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# Functional Assessment of Garcinia Cambogia Complex: Implications for Body Fat Reduction

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## Abstract

*This study evaluated the efficacy of Garcinia cambogia complex (CAC) in preventing body fat accumulation. Male SD rats were used in the experiment, which included a normal diet (ND) group and a high-fat diet (HFD) group. The HFD group was further divided into four subgroups, each receiving a different treatment: 0, 207, 414, and 828 mg/kg of the CAC (1x, 2x, 4x dose). All rats were fed anHFD for 9 weeks, and the treatments were administered from week six onwards. At the end of the experiment, the rats were sacrificed, and analyses were conducted on body weight, body fat mass, serum biochemical values, and liver lipid concentration. The results showed that theCAC significantly reduced the final body weight, body fat mass, body fat percentage, and food utilization rate in the treated rats compared to the HFD control group. Rats in the HFD control group exhibited elevated serum levels of aspartate aminotransferase, alanine aminotransferase (ALT), triglycerides, total cholesterol, low-density lipoprotein, free fatty acids, and glucose compared to the ND group. The 1x dose of the CAC improved these biochemical parameters, except for ALT and glucose levels. The 2x and 4x dose groups showed a significant reduction in blood glucose levels, while the 4x dose group notably decreased serum ALT levels. In conclusion, daily intake of the CAC significantly reduced body fat accumulation in rats, demonstrating potential health benefits in preventing obesity.*

**Keywords:** *Garcinia Cambogia, Sinetrol®, Apple, Rose, Obesity.*

## INTRODUCTION

In 2016, the World Health Organization reported that over 1.3 billion adults globally were classified as overweight, with an additional 650 million categorized as obese [1]. Obesity is a major risk factor for various health conditions, including insulin resistance, hyperlipidemia, obstructive sleep apnea, non-alcoholic fatty liver disease (NAFLD), neurological disorders, osteoarthritis, hormonal imbalances, polycystic ovary syndrome (PCOS), and infertility [2]. Evidence from both epidemiological research and dietary intervention studies indicates that phytochemicals -rich diets, such as the Mediterranean diet, are linked to antioxidant, anti-inflammatory, and anti-obesity properties, as well as a reduced likelihood of metabolic syndrome and cardiovascular diseases in humans [3]. Consequently, the development of safe and efficacious alternative weight-loss strategies beyond traditional methods has emerged as a critical area of interest in clinical and public health research.

*Garcinia cambogia*, also referred to as Malabar tamarind, is predominantly found in India, Malaysia, and Thailand, and is recognized as a medicinally significant species within the Clusiaceae family [4]. The fruit rind serves as a rich source of hydroxycitric acid (HCA), the principal bioactive constituent. Peel-derived supplements of *G. cambogia* typically contain 20–60% HCA, which is hypothesized to underlie its potential weight management properties [5]. HCA functions by inhibiting lipogenesis through the suppression of ATP-citrate lyase, a pivotal enzyme in lipid biosynthesis. Furthermore, HCA has been reported to influence serotonin regulation, leading to appetite suppression, while also enhancing fatty acid oxidation and modulating lipid metabolism-related gene expression [6].

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Ingested polyphenols undergo extensive enzymatic transformations in the small intestine, leading to the production of phenolic metabolites that are subsequently absorbed, distributed to various tissues, and confer health benefits[7]. Sinetrol®, a food-derived ingredient formulated from bioactive compounds found in citrus fruits such as grapefruit, pomelo, orange, and guarana, predominantly contains flavanones with minor amounts of caffeine [8]. Previous studies have demonstrated that Sinetrol® exhibits anti-inflammatory, antioxidant, lipid-lowering, and anti-obesity properties, including weight reduction [9]. Apple polyphenol extracts, derived from fresh whole apples, possess a distinctive composition of polyphenols, including chlorogenic acid, phlorizin, procyanidins B2 in their free forms, and conjugated quercetin[10]. Rose polyphenols, on the other hand, are rich in bioactive compounds such as eugeniin, gallic acid, quercetin, rutin, and kaempferol[11]. Evidence from previous research indicates that extracts from apples and rose petals effectively reduce fasting blood glucose levels and adipose tissue accumulation, mitigating high-fat diet (HFD)-induced obesity[12].

Extensive research has indicated that plant extract supplementation offers significant potential in addressing obesity. Accordingly, this study focuses on formulating a *G. cambogia*-based complex as a dietary supplement with anti-obesity properties. A 4-week intervention was performed on HFD-induced obese rats to evaluate its efficacy by assessing alterations in body weight, adipose tissue mass, and hepatic fat accumulation.

## MATERIAL & METHODS

### Experimental Supplement

The CAC supplement, provided by Health Take Co., Ltd. (500 mg per tablet), included *Garcinia Cambogia* extract, Sinetrol® (a blend of polyphenols from citrus and guarana), apple extract, rose petal extract, and chromium yeast. It was formulated as a suspension in 0.5% carboxymethylcellulose (CMC) solution, with 20.7, 41.4, and 82.8 mg/mL concentrations. The supplement was administered at 1 mL per 100 g of body weight. In the control group, rats received an equivalent amount of CMC solution without the active ingredients.

### Animal and Study Design

Male SD rats, aged 6 weeks, were purchased from BioLASCO Co., Ltd. (Taipei, Taiwan) and housed at the Animal Facility of China Medical University (IACUC-2023-254). A total of 70 rats were maintained at a temperature of 22 ± 2°C with a 12-hour light-dark cycle (8 AM to 8 PM). They had free access to the experimental diet and sterile water throughout the study. After one week of acclimatization, the rats were split into two groups: a normal diet (ND) group (12 rats) and a high-fat diet (HFD) group (58 rats). The ND group was fed a standard diet providing 2.85 Kcal/g (Altromin 1320, Altromin Spezialfutter GmbH & Co. KG, Germany), while the HFD group received a high-fat diet containing 5.24 Kcal/g and 34.9% fat (D12492, Research Diet Inc., USA). After 5 weeks on their respective diets, 10 rats from the HFD group, either underweight or overweight, were excluded. The remaining rats were divided into four groups of 12, and each group was treated daily with either CMC (control) or varying doses of CAC supplement (1X, 2X, or 4X; 207, 414, or 828 mg/kg). Following 4 weeks of treatment, the rats were euthanized after an overnight fast. Liver and fat tissues were collected, weighed, and rinsed, while blood samples were drawn from the abdominal aorta for biochemical testing.

### Serum Biochemistry Analysis

The blood samples were centrifuged at 1500 ×g for 15 minutes at 4°C to separate the serum for subsequent biochemical analysis. Aspartate aminotransferase (AST), alanine aminotransferase (ALT), uric acid, and creatinine levels were measured using a Roche Cobas Mira Plus biochemical analyzer. Blood glucose concentrations were assessed with a CareSens II glucometer (i-SENS, Inc., Wonju-si, Korea), employing the glucose oxidase enzyme method. Non-esterified fatty acids (NEFA) were quantified using a commercial assay kit from RANDOX (County Antrim, UK). Triglyceride (TG) levels were determined with a DiaSys® assay kit (CT, USA). Total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) concentrations were analyzed using kits from Fortress Diagnostics Limited (Antrim, UK). Finally, blood sodium and potassium levels were measured by electrode method with a Chiron 644 Electrolytes Analyzer.

### Liver Lipid Concentration Determination

Lipid extraction from the liver was performed using the method outlined by Folch et al.[13]. For this, 0.1 grams of liver tissue were homogenized in 2 mL of Folch solution (chloroform: methanol, 2:1 ratio). The mixture was left at room temperature for 60 minutes and then centrifuged at 5000 rpm for 5 minutes. The upper liquid layer was carefully transferred into a clean 1.5 mL centrifuge tube, and 0.2 mL of 0.9% NaCl was added and thoroughly mixed, causing the liquid to become cloudy. After centrifugation at 2000 rpm for 5 minutes, two distinct layers formed. The lower layer was collected, dried under nitrogen at 55°C, and then re-dissolved by adding 100 µL of solvent (tert-butyl alcohol: Triton X-100: methanol in a 2:1:1 ratio) and heating at 65°C for 15 minutes. The concentrations of total cholesterol (TC) and triglycerides (TG) in the liver were measured using commercial assay kits.

### Statistical Analysis

The experimental data were analyzed using a one-way analysis of variance (ANOVA). To further explore group differences, post hoc comparisons were performed using Duncan's multiple range test. A $p$-value $< 0.05$ was considered indicative of a statistically significant difference between groups.

## RESULTS

### Effect of CAC Supplement on Body Weight in HFD-Induced Obese Rat

Figure 1 illustrates the body weight changes during the 4-week CAC supplementation period. At the beginning of the treatment phase (week 5), rats fed an HFD had significantly higher body weight than those on an ND after 5 weeks of diet induction. By the second week of treatment, the body weights of all three CAC-supplemented groups were significantly lower than those of the HFD control group.

### Effect of CAC Supplement on the Growth Parameters in HFD-Induced Obese Rat

The growth parameters during the treatment phase are summarized in Table 1. No significant differences were observed in baseline body weight among the four HFD-fed groups. However, after 4 weeks of treatment, the final body weight and body weight changes were significantly lower in the CAC-supplemented groups compared to the HFD control group. Although feed intake and energy intake were similar across all HFD-fed groups, feed efficiency was significantly reduced in the CAC groups, demonstrating a dose-dependent effect.

### Effect of CAC Supplement on Adipose Tissue Weight in HFD-Induced Obese Rat

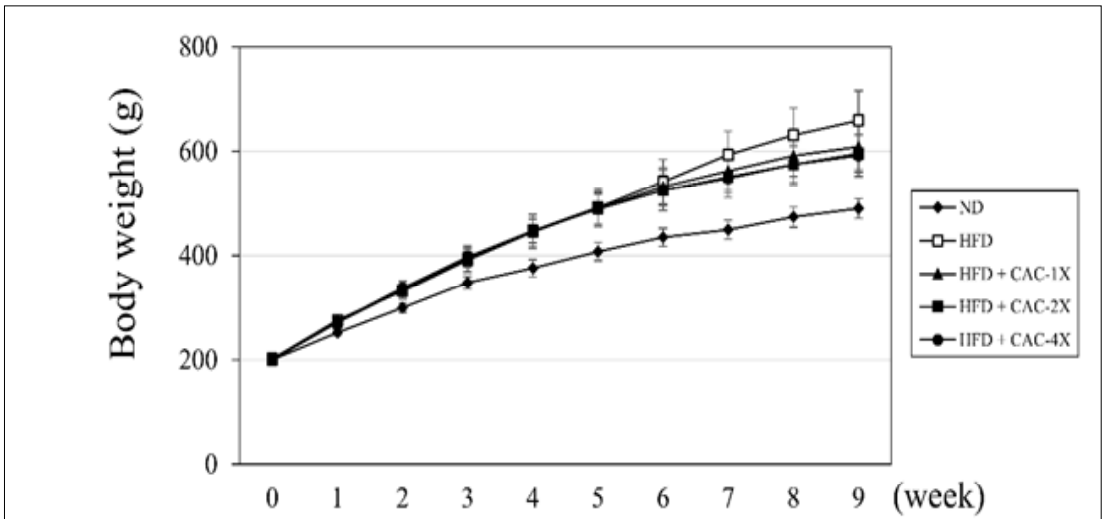
The impact of CAC supplementation on adipose tissue weight and body fat ratio is detailed in Table 2. After 4 weeks of treatment, the weights of epididymal, perirenal, and mesenteric adipose tissues, as well as total body fat and body fat ratio, were significantly lower in the CAC-supplemented groups compared to the HFD control group.

### Effect of CAC Supplement on Plasma Biochemical Parameters in HFD-Induced Obese Rat

Plasma biochemical parameters, including liver function, kidney function, lipid profile, glucose, and electrolyte balance, were analyzed after 4 weeks of treatment (Table 3). The levels of HDL-C, creatinine, uric acid, $Na^+$, and $K^+$ showed no significant changes across the three CAC doses (1X, 2X, and 4X) compared to the HFD control group. However, significant reductions were observed in AST, TC, LDL-C, TG, and FFA levels in all CAC-treated groups. Additionally, ALT levels were significantly reduced with 4X CAC supplementation, and blood glucose levels were significantly lower in the 2X and 4X CAC groups after 4 weeks of treatment.

### Effect of CAC Supplement on the Liver Lipid in HFD-Induced Obese Rat

Liver weight and lipid content were analyzed following sacrifice (Table 4). Rats supplemented with CAC at all three doses (1X, 2X, and 4X) exhibited significantly lower liver weights after 4 weeks of treatment compared to the HFD control group. Furthermore, liver TC and TG content were significantly reduced in the 2X and 4X CAC-supplemented groups.




| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| ND | a | a | a | a | a | a | a | a | a | a |
| HFD | a | b | b | b | b | b | b | c | c | c |
| HFD + CAC-1X | a | b | b | b | b | b | b | b | b | b |
| HFD + CAC-2X | a | b | b | b | b | b | b | b | b | b |
| HFD + CAC-4X | a | b | b | b | b | b | b | b | b | b |

**Figure 1.** Effect of CAC on the body weight in HFD-induced obese rats.

The reported values are the mean ± SD (n=12). Different superscript letters (a, b, and c) indicate significant differences between groups ($p< 0.05$).

**Table 1.** Effect of CAC on growth parameters in HFD-induced obese rats

| Growth parameters | ND | HFD | | | |
|---|---|---|---|---|---|
| | | Control | CAC-1X | CAC-2X | CAC-4X |
| Induced BW (g) | 407.7±17.5$^{a}$ | 492.1± 6.4$^{b}$ | 489.8± 4.1$^{b}$ | 488.8± 27.4$^{b}$ | 488.5±31.4$^{b}$ |
| Final BW(g) | 489.8±18.9$^{a}$ | 658.3± 6.8$^{c}$ | 608.6± 4.0$^{b}$ | 595.7± 36.2$^{b}$ | 591.5±40.0$^{b}$ |
| BW change (g) | 82.1 ± 9.8$^{a}$ | 166.2± 3.9$^{d}$ | 118.8± 8.7$^{c}$ | 106.8±13.1$^{bc}$ | 103.0±17.3$^{b}$ |
| Feed intake (g/rat/day) | 28.9 ± 1.4$^{b}$ | 21.3 ± 2.1$^{a}$ | 20.4 ± 2.0$^{a}$ | 20.2 ± 1.6$^{a}$ | 20.1 ± 1.3$^{a}$ |
| Energy intake (kcal/rat/day) | 102.7 ± 5.1$^{a}$ | 138.3± 4.2$^{b}$ | 134.9± 3.3$^{b}$ | 133.8± 1.2$^{b}$ | 134.7 ± 8.4$^{b}$ |
| Feed efficiency (%) | 10.2 ± 1.3$^{a}$ | 27.8 ± 2.4$^{d}$ | 20.8 ± 2.5$^{c}$ | 18.9 ± 1.9$^{b}$ | 18.3 ± 2.8$^{b}$ |

The reported values are the mean ± SD (n=12). Different superscript letters (a, b, c, and d) indicate significant differences between groups ($p$< 0.05). BW change = final BW - induced BW. Feed efficiency (%) = [Body weight change (g) ÷ total feed intake (g)] × 100%. BW, body weight.

**Table 2.** Effect of CAC on adipose tissues weight and body fat in HFD-induced obese rats

| Adipose tissues | ND | HFD | | | |
|---|---|---|---|---|---|
| | | Control | CAC-1X | CAC-2X | CAC-4X |
| Epididymal AT (g) | 4.8 ± 0.7$^{a}$ | 16.0 ± 5.1$^{c}$ | 12.8 ± 3.0$^{b}$ | 12.2 ± 3.2$^{b}$ | 10.4 ± 2.6$^{b}$ |
| Perirenal AT (g) | 4.7 ± 0.8$^{a}$ | 24.9 ± 8.1$^{c}$ | 18.6 ± 5.3$^{b}$ | 17.0 ± 4.7$^{b}$ | 16.6 ± 5.1$^{b}$ |
| Mesenteric (g) | 2.6 ± 0.7$^{a}$ | 11.4 ± 4.7$^{c}$ | 9.0 ± 3.0$^{b}$ | 8.0 ± 1.8$^{b}$ | 7.6 ± 1.9$^{b}$ |
| Total body fat (g) | 12.1 ± 1.8$^{a}$ | 52.3 ± 16.8$^{c}$ | 40.4 ± 11.0$^{b}$ | 37.2 ± 8.7$^{b}$ | 34.6 ± 9.2$^{b}$ |
| Body fat ratio (%) | 2.5 ± 0.4$^{a}$ | 7.9 ± 2.1$^{c}$ | 6.6 ± 1.4$^{b}$ | 6.2 ± 1.3$^{b}$ | 5.8 ± 1.2$^{b}$ |

The reported values are the mean ± SD (n=12). Different superscript letters (a, b, and c) indicate significant differences between groups ($p$< 0.05). AT, adipose tissue.

**Table 3.** Effect of CAC on serum biochemical parameters in HFD-induced obese rats

| Serum biochemical parameters | ND | HFD | | | |
|---|---|---|---|---|---|
| | | Control | CAC-1X | CAC-2X | CAC-4X |
| AST (U/L) | 151.5 ± 31.1$^{a}$ | 255.9±123.9$^{b}$ | 188.1 ± 48.6$^{a}$ | 176.1 ± 55.4$^{a}$ | 172.0 ± 39.1$^{a}$ |
| ALT (U/L) | 48.6 ± 6.5$^{a}$ | 136.0±174.7$^{b}$ | 68.6 ± 57.2$^{ab}$ | 65.2 ± 22.9$^{ab}$ | 59.4 ± 18.0$^{a}$ |
| TC (mg/dL) | 59.0 ± 9.2$^{a}$ | 79.7 ± 16.6$^{c}$ | 66.8 ± 8.4$^{b}$ | 65.8 ± 10.0$^{b}$ | 64.3 ± 10.6$^{b}$ |
| LDL-C (mg/dL) | 20.7 ± 5.6$^{a}$ | 28.6 ± 13.1$^{b}$ | 18.6 ± 6.1$^{a}$ | 18.4 ± 7.2$^{a}$ | 17.8 ± 8.6$^{a}$ |
| HDL-C (mg/dL) | 29.8 ± 4.5$^{a}$ | 36.7 ± 7.0$^{b}$ | 36.2 ± 10.9$^{b}$ | 36.2 ± 3.9$^{b}$ | 35.3 ± 5.3$^{ab}$ |
| TG (mg/dL) | 42.8 ± 11.2$^{a}$ | 72.2 ± 15.0$^{c}$ | 60.3 ± 12.2$^{b}$ | 56.8 ± 8.3$^{b}$ | 56.3 ± 9.7$^{b}$ |
| FFA (mg/dL) | 0.69 ± 0.07$^{a}$ | 1.04 ± 0.13$^{c}$ | 0.88 ± 0.13$^{b}$ | 0.82 ± 0.09$^{b}$ | 0.80 ± 0.08$^{b}$ |
| Glucose (mg/dL) | 122.9 ± 19.2$^{a}$ | 152.5 ± 32.8$^{b}$ | 133.8± 23.4$^{ab}$ | 128.2 ± 16.0$^{a}$ | 132.2 ± 21.5$^{a}$ |
| Creatinine (mg/dL) | 0.4 ± 0.2$^{a}$ | 0.4 ± 0.1$^{a}$ | 0.4 ± 0.1$^{a}$ | 0.4 ± 0.1$^{a}$ | 0.4 ± 0.1$^{a}$ |
| Uric acid (mg/dL) | 1.4 ± 0.5$^{a}$ | 1.4 ± 0.6$^{a}$ | 1.3 ± 0.6$^{a}$ | 1.4 ± 0.7$^{a}$ | 1.4 ± 0.5$^{a}$ |
| $Na^{+}$ (mEq/L) | 140.1 ± 2.1$^{a}$ | 141.6 ± 4.3$^{a}$ | 140.8 ± 4.2$^{a}$ | 139.8 ± 3.8$^{a}$ | 140.5 ± 2.6$^{a}$ |
| $K^{+}$ (mEq/L) | 4.5 ± 0.4$^{a}$ | 4.4 ± 0.4$^{a}$ | 4.5 ± 0.2$^{a}$ | 4.4 ± 0.2$^{a}$ | 4.5 ± 0.3$^{a}$ |

The reported values are the mean ± SD (n=12). Different superscript letters (a, b, and c) indicate significant differences between groups ($p$< 0.05). AST, aspartate amino transferase. ALT, alanine amino transferase. TC, total cholesterol. LDL-C, low density lipoprotein-cholesterol. HDL-C, high density lipoprotein-cholesterol. TG, triglyceride. FFA, free fatty acid.

**Table 4.** Effect of CAC on liver weight and liver lipid level in HFD-induced obese rats

| Parameters | ND | HFD | | | |
|---|---|---|---|---|---|
| | | Control | CAC-1X | CAC-2X | CAC-4X |
| Liver weight (g) | 11.5 ± 1.2$^{a}$ | 16.8 ± 2.0$^{c}$ | 15.0 ± 1.9$^{b}$ | 14.5 ± 2.0$^{b}$ | 14.3 ± 1.5$^{b}$ |
| Liver weight (%) | 2.4 ± 0.2$^{a}$ | 2.6 ± 0.2$^{a}$ | 2.5 ± 0.2$^{a}$ | 2.4 ± 0.2$^{a}$ | 2.4 ± 0.1$^{a}$ |
| TC (mg/g tissue) | 6.8 ± 1.5$^{a}$ | 17.4 ± 5.4$^{c}$ | 15.0 ± 4.8$^{bc}$ | 13.5 ± 1.6$^{b}$ | 13.1 ± 3.2$^{b}$ |
| TG (mg/g tissue) | 9.3 ± 5.2$^{a}$ | 65.3 ± 29.4$^{d}$ | 60.4 ± 30.0$^{cd}$ | 44.1 ± 31.1$^{bc}$ | 39.3 ± 11.7$^{b}$ |

The reported values are the mean ± SD (n=12). Different superscript letters (a, b, c, and d) indicate significant differences between groups ($p$< 0.05). TC, total cholesterol. TG, triglyceride.

## DISCUSSION

The relationship between diet and metabolic health is crucial, particularly concerning obesity and its associated complications [3]. This study examines the impact of an HFD on body fat accumulation and evaluates the potential mitigating effects of CAC supplementation. Key findings focus on weight loss, fat reduction, and liver enzyme levels, which are critical markers of liver health. Administration of the HFD resulted in a significant increase in body fat accumulation in rats. Following five weeks of feeding, subsequent treatment with CAC supplementation significantly reduced both body weight and body fat percentage (Figure 1 and Table 2). Specifically, a 1X dose led to a 7.5% reduction in body weight and a 22.8% decrease in body fat, while higher doses demonstrated even greater effects (9.5% weight loss and 28.9% fat loss for 2X doses; 10.1% weight loss and 33.8% fat loss for 4X doses). Previous studies have shown that mice fed anHFD supplemented with 1% Garcinia cambogia extract for 12 consecutive weeks exhibited significantly lower retroperitoneal, epididymal, mesenteric, perirenal, and total fat weights compared to the HFD-only group[14]. Another study reported that mice supplemented with Garcinia cambogia extract (400 mg/kg) for 8 weeks experienced significant reductions in final body weight and weight gain[15]. These findings suggest that CAC supplementation could effectively mitigate the adverse effects of HFD on body composition and obesity-related outcomes.

Feed utilization efficiency refers to an organism's ability to digest, absorb, and utilize nutrients from feed. Polyphenolic compounds have been shown to reduce feed utilization efficiency without affecting feed intake, thereby mitigating HFD-induced obesity [16]. The present study found that feed utilization efficiency was significantly lower during the CAC supplementation period compared to rats receiving only HFD (Table 1). Previous research demonstrated that apple extract (125 and 500 mg/kg) enhances the phosphorylation of hepatic *AMPK*, *ACC*, and *SIRT1* in HFD-induced obese mice, which inhibits lipogenesis and significantly reduces weight gain without altering food intake, thereby exerting anti-obesity effects [17]. Similarly, supplementation with rose extract has been reported to reduce lipid accumulation induced by HFD through downregulation of *AMPK*, *ACC*, *SIRT1*, and *PGC-1α* expression [18]. Additionally, HCA has been shown to inhibit the expression of hepatic *FAS*, *ACLY*, and *SREBP-1*, while upregulating PPARα expression, thereby suppressing lipogenesis and promoting lipolysis [19]. These findings suggest that although the rats consumed comparable amounts of feed, their ability to convert this intake into body mass was impaired by polyphenol, likely through the regulation of lipid metabolism.

The effects of HFD consumption on liver function were evident through elevated cholesterol and triglyceride levels, attributed to increased activity of HMG-CoA reductase and SREBP1c—key regulators of lipid metabolism [20]. Rats fed an HFD exhibited significantly higher hepatic cholesterol and triglyceride levels compared to the control group. However, supplementation with CAC effectively reduced these lipid levels (Table 4). Furthermore, serum markers such as ALT and AST, which were significantly elevated in HFD-fed rats and indicative of liver injury or inflammation, were mitigated by the administration of CAC supplements (Table 3). Studies have demonstrated that *G. cambogia* extract reduces hepatic triglyceride levels, enhances hepatic lipase activity, and increases ATGL mRNA expression, thereby promoting hepatic lipolysis in HFD-induced obese rats. Additionally, *G. cambogia* extract counteracts the HFD-induced elevation of ALT and AST, inhibits reactive oxygen species (ROS) production, and suppresses apoptosis by normalizing the Bcl-2/BAX ratio and reducing PARP cleavage[15]. Similarly, citrus plants, rich in polyphenols, have been shown to lower the levels of pro-inflammatory cytokines (TNF-α, IL-6, MCP-1, COX-2) in the liver, thereby inhibiting the development of HFD-induced non-alcoholic fatty liver disease (NAFLD)[21]. These findings suggest that CAC supplementation exerts protective effects against diet-induced hepatic stress and steatosis.

An HFD has been linked to increased insulin resistance, which in turn leads to elevated blood glucose levels and dyslipidemia—a common outcome of diet-induced obesity [22]. Previous studies have demonstrated that supplementation with *G. cambogia extract* in mice can prevent HFD-induced weight gain, reduce fasting blood glucose and TC levels, and improve insulin sensitivity, thereby exhibiting anti-obesity effects[5]. Similarly, supplementation with rose petal extract in mice has been shown to mitigate HFD-induced increases in ALT, AST, TG, TC, LDL-C, indicating its potential to ameliorate dyslipidemia[23]. Furthermore, dietary chromium (III) supplementation has been reported to enhance insulin sensitivity and glucose tolerance, thereby reducing the risk of hyperglycemia and atherosclerotic complications [24]. Consistent with these findings, the study revealed that CAC supplementation effectively reduced blood glucose and lipid levels (Table 3), aligning with observations from previous research.

## CONCLUSION

In summary, administering CAC supplementation for 4 weeks following obesity induction with an HFD effectively reduced final body weight, weight gain, feed efficiency, adipose tissue mass, hepatic lipid content, and blood lipid accumulation in obese rats. These findings highlight the anti-obesity potential of CAC supplementation, demonstrating a dose-dependent effect. Additionally, CAC supplementation improved abnormal blood metabolic parameters. Based on these results, CAC supplementation shows promise as a viable anti-obesity dietary supplement, with an effective dose of 207 mg/kg in rats, corresponding to a daily intake of approximately 2000 mg for a 60 kg adult.

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