No Effect of Short-Term Green Tea Extract Supplementation on Metabolism at Rest or During Exercise in the Fed State

Brian J. Martin, Rachel B. Tan, Jenna B. Gillen, Michael E. Percival, and Martin J. Gibala

Supplementation with green tea extract (GTE) in animals has been reported to induce numerous metabolic adaptations including increased fat oxidation during exercise and improved performance. However, data regarding the metabolic and physiological effects of GTE during exercise in humans are limited and equivocal.

**Purpose:** To examine the effects of short-term GTE treatment on resting energy expenditure (REE), whole-body substrate utilization during exercise and time trial performance.

**Methods:** Fifteen active men (24 ± 3 y; VO\(_2\)peak = 48 ± 7 ml·kg\(^{-1}\)·min\(^{-1}\); BMI = 26 ± 3 kg·m\(^{-2}\)) ingested GTE (3x per day = 1,000 mg/d) or placebo (PLA) for 2 day in a double-blind, crossover design (each separated by a 1 week wash-out period). REE was assessed in the fasted state. Subjects then ingested a standardized breakfast (~5.0 kcal·kg\(^{-1}\)) and 90 min later performed a 60 min cycling bout at an intensity corresponding to individual maximal fat oxidation (44 ± 11% VO\(_2\)peak), followed by a 250 kJ TT. **Results:** REE, whole-body oxygen consumption (VO\(_2\)) and substrate oxidation rates during steady-state exercise were not different between treatments. However, mean heart rate (HR) was lower in GTE vs. PLA (115 ± 16 vs. 118 ± 17 beats·min\(^{-1}\); main effect, \(p = .049\)). Mixed venous blood [glycerol] was higher during rest and exercise after GTE vs. PLA (\(p = .006\,\text{main effect for treatment}\)) but glucose, insulin and free-fatty acids were not different. Subsequent time trial performance was not different between treatments (GTE = 25:38 ± 5:32 vs. PLA = 26:08 ± 8:13 min; \(p = .75\)). **Conclusion:** GTE had minimal effects on whole-body substrate metabolism but significantly increased plasma glycerol and lowered heart rate during steady-state exercise, suggesting a potential increase in lipolysis and a cardiovascular effect that warrants further investigation.

**Keywords:** GTE, tea catechins, fat oxidation, substrate utilization

Tea is one of the most common beverages in the world (Cabrera et al., 2006; Kao et al., 2006; Khan & Mukhtar, 2007; Schneider & Segre, 2009). All teas contain polyphenols that are believed to be responsible for its purported effects on physiologic function (Kao et al., 2006). Unlike other teas, the preparation of green tea does not involve a fermentation process, resulting in a higher polyphenol concentration that has made it the preferred choice for medicinal use by Asian cultures for centuries (Cabrera et al., 2006; Khan & Mukhtar, 2007). Four specific compounds known as catechins (CAT) make up the majority of polyphenols in green tea: epigallocatechin (EGC), epicatechin gallate (ECG), epicatechin (EC), and epigallocatechin gallate (EGCG) (Cabrera et al., 2006). Of the four green tea CAT (GTC), EGCG comprises the largest percentage of the four GTC, typically accounting for ~50 to 80% (Khan & Mukhtar, 2007).

GTC administration to mice has consistently been shown to increase running distance and time to exhaustion (Call et al., 2008; Murase et al., 2005; Murase et al., 2006a, 2006b; Shimotoyodome et al., 2005). The improved endurance capacity has been attributed to increased lipid utilization during exercise, as evidenced by reductions in respiratory exchange ratio (RER) and greater muscle glycogen content following exercise (Murase et al., 2005). In contrast, data regarding the metabolic and physiological effects of GTC supplementation during exercise in humans are equivocal.

Venables et al., (2008) provided three doses of ~300mg GTE over 24 hr to healthy men (GTE = 890 mg·d\(^{-1}\) and EGCG = 366 mg·d\(^{-1}\)), and observed reductions in RER during 30 min of cycling at 50% of their maximal power (W\(_{\text{max}}\)). This was associated with a ~17% increase in fat oxidation rates compared with a placebo. The subjects also showed elevated plasma glycerol concentrations and a trend toward elevated free fatty acids (FFA). Richards et al. (2010) also found a beneficial effect of EGCG administration and reported that a dose of 405 mg·d\(^{-1}\) for 2 day to healthy men and women increased maximal oxygen uptake (VO\(_2\)max). In contrast, Eichenberger et al. (2009) observed that a daily dose of 160 mg of GTE for 3 week did not alter metabolic or physiological markers during steady-state exercise or improve subsequent time trial performance in trained subjects. Similarly, Randell
et al. (2013) examined the effects of a 1- and 7-day treatment with GTE and found no change in fat utilization during moderate intensity (~54% VO₂max) steady-state cycling in moderately-trained individuals.

The divergent findings among human studies are likely related in part to differences in experimental design. For example, Eichenberger et al. (2009) employed a relatively low dose of GTE (160 mg), administered once daily for 3 weeks. Similarly, Dean et al. (2009), examined the effects of a daily dose of EGCG (270 mg) with and without caffeine for 6 days. However, neither Eichenberger et al. (2009) nor Dean et al. (2009) observed a metabolic of physiological effect during exercise. In contrast, Venables et al. (2008) and Richards et al. (2010) both employed a larger dose of GTE administered more frequently (three times per day).

Conversely, the study by Randell et al. (2013) employed a very large dose of GTE administered two times per day. Although, considering the GTE treatment used contained caffeine, the authors discussed the potential effect the caffeine might have had on blunting fat oxidation through increased glycolysis.

In the current study, we employed dosing regimens similar to those used by both Venables et al. (2008) and Richards et al. (2010) to investigate the metabolic and physiological effects of GTE at rest, during steady-state exercise, and on time trial performance in adult humans. Previous studies have examined the effects of GTC in the fasted state, considering this we chose to examine the effects of GTE in the fed state, which is more practical to real life and would potentially blunt steady-state fat utilization allowing a difference to be observed during the GTE treatment. We hypothesized that, compared with placebo, 2 days of GTE administration would increase REE and fat oxidation during steady-state exercise, concomitantly preserving carbohydrate and improving subsequent time trial performance. These alterations would be evidenced by reductions in RER and accompanied by altered blood markers of fat catabolism.

### Methods

**Subjects**

Fifteen young healthy men between the ages of 18 and 45 completed the protocol in its entirety, except for one subject who was unable to complete the first time trial due to exhaustion from the previous 60 min of steady-state exercise. Thus, all time trial data are out of 14 subjects. Participants were healthy, nonhabitual GTC users (~two times per week), nontobacco users, and habitually performed aerobic activity on a recreational basis (≥3 times per week). The research protocol and potential risks were explained to each participant before obtaining written, informed consent. Based on differences in fat oxidation rates from a previously published study (Venables et al., 2008), with 80% power and an alpha level of 0.05, we estimated a sample size of 15 subjects per group was required to detect statistical significance.

### Overview of Research Design

A double-blind, placebo-controlled, crossover design was used to assess the effects of GTE ingestion for 2 days on REE and substrate utilization during exercise. The study design incorporated dosing protocols, which appear to be effective at improving either physiologic or metabolic processes in exercising humans (Richards et al., 2010; Venables et al., 2008). Subjects visited the laboratory on four occasions in total for baseline testing, a familiarization session, and two experimental trials. Each session was conducted in the morning at approximately the same time. Subjects ingested a standardized breakfast 90 min before exercise, which consisted of a granola bar, banana and apple juice (approximately 385 kcal which was comprised of 79 g CHO, 6 g fat, and 6 g protein). Although pharmacokinetic evidence (Lee et al., 2002) suggests rapid excretion of CAT (undetectable after 24 hr), a 1-week washout period separated the two experimental trials to eliminate any potential confounding influences or order effect.

### Supplementation

Subjects were provided a total of seven capsules containing either a high quality decaffeinated GTE powder (Sunphenon, 90D, Taiyo International Inc. Minneapolis MN) or placebo (corn flour). Subjects received 1,000 mg of total polyphenols per day, the equivalent of ~2 to 3 cups of green tea (~300 mg) three times per day. There were no adverse effects of supplementation and all subjects reported administering the treatments as prescribed. Table 1 displays the concentrations of CAT in the GTE, determined by the supplier through HPLC analysis. Capsules were opaque and of identical color, shape, and size to assure the contents of the GTE and placebo remained indiscernible to subjects and researchers. Capsules were provided in a standard prescription pill container, subjects were asked to return the empty containers to verify all capsules were consumed. Pharmacokinetic evidence suggests greater bioavailability of CAT ingested in the fasted state (Chow et al., 2005) and a half-life of ~4 hr (Lee et al., 2002). Considering this, subjects supplemented three times per day, 1 hr before meals (breakfast, lunch, and dinner) for 48 hr, ingesting each capsule with 500 ml of water. Participants were reminded via text or e-mail on the morning supplementation was to begin and to confirm they understood the instructions.

### Table 1  GTE Catechin Composition

<table>
<thead>
<tr>
<th>Item</th>
<th>Concentration %</th>
<th>mg/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polyphenols</td>
<td>95 %</td>
<td>1,000 mg</td>
</tr>
<tr>
<td>Catechins</td>
<td>90 %</td>
<td>900 mg</td>
</tr>
<tr>
<td>EGCG</td>
<td>50 %</td>
<td>450 mg</td>
</tr>
<tr>
<td>EGC</td>
<td>20 %</td>
<td>180 mg</td>
</tr>
<tr>
<td>EC</td>
<td>10 %</td>
<td>90 mg</td>
</tr>
<tr>
<td>ECG</td>
<td>7 %</td>
<td>63 mg</td>
</tr>
<tr>
<td>Caffeine</td>
<td>&lt; 1 %</td>
<td>&lt; 9 mg</td>
</tr>
</tbody>
</table>
Experimental Protocol

**Baseline Testing.** During the first visit, height (cm) and body mass (kg) were recorded. The BOD POD (COSMED Inc., Concord, CA) was used to measure body composition via air-displacement plethysmography. Subjects then completed a graded exercise test (GXT) on the cycle ergometer (Lode Excalibur Sport V 2.0, Groningen, The Netherlands) to determine the intensity eliciting maximal fat oxidation rates ($\text{Fat}_{\text{max}}$). Previous work suggests high interindividual variability in $\text{Fat}_{\text{max}}$ (Meyer et al., 2007). However, a previously established time-efficient protocol has been shown to effectively assess $\text{Fat}_{\text{max}}$ (Achten et al., 2002). Therefore, as an alternative to using a set percentage of maximal workload, a modified GXT protocol was used to individually assess the workload eliciting $\text{Fat}_{\text{max}}$. Subjects performed a 5-min warm-up, cycling at 50 W (W), after which intensity increased to 95 W for 3 min, then increased 35 W every 3 min until volitional fatigue. All participants completed the $\text{Fat}_{\text{max}}$ assessment to exhaustion, eliciting steady-state workloads for $\text{Fat}_{\text{max}}$ between 50 and 165 W, which corresponded to 44 ± 11% $\text{VO}_2^{\text{peak}}$.

Continuous heart rate (HR) was recorded throughout the session via a telemetry chest strap and wireless receiver (Polar Electro Oy, Kempele, Finland). Respiratory gases were assessed throughout the test using an on-line gas collection system (Moxus modular oxygen uptake system, AEI Technologies, Pittsburgh, PA). Oxygen consumption ($\text{VO}_2$) and carbon dioxide production ($\text{VCO}_2$) were used in stoichiometric equations to calculate fat oxidation during exercise ($1.695 \times \text{VO}_2$ to $1.701 \times \text{VCO}_2$) as previously described (Frayn, 1983; Jeukendrup & Wallis, 2005).

**Familiarization Session.** This session was conducted as an actual experimental trial (detailed below) to allow subjects to become familiarized with the trials before actual data collection. This session also allowed for verification of the previously determined intensity for eliciting $\text{Fat}_{\text{max}}$, and to determine the appropriate flow rate for the canopy system used for REE (detailed below).

**Experimental Trials.** Subjects arrived for testing, fasted, 1 hr after ingesting their last capsule. After body mass was measured, subjects lied down in a darkened and quiet room for 30 min of respiratory gas collection to assess REE. A canopy system attached to an on-line gas collection unit (Moxus modular oxygen uptake system, AEI Technologies, Pittsburgh, PA) was used for REE data collection. Subjects in a relaxed supine position, the canopy was positioned over the subject’s head and a tight seal was created using a plastic skirt attached to the canopy. The canopy flow was adjusted to the rate (± 100 ml) determined during the familiarization session. The chosen flow rate elicited mixed CO$_2$ concentrations of 0.65 to 0.85% per manufactures instructions. The first 10 min of REE data were discarded to avoid using data influenced by the initial application of the collection equipment. Stoichiometric equations were used to calculate resting EE ($0.550 \times \text{VCO}_2$ to $4.471 \times \text{VO}_2$) and fat oxidation rates ($1.67 \times \text{VO}_2$ to $1.67 \times \text{VCO}_2$) from $\text{VO}_2$ consumption and $\text{VCO}_2$ production (Frayn, 1983; Jeukendrup & Wallis, 2005).

Following REE data collection, subjects received the standardized breakfast and began the exercise portion of the trial 1.5 hr later. A 5 min warm-up (50 W) preceded 1 hr of cycling at the individually determined intensity for eliciting $\text{Fat}_{\text{max}}$. To assure hydration was maintained, water (1.5 ml·kg$^{-1}$) was provided after each blood collection. Respiratory gases were collected for 5 min at 10-min intervals throughout steady-state exercise, with the last 2 min of each collection period used for analysis. Following the 1 hr of cycling and 5 min of rest, subjects performed a 250 kJ time trial. During the time trial, resistance and cadence were self-selected, participants were instructed to give a maximal effort each time and were aware of the quantity of work completed (kJ) during the bout but blinded to other data, i.e., HR and time. Method reproducibility for the time trial (coefficient of variation) was 2.6% in our laboratory when eight active but untrained individuals were tested 1 week apart with no intervening intervention (Burgomaster, Heigenhauser, & Gibala, 2006). Both experimental trials were performed identical for each subject. Figure 1 displays the experimental trial timeline.

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**Figure 1** — Timeline of experimental protocol. GC = gas collection; BLD = blood collection; H$_2$O = water (1.5 ml/kg)
Blood Sampling

Mixed venous blood was collected during the two trials via an intravenous catheter and blood collection port (BD Angiocath 20 G x 1.25 in and Q-SYTE, Becton, Dickinson and Company, Franklin Lakes, NJ) placed in an adequate upper extremity vein. Approximately 10 ml of blood was collected preprandial, preexercise, after 15, 30, and 45 min of exercise, immediately postexercise and immediately after the performance trial. Samples were immediately placed into appropriate collection tubes (BD Vacutainer: SST, EDTA, or Fluoride, Becton, Dickinson and Company, Franklin Lakes, NJ). Catecholamines were analyzed from fasting blood samples only, and in 14 subjects only due to inadequate plasma volume in one sample.

Dietary/Exercise Controls

Subjects were asked to maintain their current activity and dietary routines throughout the study period and ceased consumption of any tea or products containing tea CAT. To maintain consistency, subjects recorded dietary intake for 48 hr before the first baseline testing then and used this to replicate nutritional intake before the familiarization session and each supplementation period. In addition, 72 hr before baseline testing, familiarization session, and experimental trials, subjects refrained from: physical activity, consuming alcohol or substances with stimulatory effects (not including caffeine), and ingesting foods and beverages containing high levels of CAT. Although caffeine might act synergistically with GTC (Dulloo et al., 1999; Dulloo et al., 2000), the withdrawal effects in habituated users suggest potential reductions in lipolysis during exercise (Hetzler et al., 1994). The results from a similar investigation (Van Soeren & Graham, 1998) disagree with the latter, however, considering the possible conflict we did not have subjects refrain from caffeine except during the 12 hr preceding each session. Lastly, subjects completed a general questionnaire to identify nutrition, supplementation, and medication patterns that might influence the effects of the GTE on fat oxidation.

Blood Analysis

Blood collected for serum (insulin and FFA) was allowed to clot for 30 min after collection then separated by centrifuging (10 min at 4000 rpm) and stored at –40°C for later analysis. Plasma obtained from either EDTA (glycerol and catecholamines) or fluoride (glucose and lactate) tubes were immediately centrifuged (10 min at 4000 rpm) and stored at –40°C for later analysis. Plasma glucose was analyzed using a glucose (hexokinase) reagent kit (Pointe Scientific, Canton MI) and serum insulin was measured through an ELISA (ALPCO Diagnostics, Salem, NH). Plasma glycerol (Sigma-Aldrich Co., St. Louis, MO) and serum FFA (WAKO Diagnostics, Richmond, VA) were determined with enzymatic colorimetric assays. Plasma lactate was measured using a lactate reagent set (Pointe Scientific, Canton MI). Lastly, the University Health Network Laboratory (Toronto, ON) analyzed plasma epinephrine (EPI) and norepinephrine (NE) using HPLC methods.

Statistical Analysis

A two-factor repeated-measures ANOVA (Time × Condition) was used to compare blood variables, HR, and fat oxidation during exercise. A student’s paired t test was used to assess differences in resting data, catecholamines, and time-trial data. Significance was accepted with a p < .05 (two-tailed). All data were analyzed using Statistical Package for Social Sciences (version 20.0, SPSS Inc, Chicago, IL).

Results

Resting Measures:

There was no difference between treatments in resting HR (GTE = 61 ± 8 vs. PLA = 60 ± 8 beats·min–1, p = .67), SBP (GTE = 121 ± 12 vs. PLA = 122 ± 11 mm·hg–1, p = .68), or DBP (GTE = 70 ± 10 vs. 71 ± 10 mm·hg–1, p = .57). Similarly, RER (GTE = 1.4 ± 0.20 vs. PLA = 1.5 ± 0.24 kcal·min–1, p = .17), fat oxidation (GTE = 0.12 ± 0.03 vs PLA = 0.12 ± 0.04 g·min–1, p = .71) and RER was not different between treatments (GTE = 0.77 ± 0.05 vs. PLA = 0.77 ± 0.06, p = .90).

Steady-State Exercise:

GTE supplementation did not alter O2 uptake (GTE = 1.66 ± 0.50 vs. PLA = 1.66 ± 0.47 l·min–1, p = .83) or CO2 production (GTE = 1.46 ± 0.47 vs. PLA = 1.46 ± 0.42 l·min–1, p = .86) during steady-state exercise. Fat oxidation rates (0.33 ± 0.05 vs. 0.33 ± 0.08 g·min–1, p = .83), and RER were also similar between GTE vs. PLA (0.88 ± 0.03 vs. 0.88 ± 0.03, p = .67). However, mean exercise heart rate was lower (Figure 2) during GTE vs. PLA (115 ± 16 vs. 118 ± 17 beats·min–1; main effect, p = .049). The within subject coefficient of variation, determined using the method error (Sale, 1991) and based on data collected during the familiarization and placebo trials, was 5% for VO2, 5% for VCO2, 2% for RER and 2.5% for HR.

Hematologic Measures:

Blood [glycerol] was higher in GTE vs. PLA during steady-state exercise (Figure 3A) and following the time trial (0.22 ± 0.17 vs. 0.17 ± 0.10 mmol·L–1, p = .02), but not different at rest (0.08 ± 0.15 vs 0.05 ± 0.05 mmol·L–1, p = .056) GTE did not affect FFA (Figure 3B) glucose (GTE = 5.71 ± 0.52 vs. PLA 6.05 ± 0.66 mmol·L–1, p = .09), insulin (GTE = 4.45 ± 2.17 vs. PLA = 4.64 ± 2.44 μIU·L–1, p = .66) or lactate (GTE = 1.96 ± 0.27 vs. PLA = 1.96 ± 0.23 mmol·L–1, p = .88) during steady-state exercise. Lastly, NE at rest was not different between treatments (GTE = 0.79 ± 0.34 vs. PLA = 1.00 ± 0.49 mmol·L–1, p = .14). EPI at rest did not rise above the reference value of 0.8 nmol·L–1 and reported as no change.
Figure 2 — Effect of GTE on steady-state HR. GTE = open circles, PLA = closed squares. All values are means ± SEM. *denotes significance ($p \leq .05$).

Figure 3 — Effects of GTE vs. PLA on blood components. GTE = open circles, PLA = closed squares. A) Free glycerol (mmol/L) B) FFA (mmol/L). *denotes significance ($p \leq .05$). All values are means ± SEM. –5, Preexercise; 15, 30, 45, 60 min during steady-state exercise.
Green Tea Extract and Exercise Heart Rate

Time Trial Performance:
There was no difference in time required to complete the 250 kJ time trial (GTE = 25:38 ± 5:32 vs. PLA = 26:08 ± 8:13 min; \( p = .75 \)), nor mean HR during this test (GTE = 160 ± 13 vs. PLA 160 ± 14, beats·min⁻¹, \( p = .65 \)).

Discussion
The current study examined the metabolic and physiological effects of short-term GTE supplementation at rest and during exercise in healthy active men. This is the first study to investigate the effects GTE during exercise in the fed state, in addition to employing a \( \text{Fat}_{\text{max}} \) protocol to individually determine steady-state exercise intensity. The main novel finding was that, while GTE did not alter oxygen uptake or substrate utilization, heart rate was slightly but significantly reduced during steady-state exercise. Based on first principles, the reduced HR response despite similar \( \text{VO}_2 \) is suggestive of either a slightly increased stroke volume or enhanced rate of skeletal muscle oxygen extraction during exercise after GTE. While we are the first to report such an effect, Richards et al. (2010) previously reported that a dose of 405 mg·d⁻¹ of EGCG for 2 day increased \( \text{VO}_2\text{max} \) by ~4.4% in healthy adult men and women. These researchers did not detect changes in HR or stroke volume (SV) and thus the potential mechanism to explain the increased \( \text{VO}_2\text{max} \) was unclear. Although the previous study would suggest a potential performance improvement, in the current study we hypothesized that improved time trial performance would occur due to increased availability of carbohydrate following 60 min of steady-state exercise. Since we did not observe improved fat utilization during the steady-state exercise, substrate availability would likely not have been influenced during the time trial, thus explaining the lack of performance improvement we have reported.

The most often-cited mechanism by which GTC exerts metabolic and physiological effects following acute supplementation is through inhibition of catechol-O-methyl transferase (COMT), an enzyme responsible for the degradation of catecholamines, such as NE (Dulloo et al. 1999; Lu et al., 2003). Inhibition of COMT could result in greater plasma concentrations of NE and thus potentially increase sympathetic stimulation through adrenergic receptors. The increased NE would increase adrenergic drive, and potentially the subsequent effects on HR and glycerol observed in the current study. Although our findings are supported by this potential mechanism, Richards et al. (2010) did not observe a concomitant effect on HR or SV with increased \( \text{VO}_2\text{max} \), and suggested the increase might have resulted from enhanced arterial-venous \( O_2 \) difference. Interestingly, GTC supplementation in mice was recently shown to promote increases in muscle capillarity after exercise training (Nogueira et al., 2011). However, these findings were observed after 15 days of supplementation, long enough to allow for potential adaptations, unlike the acute nature of this study and the one by Richards et al. (2010). Indeed there is evidence, which suggests GTC might have the potential to enhance \( O_2 \) delivery to exercising muscles. For example, GTC have been shown to increase activation of endothelia nitric oxide synthase following both acute in-vitro treatment (Ramirez-Sanchez et al., 2010) and long-term administration in mice (Ihm et al., 2012). However, supporting evidence has yet to be examined in exercising humans. Therefore, the mechanisms to explain the findings observed following acute supplementation with GTE are unclear.

Although the increased adrenergic stimulation, which might result from the potential inhibition of COMT following GTC supplementation is commonly cited as a potential mechanism, only a few studies have attempted to measure catecholamine concentrations. Dulloo et al. (1999) observed increased EE with concomitant elevation in 24-hr urinary NE excretion following a 24-hr GTE+caffeine intervention. Conversely, a study by (Berube-Parent, Pelletier, Dore, & Tremblay, 2005) also observed increased EE following administration with EGCG, yet reported no effect on 24-hr urinary NE. Consistent with another recent report (Hodgson et al., 2013), in the current study we did not observe an effect of GTE on resting catecholamine concentrations.

Previous studies suggest a sedentary lifestyle reduces sensitivity of \( \beta \)-adrenergic receptors (Bell et al., 2001; Stob et al., 2007). If GTC potentially improve this sensitivity, it might explain why resting studies, using sedentary participants, observe more consistent improvements in EE then exercise studies, which generally use trained or recreationally active subjects. These differences are summarized in Table 2, which depicts several studies that have examined the effects of various GTC at rest and during exercise in humans. In the current study we found evidence of increased lipolysis, similar to those reported previously (Venables et al. 2008), which was believed to contribute to the increased fat utilization observed during steady-state exercise in healthy men in that study. However, we, and others (Hodgson et al., 2013; Randell et al., 2013; Dean et al. 2009) did not observe any change in substrate use during steady-state exercise in active or trained subjects. It is possible that other disparities, such as exercise intensity, might have contributed to the divergent effects on fat utilization during exercise. For example, the steady-state exercise intensity was much lower in the current study (~45% \( \text{VO}_2\text{peak} \)) compared with the study conducted by Venables et al. (2008) (~60% \( \text{VO}_2\text{max} \)). However, as Randell et al. (2013) and Dean et al. (2009) had their subjects exercise at similar intensities (~54% and 60% \( \text{VO}_2\text{max} \), respectively) this is unlikely. Moreover, considering the current study largely replicated aspects of the design by Venables et al. (2008) we do not believe the conflicting results would be related to the GTE treatment regimen.

There are limitations to the current study that should be addressed. First, as previously noted, the steady-state exercise intensity was lower than used in previous studies. Considering the work by Venables et al. (2008), if fat utilization was improved through an increase in lipolysis and lipolysis is not limited at low exercise intensities,
than an increase in fat oxidation might not have occurred due to the lower exercise intensity used in the current study. In addition, although having exercise performed in the fed state was a novel aspect of the current study, it has been shown that a preexercise meal reduces fat utilization and increases carbohydrate oxidation (Coyle et al., 1985). Thus, the combined effects of feeding and relatively low intensity exercise might have blunted the potential metabolic effects of the GTE, as well the high carbohydrate feeding might have further blunted the potential effects on substrate use during the time trial. Second, since we examined the effects of GTE both at rest and during exercise in the fed state, this limited our ability to assess either condition when CAT have been shown (~2 hr). Thus, the timing from ingestion of the last capsule to collection of data might have influenced our ability to detect potential metabolic changes. Lastly, while we took steps to promote subject compliance with the supplementation protocol, we did not measure plasma catechins, which would have helped to verify the participants indeed ingested the GTE as prescribed.

In summary, the current study found that 2 day of GTE supplementation did not alter REE or substrate utilization during steady-state exercise in healthy active men. However, we did observe a lower HR during steady-state exercise and evidence of increased lipolysis. These findings, in conjunction with previous data showing that short-term EGCG supplementation increased VO_{2}\text{max} in healthy adults (Richards et al. 2010) suggest potential physiological effects of GTC that warrant further investigation.

**Acknowledgments**

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**References**


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**Table 2** GTC in Humans During Sedentary and Exercising Conditions

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<thead>
<tr>
<th>Researchers</th>
<th>Population</th>
<th>GTC (dose)</th>
<th>Frequency</th>
<th>Duration</th>
<th>Result</th>
<th>Change</th>
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</thead>
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<td>Sedentary</td>
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<tr>
<td>Dulloo et al. (1999)</td>
<td>healthy</td>
<td>GTE (125 mg)</td>
<td>3</td>
<td>24 hr</td>
<td>↑EE/↓RER</td>
<td>10%↑/24 hr</td>
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<tr>
<td>Boschmann et al. (2007)</td>
<td>overweight</td>
<td>EGCg (135 mg)</td>
<td>3</td>
<td>48 hr</td>
<td>↓RER</td>
<td>8%</td>
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<tr>
<td>Venables et al. (2008)</td>
<td>healthy</td>
<td>GTE (300 mg)</td>
<td>3</td>
<td>24 hr</td>
<td>↑ISI</td>
<td>13%</td>
</tr>
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<td>EGCg 135 mg</td>
<td>3</td>
<td>48 hr</td>
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<td>no change</td>
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<td>Exercise</td>
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<td>Richards et al. (2010)</td>
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<td>EGCg (135 mg)</td>
<td>3</td>
<td>48 hr</td>
<td>↑VO_{2}\text{max}</td>
<td>4.4%</td>
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<td>GTE (300 mg)</td>
<td>3</td>
<td>24 hr</td>
<td>↑FOX, ↑Glycerol</td>
<td>17%, p &lt; .05</td>
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<td>GTE (160 mg)</td>
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<td>3 weeks</td>
<td>_</td>
<td>no change</td>
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<td>Dean et al. (2009)</td>
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<td>EGCg (270 mg)</td>
<td>1</td>
<td>6 days</td>
<td>--</td>
<td>no change</td>
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<tr>
<td>Hodgson et al. (2013)</td>
<td>active</td>
<td>GTE (600 mg)</td>
<td>2</td>
<td>7 days</td>
<td>↑FOX – rest(^{b})</td>
<td>_</td>
</tr>
<tr>
<td>Randell et al. (2013)</td>
<td>active</td>
<td>GTE (600 mg)</td>
<td>2</td>
<td>7 days</td>
<td>↓FOX - exercise</td>
<td>_</td>
</tr>
</tbody>
</table>

Note. EE = energy expenditure, RER = respiratory exchange ratio, ISI = insulin sensitivity index, TFA = total fat area, FOX = fat oxidation.

\(^{a}\)Frequency is in times per day.

\(^{b}\)Assessed through changes in metabolite profiling.


