Blood was obtained using the retro-orbital method with specialized glass capillary tubes and transferred into clean, dry centrifuge tubes. The samples were allowed to clot for 30 minutes at room temperature in a water bath maintained at 37 °C. Subsequently, the clotted blood was centrifuged at 3000 rpm for 10 minutes to separate the serum prior to glucose analysis. The resulting serum was carefully aspirated, transferred into clean, tightly sealed polypropylene tubes, and stored at –20 °C until further analysis.

3.2.6. Biological evaluation:

Throughout the 28-day study period, daily food intake was recorded, while body weight was measured weekly. Based on these measurements, the feeding efficiency ratio (F.E.R.), body weight gain percentage (B.W.G.%), and organ weights were calculated according to the method described by [15].

3.2.7. Biochemical Analyses

• Lipid Profile

Serum total lipids were measured using a colorimetric method as described by [16]. Triglycerides were determined enzymatically according to the method of [17], while total cholesterol was measured using the method of [18]. High-density lipoprotein cholesterol (HDL-C) levels were quantified following the method of [19]. Concentrations of low-density lipoprotein (LDL-C) and very low-density lipoprotein (VLDL-C) were calculated using the Friedewald equation [20]. The atherogenic index (AI) was calculated according to [21]. All analyses were performed in triplicate, and results were expressed in mg/dL.

• Liver Function Tests

Serum alanine aminotransferase (ALT) activity was measured using the colorimetric method described by [22]. Aspartate aminotransferase (AST) activity and serum total protein and albumin concentrations were determined following the method of [23]. Alkaline phosphatase (ALP) activity was assessed according to the method of [24]. All assays were performed in triplicate and expressed in appropriate units according to standard protocols.

• Renal Function Tests

Serum uric acid concentration was determined using the colorimetric method as described by [25]. All measurements were performed in triplicate and expressed in mg/dL.

• Blood Glucose

Serum glucose concentration was determined using the enzymatic colorimetric method based on [26]. All samples were analyzed in triplicate and results expressed in mg/dL.



3.2.8. Ethical Approval

All experimental procedures involving live animals were performed in full compliance with the established institutional regulations governing the ethical care and use of laboratory animals. The complete study design and related experimental protocols received prior review and official approval from the Research Ethics Committee of Al-Baha University (Approval No. 46123022; 17 April 2025).

3.2.9. Statistical Analysis

All data are presented as mean±standard deviation (SD). Statistical analysis was performed using one-way analysis of variance (ANOVA) for a completely randomized design. Significant differences among group means were determined using Duncan's multiple range test [27] at $p < 0.05$.

4. Results

4.1. Phytochemical Profile

The quantitative phytochemical analysis of *Chlorella vulgaris* extract revealed a substantial abundance of bioactive secondary metabolites, particularly phenolic and flavonoid compounds. As illustrated in Figure 4, the Total Phenolic Content (TPC) was found to be 38.1 mg gallic acid equivalents (GAE) per 100 g of dry weight (DW). These phenolic constituents are pivotal to the algae's antioxidant capacity, as their redox properties facilitate the neutralization of reactive oxygen species (ROS) and provide a robust defense against oxidative stress.

Furthermore, the Total Flavonoid Content (TFC) was recorded at 21.7 mg catechin equivalents (CE) per 100 g DW, indicating a significant concentration of these polyphenolic antioxidants within the algal biomass. Flavonoids are well-documented for their multi-faceted biological activities, including potent anti-inflammatory and radical-scavenging effects. The elevated levels of both TPC and TFC underscore the high nutritional and therapeutic value of *Chlorella vulgaris*, supporting its potential application as a functional food ingredient and a natural source of cardioprotective agents.

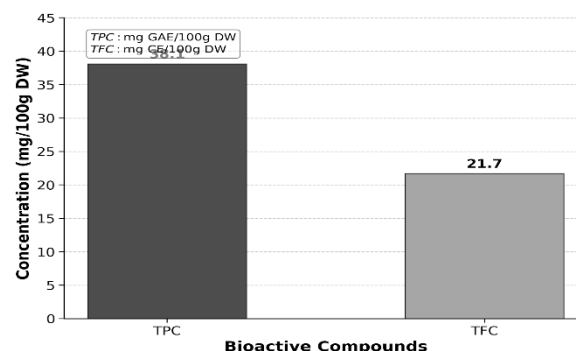


Figure 4. Phytochemical composition of *Chlorella vulgaris* extract: Total Phenolic Content (TPC) and Total Flavonoid Content (TFC).

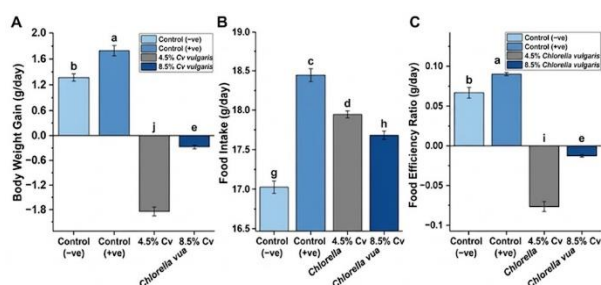
4.2 Biological Evaluation

The results presented in Figure 5 illustrate the effect of dietary supplementation with *Chlorella vulgaris* at different levels on body weight gain (BWG), feed intake (FI), and feed efficiency ratio (FER) compared with the control groups. Statistical analysis indicated that the differences among the studied groups were significant at the 5% level ($P \leq 0.05$).

The negative control group recorded a body weight gain of 1.15 ± 0.004 g/day, with a feed intake of 16.89 ± 0.200 g/day and a feed efficiency ratio of 0.067 ± 0.011 . In comparison, the positive control group showed a higher body weight gain of 1.68 ± 0.020 g/day, along with a feed intake of 18.48 ± 0.003 g/day and a feed efficiency ratio of 0.091 ± 0.008 .

In the groups supplemented with *Chlorella vulgaris*, a reduction in body weight gain was observed compared with the control groups. The group receiving 4.5% *Chlorella vulgaris* exhibited a body weight gain of -1.54 ± 0.003 g/day, with a feed intake of 17.96 ± 0.002 g/day and a feed efficiency ratio of -0.086 ± 0.002 . Meanwhile, the group fed 8.5% *Chlorella vulgaris* showed a body weight gain of -0.213 ± 0.001 g/day, a feed intake of 17.69 ± 0.010 g/day, and a feed efficiency ratio of -0.012 ± 0.001 .

Overall, these findings suggest that dietary supplementation with *Chlorella vulgaris* may contribute to reducing body weight gain and feed efficiency compared with the control groups, with statistically significant differences observed among the treatments at $P \leq 0.05$.



Impact of *Chlorella vulgaris* supplementation on growth performance and feed efficiency in hyperlipidemic rats. Values are expressed as mean \pm Standard Deviation ($n=6$). Different lowercase letters (a-j) indicate significant differences between groups ($P \leq 0.05$) using Duncan's multiple range test.

Figure 5. Impact of *Chlorella vulgaris* supplementation on growth performance and feed efficiency in hyperlipidemic rats. (A) Body weight gain (BWG; g/day), (B) Food intake (FI; g/day), and (C) Food efficiency ratio (FER). Values are expressed as mean \pm SD ($n=6$). Different lowercase letters (a-j) indicate statistically significant differences between groups at $p \leq 0.05$ according to Duncan's multiple range test.

4.3 Biochemical Analysis

The data presented in Figure 6 demonstrate the effect of dietary supplementation with *Chlorella vulgaris* on serum lipid profile parameters in experimental rats. A marked disturbance in lipid metabolism was observed in the positive control group compared with the negative control group, confirming the successful induction of hyperlipidemia. Total cholesterol (TC) levels were significantly elevated in the positive control group, reaching 238.08 mg/dL, whereas the negative control group exhibited a much lower level of 103.03 mg/dL. Dietary supplementation with *Chlorella vulgaris* resulted in a notable improvement in cholesterol levels. Rats receiving 4.5% *Chlorella vulgaris* showed a reduction in TC compared with the positive control group, while the group supplemented with 8.5% *Chlorella vulgaris* demonstrated a substantial decrease, with cholesterol values approaching those of the negative control group.

A similar pattern was observed in triglyceride (TG) concentrations. The positive control group exhibited markedly elevated TG levels relative to the negative control group. However, supplementation with *Chlorella vulgaris* significantly reduced triglyceride concentrations. The reduction was more pronounced in the group receiving the higher supplementation level (8.5%), indicating a dose-dependent improvement in lipid metabolism.

High-density lipoprotein cholesterol (HDL-C), which is considered the protective fraction of plasma lipoproteins, showed the opposite trend. The positive control group

recorded the lowest HDL-C levels, reflecting impaired lipid metabolism. In contrast, both *Chlorella*-supplemented groups exhibited increased HDL-C concentrations compared with the positive control group, with the 8.5% supplementation level producing the most noticeable improvement.

Regarding low-density lipoprotein cholesterol (LDL-C), the positive control group showed markedly elevated levels compared with the negative control group. Supplementation with *Chlorella vulgaris* significantly decreased LDL-C concentrations. Although the 4.5% supplementation level resulted in partial improvement, the 8.5% supplementation level reduced LDL-C values to levels close to those observed in the negative control group.

Very low-density lipoprotein cholesterol (VLDL-C) followed a similar trend, where the positive control group showed the highest levels. Dietary inclusion of *Chlorella vulgaris* significantly lowered VLDL-C concentrations, with the higher supplementation level producing a stronger effect.

The calculated atherogenic index (AI), which reflects the overall risk of cardiovascular complications, was substantially elevated in the positive control group. In contrast, supplementation with *Chlorella vulgaris* significantly reduced the atherogenic index. The reduction was particularly evident in the group receiving 8.5% *Chlorella vulgaris*, where the AI values approached those observed in the negative control group.

Overall, these findings indicate that dietary supplementation with *Chlorella vulgaris* effectively ameliorates hyperlipidemia by improving the lipid profile, increasing protective HDL-C levels, and reducing atherogenic lipoproteins.

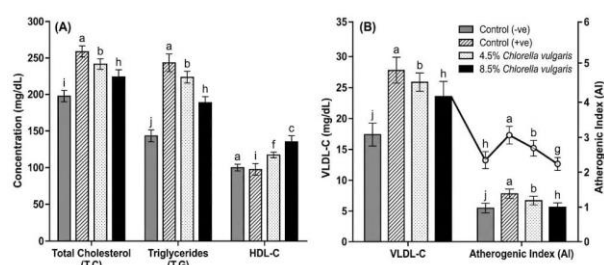


Figure 6. *Chlorella vulgaris* supplementation attenuates dyslipidemia and improves the atherogenic index in a dose-dependent manner.

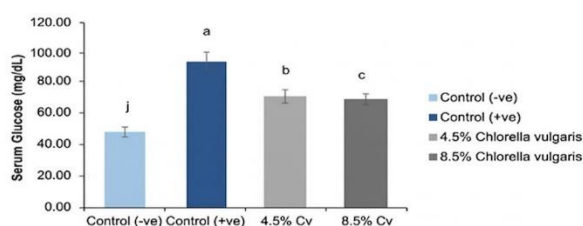


The results in Figure 7 show the effect of *Chlorella vulgaris* supplementation on blood glucose levels in hyperlipidemic rats. Statistical analysis indicated that the differences among the groups were significant at $P \leq 0.05$.

The negative control group exhibited a normal glucose level of 48.01 ± 0.020 mg/dL, reflecting baseline glycemic conditions. In contrast, the positive control group showed a substantial increase in blood glucose concentration, reaching 91.98 ± 0.060 mg/dL, which indicates hyperglycemia associated with the induced hyperlipidemic condition.

Supplementation with 4.5% *Chlorella vulgaris* led to a notable reduction in blood glucose, with levels measuring 70.50 ± 0.010 mg/dL, indicating a partial improvement compared with the positive control group. Similarly, the group receiving 8.5% *Chlorella vulgaris* showed a comparable reduction in glucose levels, recording 68.60 ± 0.035 mg/dL, suggesting that this higher supplementation level slightly enhanced glycemic regulation relative to the lower dose.

Overall, these findings suggest that dietary inclusion of *Chlorella vulgaris* contributed to a significant decrease in elevated blood glucose levels in hyperlipidemic rats, demonstrating a dose-dependent improvement in glucose metabolism compared with untreated hyperlipidemic controls.



Effect of *Chlorella vulgaris* supplementation on serum glucose levels in hyperlipidemic rats. Values are expressed as mean \pm SD ($n=6$). Bars with different lowercase letters (a, b, c, j) are significantly different ($P \leq 0.05$) based on Duncan's multiple range test.

Figure 7. Impact of *Chlorella vulgaris* supplementation on serum glucose levels in hyperlipidemic rats. Data are presented as mean \pm SD ($n=6$). Different lowercase letters (a, b, c, j) above the bars indicate statistically significant differences between groups ($p \leq 0.05$) according to Duncan's multiple range test.

The results presented in Figure 8 demonstrate the effect of *Chlorella vulgaris* supplementation on liver function enzymes in hyperlipidemic rats, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP). Statistical

analysis revealed that the differences among the experimental groups were significant at the 5% level ($P \leq 0.05$), indicating that dietary treatments had a measurable influence on liver enzyme activity.

With respect to alanine aminotransferase (ALT), the negative control group exhibited a relatively low level of 30.35 ± 4.18 U/L, reflecting normal liver function under healthy physiological conditions. In contrast, the positive control group showed a substantial elevation in ALT activity, reaching 53.80 ± 1.42 U/L, which indicates liver impairment associated with hyperlipidemia. Dietary supplementation with 4.5% *Chlorella vulgaris* markedly reduced ALT levels to 32.75 ± 2.12 U/L, bringing the value close to that observed in the negative control group. Meanwhile, the group receiving 8.5% *Chlorella vulgaris* showed a moderate decrease in ALT activity, recording 39.90 ± 5.64 U/L, which was lower than the positive control group but slightly higher than the negative control group.

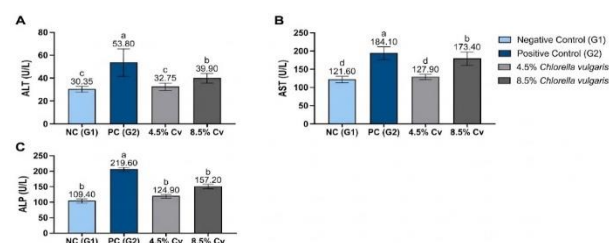
Regarding aspartate aminotransferase (AST) activity, the negative control group recorded a value of 121.60 ± 2.88 U/L, which represents the normal baseline level for this enzyme. A pronounced increase was observed in the positive control group, where AST rose to 184.10 ± 3.62 U/L, suggesting significant hepatic stress or cellular damage. Supplementation with 4.5% *Chlorella vulgaris* resulted in a considerable improvement, with AST decreasing to 127.90 ± 2.36 U/L, approaching the level of the negative control group. However, the group treated with 8.5% *Chlorella vulgaris* showed a smaller reduction in AST activity, recording 173.40 ± 8.95 U/L, which remained relatively higher than the other treated group but still lower than the positive control group.

A similar trend was observed for alkaline phosphatase (ALP) levels. The negative control group showed an ALP value of 109.40 ± 3.28 U/L, reflecting normal enzymatic activity. In comparison, the positive control group exhibited a marked increase in ALP, reaching 219.60 ± 52.90 U/L, indicating possible liver dysfunction and metabolic disturbance associated with hyperlipidemia. Treatment with 4.5% *Chlorella vulgaris* significantly reduced ALP levels to 124.90 ± 10.65 U/L, demonstrating a clear improvement toward normal physiological values. Likewise, the 8.5% *Chlorella vulgaris* group showed a reduction in ALP activity to



157.20±9.18 U/L, although the value remained higher than that observed in the negative control group.

Overall, these findings suggest that supplementation with *Chlorella vulgaris* contributed to the improvement of liver enzyme profiles in hyperlipidemic rats. The treatment groups showed reduced enzyme activities compared with the positive control group, indicating a protective effect against liver dysfunction. The statistical differences observed among the groups confirm that the dietary treatments significantly influenced liver enzyme levels at $P \leq 0.05$.



Impact of *Chlorella vulgaris* supplementation on liver enzyme activities in hyperlipidemic rats. Values are expressed as mean ± SD (n=6). Different lowercase letters (a–d) above bars indicate statistically significant differences between groups ($P \leq 0.05$) based on the LSD test.

Figure 8. Hepatoprotective effect of *Chlorella vulgaris* supplementation on serum liver enzyme activities in hyperlipidemic rats. (A) Alanine aminotransferase (ALT), (B) Aspartate aminotransferase (AST), and (C) Alkaline phosphatase (ALP). Values are expressed as mean±SD (n=6). Different lowercase letters (a–d) indicate statistically significant differences between groups ($p \leq 0.05$) according to the Least Significant Difference (LSD) test.

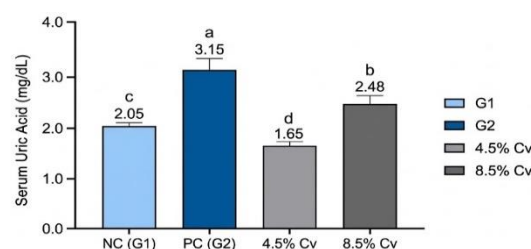
The results presented in Figure 9 illustrate the effect of *Chlorella vulgaris* supplementation on serum uric acid (UA) levels in hyperlipidemic rats. Statistical analysis indicated that the differences among the experimental groups were significant at $P \leq 0.05$.

The negative control group showed a baseline UA level of 2.05±0.15 mg/dL, representing normal physiological conditions. In contrast, the positive control group exhibited a significant increase in uric acid, reaching 3.15±0.13 mg/dL, indicating hyperuricemia associated with hyperlipidemia.

Dietary supplementation with 4.5% *Chlorella vulgaris* resulted in a marked reduction in serum UA, recording 1.65±0.02 mg/dL, which was even lower than the level observed in the negative control group. The group receiving 8.5% *Chlorella vulgaris* also showed a decrease in uric acid to 2.48±0.05 mg/dL, reflecting an

improvement compared with the positive control group, although the reduction was less pronounced than with the 4.5% supplementation.

Overall, these findings suggest that *Chlorella vulgaris* supplementation can effectively reduce elevated serum uric acid levels in hyperlipidemic rats, with significant differences observed among the groups at $P \leq 0.05$.



Effect of *Chlorella vulgaris* supplementation on serum uric acid (UA) levels in hyperlipidemic rats. Values are expressed as mean ± SD (n=6). Bars with different lowercase letters (a–d) are significantly different ($P \leq 0.05$) based on the LSD test.

Figure 8. Figure 9. Effect of *Chlorella vulgaris* (Cv) supplementation on serum uric acid (UA) levels in hyperlipidemic rats. Values are expressed as mean±SD (n=6). Different lowercase letters (a–d) above bars indicate statistically significant differences between groups ($p \leq 0.05$) according to the Least Significant Difference (LSD) test.

4.4 Immunological Results

The results in Figure 10 illustrate the effects of *Chlorella vulgaris* supplementation on serum immunoglobulin levels, including IgA and IgE, in hyperlipidemic rats. Statistical analysis indicated that the differences among the experimental groups were significant at $P \leq 0.05$.

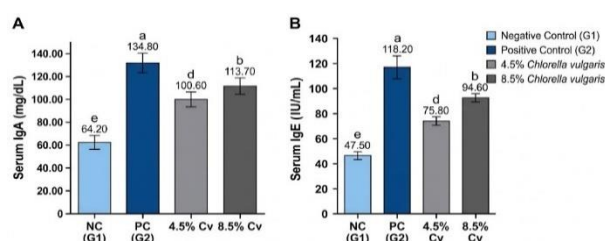
The negative control group showed baseline immunoglobulin levels, with IgA measured at 64.20±1.10 mg/dL and IgE at 47.50±1.10 IU/mL, reflecting normal immune function. In contrast, the positive control group exhibited a marked elevation in both immunoglobulins, with IgA reaching 134.80±0.90 mg/dL and IgE 118.20±1.30 IU/mL, indicating an activated immune response associated with hyperlipidemia.

Dietary supplementation with 4.5% *Chlorella vulgaris* resulted in a partial normalization of immunoglobulin levels. IgA decreased to 100.60±1.20 mg/dL, and IgE to 75.80±1.20 IU/mL, demonstrating an improvement in immune balance compared with the positive control group. Similarly, the group receiving 8.5% *Chlorella vulgaris* showed further reduction in immunoglobulin



levels, with IgA at 113.70 ± 1.30 mg/dL and IgE at 94.60 ± 2.10 IU/mL, indicating that a higher supplementation level contributed to a more pronounced modulation of immune responses.

Overall, these findings suggest that dietary inclusion of *Chlorella vulgaris* can mitigate the hyperactivation of immune markers observed in hyperlipidemic rats, with statistically significant improvements among the treated groups at $P \leq 0.05$.



Effect of *Chlorella vulgaris* supplementation on immunoglobulin levels in hyperlipidemic rats. Values are expressed as mean \pm SD ($n=6$). Bars with different lowercase letters (a, b, d, e) are significantly different ($P \leq 0.05$) based on the LSD test.

Figure 10. Modulatory effect of *Chlorella vulgaris* (Cv) supplementation on serum immunoglobulin levels in hyperlipidemic rats. (A) Serum Immunoglobulin A (IgA) levels (mg/dL) and (B) Serum Immunoglobulin E (IgE) levels (IU/mL). Data are expressed as mean \pm SD ($n=6$). Different lowercase letters (a, b, d, e) above the bars indicate statistically significant differences between groups ($p \leq 0.05$) based on the LSD test.

4.5 Histopathological Analysis of Myocardial Sections

The microscopic examination of H&E-stained cardiac sections provided definitive evidence of the cardioprotective efficacy of *Chlorella vulgaris* against hyperlipidemia-induced tissue damage. The findings are detailed as follows:

Panel (A): Negative Control Group (Normal Architecture): Photomicrographs of the heart from the negative control group revealed a pristine histological landscape. The myocardium exhibited a normal architectural pattern characterized by well-organized, branching cardiac muscle fibers (myocytes). The cardiomyocytes appeared intact with distinct, centrally located oval nuclei and clearly defined eosinophilic sarcoplasm. No evidence of inflammatory cell infiltration, edema, or myofibrillar degeneration was observed.

Panel (B): Positive Control Group (Pathological Alterations): In stark contrast, the positive control group

sections displayed profound pathological manifestations. The myocardial architecture was severely compromised, as evidenced by the presence of extensive focal inflammatory cell infiltration (indicated by black arrows). Furthermore, significant myofibrillar fragmentation and loss of transverse striations were prominent. These degenerative changes signify the detrimental impact of hyperlipidemic stress on cardiac structural integrity, potentially leading to myocarditis and functional impairment.

Panel (C): 4.5% *Chlorella vulgaris* Supplemented Group (Partial Recovery): Dietary intervention with 4.5% *Chlorella vulgaris* resulted in a noticeable attenuation of the cardiac lesions. There was a moderate restoration of the myocardial fiber arrangement compared to the positive control group. While some residual inflammatory foci remained, the intensity and distribution of the leucocytic infiltration were significantly reduced. The cardiomyocytes showed improved structural coherence, indicating the initiation of a dose-responsive protective mechanism.

Panel (D): 8.5% *Chlorella vulgaris* Supplemented Group (Optimal Restoration): The most striking therapeutic response was observed in the 8.5% *Chlorella vulgaris* group. The myocardial tissue exhibited remarkable histological recovery, closely resembling the normal architecture of the negative control. The muscle fibers were densely packed and properly aligned, with a near-complete disappearance of inflammatory cells. This finding suggests that the higher concentration of *Chlorella vulgaris* exerts a potent cytoprotective effect, effectively neutralizing the lipotoxic damage and preserving the cardiac muscle's micro-anatomy.

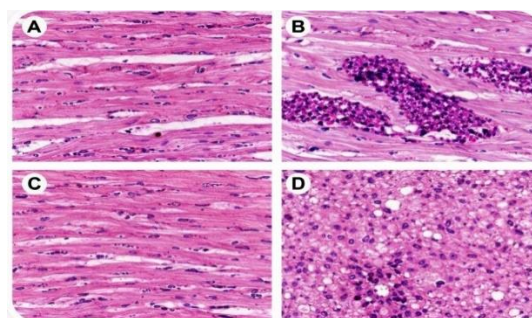


Figure 11. Histopathological sections of myocardial tissues in different experimental groups (H&E, $\times 400$). (A) Negative Control group showing normal cardiac myofibrils with central nuclei. (B) Positive Control group showing severe inflammatory cell infiltration (arrows) and myofibrillar



degeneration. (C) 4.5% *Chlorella vulgaris* group showing moderate improvement with persistent mild inflammation. (D) 8.5% *Chlorella vulgaris* group showing significant restoration of myocardial architecture and minimal inflammatory changes.

5. Discussion

The present study demonstrated that *Chlorella vulgaris* supplementation exerted significant protective and metabolic regulatory effects in hyperlipidemic rats, as evidenced by improvements in growth performance, lipid profile, glycemic status, liver function, immune markers, and myocardial histology. These beneficial effects can largely be attributed to the rich phytochemical composition of *Chlorella vulgaris*, particularly its phenolic and flavonoid constituents, which possess strong antioxidant and anti-inflammatory activities.

5.1 Phytochemical Profile and Antioxidant Potential

The phytochemical analysis revealed considerable levels of total phenolic compounds (TPC) and total flavonoids (TFC) in *Chlorella vulgaris*. These bioactive compounds are well known for their ability to neutralize reactive oxygen species and reduce oxidative stress, which plays a crucial role in the development of metabolic disorders such as hyperlipidemia and cardiovascular disease. Phenolic compounds can donate hydrogen atoms or electrons to stabilize free radicals and inhibit lipid peroxidation processes [28]. Similarly, flavonoids contribute to antioxidant defense systems by scavenging free radicals and modulating cellular signaling pathways associated with inflammation and oxidative damage [29]. Previous studies have reported that microalgae such as *Chlorella vulgaris* contain abundant bioactive molecules including carotenoids, polyphenols, vitamins, and essential fatty acids that collectively contribute to their therapeutic. Therefore, the high levels of TPC and TFC observed in the present study may explain the protective biological effects observed in the experimental animals [30].

5.2 Effects on Growth Performance and Body Weight

The current findings revealed that supplementation with *Chlorella vulgaris* significantly reduced body weight gain and feed efficiency ratio in hyperlipidemic rats. This reduction in weight gain may be attributed to the metabolic regulatory effects of bioactive compounds present in the algae. Microalgae-derived bioactive compounds have been shown to influence lipid metabolism, improve energy utilization, and reduce fat

accumulation in tissues [28]. In addition, the high fiber content and presence of bioactive polysaccharides in *Chlorella* may enhance satiety and reduce lipid absorption in the gastrointestinal tract [31]. These findings are consistent with previous research indicating that *Chlorella* supplementation can contribute to weight control and metabolic improvement by modulating lipid metabolism and energy balance [32].

5.3 Effects on Lipid Profile and Atherogenic Index

Hyperlipidemia is characterized by elevated levels of total cholesterol, triglycerides, LDL-cholesterol, and VLDL-cholesterol, accompanied by reduced HDL-cholesterol. In the present study, the positive control group exhibited severe dyslipidemia, whereas supplementation with *Chlorella vulgaris* significantly improved all lipid parameters and reduced the atherogenic index [4]. These results agree with previous findings indicating that *Chlorella* possesses hypolipidemic properties. The lipid-lowering effect of *Chlorella* may be explained by several mechanisms, including inhibition of cholesterol absorption in the intestine, increased bile acid excretion, and regulation of hepatic lipid metabolism. Furthermore, *Chlorella* contains dietary fiber, phytosterols, and polyunsaturated fatty acids that contribute to reducing plasma lipid levels and improving cardiovascular health [4]. The improvement in HDL-cholesterol observed in the treated groups is particularly important, as HDL plays a protective role by facilitating reverse cholesterol transport and reducing the risk of atherosclerosis. Consequently, the reduction in the atherogenic index observed in this study suggests a cardioprotective effect of *Chlorella vulgaris* supplementation [33].

5.4 Effects on Blood Glucose Levels

The present study also demonstrated that *Chlorella vulgaris* supplementation significantly reduced elevated blood glucose levels in hyperlipidemic rats. Hyperglycemia is often associated with metabolic syndrome and dyslipidemia due to impaired insulin sensitivity and altered glucose metabolism [32]. The hypoglycemic effect of *Chlorella* may be attributed to its antioxidant compounds, which improve pancreatic β -cell function and enhance insulin sensitivity. Additionally, the dietary fiber present in *Chlorella* may slow glucose absorption in the intestine, leading to improved glycemic control [28]. These findings support



the potential role of *Chlorella vulgaris* as a functional food ingredient capable of improving metabolic health.

5.5 Effects on Liver Function Enzymes

Liver enzymes such as ALT, AST, and ALP are commonly used indicators of hepatic injury. The marked elevation of these enzymes observed in the positive control group suggests liver damage induced by hyperlipidemia. [40]. The hepatoprotective effect of *Chlorella* may be related to its antioxidant and anti-inflammatory properties. Oxidative stress is a major contributor to hepatic damage in hyperlipidemic conditions, leading to lipid peroxidation and hepatocyte injury. Bioactive compounds in *Chlorella*, such as carotenoids, chlorophyll, and polyphenols, can reduce oxidative stress and protect liver cells from damage [34]. These findings are supported by previous studies demonstrating that *Chlorella vulgaris* supplementation improves liver function and reduces hepatic lipid accumulation in experimental models of metabolic disorders [28].

5.6 Effects on Uric Acid Levels

The study also demonstrated that *Chlorella vulgaris* significantly reduced serum uric acid levels in hyperlipidemic rats. Elevated uric acid is commonly associated with oxidative stress, metabolic syndrome, and cardiovascular diseases [34]. The reduction in uric acid levels observed in this study may be attributed to the antioxidant activity of *Chlorella*, which helps reduce oxidative stress and improves metabolic regulation. Furthermore, bioactive compounds in microalgae may influence purine metabolism and enhance renal excretion of uric acid [35].

5.7 Immunomodulatory Effects

The results of the immunological analysis showed that hyperlipidemia significantly increased serum levels of IgA and IgE, indicating immune system activation and inflammatory responses [39]. However, supplementation with *Chlorella vulgaris* resulted in a noticeable reduction in these immunoglobulins. Microalgae such as *Chlorella* are known to possess immunomodulatory properties due to their content of polysaccharides, peptides, and antioxidants. These compounds can regulate immune responses by reducing inflammatory cytokine production and improving immune balance [28]. Previous studies have also reported that *Chlorella* supplementation

enhances immune function while preventing excessive inflammatory responses [36].

5.8 Histopathological Findings

Histopathological examination of myocardial tissues provided further evidence supporting the protective role of *Chlorella vulgaris*. The positive control group exhibited severe myocardial degeneration and inflammatory cell infiltration, indicating structural damage induced by hyperlipidemia. In contrast, the treated groups showed progressive improvement in cardiac tissue architecture, with the highest recovery observed in the group receiving 8.5% *Chlorella vulgaris*. These findings suggest that *Chlorella* may protect cardiac tissues against lipotoxicity and oxidative damage. Antioxidants present in *Chlorella* can inhibit lipid peroxidation and reduce inflammatory processes within cardiac tissues [37]. Consequently, the histological restoration observed in this study further supports the cardioprotective potential of *Chlorella vulgaris*[38].

5.9 Overall Implications

The findings of this study provide compelling evidence that *Chlorella vulgaris* exerts comprehensive protective effects against hyperlipidemia-induced metabolic disturbances. By improving lipid profiles, reducing oxidative stress, modulating immune responses, and preserving hepatic and cardiac function, *Chlorella* demonstrates potential as a natural therapeutic agent for managing cardiovascular and metabolic disorders. Its rich composition of bioactive compounds including polyphenols, carotenoids, chlorophyll, and polysaccharides underpins these multifaceted benefits, highlighting the algae's capacity to act synergistically on multiple physiological pathways. These results suggest that dietary supplementation with *Chlorella* may serve as a safe and effective adjunct strategy for the prevention and management of metabolic syndrome, dyslipidemia, and associated inflammatory and oxidative complications. Furthermore, the dose-dependent effects observed emphasize the importance of optimizing supplementation levels to maximize health benefits. Future research should focus on elucidating the molecular mechanisms of action in human models and evaluating long-term safety and efficacy, which could pave the way for the incorporation of *Chlorella vulgaris* into functional foods and nutraceutical formulations targeting cardiometabolic health.

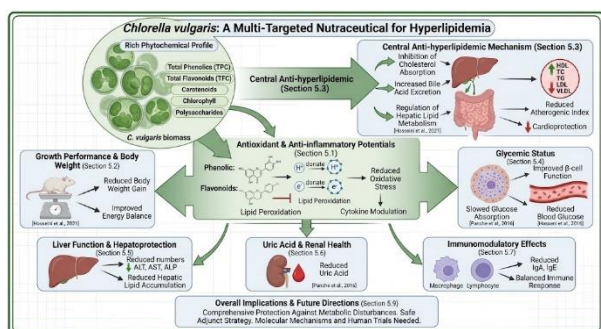


Figure 12. Protective and Metabolic Regulatory Effects of *Chlorella vulgaris* Supplementation in Hyperlipidemic Rat Models: A Mechanistic Overview.

6. Conclusion

In conclusion, dietary supplementation with *Chlorella vulgaris* showed significant beneficial effects in hyperlipidemic rats, including improved growth performance, reduced body weight gain, enhanced feed efficiency, and normalization of serum lipid profiles and atherogenic index. The algae also improved liver function, mitigated hyperglycemia, modulated immune markers, and demonstrated cardioprotective effects by restoring myocardial architecture and reducing inflammation. These benefits are likely due to its rich content of bioactive compounds with antioxidant, anti-inflammatory, and metabolic regulatory properties. Overall, these findings highlight the potential of *Chlorella vulgaris* as a natural functional food or nutraceutical for hyperlipidemia management, although further research is needed to elucidate its molecular mechanisms and confirm efficacy in clinical settings.

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