Original Research

The Effects of Epigallocatechin-3-Gallate on Thermogenesis and Fat Oxidation in Obese Men: A Pilot Study

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Key words: EGCG, epigallocatechin gallate, fat oxidation, energy expenditure

Objectives: The development of obesity is characterized by an increase in adipose tissue mass and by concomitant and profound changes in almost all organ functions leading to diseases such as hypertension, diabetes mellitus and coronary heart disease. Recent data from human studies indicate that the consumption of green tea and green tea extracts may help reduce body weight, mainly body fat, by increasing postprandial thermogenesis and fat oxidation. However, human studies investigating the metabolic effects of the most predominant tea catechin, EGCG, alone are absent.

Methods: In a randomized double blind, placebo-controlled, cross-over pilot study, six overweight men were given 300 mg EGCG/d for 2d. Fasting and postprandial changes in energy expenditure (EE) and substrate oxidation were assessed.

Results: Resting EE did not differ significantly between EGCG and placebo treatments, although during the first postprandial monitoring phase, respiratory quotient (RQ) values were significantly lower with EGCG compared to the placebo.

Conclusions: These findings suggest that EGCG alone has the potential to increase fat oxidation in men and may thereby contribute to the anti-obesity effects of green tea. However, more studies with a greater sample size and a broader range of age and BMI are needed to define the optimum dose.

INTRODUCTION

Obesity is increasingly recognized as public health burden, because it is associated with an increased risk for many diseases, including the metabolic syndrome, i.e., hypertension, insulin resistance or type 2 diabetes mellitus, arteriosclerosis and coronary heart disease [1]. For example, in the San Antonio Heart Study [2], 80% of obese subjects were hypertensive and diabetic, 85% of diabetics were hypertensive and obese, and 67% of the hypertensives were diabetic and obese. In 1999–2000, the age-adjusted prevalence of overweight and obesity in the US was estimated at 64.5% and 30.5%, respectively [3]. An important link between hypertension and obesity appears to be insulin resistance.

Insulin resistance develops as physiological fat stores (adipose tissue) expand. When fat (triacylglycerides) is routed into the visceral adipose tissue and into liver, muscle and pancreas, organ function worsens in a complex manner resulting in insulin resistance [4]. Increases in adipocyte number (hyperplasia) and size (hypertrophy) are also associated with the accumulation and activation of macrophages in the adipose tissue [5]. A number of inflammatory factors are known to interact with the insulin signaling cascade with a concomitant reduction in insulin sensitivity [5].

Due to the complex nature of obesity (genetic factors contribute about 66% and environmental factors 33%) treatment of obesity is also a complex issue. An optimal therapeutic approach should address both factors by: 1) changing energy
balance in a more negative direction by increasing energy expenditure and/or decreasing energy intake; and (2) improving insulin signaling and metabolism. Over the last two decades, numerous strategies, both non-pharmacological and pharmacological, have been developed in order to achieve long-term body-weight reduction and improve risk factors. Non-pharmacological approaches aim to: (1) change eating behavior, i.e., reduce caloric intake in general and reduce intake of fat and simple sugars in particular; and to (2) increase energy expenditure, mainly by increasing physical activity. Pharmacological approaches aim to: (1) change eating behavior, i.e., reduce hunger or appetite and increase satiety; and to (2) increase energy expenditure, mainly by increasing thermogenesis. Since conventional weight management programs have had only limited success, particularly in long-term efficacy, there is growing interest in alternative strategies for weight management, including pharmacological interventions.

One potential anti-obesigenic agent currently in focus is green tea. For centuries, green tea has been a widely consumed beverage in Asia. Green tea has also been reported to have medicinal efficacy in the prevention and treatment of many diseases. Around 2700 BC, discovery of the detoxifying and health-maintaining effects of green tea was attributed to the legendary Chinese emperor Shen Nung [7]. Later, Chen Zang, a noted pharmacist of the Tang Dynasty (618–907 AD) highlighted a broad range of health-promoting effects induced by tea intake. Although the Chinese pharmacist Wang Ang (1615–1695 AD) stated that chronic consumption of tea eliminated fat, the scientific and medical evaluation of tea as an anti-obesigenic agent began over the last 12 years [8]. Meanwhile, a large number of publications have reported on its anti-inflammatory, anti-arthritis, antibacterial, anti-angiogenic, antioxidative, antiviral, neuroprotective, and cholesterol-lowering effects [9]. These effects might all contribute to the prevention of cancer and obesity-associated diseases such as type 2 diabetes mellitus, hypertension, and cardiovascular diseases [9].

EGCG, the most abundant catechin in green tea, representing approximately 35% of total catechins, has received the most attention as a potential anti-obesigenic agent [10,11]. Some evidence suggests green tea extracts favorably affect body weight and body fat. Long term treatment (12 wk) with green tea extract containing 115 mg EGCG daily significantly reduced body fat (7%), body weight (2%), and BMI (2%) in men and women [12]. These findings are supported by other studies in which healthy volunteers received a green tea extract containing 270–300 mg EGCG, and reduced body weight by 1.2% [13] to 1.5% [14]. In one study, total body fat was reduced by 6.5%, mainly due to a reduction of visceral fat (8.7%) [14].

As mentioned above, an optimum strategy to prevent/treat obesity is to reduce energy intake/storage and increase energy expenditure. According to Kao et al. [8], complex alterations in the activities of fat, liver muscle, and intestinal cells may contribute to this goal. Possible mechanisms include: 1) decreasing digestive activity, 2) increasing lipolytic activity, 3) decreasing lipogenic activity, 4) increasing fat oxidation and thermogenesis, 5) modulation of the activity and expression of lipoprotein lipase, 6) decreasing the cell number and size of adipocytes, and 7) decreasing hormone-stimulated proliferation of preadipocytes and their differentiation to adipocytes [8]. Indeed a large body of evidence from in vitro and animal model studies suggests that all these activities can be positively affected by green tea extracts or EGCG (Fig. 1). Detailed information about these mechanisms is found in reviews by Kao et al. [8] and Wolfram et al. [9]. However, despite the numerous in vitro and animal studies, there are only a few publications regarding the mechanism of the anti-obesity properties of green tea and its main catechin, EGCG, in humans (Table 1) [15–18].

Evidence from human studies suggests that the anti-obesity properties of extracts from green or oolong tea may, at least in part, be due to increased fat oxidation [15,16]. In a randomized, double blind, placebo-controlled trial, fat oxidation increased by 35%, and 24-h respiratory quotient (RQ) decreased by 3.4% after supplementation with encapsulated green tea extract [15]. Male volunteers consumed either placebo, 150 mg caffeine or a green tea extract containing 270 mg EGCG plus 150 mg caffeine. As expected, caffeine stimulated the sympathetic nervous system and, thus, increased energy expenditure (EE). The consumption of the green tea extract increased EE above that observed with caffeine alone, suggesting other components in green tea extract contributed to increased fat oxidation. In a randomized cross-over trial of oolong tea, fat oxidation also increased (12%) above control among 12 healthy volunteers who consumed this tea, which contained 244 mg EGCG and 270 mg caffeine, daily [16]. After single administration of either oolong tea (77 mg caffeine, 81 mg EGCG) or green tea (161 mg caffeine, 156 mg EGCG), Komatsu et al. [17] reported cumulative increases in EE of about 111 and 50 kcal, respectively, over 2 h. Bérubé-Parent et al. [18] reported an increase of 8% in 24-h EE but no significant increases in fat oxidation after administration of green tea and Guarana extracts containing 600 mg caffeine and 270 mg EGCG. Interestingly, higher doses of EGCG (600, 900, and 1200 mg) at a fixed caffeine dose (600 mg) did not result in a further increase in 24-h EE [18].

To date, clinical studies investigating the anti-obesity effects of green tea have used either green tea, green tea extract or oolong tea. Most of the putative health benefits are attributed to EGCG, the most abundant catechin in green tea. However, the anti-obesigenic effects of pure EGCG on fat oxidation in humans have not been studied. Thus, we conducted a pilot study to examine the thermogenic and fat oxidation potential of EGCG in overweight men.
Fig. 1. Mechanisms by which EGCG may decrease energy intake, increase energy expenditure, and reduce adipose tissue mass and prevent or treat obesity and its associated diseases, diabetes and hypertension (adapted from [8]). Abbreviations: HDL, high density lipoprotein; LDL, low density lipoprotein; VLDL, very low density lipoprotein; IGF, insulin-like growth factor; CCK, cholecystokinin; GLUT4, glucose transporter 4; ACC, acetyl-CoA carboxylase; FAS, fatty acid synthase; GPDH, α-glycerophosphate dehydrogenase; SCD1, stearoyl-CoA desaturase 1; HSL, hormone sensitive lipase.

MATERIALS AND METHODS

Subjects

Six healthy overweight/obese men with a sedentary lifestyle (mean age 40 ± 1 y; BMI 29.9 ± 1.6 kg/m²) were recruited for this randomized, double blind, placebo-controlled, cross-over study. Additional exclusion criteria were medication or dietary supplement use in the week prior to the study or during the study itself, use of any medications containing caffeine (e.g. analgesics, anorectics, anal ogics), habitual caffeine intake of ≥300 mg/d (approximately ≥3 cups) of habitual green tea consumption ≥5 cups, and smoking.

All measurements were carried out at the Franz Volhard Centre for Clinical Research, Charité, University Medicine Berlin, Germany. All participants gave written informed consent. The study protocol was approved by the Institutional Review Board of the Charité Campus Buch, Berlin.

Experimental Procedure

Volunteers were asked to consume two capsules of either placebo or 150 mg EGCG daily for 2 d prior to testing.

Volunteers arrived at the clinical research center at 18:00 on the evening before the testing day, i.e., during the second day of supplementation. One hour later, a standardized dinner was administered followed by a 12-h fast. On the following day, capsules were administrated to the fasted volunteers at 07:00. Testing was conducted in the supine position. Oxygen uptake and carbon dioxide production were measured by indirect calorimetry with a canopy device to assess fasting and postprandial changes in EE, RQ (VCO₂ produced/VO₂ consumed), and substrate oxidation rates. One hour after taking the capsule, fasting metabolic rate was measured for the next 2 h followed by a 30 min break and another 2-h metabolic monitoring. Monitoring was then interrupted for 60 min: 30 min for recovery and 30 min for intake of the test meal containing 5 kcal/kg body weight with 50%, 35%, and 15% energy from carbohydrates, lipids, and proteins, respectively. Metabolic monitoring was then continued for 2 h followed by a 30 min break and another 2-h period of monitoring.

EE (metabolic rate) and rates of carbohydrate and fat oxidation were calculated according to the equations of Ferrannini et al. 1988 [19]. RQ, defined as carbon dioxide production divided by oxygen consumption, was also calculated. Under
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Table 1. Effects of Green Tea, Green Tea Extract High in EGCG, Oolong Tea, or Caffeine on 24-h Energy Expenditure (EE), Respiratory Quotient (RQ), and Fat Oxidation (FOX)

<table>
<thead>
<tr>
<th>Study reported by</th>
<th>Study type</th>
<th>Study population</th>
<th>Study duration</th>
<th>Components tested</th>
<th>Main outcome</th>
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<tbody>
<tr>
<td>Dollo et al. (1999)</td>
<td>Randomized, double-blind, placebo-controlled, cross-over, respiratory chamber</td>
<td>Healthy men, n = 10, sedentary Age: 25 yrs. (mean) BMI: 25 kg/m² (mean)</td>
<td>1 day</td>
<td>1) 150 mg caffeine, 270 mg EGCG, (green tea extract, 375 mg catechins)</td>
<td>1) 24h EE: +4.0%, 24h RQ: −3.4%, 24h FOX: +35%</td>
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<td>2) 150 mg caffeine</td>
<td>2) 24h EE: n.s., 24h RQ: n.s.</td>
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<td>2) 270 mg caffeine</td>
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<td>3) oolong tea, half strength 135 mg caffeine, 122 mg EGCG, (306 mg catechins)</td>
<td>3) 24h EE: +3.4%, 24h FOX: +8%</td>
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<td>4) oolong tea, full strength 270 mg caffeine, 244 mg EGCG, (612 mg catechins)</td>
<td>4) 24h EE: +2.9%, 24h FOX: +12%</td>
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<td>Rumpler et al. (2001)</td>
<td>Randomized, controlled (water), cross-over, respiratory chamber</td>
<td>Healthy men, n = 12, sedentary Age: 44 yrs. (mean) BMI: 26 kg/m² (mean)</td>
<td>3 days</td>
<td>1) water</td>
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<td>2) oolong tea, 77 mg caffeine, 81 mg EGCG</td>
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<td>3) green tea, 161 mg caffeine, 156 mg EGCG</td>
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<td>1) Green tea and Guarana extracts</td>
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<td>1) 600 mg caffeine, 270 mg EGCG</td>
<td>1) 24h EE: +8% 24h RQ: n.s.</td>
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<td>2) 600 mg caffeine, 600 mg EGCG</td>
<td>2) 24h EE: +8% 24h RQ: n.s.</td>
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<td>3) 600 mg caffeine, 900 mg EGCG</td>
<td>3) 24h EE: +8% 24h RQ: n.s.</td>
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<td>4) 600 mg caffeine, 1200 mg EGCG</td>
<td>4) 24h EE: +8% 24h RQ: n.s.</td>
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<td>Komatsu et al. (2003)</td>
<td>Randomized, controlled (water), cross-over, sedentary Age: BMI: 21 kg/m² (mean)</td>
<td>Healthy women, n = 11 sedentary Age:</td>
<td>Single treatment</td>
<td>1) water</td>
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<td>2) oolong tea, 77 mg caffeine, 81 mg EGCG</td>
<td>2) 24h EE: +11 ± 1 kJ</td>
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<td>3) green tea, 161 mg caffeine, 156 mg EGCG</td>
<td>3) 24h EE: +0 ± 0 kJ</td>
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<td>1) 600 mg caffeine, 270 mg EGCG</td>
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<td>4) 600 mg caffeine, 1200 mg EGCG</td>
<td>4) 24h EE: +11 ± 1 kJ</td>
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<td>Bétrubé-Parent et al. (2005)</td>
<td>Randomized, double-blind, placebo-controlled, cross-over, respiratory chamber</td>
<td>Healthy men, n = 14, sedentary Age: 35 yrs. (mean) BMI: 26 kg/m² (mean)</td>
<td>1 day</td>
<td>1) water</td>
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<td>2) oolong tea, 77 mg caffeine, 81 mg EGCG</td>
<td>2) 24h EE: +11 ± 1 kJ</td>
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Fasting conditions, RQ can theoretically vary between 1.0 (total carbohydrate oxidation) and 0.7 (total lipid oxidation). Plasma insulin, glucose, and non-esterified fatty acids were determined with an automated analyzer. Heart rate, as well as systolic and diastolic blood pressures, was measured continuously.

Supplement

EGCG was provided as TEAVIGO™ by DSM Nutritional Products (Basel, Switzerland). TEAVIGO™ is a highly purified extract from the leaves of green tea (Camellia sinensis) containing at a minimum 94% EGCG and at a maximum 0.1% caffeine.

RESULTS

Resting EE did not differ significantly between EGCG and placebo treatments. After intake of the meal, EE changed in the same way with both treatments, i.e., a strong initial increase followed by a slow decrease towards the end of the measurement (Fig. 2, upper panel).

With the placebo, fasting RQ was 0.87 ± 0.03 and remained almost unchanged over the following 90 min, then decreased to 0.79 ± 0.01 at t = 280 min, and returned to fasting values at t = 330 min (Fig. 2, lower panel). After the intake of the test meal, RQ increased to 0.91 ± 0.07 at t = 460 min, remained at that level until t = 570 min, and returned nearly to the fasting value at the end of the test.

With EGCG treatment, fasting RQ was 0.87 ± 0.04 and oscillated around that value for the following 90 min, then decreased slightly, but not significantly, to 0.81 ± 0.06 at t = 330 min. After the test meal, RQ increased to 0.84 ± 0.03 at t = 420 min and remained at this fasting value until the end of the test. During the first postprandial monitoring phase, RQ values were significantly lower with EGCG compared to the placebo.

DISCUSSION

This pilot study was designed to test whether EGCG alone increased EE and fat oxidation in overweight/obese volunteers and to differentiate the effects of EGCG between fasting and postprandial conditions. EGCG alone at a daily dose of 300 mg has the potential to increase fat oxidation, at least in the postprandial state, with a magnitude similar to that found by
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Dulloo et al. [15] who examined the efficacy of a green tea extract rich in catechins and caffeine on 24-h EE and fat oxidation in humans. On three separate occasions, subjects received either a green tea extract (50 mg caffeine/90 mg EGCG), 50 mg caffeine or a placebo 3 times/d. Fat oxidation increased significantly with the green tea extract when compared to the control group, whereas caffeine alone resulted in a higher, but statistically insignificant, increase in fat oxidation [15]. Rumpler et al. [16] studied the efficacy of oolong tea, high in EGCG on 24-h EE and fat oxidation in normal weight and overweight men. Subjects received for 3 d each either water, water + 270 mg caffeine, half-strength oolong tea (122 mg EGCG/135 mg caffeine) or full-strength oolong tea (244 mg EGCG/270 mg caffeine). Relative to water alone, EE was significantly increased by 2.9% (+281 kJ/d) and 3.4% (+331 kJ/d) for the full-strength tea and caffeine-treated water treatments, respectively. Fat oxidation was significantly higher (12%) when subjects consumed the full-strength tea compared to water. Rumpler et al. [16] and Bérubé-Parent et al. [18] both reported an increase in EE with consumption of green tea extract or oolong tea. No increase in EE was observed with EGCG alone in our pilot study.

The potential of EGCG to increase fat oxidation without significantly affecting total energy expenditure has recently been reported in mice by Klaus et al. [20], who suggested the changes in EE might result from the caffeine present in tea beverages and extracts. There seems to be an optimal dose of EGCG plus caffeine that has the potential to increase fat oxidation. Bérubé-Parent et al. [18] also investigated the effects of a constant intake level of caffeine (600 mg) combined with varying amounts of a green tea extract; 270, 600, 900, or 1200 mg EGCG, respectively. The administration of 600 mg caffeine plus 270 mg EGCG increased 24-h EE by ≈750 kJ, although fat oxidation was not altered. Increasing amounts of green tea extract did not exert additional effects. It is possible that tachyphylaxia develops when high doses of pure EGCG or EGCG combined with caffeine are administered. Alternatively, the dose of caffeine may reach a level at which the effects of EGCG are masked [21].

EGCG and caffeine modulate EE and substrate oxidation rates via different targets (Fig 3). EGCG can inhibit catechol O-methyltransferase (COMT), an enzyme involved in the degradation of norepinephrine [22]. As a consequence, once released, norepinephrine remains in the synaptic cleft longer and provides a prolonged stimulation of adrenergic receptors. Caffeine also inhibits the phosphodiesterase-induced degradation of intracellular cyclic AMP (cAMP) [23]. Both a prolonged stimulation of adrenergic receptors, specifically β-adrenergic receptors, and an increased intracellular cAMP concentration result in an increase in EE and fat oxidation.

A number of studies have reported an increase in EE after the ingestion of caffeine [24–29]. Interestingly, Dulloo et al. [15] found no significant increase in EE when 150 mg of caffeine was administered in capsule form. In contrast, Bérubé-Parent et al. [18] showed that capsule preparations containing 600 mg caffeine and varying amounts of EGCG did significantly increase EE. In addition, Rumpler et al. [16] found a significant increase in EE when 270 mg caffeine was administered in 300 mL water. Water may have an impact on the magnitude of the thermogenic effect of caffeine. It appears that water may potentiate the pressor effect of ephedra alkaloids [30]. Recently, we reported a thermogenic effect of water [31]. After drinking 500 mL tap water (22°C), there was a 30% increase in fasting metabolic rate within 60 min. A total thermogenic response of ≈100 kJ was achieved for both men and women. The thermogenic response was attenuated under β-adreno-receptor blockade or when 500 mL isotonic saline was provided instead of tap water. Drinking 500 mL tap water also elicited a decrease in blood osmolality by about 5 mosmol/L [32]. It is widely accepted that a number of organs can act as an "osmostat", i.e., a decrease in extracellular osmolality leads to water movement from the extracellular into the intracellular compartment and an increase in extracellular osmolality induces water movement from the intracellular into the extracellular compartment. The former process results in an osmotic swelling, the latter to an osmotic shrinkage of cells in the respective organs [33]. Interestingly, cell volume swelling is associated with an increase in anaerobic metabolic routes such as synthesis of carbohydrates, proteins and lipids, while cell volume shrinkage is associated with an increase in catabolic metabolic routes such as degradation of carbohydrates and proteins.
and acceleration of aging processes. Sufficient water intake should precede the ingestion of a test meal in order to observe an increase in energy expenditure after caffeine administration. However, these considerations are valid for changes in energy expenditure only.

Fat oxidation is mainly under the control of the sympathetic nervous system (SNS). Therefore, there are a number of approaches to increase SNS activity either directly (by β-adrenergic agonists) or indirectly (by norepinephrine releasers and reuptake inhibitors) [34–36]. Interestingly, a number of compounds extracted from plants such as caffeine from coffee and tea, ephedrine from ephedra, and capsacin from pungent spices can modulate catecholamine release and activity [37]. For example, adding capsacin (derived from chili peppers or red peppers) to a fat-rich food has been shown to stimulate fat oxidation and thermogenesis in humans [38,39]. Caffeine, on the other hand, can potentiate thermogenesis induced by physiological (mild or moderate exercise) or pharmacological (ephedrine) sympathetic stimuli [40]. Because of this synergism, losses of body weight and body fat have been found to be greater in obese patients treated for a longer time with a combination of caffeine and ephedrine than in those treated with placebo, caffeine or ephedrine alone [41].

CONCLUSION

There is some evidence that EGCG can increase fat oxidation. The effect of EGCG on fat oxidation is higher under postprandial than fasting conditions. An EGCG dose of 300 mg daily may be able to induce favorable metabolic effects. However, more studies with a larger sample size and a broader range of age and BMI are needed to define the optimum dose. We conclude that EGCG supplementation has the potential to increase fat oxidation in men, an action that may contribute to the apparent anti-obesity effects of green tea.

REFERENCES


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