The Effects of EGCG on Fat Oxidation and Endurance Performance in Male Cyclists

Sara Dean, Andrea Braakhuis, and Carl Paton

Researchers have long been investigating strategies that can increase athletes’ ability to oxidize fatty acids and spare carbohydrate, thus potentially improving endurance capacity. Green-tea extract (epigallocatechin-3-gallate; EGCG) has been shown to improve endurance capacity in mice. If a green-tea extract can stimulate fat oxidation and as a result spare glycogen stores, then athletes may benefit through improved endurance performance. Eight male cyclists completed a study incorporating a 3-way crossover, randomized, placebo-controlled, double-blinded, diet-controlled research design. All participants received 3 different treatments (placebo 270 mg, EGCG 270 mg, and placebo 270 mg + caffeine 3 mg/kg) over a 6-day period and 1 hr before exercise testing. Each participant completed 3 exercise trials consisting of 60 min of cycling at 60% maximum oxygen uptake (VO$_{2\text{max}}$) immediately followed by a self-paced 40-km cycling time trial. The study found little benefit in consuming green-tea extract on fat oxidation or cycling performance, unlike caffeine, which did benefit cycling performance. The physiological responses observed during submaximal cycling after caffeine ingestion were similar to those reported previously, including an increase in heart rate (EGCG 147 ± 17, caffeine 146 ± 19, and placebo 144 ± 15 beats/min), glucose at the 40-min exercise time point (placebo 5.0 ± 0.8, EGCG 5.4 ± 1.0, and caffeine 5.8 ± 1.0 mmol/L), and resting plasma free fatty acids and no change in the amount of carbohydrate and fat being oxidized. Therefore, it was concluded that green-tea extract offers no additional benefit to cyclists over and above those achieved by using caffeine.

Keywords: epigallocatechin-3-gallate, caffeine, cycling, athletes

In the search for strategies to enhance athletic performance, research has focused on nutritional techniques that promote fat oxidation leading to slower rates of muscle glycogen depletion and thereby improving exercise performance. The purpose of this research was to investigate the effects of epigallocatechin-3-gallate (EGCG) consumption on fat oxidation at rest and during submaximal exercise and on endurance performance in male cyclists.

Optimal performance relies on the supply and utilization of energy before, during, and after exercise, such that the better athletes are able to store and use...
fuel, the better they will perform. This is particularly true for endurance athletes such as cyclists. During aerobic exercise, both free fatty acids and glucose are important energy sources. With increasing exercise intensities, particularly above 70–80% of $\text{VO}_{2\text{max}}$, there is a progressive shift from fat to carbohydrate utilization, indicating limitations in the rate of fatty-acid oxidation (Hawley, Brouns, & Jeukendrup, 1998). Therefore, by enhancing the availability and use of free fatty acids, carbohydrate use is thought to be reduced, which in turn spares glycogen, suppresses lactate production, and results in increased endurance. Based on this theory attempts have been made to improve endurance performance by nutritional regimens that aim to promote fatty-acid oxidation.

The most common supplement used by athletes to enhance fat oxidation and endurance performance is caffeine. In several studies caffeine has been shown to enhance endurance performance and can act as a potent fat metabolizer and central nervous stimulant (Cox et al., 2002; Hunter, St. Clair Gibson, Collins, Lambert, & Noakes, 2002; Jacobson, Febbraio, Arkinson, & Hawley, 2001). Recently, research has shown that supplementing with green tea, in particular the green-tea extract EGCG, can increase energy expenditure and stimulate fat oxidation in animals and humans (Ashida et al., 2004; Bérubé-Parent, Pelletier, Doré, & Tremblay, 2005; Choo, 2003; Dulloo, Seydoux, Girardier, Chantre, & Vandermander, 2000; Kao, Hipakka, & Liao, 2000; Murase, Nagasawa, Suzuki, Hase, & Tokimitsu, 2002; Rumpler et al., 2001; Tokimitsu, 2004; Wolfram et al., 2005; Yang & Landau, 2000). Researchers have shown that consumption of EGCG improves endurance performance and stimulates fat oxidation in mice (Murase, Haramizu, Shimotoyodome, Nagasawa, & Tokimitsu, 2005; Murase, Haramizu, Shimotoyodome, Tokimitsu, & Hase, 2006). However, there is limited research in this area, particularly studies investigating green tea and EGCG, fat oxidation, energy metabolism, and endurance performance, in humans (Mukhtar & Ahmad, 2000). It is unclear whether the improvement in endurance performance seen in mice with green-tea supplementation occurs in human athletes. Most EGCG studies on humans have focused on diet-induced obesity. Currently, there is no published research on the effects of EGCG or green tea in athletes. There is also a lack of research investigating the possible mechanisms responsible for EGCG enhancement of fat oxidation, so it is unclear how EGCG works to increase fat oxidation in humans and improve endurance performance in mice. One theory is that green-tea extract increases fat oxidation via the stimulation of the adipocyte hormone leptin (Shimotoyodome, Haramizu, Inaba, Murase, & Tokimitsu, 2005; Wolfram, Wang, & Thielecke, 2006). However, the impact leptin has on fat oxidation is still unclear.

Therefore, this research was undertaken to compare the effects of EGCG with those of caffeine and placebo on endurance performance in trained male cyclists. In addition, this research aimed to provide insight into the effects EGCG supplementation has on the hormones and biochemical markers involved in energy metabolism and fat oxidation at rest and during submaximal exercise.
Methods

Participants

Ten moderately well-trained male cyclists able to cycle at a B-grade level or better volunteered for this study. To be classified as a B-grade or better cyclist a participant needed to attain a relative VO$_{2\text{max}}$ of 45–60 in the preexperimental testing, be regularly competing in races, and be training 10–12 hr/week. Only 8 participants completed the full trial (age 36.4 ± 6.1 years, mass 81.7 ± 7.7 kg, VO$_{2\text{max}}$ 52.5 ± 6.1 ml · kg$^{-1}$ · min$^{-1}$, maximal power output [W$_{\text{max}}$] 393.8 ± 25.6 W/kg; $M \pm SD$); 2 failed to finish because of illness and personal commitments. All participants were nonsmokers. They were recruited via local cycle clubs and cycle shops in Hamilton, New Zealand. Each participant was fully informed about the nature of the investigation, the experimental procedures, and possible risks before giving informed, written consent. The research proposal and protocol were approved by the Research and Ethics Committee of the Waikato Institute of Technology, Hamilton, New Zealand.

Experimental Procedures and Design

This study was designed as a three-way crossover, randomized, placebo-controlled, double-blinded study with three supplementation periods of 6 days and an exercise trial conducted on Day 6 of each supplementation period. Supplementation periods were separated by a washout period of 6–10 days. Each participant completed three exercise trials. Each trial consisted of 60 min of steady-state cycling at a workload equivalent to 60% VO$_{2\text{max}}$, immediately followed by a maximal-effort 40-km cycling time trial.

Preexperimental Protocol. Each participant’s W$_{\text{max}}$ and VO$_{2\text{max}}$ were determined before the actual exercise trials via an incremental cycle exercise test to volitional exhaustion. Participants also completed a familiarization 40-km cycling time trial before the experimental trials.

Before the preexperimental testing, participants were weighed in their cycle gear using electronic body-mass scales (Wedderburn, Hamilton, New Zealand), and their height was also measured. A Polar heart-rate monitor (Polar Electro, Finland) was worn by the participants to measure heart rate during each test. The VO$_{2\text{max}}$ and W$_{\text{max}}$ tests were conducted on a Kingcycle ergometer (Kingcycle, High-Wycombe, England) using a Cortex Metalyzer 3b gas-analysis system (Cortex Biophysik Metalyzer, Germany). Before each maximal test and all experimental trials, the gas analyzer was calibrated with commercially available gases of known O$_2$ and CO$_2$ content.

Participants began the incremental test at a power output of 100 W. Thereafter power output was increased in accordance with the testing guidelines of BikeNZ by 33 W every minute until participants reached volitional exhaustion. Expired-gas samples were taken and analyzed by the Cortex Biophysik Metalyzer for the duration of the test. VO$_{2\text{max}}$ was determined as the highest 30-s average recorded during the test. Each participant’s W$_{\text{max}}$ was calculated by the Kingcycle software as the highest mean work rate sustained for a period of 60 s. Based on the data obtained from the VO$_{2\text{max}}$ and W$_{\text{max}}$ tests, a calculation was used to determine a
workload (W) equivalent to 60% of each participant’s VO$_{2\text{max}}$, which was later employed in the steady-state exercise trials. The calculation used was 100 W plus 33 times the time in minutes taken for a participant to reach 60% of his VO$_{2\text{max}}$.

Fifteen minutes after the incremental cycle test to exhaustion, participants completed a 40-km familiarization time trial on the Kingcycle to establish their baseline performance. They were instructed to complete the 40-km distance as quickly as possible at a maximal self-selected intensity. This 40-km time trial served as a practice ride, and no measurements were performed on the participants before or during the session. During the time trial, participants were only able to view the distance they had covered in kilometers. No other information was provided to them during the trial.

**Treatments.** The three treatments were administered in a randomized, placebo-controlled, double-blind manner. The treatments were placebo (270 mg glucose powder), caffeine (3 mg/kg body mass), and green-tea extract (270 mg TEAVIGO). TEAVIGO is a highly purified extract from the leaves of green tea (*Camellia sinensis*) containing a minimum of 90% EGCG and a maximum of 0.1% caffeine. The polyphenolic compositions of green tea and TEAVIGO are shown in Table 1. During each 6-day supplementation period participants consumed five capsules over 5 days (one capsule per day) and two capsules on Day 6. During the placebo period this equated to seven capsules of glucose powder, during the caffeine period it equated to five capsules of glucose powder and two caffeine capsules taken on Day 6, and during the green-tea period it was seven capsules of TEAVIGO EGCG. Absorption, distribution, and elimination profiles of green-tea polyphenols reveal that EGCG has a lower plasma half-life and slower clearance rates, indicating that it could stay in the body for a longer period than other green-tea catechins (Chen, Lee, Li, & Yang, 1997). Therefore, using a green-tea extract high in EGCG may allow a shorter supplementation period than may be necessary with actual green tea, which is biologically less available.

**Training and Nutritional Control.** For the duration of the study, participants were asked to refrain from consuming any products containing green tea and limit their consumption of caffeine-containing products to two per day. They were asked to refrain from heavy physical exercise for the 24-hr period before each exercise trial. They were also required to complete a nutritional and exercise information sheet on which they recorded their exercise and food and fluid intake for the 5 days preceding each exercise trial. Participants were asked to maintain a similar diet and training schedule for each of the supplementation weeks. Twenty-four hours before each exercise trial participants’ dietary intake was controlled. Each participant was provided with a standardized diet composed of 9 g carbohy-

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**Table 1 Polyphenolic Composition of Green Tea and TEAVIGO**

<table>
<thead>
<tr>
<th></th>
<th>EGCG</th>
<th>EGC</th>
<th>ECG</th>
<th>EC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Green tea</td>
<td>40.6%</td>
<td>23.1%</td>
<td>12.4%</td>
<td>9.2%</td>
</tr>
<tr>
<td>TEAVIGO</td>
<td>99.9%</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

*Note. EGCG = epigallocatechin-3-gallate; EGC = epigallocatechin; ECG = epicatechin gallate; EC = epicatechin.*
Green-Tea Extract

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drake/kg body mass, 1.2 g protein/kg body mass, and 1 g fat/kg body mass, which was consumed over the 24-hr period before each exercise trial. Such a diet-exercise protocol has previously been shown to result in standardized preexercise muscle glycogen stores (Jacobson et al., 2001). The standardized diet was repeated before each of the three exercise trials. To ensure participant compliance the food was provided to the participants and they were asked to complete a menu sheet and record any deviations from the prescribed diet. Participants were also asked to maintain a similar training schedule and diet throughout the duration of the study.

**Experimental Protocol.** Each participant completed three exercise trials. Each trial consisted of 60 min of cycling at a workload equivalent to 60% VO$_{2\text{max}}$ immediately followed by a self-paced 40-km cycling time trial.

For a given participant, each exercise trial was conducted at the same time of day to control for circadian rhythm. One hour before each exercise trial the participants consumed their treatment capsules. All exercise trials were conducted at the Waikato Institute of Technology, Hamilton, New Zealand, in a temperature-controlled exercise-testing laboratory (20 °C, ~50% relative humidity). Participants were allowed to consume only water ad libitum for the duration of each test.

During each exercise trial participants initially completed 60 min of cycling at a power output (W) eliciting ~60% VO$_{2\text{max}}$ as previously calculated from the preexperimental testing. Immediately before the start of the 60-min cycling phase, blood samples were taken to measure levels of glucose and lactate. To measure lactate, blood was drawn from the fingertip and immediately analyzed using an enzymatic lactate analyzer (YSI 1500 Sport, Yellow Springs International, OH). From the same fingertip another drop of blood was analyzed for glucose using an Advantage Accu-chek glucose monitor 849 glucose strip (Roche, Basel, Switzerland). After these blood samples were taken participants were given a 5-min warm-up before starting the 60-min cycling phase. During the 5-min warm-up period the workload (W) was gradually increased to the workload equivalent to 60% VO$_{2\text{max}}$, at which the participants were required to cycle for 60 min. Expired-breath samples were collected by the Cortex Metalyzer during two 10-min intervals at 25 and 50 min. From these samples, respiratory-exchange ratio (RER) was calculated by the Cortex Metalyzer. Glucose and lactate levels were sampled again at 20, 40, and 60 min after the start of this steady-state cycling phase.

After the 60-min cycling phase, participants were given 5 min rest before starting the 40-km time trial. They were asked to complete the 40-km distance in the fastest time possible. A 2-min warm-up was completed before the start of each 40-km time trial. During this 2-min period participants were required to keep their output below 100 W to prevent a rolling start to the time trial. During each time trial the participants could see on the computer display the distance they had traveled in kilometers, but no other information was provided to them during the trial.

**Collection and Measurement of Carbohydrate and Fat Oxidation.** RER data provided by the Cortex Metalyzer were used to calculate fat- and carbohydrate-oxidation rates. The following formulas were used. Energy expenditure was calculated as VO$_2$ (L/min) × energy equivalent (kcal/L O$_2$) for specified RER. Net
energy expenditure was calculated as Gross energy expenditure – average resting energy expenditure and grams of fat/carbohydrate consumed = g carbohydrate/fat L O\(_2\) consumed × VO\(_2\) (L/min; Zuntz, 1901).

**Blood Sampling and Analysis.** On the morning of each exercise trial, the participants reported for blood testing to the Waikato District Health Board, Health Waikato Division Laboratory, in Hamilton, New Zealand, between the hours of 8.00 and 9.30 a.m. after an 8- to 12-hr overnight fast. Before each blood test, the participants consumed one of their treatment capsules. For the determination of free-fatty-acid concentration, a 5-ml blood sample was drawn by venipuncture into an EDTA tube. The specimens were collected on ice and the plasma separated from the red cells within 30 min of collection and then immediately frozen. Enzymatic determination by acyl-CoA synthetase substrate was used to determine plasma free-fatty-acid concentration. A 2-ml blood sample was drawn by venipuncture for determination of insulin concentration. Serum was separated off from the red blood cells before analysis with an Immulite insulin kit (Euro/DPC, Gwynedd, UK). The human leptin radioimmunoassay kit (Linco Research, St. Charles, MO) was used to measure leptin concentrations. A 100-µl blood sample was taken from each participant and equilibrium-incubated at 4 °C to measure leptin concentration by way of the human leptin radioimmune assay kit. Plasma insulin, leptin, and free fatty acid were determined in duplicate. The uncertainty of measurement for insulin is 12% at 68 pmol/L and 22% at 311 pmol/L. The uncertainty of measurement for free fatty acids is 3.4% at 1.5 milliequivalents (mEq)/L and 3.8% at 20.9 mEq/L. For leptin the uncertainty of measurement at 4.9 is ±6%.

**Statistical Procedures**

Measures of centrality and spread are shown as \(M \pm SD\). Uncertainties in the estimates of effects are expressed as 90% confidence limits and as chances that the true value of the effect is practically beneficial, trivial, or harmful in relation to performance (Batterham & Hopkins, 2005). To calculate these chances, we assumed that the smallest worthwhile change in time-trial performance for a competitive cyclist is 1.0%. Thresholds for assigning qualitative terms to chances were as follows: <1%, almost certainly not; <5%, very unlikely; <25%, unlikely or probably not; <50%, possibly not; >50%, possibly; >75%, likely or probable; >95%, very likely; and >99%, almost certain. In addition, two-way analyses of variance were used to determine significant differences between variables at an alpha level of .05.

**Results**

**Training and Nutritional Control**

**Food Diaries.** Food and exercise diaries completed for 5 days before each test showed that all participants complied with research restrictions regarding pretrial exercise and caffeine and green-tea consumption. No exercise was undertaken during the 24-hr period before each trial, and complete green-tea withdrawal and
limited caffeine intake were reported during each supplementation period. The average intake of protein, fat, and carbohydrate was similar during each of the three supplementation periods. Analysis of the participants’ food diaries is shown in Table 2.

**Standardized Diet.** No significant differences were found in the mean intake of carbohydrate, protein, and fat compared with the planned 24-hr diet between the three treatments. Analyses of the reported intakes of the 24-hr standardized diet are shown in Table 2.

**Submaximal Cycling Phase**

**Carbohydrate and Fat Oxidation.** RER data were collected over two separate 10-min periods of steady-state cycling at 60% VO\(_{2\text{max}}\), except that an equipment malfunction during the steady-state cycling phase prevented the collection of RER data for Participant 8. These RER data were converted into grams of carbohydrate and fat oxidized during each of the 10-min periods for each treatment. The average amounts oxidized and analyses are shown in Tables 3 and 4.

During the first 10-min interval more carbohydrate was being oxidized for energy during the EGCG and placebo treatments than during the caffeine treatment, as shown in Table 3.

During the second 10-min interval the caffeine and placebo treatments resulted in more fat on average being oxidized for energy than carbohydrate, whereas the EGCG treatment had a greater oxidation of carbohydrate than of fat.

**Plasma Metabolites—Glucose and Lactate.** Mean blood glucose and lactate concentrations at rest and during 60 min of steady-state cycling at 60% VO\(_{2\text{max}}\) are presented in Figure 1.

Intake of caffeine in the hour before exercise caused a greater increase in resting mean blood glucose than did placebo or EGCG. During the first 20 min of cycling, blood glucose declined across all three treatments. Mean blood glucose concentrations plateaued for placebo during the 20- to 40-min period and increased for caffeine and EGCG. During the last 20 min of cycling, mean blood glucose decreased for caffeine and increased for placebo and EGCG. Throughout the 60 min of cycling mean blood glucose concentrations remained highest for caffeine and EGCG compared with placebo at all time points except at rest. At the end of

| Table 2 Mean Intake of Protein, Carbohydrate (CHO), and Fat During the Placebo, Green-Tea-Extract (EGCG), and Caffeine Supplementation Periods and During the 24 hr Before Each Trial, \(M \pm SD, N = 8\) |
|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
| Supplementation Period          | 24 hr Before Each Trial         |                                  |                                  |
|                                 | Placebo                         | EGCG                            | Caffeine                        |
| Protein (g)                     | 95 ± 33                         | 94 ± 27                         | 99 ± 28                         |
| CHO (g)                         | 292 ± 83                        | 294 ± 99                        | 283 ± 87                        |
| Fat (g)                         | 101 ± 40                        | 93 ± 21                         | 91 ± 25                         |
|                                 | 94 ± 11                         | 98 ± 9                          | 95 ± 11                         |
|                                 | 673 ± 63                        | 688 ± 38                        | 674 ± 72                        |
|                                 | 36 ± 2                          | 35 ± 4                          | 34 ± 4                          |
### Table 3  Average Grams of Carbohydrate (CHO) Oxidized During the 25- to 35-min and 50- to 60-min Intervals of Steady-State Cycling at 60% VO\textsubscript{2max} After the Ingestion of Placebo, Green-Tea Extract (EGCG), and Caffeine 1 hr Before the Start of Exercise, $N = 7$

<table>
<thead>
<tr>
<th>Cycling interval</th>
<th>Treatment</th>
<th>g CHO/L O\textsubscript{2}, M ± SD</th>
<th>Mean effect vs. placebo (90%CI)</th>
<th>$p$</th>
<th>Chances, %, qualitative</th>
</tr>
</thead>
<tbody>
<tr>
<td>25–35 min</td>
<td>placebo</td>
<td>0.93 ± 0.33</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<tr>
<td></td>
<td>EGCG</td>
<td>0.96 ± 0.27</td>
<td>3.62 (–3.7 to 11.5)</td>
<td>.4</td>
<td>56, possibly not</td>
</tr>
<tr>
<td></td>
<td>caffeine</td>
<td>0.90 ± 0.28</td>
<td>–2.39 (–9.0 to 4.8)</td>
<td>.5</td>
<td>85, likely</td>
</tr>
<tr>
<td>50–60 min</td>
<td>placebo</td>
<td>0.84 ± 0.13</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>EGCG</td>
<td>0.90 ± 0.15</td>
<td>6.90 (–1.6 to 16.2)</td>
<td>.2</td>
<td>88, likely</td>
</tr>
<tr>
<td></td>
<td>caffeine</td>
<td>0.84 ± 0.15</td>
<td>–0.01 (–10.5 to 11.7)</td>
<td>1.0</td>
<td>43, possibly</td>
</tr>
</tbody>
</table>
Table 4  Average Grams of Fat Oxidized During the 25- to 35-min and 50- to 60-min Intervals of Steady-State Cycling at 60% VO_{2\text{max}} After the Ingestion of Placebo, Green-Tea Extract (EGCG), and Caffeine 1 hr Before the Start of Exercise, \( N = 7 \)

<table>
<thead>
<tr>
<th>Cycling interval</th>
<th>Treatment</th>
<th>g fat/L O_{2}, M ± SD</th>
<th>Mean effect vs. placebo (90%CI)</th>
<th>( p )</th>
<th>Chances, %, qualitative</th>
</tr>
</thead>
<tbody>
<tr>
<td>25–35 min</td>
<td>placebo</td>
<td>0.12 ± 0.05</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>EGCG</td>
<td>0.11 ± 0.06</td>
<td>−19.35 (–36.9 to 3.1)</td>
<td>.1</td>
<td>6, unlikely</td>
</tr>
<tr>
<td></td>
<td>caffeine</td>
<td>0.13 ± 0.05</td>
<td>7.16 (–11.8 to 30.2)</td>
<td>.5</td>
<td>71, possibly</td>
</tr>
<tr>
<td>50–60 min</td>
<td>placebo</td>
<td>0.16 ± 0.05</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>EGCG</td>
<td>0.13 ± 0.06</td>
<td>−18.28 (–30.1 to –4.5)</td>
<td>.05</td>
<td>2, very unlikely</td>
</tr>
<tr>
<td></td>
<td>caffeine</td>
<td>0.16 ± 0.06</td>
<td>−2.42 (–21.3 to 21.0)</td>
<td>.8</td>
<td>38, possibly not</td>
</tr>
</tbody>
</table>
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60 min, mean blood glucose was highest for EGCG and lowest for placebo. Mean blood glucose concentrations were significantly higher at the 40-min time point with caffeine than with placebo \( (p = .04) \). No other significant differences were found in mean blood glucose concentrations at rest or the 20-, 40-, and 60-min time points between the three treatments (Figure 1).

At rest, mean blood lactate concentrations were similar across the three treatments. Throughout the exercise period mean blood lactate increased for all three treatments. It reached peak levels after 40 min of exercise before decreasing during the last 20 min of exercise. At the 20-, 40-, and 60-min time points mean blood lactate was lowest for the caffeine trials compared with placebo and EGCG. At the end of 60 min mean blood lactate was highest for placebo. There were no significant differences found in mean blood lactate concentrations at rest or during or at the end of the 60 min of cycling between the three treatments (Figure 2).

**Time Trial**

**Heart Rate.** The mean average heart rates recorded during the 40-km time trial after 60 min of steady-state cycling at 60% \( \text{VO}_2\text{max} \) are shown in Figure 3. Mean heart rate was highest for caffeine, followed by EGCG, then placebo. Mean heart rate was significantly higher with caffeine than with EGCG \( (p = .03) \). There were no significant differences found in mean average heart rate between the other treatments (EGCG vs. placebo, \( p = .73 \), and caffeine vs. placebo, \( p = .09 \)).
Figure 2 — The effect of placebo (PL), EGCG (GT), and caffeine (CA) taken 1 hr before exercise on mean blood lactate (LA) at rest and during and at the end of 60 min of steady-state cycling at $60\% \text{VO}_{2\text{max}}$, $M \pm SD$, $N = 8$ participants.

Figure 3 — Mean average heart rate (HR) recorded during the 40-km time trial after 60 min of steady-state cycling at $60\% \text{VO}_{2\text{max}}$ after placebo, green-tea-extract, and caffeine ingestion 1 hr before the start of exercise, $M \pm SD$, $N = 6$ participants.
Maximum heart rate was highest for caffeine, followed by placebo, then EGCG (caffeine 188 ± 8, placebo 183 ± 7, and EGCG 182 ± 7 beats/min). Maximum heart rate was significantly higher with caffeine than with EGCG \((p = .04)\). There were no significant differences in maximum heart rate between the other treatments (EGCG vs. placebo, \(p = .63\), and caffeine vs. placebo, \(p = .05\)).

**Time.** Table 5 shows the mean time-trial times (s) and related measures for the observed effect of placebo, EGCG, and caffeine on time-trial performance.

The caffeine treatment resulted in the fastest mean time-trial time, followed by placebo, then EGCG (caffeine 3,576 ± 233, placebo 3,627 ± 211, and EGCG 3,652 ± 295 s). The observed effect shows that time-trial performance was improved with caffeine compared with placebo and EGCG. However, time-trial performance with EGCG declined compared with placebo. In terms of magnitude-based inferences, the likelihood that the true practical outcome in 40-km cycling time-trial performance will have the observed effect with the use of EGCG is unclear, possible in terms of caffeine, and also possible with the use of caffeine compared with EGCG. Five participants performed a faster time trial on EGCG than placebo, with the biggest improvement in time of ~5%, while 3 participants had a slower time-trial time on EGCG than placebo, with 1 participant performing ~9% worse on EGCG.

**Average Power.** Figure 4 shows the mean average power (W) and related measures for the observed effect of EGCG and caffeine on time-trial performance.

Mean power output was highest for caffeine, followed by placebo, then EGCG. The observed effect shows that average power output during the time trial was 4.1% greater with caffeine than with placebo \((p = .2)\) and 1.2% greater with placebo than with EGCG \((p = .7)\). In terms of magnitude-based inferences, the likelihood that a true enhancement of average power over the placebo condition during a 40-km time trial will occur with the use of EGCG is unclear, likely probable in terms of caffeine, and also likely probable with the use of caffeine compared with EGCG. No significant differences in average power (W) were found between the three treatments (EGCG vs. placebo, \(p = .72\); caffeine vs. placebo, \(p = .24\); and EGCG vs. caffeine, \(p = .21\)).

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Time-trial time (s)</th>
<th>Mean effect vs. placebo (90%CI)</th>
<th>(p)</th>
<th>Chances, %, qualitative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>3,627 ± 211</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Green-tea extract</td>
<td>3,652 ± 295</td>
<td>0.6 (~2.3 to 3.5)</td>
<td>.7</td>
<td>17, unlikely</td>
</tr>
<tr>
<td>Caffeine</td>
<td>3,576 ± 233</td>
<td>−1.4 (~3.7 to 0.9)</td>
<td>.3</td>
<td>64, possibly</td>
</tr>
</tbody>
</table>
Blood Tests

The effects of placebo, EGCGs, and caffeine on fasted, resting plasma free-fatty-acid concentrations and related measures are presented in Table 6. Resting mean plasma free-fatty-acid concentrations were the highest after caffeine ingestion compared with placebo and EGCG. Resting plasma free-fatty-acid concentrations were significantly higher after caffeine ingestion than after placebo or EGCG. No significant difference in resting plasma free-fatty-acid concentrations was found between EGCG and placebo. In terms of magnitude-based inferences, the likelihood that the true practical effect on resting plasma free-fatty-acid concentrations will be the observed effect after the ingestion of caffeine is very likely compared with the intake of EGCG and placebo.

The effects of placebo, EGCG, and caffeine on fasted, resting plasma insulin concentrations and related measures are presented in Table 7. Resting mean
plasma insulin concentrations were highest after the ingestion of EGCG. Mean resting plasma insulin concentrations were lowest after caffeine ingestion. No significant differences in resting plasma insulin concentrations were found between the three treatments. In terms of magnitude-based inferences, the likelihood that the true practical outcome will be that the intake of EGCG and/or caffeine increases resting plasma insulin concentrations compared with placebo is unclear.

The effects of placebo, EGCG, and caffeine on fasted, resting plasma leptin concentrations and related measures are presented in Table 8. Resting mean plasma leptin concentrations were highest after EGCG ingestion compared with placebo and caffeine. No significant differences in resting plasma leptin concentrations were found between the three treatments. In terms of magnitude-based inferences, the likelihood that the true practical outcome will be that the intake of EGCG and/or caffeine increases resting plasma leptin concentrations compared with placebo is possibly not.

**Table 7** Effect of Placebo, Green-Tea Extract (EGCG), and Caffeine on Fasted, Resting Plasma Insulin Concentrations With Treatment Taken 1 hr Before Blood Test, $N = 8$

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Insulin (mmol/L)</th>
<th>Mean effect vs. placebo (90%CI)</th>
<th>$p$</th>
<th>Chances, %, qualitative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>50 ± 23</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>EGCG</td>
<td>56 ± 36</td>
<td>−1.44 (−23.6 to 27.1)</td>
<td>.9</td>
<td>43, possibly not</td>
</tr>
<tr>
<td>Caffeine</td>
<td>49 ± 20</td>
<td>−1.34 (−21.7 to 24.3)</td>
<td>.9</td>
<td>43, possibly not</td>
</tr>
</tbody>
</table>

**Table 8** Effect of Placebo, Green-Tea Extract (EGCG), and Caffeine on Fasted, Resting Plasma Leptin Concentrations With Treatment Taken 1 hr Before Blood Test, $N = 6$

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Leptin ($\mu$g/L)</th>
<th>Mean effect vs. placebo (90%CI)</th>
<th>$p$</th>
<th>Chances, %, qualitative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>5 ± 3</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>EGCG</td>
<td>6 ± 4</td>
<td>2.35 (−15.3 to 23.6)</td>
<td>.8</td>
<td>55, possibly not</td>
</tr>
<tr>
<td>Caffeine</td>
<td>5 ± 3</td>
<td>−4.00 (−15.8 to 9.5)</td>
<td>.6</td>
<td>24, probably not</td>
</tr>
</tbody>
</table>

**Discussion**

To the best of our knowledge this is the first study of its kind to measure the effects of EGCG intake on fat oxidation at rest, during 60 min of steady-state cycling at 60% $VO_{2\text{max}}$, and during endurance performance during a subsequent 40-km cycling time trial in male cyclists. Intake of 270 mg/day of EGCG for 6 days and 1 hr before exercise did not significantly affect fat oxidation at rest or during steady-state cycling. In addition, this study found that EGCG had little if any effect on endurance performance during a 40-km time trial undertaken at the end
of 60 min of steady-state cycling. In contrast, caffeine intake of 3 mg/kg was beneficial in improving performance in the 40-km time trial. Therefore, these results do not support the hypothesis that ingesting EGCG will result in higher fat-oxidation rates than ingesting the same amount of placebo and 3 mg/kg of caffeine. Furthermore, the current results do not support the hypothesis that EGCG would elicit an ergogenic effect resulting in a faster overall time-trial performance.

A finding of the current study was the beneficial effect of 3 mg/kg of caffeine on improving times and average power output during a 40-km time trial undertaken at the end of 60 min of steady-state cycling at 60% VO$_2$max. The caffeine trials produced the fastest mean time-trial time and mean power output compared with placebo and EGCG. These results are consistent with the findings from previous research reporting enhanced capacity for submaximal exercise and endurance performance with intakes of caffeine in the hour before exercise (Bell & McLellan, 2002, 2003; Conway, Orr, & Stannard, 2003; Cox et al., 2002; Graham, 2001; Greer, Friars, & Graham, 2000). Cox et al. enhanced the performance of a time trial undertaken at the end of a prolonged cycling bout, and Kovacs, Stegen, and Brounds (1998) found that a 1-hr cycling time trial was improved after caffeine consumption (Cox et al.; Kovacs et al., 1998).

In the current study a single component of green tea—the most pharmacologically active component, EGCG—was investigated. The major active component of green tea was used in isolation to determine whether it increased fat oxidation independently of caffeine. Murase et al. (2005) investigated the ability of mice to swim to exhaustion when taking EGCG and found that it enhanced endurance capacity, suggesting that the endurance-improving effects of green tea are mediated, at least in part, by EGCG rather than caffeine. As yet, the clinical efficacy of EGCG has not been confirmed in a human athletic population. However, most other research has used a combination of ingredients including EGCG and caffeine. Sale, Harris, Delves, and Corbett (2006) used a combination of ingredients including caffeine and catechin polyphenols, which may act synergistically and therefore potentially affect metabolism in several different ways. A number of studies reporting positive effects of green tea on thermogenesis and fat oxidation have attributed effects to an interaction between the green tea’s high content of catechin polyphenols and caffeine on sympathetic activity rather than being attributed to the individual catechin components (Belza & Jessen, 2005; Kao, Chang, Lee, & Chen, 2006; Kao et al., 2000; Murase et al., 2002; Murase et al., 2005; Murase et al., 2006; Rumpler et al., 2001; Wolfram et al., 2005; Wolfram et al., 2006; Zheng, Sayama, Okubo, Juneja, & Oguni, 2004). Kao et al. (2006) have suggested that the EGCG-containing green-tea extracts are more potent than caffeine alone at stimulating lipid metabolism in humans. In addition, the combination of caffeine and tea catechins in green tea, including EGCG, induced stronger suppression of body weight and fat accumulation than caffeine alone (Zheng et al.). Because tea catechins are known to inhibit catechol-O-methyltransferase (the enzyme that degrades noradrenaline), and caffeine is known to inhibit phosphodiesterase (an enzyme that degrades intracellular cyclic adenosine monophosphate and antagonizes the negative effect of adenosine on noradrenaline release), it has been proposed that these compounds synergistically prolong and augment the sympathetic stimulation of fat oxidation (Klaus, Pultz, Ghone-Reineke, & Wolfram, 2005; Murase et al., 2002; Zheng et al.). However, these research findings
are conclusions based on mice or inactive human population groups, and it was
our aim to investigate whether EGCG in isolation was equal to or more effective
than caffeine at increasing the lipid metabolism-modulating effects in an athletic
male population. In addition, studies by Kao et al. (2000) found acute weight loss
using EGCG but not other green-tea catechins in as little as 2–7 days. The weight
loss was reversed on removal of EGCG from the diet. It was also found that after
7 days rats adapted to EGCG and higher doses were required. This suggests that
when using EGCG a supplement period as short as 7 days is sufficient, unlike the
complex green-tea products. It may also explain differing results in the
literature.

Insulin influences on lipolysis and resting mean plasma insulin levels were
increased with the intake of EGCG and decreased after caffeine intake. Elevated
insulin levels, as observed after EGCG intake in the current study, may have pre-
vented an increase in the mobilization of lipids during the steady-state cycling and
subsequent time trial, leading to a reduced availability of nonesterified fatty acids
as a fuel source. High insulin and glucose levels prevent an increase in the mobi-
lization of fats during exercise by inhibiting the activity of enzyme hormon-
sensitive triglyceride lipase and thereby reducing lipolysis (Bennard, Imbeault, &
Doucet, 2005; Hawley et al., 1998; McMurray & Hackney, 2005). Anderson and
Polansky (2002) found tea, in particular EGCG, to enhance insulin sensitivity.

Leptin is a hormone that has a direct effect on the peripheral tissues and
stimulates fatty-acid oxidation. Adipose tissue secretes leptin in response to an
abundance of glucose as an energy substrate, so measuring levels of leptin and
glucose and respiratory quotient values may serve to gauge substrate availability
and oxidation (Bouassida et al., 2006). Mice administered a 2% green-tea diet for
16 weeks showed a decrease in serum leptin and lower fat accumulation and body
weight, suggesting suppressed lipid metabolism in mice (Sayama, Lin, Zheng, &
Oguni, 2000). Leptin has a profound effect on fuel selection: It is associated with
a lower respiratory quotient, indicating a reduction in carbohydrate oxidation and
an increase in fat oxidation. These acute effects of leptin on metabolic parameters
are consistent with the selective loss of body fat observed with chronic leptin
treatment and suggest that increased energy utilization plays an important role in
the antiobese actions of leptin (Hwa, Gaivin, Porter, & Perez, 1997). In the current
study, mean resting leptin concentrations were shown to increase across each of
the three treatments as the body weight of the participants increased. Tanizawa et
al. (1997) noted that leptin acts directly on the pancreatic beta cells stimulating
insulin secretion at rest (McMurray & Hackney, 2005). In the current study, rest-
ing mean leptin concentrations were highest after EGCG consumption versus placebo and caffeine, but statistically the results are unclear. In the study by
Pérusse et al. (1997) leptin levels were significantly correlated with fasting insulin
for men. However, although there is increasing evidence that leptin production is
regulated by insulin, probably via changes of adipocyte glucose metabolism, the
effects of leptin on insulin secretion are more controversial because of a number
of conflicting reports in the literature (Ahren & Havel, 1999).

The effect of plasma fatty-acid level on endurance performance is still con-
troversial (Murase et al., 2005). However, an increased supply of fatty acids in
circulation would induce the uptake of fatty acids and thereby stimulate lipid
metabolism in muscle (Murase et al., 2005). Resting mean plasma free-fatty-acid
levels were increased after the intake of caffeine, with lower levels observed after the intake of EGCG and placebo. One of the more consistent research findings on caffeine administration is an increased plasma fatty-acid concentration at rest, and this has been attributed to a stimulation of lipolysis because of caffeine’s directly antagonizing adenosine A1 receptors on adipocytes (Chesley, Howlett, Heigenhauser, Hultman, & Spriet, 1998; Graham, Helge, MacLean, Kiens, & Richter, 2000). Others have reported that intake of caffeine in the hour before exercise resulted in increased plasma free-fatty-acid concentrations and increased fat oxidation from both blood-borne and intramuscular stores in a subsequent exercise task. In the current study the rise in mean plasma free-fatty-acid concentrations at rest after caffeine intake suggests that the caffeine resulted in increased adipose-tissue lipolysis. Fat-oxidation rates were also higher during the steady-state cycling after caffeine intake than with EGCG and placebo, and endurance performance in the time trial was improved with caffeine intake, indicating there may have been some glycogen sparing during the submaximal phase.

Lactate is known to be a strong inhibitor of lipolysis. Throughout the 60 min of steady-state cycling at 60% VO$_{2_{\text{max}}}$, mean blood lactate concentrations were the lowest after caffeine intake at all time points compared with placebo and EGCG. These findings suggest that the intake of caffeine suppressed carbohydrate utilization and that there was decreased glycogen breakdown and glycolytic flux, thus sparing glycogen, possibly resulting in improved endurance performance during the subsequent 40-km time trial. These results suggest that the intake of caffeine enhanced fatty-acid availability, catabolism, and use in muscle, and this was accompanied by a reduction in carbohydrate use, which together may have resulted in the fastest mean time and mean average power during the 40-km time trial.

In addition, higher average and maximum heart rates were observed during the caffeine time trials than with placebo and EGCG. Therefore, in the current study time-trial performance was improved after caffeine intake even though participants’ heart rates were higher than with placebo and EGCG intake, which may have affected their perception of effort. It is possible, then, that the participants’ perception of effort was affected by caffeine’s directly stimulating the central nervous system, but the exact mechanism remains unclear. Some research has implied that caffeine may become increasingly capable of reducing the sensation of effort as more strenuous activity is performed (Magkos & Kavouras, 2004). A 40-km time trial after 60 min of cycling at 60% VO$_{2_{\text{max}}}$ would probably be classified as strenuous activity, but ratings of perceived exertion were not used in the current study, and they may have helped determine whether the performance enhancement observed with caffeine was a central nervous system effect.

The current investigation found an unclear effect of EGCG intake on endurance performance during a 40-km time trial undertaken at the end of 60 min of cycling at 60% VO$_{2_{\text{max}}}$. Kovacs, Lejeune, Nijs, and Westerterp-Plantenga (2004) claimed that green-tea supplementation is only effective when habitual caffeine intake is low, and a much higher dose of green tea is required when habitual caffeine intake is high. Kovacs et al. (2004) found a stronger weight maintenance with green tea in low caffeine consumers than in high caffeine consumers, proposing that the magnitude of habitual caffeine intake may affect the effectiveness of green-tea administration (Kovacs et al., 2004). A mixture of green tea with caffeine was associated with greater weight maintenance in habitually low caffeine
consumers, supported by relatively greater thermogenesis and fat oxidation (Westerterp-Plantenga, Lejeune, & Kovacs, 2005). Although the current study did not look specifically at the effects of EGCG on fat oxidation and endurance performance in caffeine users versus nonusers, the results suggest there may be some individual variability in the response to EGCG in caffeine users. Furthermore, a greater effect on fat oxidation and endurance performance may be observed when EGCG and caffeine are combined. However, uncertainty remains regarding the mechanisms by which EGCG or green tea may or may not exert any influence over the metabolic and/or neural pathways involved in fat oxidation and endurance performance.

Long-term supplementation with EGCG or green tea appears to be associated with enhanced energy expenditure and fat oxidation. The improved endurance capacity that Murase et al. (2005, 2006) found in mice was observed after long-term intake of a green-tea extract, and no marked effects were observed after a single dose, suggesting that some biochemical changes induced by habitual green-tea extract intake, such as up-regulation of muscular beta oxidation, contribute to the improvement in endurance capacity (Murase et al., 2005; Murase et al., 2006). Wolfram et al. (2005, 2006) also observed that extending the supplementation time of green tea to more than 12 weeks may be more beneficial and therefore more likely to produce an effect (Wolfram et al., 2005; Wolfram et al., 2006). In the current study, participants were supplemented with EGCG for 6 days, and there were no significant effects of EGCG on fat-oxidation measures at rest or during steady-state cycling at 60% $V_{O_2}^{\text{max}}$ or on time-trial performance.

There is general agreement that caffeine ingestion increases heart rate during exercise because of its powerful effect on the sympathetic nervous system (Jacobson et al., 2001). In the current study, higher heart rates during the 40-km time trial after caffeine ingestion confirm that the ingested caffeine was physiologically active (Jacobson et al.). A higher mean average heart rate during steady-state cycling with EGCG than with caffeine and placebo also suggests that the intake of EGCG is physiologically active and may exert some effect on the nervous systems similar to caffeine but not to the same extent, which may explain individual performance improvements in the EGCG time trials without change in fat-oxidation measures. Most evidence shows no effect on heart rate during exercise with the isolated use of caffeine, although some have reported an increase in heart rate (Engels, Wirth, Celik, & Dorsey, 1999; Magkos & Kavouras, 2004).

**Conclusion**

In summary, the results of the current investigation show that there were no significant effects of EGCG on fat oxidation during 60 min of steady-state cycling at 60% $V_{O_2}^{\text{max}}$. There was an unclear effect of EGCG intake on endurance performance during the 40-km time trial that followed the steady-state cycling. Some individuals performed better on EGCG than with placebo and caffeine, and the mechanisms are unclear, possibly a synergism between caffeine and EGCG or an individual variability in the response to EGCG. Because this was the first study to our knowledge to investigate the effects of EGCG on fat oxidation during exercise and endurance performance in humans, future research may be warranted to
expand the current knowledge base in the area. The current investigation also found that the intake of 3 mg/kg of caffeine 1 hr before exercise enhanced performance in a 40-km cycling time trial. However, the mechanism of action was unclear and the findings lend support to the argument that caffeine’s purported ergogenicity is likely to have more than one mechanism of action, possibly metabolic and via central nervous system effects (Doherty & Smith, 2005). The cardiorespiratory and metabolic responses seen in the current study during exercise after caffeine intake were similar to those reported previously, generally either no change or a slight increase in heart rate, glucose, and resting plasma free fatty acids or no change in RER (Conway et al., 2003).

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References


