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Green Tea Polyphenol Epigallocatechin Gallate Promotes Autophagy and Lipid Droplet Degradation in HepG2 Cells

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Abstract

Epigallocatechin gallate (EGCG), a major polyphenol in green tea, exerts beneficial effects on lipid metabolism and liver health. However, its underlying action mechanisms remain unclear. Therefore, in this study, we investigated the effects of EGCG on autophagy and lipid metabolism using HepG2 cells. Autophagy induction was evaluated based on light chain (LC)-3B-II protein expression and green fluorescent protein-LC3 puncta formation. Autophagic flux was assessed using tandem red fluorescent protein-green fluorescent protein-LC3. Lipophagy was examined by analyzing the co-localization of lipid droplets with the lysosomal marker, lysosomal-associated membrane protein-1. Oil red O staining was used to quantify intracellular lipid accumulation. EGCG treatment increased LC3B-II levels in a concentration-dependent manner and induced LC3 puncta formation, indicating enhanced autophagosome formation. Tandem red fluorescent protein-green fluorescent protein-LC3 analysis revealed that EGCG enhanced the autophagic flux, as evidenced by the increased proportions of autolysosomes. Furthermore, EGCG enhanced the co-localization of lipid droplets with lysosomes, suggesting the activation of lipophagy. Oil Red O staining revealed that EGCG significantly reduced intracellular lipid accumulation in HepG2 cells. These findings suggest that EGCG activates autophagy and lipophagy, leading to reduced lipid accumulation in HepG2 cells. Overall, our results provide mechanistic insights into the lipid-lowering effects of EGCG and highlight its potential as a dietary compound to prevent steatotic liver disease.

Keywords

EGCG; Obesity; Autophagy; Lipophagy

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1. INTRODUCTION

Lifestyle-related diseases have become a global epidemic, with obesity posing a serious health threats to modern society [1,2]. Obesity is particularly acute in developed countries, where changes in diet and physical activity patterns in recent decades have created a weight gain-promoting environment.

Visceral obesity leads to a cascade of metabolic disturbances including insulin resistance, abnormal blood lipid levels, and elevated blood pressure [3–6]. When these conditions occur together, they constitute what clinicians refer to as a “metabolic syndrome”. Patients with metabolic syndromes exhibit drastically increased risks of type 2 diabetes and cardiovascular disease. Cardiovascular disease

remains the leading cause of death worldwide, accounting for approximately 17.9 million deaths annually [7,8]. Visceral fat is associated with heart disease via several mechanisms; when the liver produces more glucose, cholesterol metabolism is disrupted, blood vessel's function is impaired, and oxidative stress accelerates arterial plaque formation [9,10].

Although reducing dietary fat intake is the most logical approach to prevent obesity, implementing lasting dietary changes is extremely difficult in practice [11,12]. Most people struggle to follow a long-term calorie-restricted diet due to various biological, psychological, and social factors. Therefore, daily consumption of visceral fat accumulation-suppressing bioactive compounds is a potential strategy to prevent obesity and reduce mortality related to atherosclerotic disease.

Tea consumption has various advantages. Tea contains several bioactive compounds that are potentially beneficial to human health. Green tea is particularly rich in catechins, with epigallocatechin gallate (EGCG) being the most abundant and well-studied catechin [13]. EGCG typically accounts for 50–80% of total catechins in green tea and exhibits antioxidant activity superior to that of vitamins C and E [14]. Notably, EGCG consumption is associated with reduced risks of cancer, neurodegenerative diseases, and importantly for this discussion, cardiovascular diseases, and obesity [15–17].

Substantial clinical evidence supports the weight management properties of EGCG. Meta-analyses of randomized controlled trials have revealed that EGCG supplementation considerably reduces the body weight, body mass index, and waist circumference [18–20]. Its effects are possibly the most pronounced in Asian populations and when combined with modest calorie reduction. In addition to causing weight loss, regular EGCG intake improves the cholesterol profiles, lowers the blood pressure, and enhances blood vessel functions. The mechanisms underlying these benefits possibly include increased energy expenditure, enhanced fat burning, reduced fat synthesis, and favorable changes in the gut bacterial composition.

Autophagy, a fundamental cellular process degrading dysfunctional proteins and organelles, also eliminates lipid droplets [21,22]. When autophagy is impaired in liver cells, fat accumulates and contributes to metabolic dysfunction-associated steatotic liver disease, insulin resistance, and other metabolic disturbances [23–26]. Therefore, promoting autophagy is a potential therapeutic approach for obesity-related disorders.

The relationship between EGCG and autophagy is complex and varies under different conditions. A previous study demonstrated that EGCG stimulates autophagy in liver cells [27]. In contrast, another study using diabetic model mice reported that EGCG suppresses autophagy [28], suggesting that the metabolic state of cells and disease context significantly influence the impact of EGCG on autophagy.

Despite growing evidence suggesting that EGCG prevents obesity, its action mechanisms remain unclear. Particularly, effect of EGCG on autophagy in fat-rich liver cells represents a notable knowledge gap. Considering the involvement of fat-loaded hepatocytes, determining whether EGCG promotes autophagy in obesity and steatotic liver disease is crucial to clarify its therapeutic potential.

This study aimed to address this knowledge gap by examining the mechanisms by which EGCG treatment affects autophagy and fat accumulation using HepG2 cells, a human liver cell line commonly used to study liver metabolism. Specifically, we aimed to determine whether EGCG stimulates autophagy and reduces intracellular lipid accumulation in hepatocytes.

2. MATERIALS AND METHODS

2-1. Materials

Immunostar Zeta chemiluminescence reagents and oil red O were obtained from FUJIFILM Wako Pure Chemical Corporation (Osaka, Japan), ProLong Diamond Antifade Mountant from ThermoFisher Scientific (Waltham, MA, USA), Alexa555-conjugated donkey anti-Mouse IgG (H + L) polyclonal antibody from Abcam (Cambridge, UK), (-)-EGCG and complete protease inhibitor cocktail from Sigma-Aldrich (St. Louis, MO, USA), Bodipy 493/503 from Tokyo Chemical Industry (Tokyo, Japan), anti-LC3 antibody (Cat. No. PM036), and anti-b-actin antibody (Cat. No. M177-3) antibodies from MBL (Nagoya, Japan), anti-lysosomal associated membrane protein (LAMP)-1 antibody (Cat. No. NB120-19294) from Novus Biologicals (Centennial, CO, USA), horseradish peroxidase (HRP)-conjugated goat anti-rabbit IgG (H + L) polyclonal antibody (Cat. No. 611-1302) from Rockland Immunochemicals (Gilbertsville, PA, USA), and HRP-conjugated goat anti-mouse IgG (H + L) polyclonal antibody (Cat. No. A90-116P) from Bethyl (Montgomery, TX, USA). Additionally, human hepatoma cell line, HepG2 (RCB1648), was provided by the RIKEN BRC through the National Bio-Resource Project of the MEXT/AMED, Japan.

2-2. Cell culture and transfection

HepG2 cells were cultured in the Dullbecco's modified Eagle's medium supplemented with 10%

fetal bovine serum, 100 U/mL penicillin, 100 µg/mL streptomycin, and non-essential amino acids at 37 °C in 5% CO2. For DNA transfection, green fluorescent protein (GFP)-LC3 and tandem-GFP-red fluorescent protein (RFP)-LC3 (tf-LC3) plasmids were each transfected into HepG2 cells using Polyethyleneimine MAX.

2-3. Western blotting

HepG2 cells were seeded at a density of 3 × 10^5 cells/well in a 12-well plates. The next day, the cells were treated with EGCG in 10% fetal bovine serum/Dullbecco's modified Eagle's medium for 16 h. After washing with cold phosphate-buffered saline (PBS), the cells were lysed with a lysis buffer (62.5 mM Tris-HCl [pH 6.8], 2% sodium dodecyl sulfate, 10% glycerol, 5% 2-mercaptoethanol, 0.02% bromophenol blue, and complete protease inhibitor cocktail) and transferred to microcentrifuge tubes. The cell lysates were sonicated, boiled for 5 min, and subjected to sodium dodecyl sulfate-polyacrylamide gel electrophoresis, followed by protein transfer to polyvinylidene difluoride membranes. The membranes were blocked with Tris-buffered saline with Tween-20 (20 mM Tris-HCl [pH 7.4], 150 mM NaCl, and 0.1% Tween 20) containing 1% skim milk and probed with primary antibodies overnight at 4 °C, followed by incubation with HRP-conjugated secondary antibodies for 1 h at room temperature. The signals were visualized using the Immunostar Zeta chemiluminescence reagents, and signal intensities were analyzed using the LuminoGraph II imaging analyzer (ATTO, Tokyo, Japan).

2-4. Immunofluorescence imaging

For ectopic GFP–LC3 puncta imaging, GFP–LC3 plasmid was transfected into HepG2 cells, and the cells were observed under a laser-scanning microscope (FV3000; Olympus). For autophagic flux analysis, tf-LC3 plasmid was transfected into HepG2 cells, and the cells were visualized under a laser-scanning microscope (FV3000; Olympus). For lysosome and lipid droplet imaging, HepG2 cells were treated with 0.2 mM oleic acid-bovine serum albumin (BSA) and 40 µM EGCG for 16 h and incubated with 2 µM BODIPY 493/503 solution for 15 min. After washing with PBS, the cells were fixed with 4% paraformaldehyde at room temperature for 10 min, washed again with PBS, permeabilized with 50 µg/mL digitonin for 5 min, and blocked with 3% BSA/PBS buffer for 30 minutes. Subsequently, the cells were incubated with anti-LAMP1 antibody at room temperature for 1 h, followed by incubation with Alexa Fluor 555-labelled anti-mouse antibody at room temperature for 1 h. Coverslips were washed with PBS and mounted with the ProLong Diamond Antifade Mountant. Confocal images were acquired using a laser-scanning microscope (FV3000; Olympus).

2-5. Oil red O staining

Oil red O powder (30 mg) was dissolved in isopropyl alcohol (10 mL) and diluted with deionized water at a 3:2 ratio. The mixture was filtered to obtain the staining solution. HepG2 cells were seeded in culture dishes and treated with 0.2 mM oleic acid-BSA and 40 µM EGCG for 16 h. After washing with PBS, the cells were fixed with 4% paraformaldehyde and stained with oil red O solution for 15 min. Then, the cells were subsequently washed with 60% isopropyl alcohol and deionized water. Stained lipid droplets were eluted with 100% isopropyl alcohol, and optical density at 520 nm was determined using a spectrophotometer.

2-6. Statistical analyses

Data are represented as the mean ± standard deviation. Statistical analyses were conducted via one-way analysis of variance, followed by Tukey's post-hoc test. The sample size is indicated in each figure legend. Statistical significance was set at $p < 0.05$.

3. RESULTS

3-1. Effect of EGCG on light chain (LC)-3 levels in HepG2 cells

We investigated the effect of EGCG on autophagy in HepG2 cells. We measured the levels of microtubule-associated protein LC3B-II after EGCG addition via western blotting. LC3B-II is an autophagy marker. During autophagosome formation, phosphatidylethanolamine binds to LC3B-I, leading to the conversion of LC3B-I to LC3B-II. Upon treatment with EGCG, HepG2 cells showed an increase in LC3B-II levels in a concentration-dependent manner (Figure 1a and b).

Next, we examined LC3 puncta formation via confocal microscopy. Cytoplasmic LC3 puncta are characteristic features of autophagosome membrane formation. HepG2 cells were transfected with the GFP–LC3 plasmid to observe the effects of EGCG treatment. As shown in Figure 1c, GFP–LC3 diffused throughout the cytoplasm in control cells; however, treatment with 40 µM EGCG caused GFP–LC3 to adopt a punctuate dot distribution in the cytoplasm.

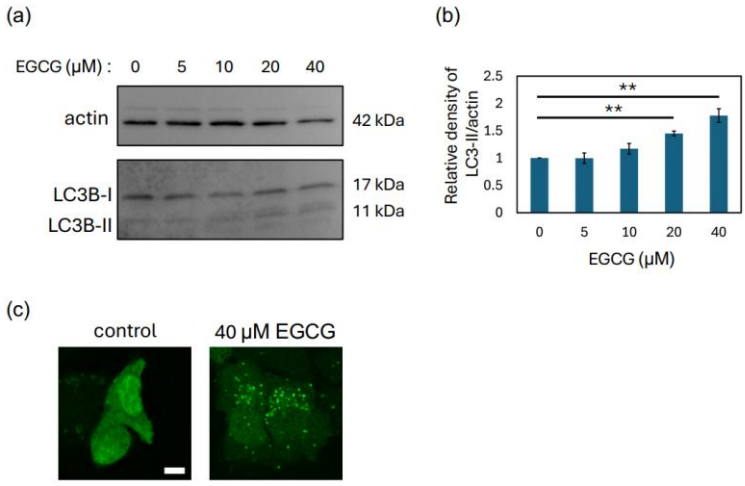

Figure 1: Effects of epigallocatechin gallate (EGCG) on light chain (LC)-3 levels in HepG2 cells.
(a) Immunoblotting analysis revealed the dose–response relationship of LC3B-II accumulation in HepG2 cells treated with the indicated concentrations of EGCG. (b) Bar chart shows the results of densitometric analysis in (a). Bars represent the mean of the respective individual ratios ± standard deviation (SD; n = 3). Significant differences compared to control cells are indicated by $*p < 0.05$ and $**p < 0.01$ (Tukey's post-hoc test). (c) Representative confocal image of green fluorescent protein (GFP)–LC3 puncta in transfected HepG2 cells. Scale bar, 20 μm.

3-2. EGCG stimulates HepG2 cell autophagy

Next, we investigated the effect of EGCG on the autophagic flux. We transfected the tandem RFP–GFP–LC3 (tfLC3) plasmid into HepG2 cells to measure the autophagic flux. Notably, tfLC3 emitted both RFP and GFP fluorescence in autophagosomes, whereas GFP was denatured in the acidic environment of autolysosomes, resulting in RFP fluorescence alone. Autophagosomes appeared as yellow dots, whereas autolysosomes appeared as red dots. HepG2 cells treated with 40 μM EGCG exhibited several autolysosomes (red dots, Figure 2). Overall, autophagic flux was enhanced following EGCG treatment.

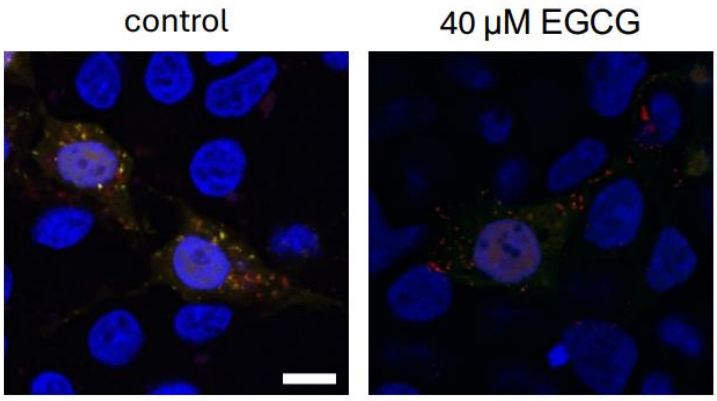

Figure 2: Confocal microscopic images of tandem red fluorescent protein (RFP)–GFP–LC3 (tfLC3) puncta in transfected HepG2 cells. Scale bar, 20 μm.

3-3. EGCG promotes the fusion of lipid droplets and lysosomes

We investigated whether autophagy promoted by EGCG contributes to the removal of intracellular lipid droplets. Lipid droplets were accumulated in oleic acid-treated HepG2 cells. To determine whether autophagy promoted by EGCG is involved in intracellular lipid droplet degradation, we examined the co-localization of LAMP-1, a lysosomal marker protein, with lipid droplets. Lipid droplets and lysosomes did not co-localize in control cells; however, their co-localization was observed in EGCG-treated cells (Figure 3). These results suggest that EGCG promotes intracellular lipid droplet degradation.

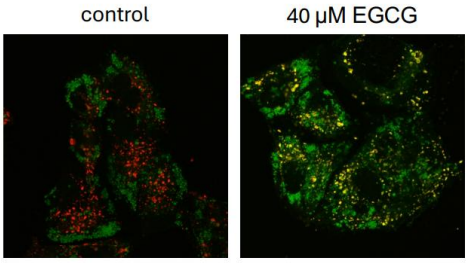

Figure 3: Co-localization of lipid droplets and lysosomes. Lipid droplets were stained with BODIPY 493/503 (green), and lysosomes were stained with the lysosomal-associated membrane protein 1 (LAMP-1) antibody (red). Scale bar, 20 µm.

3-4. EGCG reduces lipid droplet accumulation in HepG2 cells

To investigate the effect of EGCG on intracellular lipid accumulation, oil red O staining was performed to visualize and quantify neutral triacylglycerides. The dye was extracted and measured spectrophotometrically for the semi-quantitative assessment of lipid content. EGCG treatment significantly reduced intracellular lipid accumulation, showing potential to modulate cellular lipid metabolism and prevent lipotoxicity (Figure 4).

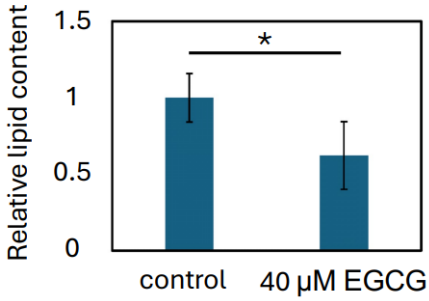

Figure 4: Semi-quantitative assessment of lipid levels in HepG2 cells. Bars represent the mean of the respective individual ratios ± SD (n = 3). Significant differences compared to control cells are indicated by * $p < 0.05$ and ** $p < 0.01$ (Student's t-test).

4. DISCUSSION

In this study, we found that EGCG induced autophagy and promoted lipid degradation by enhanceing lipophagy in HepG2 cells. Our findings provide a mechanistic basis for the lipid-lowering effects of EGCG, supporting its use for the prevention and treatment of metabolic liver disorders.

EGCG treatment significantly increased LC3B-II levels and induced LC3 puncta formation in HepG2 cells. These results suggest that EGCG activates the autophagic machinery in a concentration-dependent manner. Consistently, tfLC3 assay showed that EGCG not only promoted autophagosome formation but also facilitated their maturation into autolysosomes, suggesting that EGCG stimulates the autophagic flux, rather than merely blocking autophagosome degradation. This comprehensive activation of autophagy provides strong evidence of the role of EGCG as an autophagy inducer.

EGCG also enhanced the co-localization of lipid droplets with lysosomes, potentially promoting lipophagy. Lipophagy, a selective form of autophagy, is critical for lipid turnover and energy homeostasis [29,30]. The observed co-localization of lipid droplets with LAMP-1 following EGCG treatment suggests that EGCG facilitates the delivery of lipid droplets to lysosomes for degradation. In addition to its previously reported antioxidant and anti-inflammatory activities [27,31,32], our findings suggest a potential mechanism by which EGCG contributes to lipid metabolism. Furthermore, EGCG treatment markedly reduced intracellular lipid accumulation, as indicated by oil red O staining. This reduction in lipid accumulation confirmed that EGCG-mediated autophagy contributed to the breakdown of stored lipids. Excessive lipid accumulation in hepatocytes is a hallmark of metabolic dysfunction-associated steatotic liver disease [23]. Collectively, these findings highlight the therapeutic potential of EGCG to ameliorate hepatic steatosis.

Despite providing mechanistic insights into the lipid-lowering effects of EGCG, this study has some limitations. First, the upstream signaling pathways through which EGCG activates autophagy, such as the AMP-activated protein kinase activation or

mechanistic target of rapamycin inhibition pathways, remain unclear [31]. Therefore, further investigation is necessary to determine whether similar effects occur in vivo under various physiological and pathophysiological conditions. Second, whether EGCG specifically enhances lipophagy or broadly stimulates various autophagy pathways that secondarily influence lipid metabolism warrants further investigation. Comparative studies with other dietary polyphenols are also necessary to determine whether the observed effects are unique to EGCG.

5. CONCLUSIONS

In conclusion, EGCG promoted autophagy and lipophagy, reducing intracellular lipid accumulation in HepG2 cells. Our results provide insights into the mechanisms underlying the beneficial effects of EGCG on hepatic lipid metabolism and highlight its potential as a dietary compound for the prevention of steatotic liver disease. However, further investigations in animal models and clinical settings are essential to clarify its therapeutic relevance.

6. ACKNOWLEDGEMENT

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7. CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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