Camellia oil Enhances Plasma Antioxidant Metabolism and Improves Plasma Lipid Metabolism in High-fat Diet-fed Rats

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Abstract
Living on a high-fat, high-calorie, and high-protein diet for a long period may compromise human immunity due to the long-term accumulation of free radicals and plasma lipids. The antioxidant and lipid-lowering compounds (ie polyphenols and vitamin E) in Camellia oil help to decrease the risk of numerous ailments, including cardiovascular disease (CVD), and obesity.

The aims of this study were to study the hypolipidemic and antioxidant effects of Camellia oil in high-fat-fed rats and to promote the high-value use of camellia resources. The high-fat-fed rats were administrated with 2.5, 7.5, and 15 mL/kg BW Camellia oil (Camellia oil group), and 10 mg/kg BW atorvastatin (atorvastatin group), respectively, and compared with a model group (only fed with high fat) and a control group (fed with basal diet). Several parameters were measured, including (1) body weight (BW), liver-to-BW ratio; (2) plasma total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C); and (3) alanineaminotransferase (ALT), alanine aminotransferase (AST), superoxide dismutase (SOD), glutathione peroxidase (GSH-Px) activity, model driven architecture (MDA) content, lipid metabolism-related genes, and antioxidant-related genes in liver tissue.

Compared with the model group, the high-fat-fed rats in the Camellia oil and atorvastatin group had significantly lower BW and liver-to-BW ratio (P<.01), plasma TC, TG, and LDL-C levels and ALT and AST activities, but higher HDL-C levels. The relative expressions of ACAT1, DGAT2, FAS, and SREBP genes were significantly reduced in the Camellia oil and atorvastatin groups, while the relative expressions of LCAT, UCP2, MCD, and CPT-1 genes were significantly increased. The rats in the Camellia oil group showed significantly higher SOD and GSH-Px activities, significantly lower MDA content, and significantly higher relative expression of antioxidant genes (eg SOD1, GPx1, CAT, and Gclm). Thus, atorvastatin and Camellia oil exhibited significant hypolipidemic and antioxidant effects, which were better at a dose of 7.5 mL/kg (BW) of Camellia oil. Therefore, Camellia oil becomes a potential new natural resource for future research and development of antioxidant and hypolipidemic drugs, nutraceuticals, and additives.

Keywords
camellia oil, rat, hypolipidemic, antioxidant, liver protection

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Introduction
Camellia oleifera Abel, family Theaceae, which is found in mountainous and hilly areas around the world, is one of the 4 major woody oil species in the world and one of the oldest woody edible vegetable oils in China.1,2 The oil of C. oleifera, which is extracted from the seeds, was the first hygienic vegetable edible oil promoted by Food and Agriculture Organization (FAO). It contains a variety of substances, such as camellia saponins, polyphenols, squalene, VE, and unsaturated fatty acids,3–6 which have health benefits, including improving blood...
circulation, softening blood vessels, lowering blood lipids, and scavenging oxygen free radicals.7–9

With the improvement in people’s living standards, the consumption of high-fat, high-calorie, and high-protein food in large quantities promotes a rapid increase in oxygen free radicals and blood lipid levels in the human body.10–12 Free radicals have a strong oxidizing ability that breaks down cells, producing a series of irreversible damage and affecting normal cell metabolism, accelerating the aging of the body.13 Meanwhile, hyperlipidemia promotes the deposition of fat in the blood vessels, decreasing the speed of blood flow through the arteries and increasing the risk of CVD and related mortality.14,15 Previous studies have shown that a 70% ethanol extract of C. oleifera has antioxidant and antimelanogenic activities.16 Moreover, water-soluble polysaccharides of C. oleifera have antitumor and antioxidant activities.17 However, references to the antioxidation and hypolipidemic effect of C. oleifera fruit oil remain unavailable. Therefore, in this study, the effects of the oil on hypolipidemic and antioxidant capacity in rats fed a high-fat diet were analyzed and the related mechanisms of action were explored.

This study had three aims: (1) to evaluate the hypolipidemic and antioxidant capacity of Camellia oil in rats; (2) to provide a theoretical basis for the future development of health foods and pharmaceutical products using Camellia oil, and to enrich the research base, both nationally and internationally; and (3) to improve the comprehensive benefits of Camellia oil, promote the sustainable and efficient development of the camellia industry and promote its economic value.

Methods

Chemicals

TC, triglyceride (TG), HDL-C, LDL-C, ALT, and AST kits were purchased from Mairui Medical Procedure Electronics Co. Ltd, Shenzhen, China, MDA, SOD, and GSH-Px kits from Nanjing Jiancheng Institute of Bioengineering, Changsha, China, Atorvastatin from Beida Weixin Biotechnology Co. Ltd, Beijing, China, DEPC and Trizo land RNA reverse transcription kits from Full-style Gold Biotechnology Co. Ltd, Beijing, China, Real-time Quantitative PCR Detecting System (qPCR) primers from Bioengineering Technology Co. Ltd, Shanghai, China, and basal and high-fat diets from Slake Jingda Laboratory Animal Co. Ltd, Changsha, China. The detailed ingredients of the diet are shown in Table 1.

Preparation of Camellia oil

30 g C. oleifera powder and 300 mL distilled water were added in a 500 mL conical bottle, and ultrasonically extracted under 400 W and a pH of 9 for 30 minutes. Enzyme hydrolysis was achieved with 2% alkaline procamelliase at 53 °C for 2.5 h. The enzymes were then inactivated by heating in a water bath at 90 °C for 10 min. The mixture was then centrifuged for 20 min at 5000 r/min. The supernatant, in a separating funnel, was extracted with 25 mL light petroleum, and the extract was evaporated under vacuum to remove the solvent. The composition of the oil is shown in Table 2.

Animals

Male Sprague Dawley (SD) rats of SPF grade, BW about 125 g, were purchased from Slake Jingda Laboratory Animal Co. Ltd, Changsha, China. Sixty healthy male rats were randomly divided into 6 groups according to their BW after 1 week of adaptive feeding. Animals were grouped and treated as described by Chou et al (Table 3).18,19 Rats were fed in individual cages on either a basal or high-fat diet. Feeding conditions were as followed: room temperature 22 to 25 °C (humidity: 40%-70%), 12-hour cycle alternating light and dark, free drinking water, good ventilation, change of bedding every other day, weekly cleaning, and disinfection of cages. The atorvastatin and Camellia oil groups were dosed orally once a day at 9:30 am. For the rest of the day, the animals could drink (water) and eat ad libitum for 6 weeks.20 After the last administration, the animals were fasted for 12 h, but could drink freely. After that, blood and liver tissues were taken to determine their enzyme

Table 1. Composition of the Experimental Diets (%).

| Ingredient       | Basal diet | High fat diet |
|------------------|------------|--------------|
| Corn starch      | 43%        | 30%          |
| Fiber            | 7%         | 5%           |
| Mineral mix      | 6%         | 4%           |
| Casein           | 29%        | 20%          |
| Vitamin mix      | 1%         | 0.8%         |
| Sucrose          | 7%         | 5%           |
| Cholesterol      | 0          | 4%           |
| Yolk powder      | 0          | 6%           |
| Lard             | 0          | 10%          |
| Bile salt        | 0          | 0.2%         |
| Total            | 100%       | 100%         |

Table 2. Composition of Camellia oleifera.

| Composition       | Protein | Cellulose | Starch | Water | Fatty acid | Ash | Other | Selenium |
|-------------------|---------|-----------|--------|-------|------------|-----|-------|----------|
| Camellia Seeds    | 13.3%   | 11.3%     | 14.5%  | 6.7%  | 35.1%      | 5.1%| 14.2% | 0.08 mg/kg |
| Camellia oil      | Palmitic acid | 8.8% | Stearic acid | 2.1% | Oleic acid | 10.2% | Linoleic acid | 8.0% | α-linolenic acid | 60.6% | Squalene | 0.6% | Sterol | 0.8% | VE |
activity and gene expression level. All investigations were carried out with reference to the Technical Specification for the Inspection and Evaluation of Health Food issued by the National Health Commission of China in 2003 under license [2003] No. 42.

**Determination of Body Condition**

The SD rats were observed daily for hair color, activity, dietary status, and mental condition. Liver weight and BW were measured and recorded.

**Determination of Serum Indicators**

The levels of TG, TC, HDL-C, and LDL-C, and the activities of ALT and AST were measured in the blood of rats, according to the kit instructions.

**Determination of Antioxidant Indicators**

The liver homogenate was frozen, centrifuged at 4000 r/min for 10 min at 4 °C, and the supernatant was collected. The SOD and GSH-Px activities and MDA content were measured according to the kit instructions.

**Real-Time Quantitative PCR Analysis**

RNA was extracted using a trizol kit. RNA was reverse transcribed into cDNA and then PCR amplified using genomic DNA as the template (see Table 4 for primer sequences). RT-PCR reaction system: 10 µL SYBR Green qPCR SuperMix, 1 µL template, 1 µL upstream primer (10nM), 1 µL downstream primer (10nM), supplemented with double distilled water to 20 µL. Reaction conditions were as follows: pre-denaturation at 94 °C for 30 s, denaturation at 94 °C for 5 s, annealing at 60 °C for 15 s, fluorescence detection for 15 s, 40 cycles. Relative quantitative 2^ΔΔCt analysis (CT value comparison method) was performed using β-actin as an internal reference to calculate the relative expression of each gene in the samples.

**Statistical Analysis**

Data were analyzed by SPSS 16.0. All data are presented as mean values with SD (mean ± SD, n = 3). Statistical analyses were performed using Student’s t-test, ANOVA, and LSD multiple comparison tests at the 95% confidence level. Statistical differences were considered to be *P < .01 and **P < .01.

**Results**

**Effect of Camellia oil on BW of Rats Fed With High-fat Diet**

The effects of Camellia oil and atorvastatin on the BW and liver weight of rats are shown in Table 5. The initial body mass of rats was 159 to 166 g in each group. The BW of rats was significantly increased in each group when they were treated with feed for 6 weeks. The BW and liver to BW ratio of rats were highest in the model group, reaching 402.6g and 4.4%, respectively. There was no significant difference in the liver-to-BW ratio between the model group and low-dose group, but a significant difference

**Table 3.** Design of Experimental Animal Groups.

| Group       | Number | Status | Feed           | Dose          |
|-------------|--------|--------|----------------|---------------|
| Control group | 10     | Normal | Basal diet     | 10 mL/kg/BW   |
| Model group | 10     | Normal | High-fat diet  | 10 mL/kg/BW   |
| Atorvastatin group | 10 | Normal | High-fat diet  | 10 mg/kg/BW   |
| Low-dose group | 10  | Normal | High-fat diet  | 2.5 mL/kg/BW  |
| Medium-dose group | 10 | Normal | High-fat diet  | 7.5 mL/kg/BW  |
| High-dose group | 10   | Normal | High-fat diet  | 15 mL/kg/BW   |

**Table 4.** Primer Sequences Used in qPCR Analysis.

| Gene name | Upstream primers (5’-3’) | Downstream primers (5’-3’) |
|-----------|--------------------------|----------------------------|
| β-actin   | CCACCATGTACCAGGCATTT    | AGGGTGTAAGGACGCTCA         |
| ACAT1     | CAGCTCTACCACCAGACCCTCT  | ACCATATGGTGGTCCTCT         |
| DGAT2     | CCTAAGGTGGTGCACTGCATTT  | CTGCCACACGGTGCACTGGT       |
| LCAT      | CTGGCTTTGCCAAGACCTAC    | TACCATCCTGCCAGCTTT         |
| UCP2      | CGGAGATACCAAGAGACCTGTC  | TGGCAATTCGCCAAGGCAACTGG    |
| MCD       | CGGCACCTCTCTAAGAAC      | TGGAAGGCCCCCAGAT           |
| CPT-1     | CACTGCGCCGCACTGCAAAG    | CCCAGAGGCTGACCAAGAG       |
| FAS       | AGGCCTAGGCTGAGGCTGAAA   | GATGAGGCTCAGCGACAGG        |
| SREBP     | GCTCTCTCTCTCTCTCTCTGG   | CAGGCCTAGGATGTCGAGT       |
| SOD1      | CCACCTCAGGACCTCAATTTTA  | TCCACTCAATCCTCTCTCTCATC    |
| GPx1      | CATGAGGAGGAGGCAAAGAA    | TCCACCATGCCAATCCTCTCTAA    |
| CAT       | CCCGAGTCAGGCAGGCCCATCT  | CGGCCATGATGAGGGAAGT       |
| Gelm      | TGTGATGACCAGGATTTT      | GTTCCTACGGATGACGAGT        |
was shown between the model group and medium and high-dose groups \((P<.01)\). Compared to the model group, the BW of rats fed with a high-fat diet was significantly decreased in the Camellia oil and atorvastatin groups, with the effect of the Camellia oil group being slightly better than that of the atorvastatin group. The medium dose \((7.5\, \text{mL/kg.BW})\) of Camellia oil produced the best effect.

### Histopathological Evaluation

Representative images of the liver tissue pathology of each group are shown in Figure 1. Figure 1B and D clearly depict the accumulation of lipid droplets in the hepatocytes. Moreover, a mild dysregulation of liver structure appeared to be evident in the rats. Interestingly, the weight loss treatment appeared to reverse the aforementioned steatosis. Little difference was observed in the histopathological specimens of the control (Figure 1A), medium dose (Figure 1E), and atorvastatin groups of rats (Figure 1C).

### Effect of Camellia oil on Blood Lipid in Rats Fed with High-fat Diet

The effects of Camellia oil and atorvastatin on blood lipids in rats fed with a high-fat diet are shown in Table 6. When compared to the model groups, the TC levels in the Camellia oil and atorvastatin groups decreased to some extent \((P<.01)\), while there was no significant difference between the low-dose Camellia oil group and the model group \((3.11\, \text{mmol L}^{-1})\). Atorvastatin and medium-dose Camellia oil treatments were the most effective and were not significantly different from the control group \((P>.01)\). Compared to the model group, TG levels decreased to some extent in both the Camellia oil and atorvastatin groups, but both were higher than the control group \((P<.01)\). Although Camellia oil and atorvastatin had some effect on reducing TG levels, they were still higher than those of the control. HDL-C levels were

### Table 5. Effect on BW of Rats.

| Group            | Initial weight (g) | Final weight (g) | Weight gain (g) | Liver body ratio (%) |
|------------------|--------------------|------------------|-----------------|----------------------|
| Control group    | 163.3 ± 5.2        | 361.6 ± 7.8      | 198.3 ± 8.5     | 3.6 ± 0.5            |
| Model group      | 159.6 ± 4.3        | 402.6 ± 6.7      | 243.1 ± 9.4     | 4.4 ± 0.9            |
| Atorvastatin     | 166.8 ± 4.3        | 369.5 ± 6.7      | 202.7 ± 9.4     | 3.9 ± 0.7*           |
| Low-dose group   | 160.9 ± 5.4        | 359.7 ± 10.5#    | 198.9 ± 11.1#   | 4.1 ± 0.5#           |
| Medium-dose group| 162.8 ± 4.2        | 354.4 ± 14.0*    | 191.6 ± 11.1*   | 3.7 ± 0.5*           |
| High-dose group  | 161.5 ± 4.8        | 366.8 ± 10.4#    | 203.3 ± 10.6*#  | 3.9 ± 0.6*           |

Note: Compared with model group, *\(P<.01\); Compared with control group, #\(P<.01\).

Figure 1. Histopathological analysis of liver. The photos were made at 100 magnifications. (A) Represents the control group, (B) the model group (lipid droplets accumulating in the hepatocytes, arrows), (C) the atorvastatin group, (D) the low-dose group, (E) the medium-dose group, and (F) the high-dose group.
The effects of Camellia oil and atorvastatin on the expression of lipid metabolism-related genes in liver tissues of rats fed with high-fat diet are shown in Figure 2. The relative expression of ACAT1 gene was very high (1.57) in the model group and lowest in the atorvastatin group at only 61.8% of the model group and also lower than the Camellia oil group. The relative expression of DGAT2 gene was the highest (1.48) in the model group and lowest in the atorvastatin and medium-dose Camellia oil groups (1.13 and 1.15, respectively). The relative expression of LCAT gene was the lowest (0.46) in the model group and increased significantly in the Camellia oil and atorvastatin groups ($P < .01$), with the atorvastatin group showing the best effect. The relative expression of UCP2 gene was the lowest (0.53) in the model group and increased significantly in the Camellia oil and atorvastatin groups ($P < .01$), with the medium-dose Camellia oil group showing the best effects. Compared to the model group, the relative expression of MCD gene was significantly increased in the Camellia oil and atorvastatin groups. Upregulation of the MCD gene would help to reduce free fatty acid (FFA) in vivo and TG in the liver. The relative expression of CPT-1 gene was lowest (0.58) in the model group and significantly increased in the Camellia oil and atorvastatin groups ($P < .01$). CPT-1 plays a regulatory role in the whole process of lipid oxidation. The relative expression of FAS gene was highest (2.41) in the model group and significantly decreased in the Camellia oil and atorvastatin groups ($P < .01$). The expression of FAS gene in the medium-dose Camellia oil group was only 55.6% of that in model group, and the downregulation of FAS gene expression could reduce fat synthesis. The relative expression of SREBP gene was highest (1.90) in the model group and significantly decreased in the Camellia oil and atorvastatin groups ($P < .01$). The expression of SREBP gene in the medium-dose Camellia oil group was only 61.1% of that in the model group; reducing SREBP gene expression would effectively reduce the total cholesterol level in cells.

### Effect of Camellia oil on Oxidative Indicator of Rats Fed With High-fat Diet

The effects of Camellia oil and atorvastatin on antioxidants in liver tissues of rats fed a high-fat diet are shown in Table 8. The lowest activities of SOD and GSH-Px were shown in the model group, with only 290.2 U/mg and 520.7 U/mg, respectively. When compared to the model group, the activity of SOD was increased the most in the medium-dose Camellia oil group, which was significantly higher (1.94) than the model group with no significant differences between the Camellia oil and atorvastatin groups.

### Table 6. Effects on Serum Lipid Levels of Rats.

| Group           | TC (mmol·L−1) | TG (mmol·L−1) | HDL-C (mmol·L−1) | LDL-C (mmol·L−1) |
|-----------------|--------------|---------------|------------------|------------------|
| Control group   | 2.06 ± 0.23  | 1.07 ± 0.26   | 1.64 ± 0.08      | 0.61 ± 0.11      |
| Model group     | 3.11 ± 0.21  | 2.58 ± 0.21   | 1.42 ± 0.14      | 1.26 ± 0.10      |
| Atorvastatin    | 2.29 ± 0.26* | 1.91 ± 0.34*# | 1.57 ± 0.13      | 0.72 ± 0.14*     |
| Low-dose group  | 2.79 ± 0.31# | 2.02 ± 0.28#  | 1.51 ± 0.12      | 0.76 ± 0.13*     |
| Medium-dose group | 2.38 ± 0.33* | 1.69 ± 0.46#  | 1.69 ± 0.21*     | 0.66 ± 0.08*     |
| High-dose group | 2.60 ± 0.18*#| 1.98 ± 0.23*# | 1.49 ± 0.10      | 0.79 ± 0.12*     |

Abbreviation: TG: triglyceride.

### Table 7. Effect on ALT and AST in Serum of Rats.

| Group           | Animals number (n) | ALT (U·L−1) | AST (U·L−1) |
|-----------------|--------------------|-------------|-------------|
| Control group   | 10                 | 57.4 ± 7.7  | 84.6 ± 10.4 |
| Model group     | 10                 | 81.6 ± 10.3 | 125.7 ± 12.6|
| Atorvastatin    | 10                 | 63.2 ± 5.9* | 101.3 ± 17.1#|
| Low-dose group  | 10                 | 67.7 ± 6.8* | 107.5 ± 9.2*#|
| Medium-dose group | 10              | 60.3 ± 11.2*| 98.2 ± 8.3*#|
| High-dose group | 10                 | 67.8 ± 8.2*#| 112.5 ± 11.1*#|

The effects of Camellia oil and atorvastatin on ALT and AST activities in high-fat-fed rats are shown in Table 7. The highest activities of the two enzymes in the model group reached 81.6 U·L−1 and 125.7 U·L−1, respectively. The activities of the two enzymes in the Camellia oil and atorvastatin groups were significantly decreased and were significantly different from those in the model group ($P < .01$). The ALT activity of the medium-dose Camellia oil group was not significantly different from that of the control group, but was much higher than that of the control group in the other doses of Camellia oil and atorvastatin groups. AST activity was much higher in the Camellia oil and atorvastatin groups than in the control group. These results suggest that Camellia oil and atorvastatin can alleviate and repair liver damage in rats fed a high-fat diet, but cannot completely prevent the damage from occurring.
oil group, and the activity of GSH-Px was increased the most in the atorvastatin group (\(P < .01\)). However, the activity of SOD and GSH-Px in both the Camellia oil and atorvastatin groups were lower than those of the control group. The highest MDA level of 3.2 \(\text{nmol/mg}\) was reported in the model group, but the atorvastatin and medium-dose Camellia oil groups had lower levels. The MDA content of the medium-dose Camellia oil group was close to that of the control, which could largely reduce the oxidative damage of membrane lipids.

**Effect of Camellia oil on Antioxidant Gene Expression in Rats Fed With High-fat Diet**

The effects of Camellia oil and atorvastatin on the expression of antioxidant genes in liver tissues of rats fed with a high-fat diet are shown in Figure 3. The relative expression of SOD1 gene was lowest (only 0.52) in the model group and increased significantly in liver tissues after treatment with either Camellia oil or atorvastatin (\(P < .01\)). The best effect was observed in the medium-dose Camellia oil group compared to the model group, with a 2.09-fold increase. The relative expression of CAT gene in the model group was also very low (only 0.43), and the atorvastatin group had the best effect with a 2.88-fold increase compared to the model group. GPx1 can regulate the activity of GSH-Px and attenuate further damage caused by ROS. The relative expression of GPx1 gene in the model group was very low (only 0.68), and the effect in the medium-dose Camellia oil group was the best, with a 1.37-fold increase compared to the model group. Glutamyl cysteine ligase is the rate-limiting enzyme for GSH-Px biosynthesis. The relative expression of Gclm gene was very high in the Camellia oil and atorvastatin groups, much higher than that in the model and control groups. The atorvastatin group showed the best effect (1.79), which was 0.79 times higher than that of the control group.

**Discussion**

Camellia oil and atorvastatin had strong hypolipidemic and antioxidant effects on high-fat diet-induced obesity in rats, while medium doses of Camellia oil (7.5 ml/kg BW) were generally more effective. Compared to the model group, rats given Camellia oil orally showed a decrease in BW and liver weight ratio, and the activities of AST and ALT were significantly decreased (\(P < .01\)), indicating that Camellia oil has some protective effect on the liver, which was consistent with the findings of Li X et al.\(^21\) Meanwhile, the levels of TC, TG, and LDL-C in the Camellia oil group were significantly lower than those in the model group (\(P < .01\)), while the level of HDL-C was increased, which is in good agreement with the results of Komprda et al.\(^22\)

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**Table 8. Effect on Antioxidant Indicators in Liver of Rats.**

| Group          | SOD (U/mg) | GSH-Px (U/mg) | MDA (nmol/mg) |
|----------------|------------|---------------|---------------|
| Control group  | 411.6 ± 17.4| 712.3 ± 22.7  | 1.7 ± 0.3     |
| Model group    | 290.2 ± 17.8| 520.7 ± 19.3  | 3.2 ± 0.7     |
| Atorvastatin group | 371.4 ± 21.9| 658.9 ± 21.3  | 2.1 ± 0.4*    |
| Low-dose group | 360.3 ± 17.8| 617.2 ± 19.3  | 2.7 ± 0.5     |
| Medium-dose group | 391.1 ± 13.5| 624.3 ± 26.2  | 2.2 ± 0.5*    |
| High-dose group | 372.2 ± 19.2| 616.6 ± 15.2  | 2.5 ± 0.2*    |

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![Figure 2. Effect on relative expression of lipid metabolism-related genes in liver of rat.](image)
Camellia oil is rich in linoleic acid, other unsaturated fatty acids, and many substances with hypolipidemic effects (ie sterols, squalene). Oleic acid can reduce LDL-C level, increase HDL-C level, reduce the extent of platelet aggregation and improve blood circulation. Linoleic acid plays a role in softening blood vessels and has a strong effect on reducing TG, LDL, and very low density lipoprotein (VLDL). Squalene can neutralize H⁺, reduce acidity, and enhance blood circulation. Sterols lower blood cholesterol levels and prevent cardiovascular and cerebrovascular diseases.

Compared with the model group, the relative expressions of ACAT1, DGAT2, FAS, and SREBP genes in the liver of rats were significantly decreased after treatment with Camellia oil. ACAT1 is a catalytic polymer allosteric enzyme, which is the only key enzyme for cholesterol esters formation. The downregulation of ACAT1 gene is beneficial for reducing TC level. DGAT2 promotes fat absorption in the intestine, catalyzes the synthesis of TG from glycerol diester, and promotes the formation of LDs. The formation and accumulation of TGs and cholesterol in the liver are avoided by downregulating the DGAT2 gene expressions. SREBP is an important transcriptional regulator leading to the TG biosynthesis pathway and also regulates lipid metabolism. By reducing SREBP gene expression, intracellular cholesterol levels should be effectively reduced. Furthermore, by reducing the expression of FAS gene, the synthesis of fatty acids in liver should be inhibited and the accumulation of lipids should be reduced.

The relative expression levels of LCAT, UCP2, MCD, and CPT-1 genes were significantly increased after treatment with Camellia oil. LCAT gene expression promotes the esterification of free cholesterol on the surface of HDL. The upregulation of LCAT gene expression enhances the ability of HDL to remove cholesterol from peripheral tissues and reduce the cholesterol level. Excessive accumulation of white fat and obesity should be avoided by increasing the expression of the UCP2 genes to increase energy expenditure, which explains why UCP2 gene expression enhanced the effect of weight loss. MCD is an important factor in regulating fatty acid oxidation since it can catalyze the conversion of coenzyme A to acetyl coenzyme A and carbon dioxide. Upregulation of MCD gene expression is beneficial to reduce the accumulation of FFA and TG in the liver. CPT-1 is the rate-limiting enzyme of mitochondrial-mediated fatty acids β-oxidation, which facilitates the mitochondrial uptake of long-chain fatty acids. Upregulation of the CPT-1 gene expression increases the oxidation of fat in hepatocytes and reduces the accumulation of lipid in cells.

Liver, the main metabolic organ of food, drugs, and even toxins, contains abundant antioxidant enzymes with comprehensive functions. Compared with the model group, the antioxidant enzyme activity was significantly increased in the liver of the rats after treatments with Camellia oil (P < .01). Camellia oil contains many antioxidants, such as selenium, squalene, and polyphenols (Table 2), which improve the activity of antioxidant enzymes in vivo and enhance the antioxidant capacity of the body. For example, selenium is an important component of the glutathione peroxidase system (GPx), whose activity is closely related to selenium level. Selenium scavenges lipid peroxides, free radicals and other harmful substances deposited on the vascular wall, keeping them open and ensuring the normal circulation of blood lipids. Furthermore, the relative expression of SOD1, GPx1, CAT, and Gclm genes in the liver of rats were significantly increased after being treated with Camellia oil (P < .01). SOD rapidly decomposes O₂⁻ to H₂O₂ and scavenges superoxide radicals, while H₂O₂ is reduced to H₂O by GSH-Px and CAT. Moreover, Gclm and Gclc are important factors in regulating fatty acid oxidation since they can catalyze the conversion of coenzyme A to acetyl coenzyme A and carbon dioxide. Upregulation of MCD gene expression is beneficial to reduce the accumulation of FFA and TG in the liver. CPT-1 is the rate-limiting enzyme of mitochondrial-mediated fatty acids β-oxidation, which facilitates the mitochondrial uptake of long-chain fatty acids. Upregulation of the CPT-1 gene expression increases the oxidation of fat in hepatocytes and reduces the accumulation of lipid in cells.

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antioxidant proteases, and upregulation of Gclm expression facilitates free radical scavenging.38

Conclusion
Camellia oil regulated lipid and free radical metabolism in rats fed a high-fat diet. This study provides a theoretical basis for the future development of lipid-lowering and antioxidant-related products and further promotes the development and utilization of oil tea resources.

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