Improved 2000-Meter Rowing Performance in Competitive Oarswomen After Caffeine Ingestion

Megan E. Anderson, Clinton R. Bruce, Steve F. Fraser, Nigel K. Stepto, Rudi Klein, William G. Hopkins, and John A. Hawley

Eight competitive oarswomen (age, 22 ± 3 years; mass, 64.4 ± 3.8 kg) performed three simulated 2,000-m time trials on a rowing ergometer. The trials, which were preceded by a 24-hour dietary and training control and 72 hours of caffeine abstinence, were conducted 1 hour after ingesting caffeine (6 or 9 mg · kg⁻¹ body mass) or placebo. Plasma free fatty acid concentrations before exercise were higher with caffeine than placebo (0.67 ± 0.34 vs. 0.72 ± 0.36 vs. 0.30 ± 0.10 mM for 6 and 9 mg · kg⁻¹ caffeine and placebo, respectively; p < .05). Performance time improved 0.7% (95% confidence interval [CI] 0 to 1.5%) with 6 mg · kg⁻¹ caffeine and 1.3% (95% CI 0.5 to 2.0%) with 9 mg · kg⁻¹ caffeine. The first 500 m of the 2,000 m was faster with the higher caffeine dose compared with placebo or the lower dose (1.53 ± 0.52 vs. 1.55 ± 0.62 and 1.56 ± 0.43 min; p = .02). We concluded that caffeine produces a worthwhile enhancement of performance in a controlled laboratory setting, primarily by improving the first 500 m of a 2,000-m row.

Key Words: ergogenic, ergometer, dose-response

Many well-controlled laboratory studies have demonstrated an ergogenic effect of caffeine on moderate intensity endurance exercise capacity (for review, see 20). For the most part, enhancement of performance after caffeine ingestion has been attributed to alterations in the rate of substrate utilization. the so-called “metabolic theory” (19). However, there is a growing body of evidence to suggest that there are other (non-metabolic) mechanism(s) underlying the caffeine-induced improvements in exercise capacity, particularly in those exercise situations that do not lead to glycogen depletion. For example, Jackman et al. (15) reported that caffeine ingestion increased exercise time to exhaustion, which lead to fatigue in ~5 min, and that this effect was not associated with a reduction in the rate of glycogenolysis. Cole et al. (6) reported increased work output after caffeine ingestion for the same rating of perceived exertion. Taken collectively, these studies suggest that caffeine might be exerting a direct effect on muscle contractility (15) and/or altering neurological

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function (6) independent of changes in substrate metabolism. To date, however, few studies have investigated the effect of caffeine ingestion on short-term (<10 min) endurance exercise. Events of this duration are not likely to be limited by muscle glycogen availability and thus provide a valuable research tool to further explore the actions of caffeine.

There have been many studies that have evaluated the ergogenicity of caffeine on metabolism and performance in trained and untrained male subjects (see 19, 20). While several investigations have included male and female subjects in the same study (7, 15), they have usually failed to report individual data, making it difficult to determine whether there were gender differences in response to caffeine ingestion. Indeed, we were unable to find a single investigation examining the effect of caffeine ingestion on metabolism and short-term endurance performance in competitive female athletes.

Accordingly, the aims of the current investigation were to determine the effects of varying doses of caffeine on substrate metabolism and short-term, high-intensity endurance performance in well-trained female rowers utilizing a highly reliable laboratory test that simulates the demands of competition.

Methods

Subjects and Preliminary Testing

Eight competitive female rowers (age, 22.4 ± 3 years; height, 172 ± 6 cm; mass 64.4 ± 4 kg; peak oxygen uptake [VO\textsubscript{peak}] 3.13 ± 0.14 L · min\textsuperscript{-1}; values are mean ± SD) were recruited to participate in the study, which was approved by the Human Research Ethics Committee of RMIT University. Each subject was fully informed about the nature of the investigation and the possible risks involved prior to giving written consent.

All subjects completed three preliminary familiarization 2,000-m time trials separated by 7 days on a Concept II rowing ergometer (Model b, Morrisville, VT) in order to determine the reliability of each rower’s performance. For these trials, subjects performed a standardized warm-up that consisted of rowing at 60% peak heart rate (HR\textsuperscript{peak}) for 4 min, followed by 1 min of rest, then rowing at a power output which elicited ~80% of HR\textsuperscript{peak} (~74% VO\textsubscript{2peak}) for a further 6 min. This warm-up was chosen after discussion with the rower’s coach. It represented the best compromise between the rower’s normal competition warm-up routine and a protocol that enabled the collection of valid submaximal steady-state metabolic data at an exercise intensity that would not excessively perturb the acid base status of subjects (as reflected by individual post warm-up blood lactate concentration) thereby potentially compromising subsequent time trial performance. Following this warm-up, subjects rested for 3 min before completing a 2,000-m time trial.

Nutritional, Training, and Menstrual Control

One day prior to each experimental trial (described subsequently), the nutritional status of each subject was controlled. Subjects were provided with a standard diet of 50 kcal · kg\textsuperscript{-1} body mass (BM), composed of 63% carbohydrate (8 g · kg\textsuperscript{-1} BM), 20% fat, and 17% protein, which was consumed during the day prior to a trial. Such a diet is likely to result in a positive energy balance in lightweight female rowers (L.M.
Burke, personal communication). In addition, subjects were required to abstain from ingesting caffeine-containing products and alcohol 72 hours prior to each experiment. Upon finishing the study, subjects completed a questionnaire regarding their habitual daily caffeine consumption. For 48 hours prior to the first trial, subjects kept a record of all training and physical activity. They then attempted to replicate this regimen for the subsequent trials. Every effort was made to control for phase of the menstrual cycle at the time of testing. Six subjects were tested during the follicular phase of the cycle; their experimental trials were separated by 3 days. Two subjects were oligomenorrheic, and their experimental trials were conducted 7 days apart due to training commitments. In addition to the timing of the phase of the menstrual cycle, training and competition commitments had to be taken into consideration during the investigation. Such practical issues need to be acknowledged and recognized as possible limitations of studies using competitive (female) athletes.

**Experimental Trials**

Each subject completed three double-blind experimental trials in a random order separated by at least 3 but no more than 7 days. For each subject, all three trials were completed at the same time of day. On the day of an experimental trial, subjects reported to the laboratory after an 8–12-hour fast. Seven of the 8 subjects were overnight fasted and undertook their trials between 0700–0800 hours. One subject performed the experimental trials later (1200 hours) in the day. However, for each subject, experimental trials were conducted after the same fasting period and at the same time of day. On arrival, a resting urine sample was collected. Subjects were then weighed on electronic scales (Wedderburn Tanita BWB-620, Japan) before a 22-gauge Teflon cannula (Terumo, Tokyo, Japan) was inserted into a forearm vein. A 10 ml blood sample was then drawn and the cannula was flushed with 5 ml of 0.9% sterile saline solution to maintain potency, a process that was repeated after all subsequent blood sampling.

Subjects then ingested 3 ml · kg⁻¹ BM of water with a capsule containing either 6 ml · kg⁻¹ BM caffeine, 9 ml · kg⁻¹ BM caffeine, or a placebo containing ~500 mg of glucose. Further blood samples (10 ml) were collected 30 and 45 min after ingestion of the test capsule. Subjects then commenced the standardized warm-up (described previously) on the rowing ergometer. For the final 6 min of the warm-up, subjects breathed through a mouthpiece attached to a Quark b2 metabolic cart (Cosmed, Rome, Italy). Expired air was passed through a flowmeter, an O₂ analyzer, and a CO₂ analyzer. The flowmeter was calibrated before each experiment with a 3-L syringe. The gas analyzers were calibrated with α-rated gas standards of known concentration (16.0% O₂ and 4.0% CO₂). The flowmeter and gas analyzers were connected to an IBM computer, which calculated minute ventilation (V̇E), oxygen consumption (VO₂), carbon dioxide production (VCO₂), and the respiratory exchange ratio (RER) from conventional equations. Immediately after the warm-up, a further blood sample (5 ml) was drawn, and a rating of perceived exertion (RPE) was obtained (3) before subjects rested for 3 min prior to commencing an all-out 2,000-m time trial.

Throughout the 2,000-m time trial, expired air was collected using the same procedure as previously described. HR during all trials was measured every 5 s by telemetry and stored using a Polar Accurex Plus (Polar Electro OY, Kempele, Finland). The time to complete the 2,000-m row was recorded along with each rower’s stroke rate and the time taken to complete each 100 m of the time trial.
Average power output sustained throughout the 2,000-m time trial was obtained upon the completion of each row. RPE was taken immediately after a subject had completed the 2,000 m. Post exercise blood samples (5 ml) were drawn 3, 5, 10, 15, and 30 min after each time trial. A second urine sample was collected after the final blood sample had been drawn.

**Analytical Techniques**

Blood samples were placed on ice after removal of ~5 ml for the immediate analysis of blood glucose and blood lactate concentrations (in triplicate) using an automated analyzer (Yellow Springs Instruments 2300 Stat Plus Glucose and L-Lactate Analyzer, Yellow Springs, OH, USA). All analytical instruments were calibrated before and during the analyses with known standards. Plasma free fatty acid concentration (FFA) was determined by placing whole blood (5 ml) in tubes containing 60 µL of ethylene glycol-bis (β-aminoethyl ether)—N,N,N',N'-tetraacetic acid and reduced glutathione, and spun at 4,000 rev · min⁻¹ for 15 min in a refrigerated centrifuge (J6-MC Beckman Centrifuge, Beckman Instruments) at 4 °C. The plasma was then stored at −80 °C until analyses. FFA concentration was determined by a spectrophotometer using an enzymatic colorimetric method (half-micro test, Boehringer Mannheim, 1383175, GmbH, Germany).

Pre- and post-exercise urine samples were stored at −80 °C, and later analyzed for urinary caffeine concentration using high performance liquid chromatography (HPLC; ICI Instruments, Australia) using a method modified from Aldridge et al. (1). A urine sample (2 ml) was thawed and transferred to a non-treated tube, and the pH was adjusted to > 9.0 by adding NaOH. Dichloromethane was added to the 1-ml urine samples, and this solution was then mixed for 20 min and centrifuged for 10 min at 4,000 rev · min⁻¹ at 4 °C. The organic phase was dried at 45 °C under a constant stream of oxygen-free N₂. The samples were then resuspended in 1 ml of HPLC mobile phase solvent (65% distilled water, 25% methanol, 10% acetonitrile, 1% glacial acetic acid) and injected onto an Alltech column (Platinum C18, 100A, 5 u, 250 × 4.6 mM). Caffeine was measured at a wavelength of 273 nm.

**Statistical Analyses**

Changes in the mean of variables and/or treatments, and measures of within-subject variability were estimated using a mixed modeling procedure (Proc Mixed) in the Statistical Analysis System (SAS Institute, Cary, NC). The same procedure provided 95% confidence limits (the likely range of true value) for all estimates. For all analyses, the identity of subjects was a random effect. For the analysis of reliability of performance time in the three familiarization trials, the identity of the trial (first, second, third) was included as a fixed effect to account for any learning or habituation effects between trials. For the analysis of all outcome variables in the crossover trials, the identity of the caffeine or placebo treatment was a fixed effect; identity of the trial was included in these analyses to account for any learning or habituation effects. An extra random effect was included for the caffeine trials to account for individual differences in the effect of caffeine. We modeled the effect of dose of caffeine as a linear repeated-measures covariate. By declaring the interaction of dose and subject as a random effect, we also obtained an estimate of individual differences in the effect of dose, represented by a between-subject standard deviation
for the estimate of response per unit dose of caffeine. Changes in performance time between the last familiarization trial (baseline) and the three treatment trials were also analyzed by including an extra random effect to account for individual differences in the change in performance between the baseline and treatment trials.

The centrality and spread of values of variables are shown throughout as mean ± standard deviation. Individual differences in treatment or learning effects are shown as standard deviations of the change in the mean. These standard deviations represent typical variation in the effect between subjects, and they are free of random error of measurement. The precision of estimates of outcome statistics are shown as 95% confidence limits (which define the likely range of the true value in the population from which the sample was drawn).

Results

Familiarization Time Trials

The mean performance time for the three 2,000-m familiarization trials was 473 ± 15, 478 ± 16, and 476 ± 15 s. Reproducibility calculated for trials two and three alone was 0.7% (95% confidence interval [CI] = 0.4 to 1.3%), which was somewhat better than that of trials one and two alone (coefficient of variation = 0.9%, 95% CI = 0.6 to 1.8%).

Submaximal Steady State Row

Table 1 summarizes the metabolic data obtained during the last 5 min of the steady-state submaximal row. As intended, the power output (~135 W) sustained by the subjects for the submaximal row was similar between trials. There were no differences between treatments for \( \dot{V}O_2 \), \( \dot{V}CO_2 \), or HR. \( V_{ESPD} \) was significantly elevated with the higher caffeine dose compared with placebo (56.5 ± 5.9 vs. 51.7 ± 6.0

<table>
<thead>
<tr>
<th>Treatment</th>
<th>( \dot{V}O_2 ) (L·min(^{-1}))</th>
<th>( \dot{V}CO_2 ) (L·min(^{-1}))</th>
<th>RER</th>
<th>( V_{ESPD} ) (L·min(^{-1}))</th>
<th>HR (beats·min(^{-1}))</th>
<th>Power output (W)</th>
<th>RPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>2.20 ± 0.94</td>
<td>1.96 ± 0.52</td>
<td>0.89</td>
<td>51.7 ± 6.1</td>
<td>152 ± 6</td>
<td>133 ± 15</td>
<td>10.8</td>
</tr>
<tr>
<td>6 mg · kg(^{-1}) caffeine</td>
<td>2.30 ± 0.41</td>
<td>1.93 ± 0.92</td>
<td>0.84</td>
<td>53.8 ± 6.1</td>
<td>153 ± 9</td>
<td>133 ± 15</td>
<td>11.1</td>
</tr>
<tr>
<td>9 mg · kg(^{-1}) caffeine</td>
<td>2.25 ± 0.64</td>
<td>1.98 ± 0.08</td>
<td>0.88</td>
<td>56.5* ± 5.9</td>
<td>153 ± 8</td>
<td>135 ± 16</td>
<td>11.3</td>
</tr>
</tbody>
</table>

Note. \( \dot{V}O_2 \), oxygen uptake; \( \dot{V}CO_2 \), carbon dioxide production; RER, respiratory exchange ratio; \( V_{ESPD} \), ventilation; HR, heart rate; RPE, rating of perceived exertion. Values are mean ± SD. *Significantly different to placebo (p < .01).
L \cdot \text{min}^{-1}; p < .01). RER values (Table 1) tended to be lower with the lower dose of caffeine (0.84 ± 0.04) but, due to the large inter-individual variability, were not statistically different from either the placebo (0.89 ± 0.06) or the higher caffeine dose (0.88 ± 0.05).

### 2,000-Meter Time Trial

The percentage changes in the time to complete the 2,000-m time trials for the moderate and high caffeine doses compared with the placebo condition are presented in Figure 1. Performance time improved significantly following the ingestion of the high dose of caffeine (1.3%; 95% CI = 0.5 to 2.0%; \( p = .005 \)). Although the lower dose was not statistically significant compared with placebo (\( p = .06 \)), it did result in a 0.7% improvement in time to complete the 2,000-m time trial (95% CI = 0.0 to 1.5%). The higher caffeine dose resulted in a 0.5% faster time to complete the 2,000 m than the lower caffeine dose (95% CI = -0.2 to 1.3%). The average of the two caffeine trials was 1% faster than the placebo trial (95% CI = 0.4 to 1.7%, \( p = .008 \)). The mean power output during the 2,000-m time trial was 2.7% greater following the ingestion of the higher caffeine dose compared with the placebo trial (95% CI = 4.9 to 0.1, \( p = .02 \)). While no significant difference in the mean power output existed between the lower caffeine dose and the placebo, this caffeine dose increased the average power by 1.4% (95% CI = 3.7 to -0.8, \( p = 0.18 \)). The average of the two caffeine trials resulted in a 2.1% greater mean power output (95% CI = 4.0 to 0.1, \( p = .04 \)). The higher caffeine dose resulted in a 1.3% greater power output than lower dose (95% CI = 3.4 to -0.9%, \( p = .21 \)). There was a 0.15% reduction in time per mg \cdot kg^{-1} \text{ of caffeine (likely range, 0.06 to 0.25, p = .01)}. Individual differences in this effect were ±0.04% per mg \cdot kg^{-1} \text{ (likely range, 0.15 to -0.13, p = .85)}. The time to complete each 500-m segment of the 2,000-m time trial for the experimental trials are presented in Figure 2. The ingestion of the high caffeine dose resulted in a significant improvement in the time to complete the first 500 m (113.4 ± 4.3 s) of the 2,000-m time trial compared with both placebo (116.6 ± 4.3 s; \( p < .01 \)) and lower caffeine dose (115.8 ± 5.9 s; \( p < .05 \)). However, there was no significant difference between the treatments for the second 500-m split. The lower dose of

![Figure 1](image_url)

**Figure 1** — The percentage change in 2,000-m time trial performance following caffeine ingestion compared with placebo (zero point). Values are means; bars represent the 95% confidence intervals (CI). ⬠ 6 mg \cdot kg^{-1} \text{ BM caffeine; } \Delta 9 mg \cdot kg^{-1} \text{ BM caffeine.}
caffeine resulted in a significantly faster third 500-m split compared with placebo (p < .05).

Caffeine ingestion had no effect on the mean $\dot{V}O_2$, $\dot{V}CO_2$, or HR measured throughout the 2,000-m time trial, or RPE taken immediately after the experimental trials (Table 2). However, the higher caffeine dose significantly elevated $\dot{V}E_{STPD}$ during the 2,000-m time trial compared to placebo (103 ± 12.5 L/min vs. 98.7 ± 10.9 L/min; p < .05).

#### Table 2 Metabolic Data Determined During or Immediately After the 2,000-Meter Time Trial

<table>
<thead>
<tr>
<th>Treatment</th>
<th>$\dot{V}O_2$ (L/min)</th>
<th>$\dot{V}CO_2$ (L/min)</th>
<th>RER</th>
<th>$\dot{V}E_{STPD}$ (L/min)</th>
<th>HR (beats/min)</th>
<th>RPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>2.94 ± 0.35</td>
<td>3.08 ± 0.17</td>
<td>1.05</td>
<td>98.7 ± 10.9</td>
<td>180 ± 11</td>
<td>18.1</td>
</tr>
<tr>
<td>6 mg · kg⁻¹ caffeine</td>
<td>3.05 ± 0.40</td>
<td>2.98 ± 0.61</td>
<td>0.98</td>
<td>100.0 ± 13.8</td>
<td>181 ± 8</td>
<td>17.9</td>
</tr>
<tr>
<td>9 mg · kg⁻¹ caffeine</td>
<td>2.92 ± 0.61</td>
<td>3.04 ± 1.04</td>
<td>1.04</td>
<td>103.0* ± 12.5</td>
<td>182 ± 7</td>
<td>18.1</td>
</tr>
</tbody>
</table>

*Note.* $\dot{V}O_2$, oxygen uptake; $\dot{V}CO_2$, carbon dioxide production; RER, respiratory exchange ratio; $\dot{V}E_{STPD}$, ventilation; HR, heart rate; RPE, rating of perceived exertion. Values are mean ± SD obtained throughout the 2,000-m time trial. *Significantly different to placebo (p < .05).

![Figure 2](image-url)

Figure 2 — The time to complete each successive 500 m of the 2,000-m time trials. Values are mean ± SD. □ placebo; ■ 6 mg · kg⁻¹ caffeine; □ 9 mg · kg⁻¹ caffeine. *9 mg · kg⁻¹ caffeine significantly different from placebo (p < .01). †9 mg · kg⁻¹ caffeine significantly different from 6 mg · kg⁻¹ caffeine (p < .05). ‡6 mg · kg⁻¹ caffeine significantly different from placebo (p < .05).
**Blood Metabolites**

Blood lactate concentration was similar at rest for all treatments (~1 mM). Following submaximal exercise, lactate concentration rose to ~2 mM, and following the time trial blood lactate concentration peaked at ~11 mM. While there was a trend for higher blood lactate concentrations after caffeine ingestion, there was no significant difference between any of the treatments. Blood glucose concentrations were similar between all experimental trials prior to the administration of a treatment (~4.5 mM) and remained stable during the 45-min time period following ingestion. Although there was a marked hyperglycemia following the 2,000-m time trial for all treatments (~9 mM), there was no difference between treatments during the 30 min of recovery. Resting plasma FFA concentrations were similar for all the experimental trials (Table 3). The ingestion of caffeine resulted in an elevation of FFA concentrations compared with the placebo. However, this increase in FFA concentration was only significant with the higher caffeine dose compared with placebo 45 min after ingestion ($p = .008$). Figure 3 presents the resting FFA concentrations as a percentage of the pre-caffeine ingestion concentration. The percentage change from

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Rest (mM)</th>
<th>30 min (mM)</th>
<th>45 min (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>0.22 ± 0.12</td>
<td>0.24 ± 0.10</td>
<td>0.30 ± 0.10</td>
</tr>
<tr>
<td>6 mg · kg⁻¹ caffeine</td>
<td>0.34 ± 0.16</td>
<td>0.47 ± 0.34</td>
<td>0.67 ± 0.34</td>
</tr>
<tr>
<td>9 mg · kg⁻¹ caffeine</td>
<td>0.23 ± 0.17</td>
<td>0.46 ± 0.41</td>
<td>0.72 ± 0.36*</td>
</tr>
</tbody>
</table>

*Note. Values are mean ± SD. *9 mg · kg⁻¹ caffeine significantly greater than placebo ($p = .008$).

Figure 3 — Plasma free fatty acid (FFA) concentrations as a percentage of the pre-caffeine ingestion concentrations. ◆ placebo; ■ 6 mg · kg⁻¹ BM caffeine; ▲ 9 mg · kg⁻¹ BM caffeine.
pre-caffeine concentration 45 min after ingestion was −200% for the lower caffeine dose and −300% for the higher caffeine dose.

**Urinary Caffeine Concentration**

Due to technical problems, the data for urinary caffeine concentrations are reported for 4 subjects. Resting urinary caffeine concentration was similar before all trials (0.78 ± 0.2, 1.37 ± 1.5, and 1.45 ± 1.81 μg · ml⁻¹ for placebo, 6, and 9 mg · kg⁻¹ caffeine, respectively). Post-exercise urinary caffeine concentration was elevated to 5.43 ± 0.94 and 8.23 ± 1.22 μg · ml⁻¹ after the 6 and 9 mg · kg⁻¹ caffeine doses, while they remained similar after placebo (0.76 ± 0.34 μg · ml⁻¹).

**Discussion**

The first major finding of the present study was that, under well-controlled laboratory conditions, the ingestion of caffeine resulted in a dose-dependent improvement in simulated 2,000-m rowing performance in well-trained female athletes. The high caffeine dose (9 mg · kg⁻¹) increased average power output by 2.7% (95% CI 0.1 to 4.9%) and resulted in a 1.3% (95% CI 0.5 to 2.0%) reduction in time to complete the 2,000-m time trial. The moderate dose of caffeine (6 mg · kg⁻¹) increased average power by 1.4% (95% CI −0.8 to 3.7%) and correspondingly reduced the time to complete the 2,000 m by 0.7% (95% CI 0.0 to 1.5%). The observed individual differences in the effect of dose of caffeine were modest relative to the average effect, but we cannot exclude the possibility that the individual differences were as large as the average effect itself (i.e., typically some subjects would have no response to caffeine while others would have twice the average dose-response) or were nonexistent (i.e., all subjects would have the same dose-response). Adequate precision for estimating the individual differences in the dose-response clearly requires larger samples or even more trials per subject than we currently employed.

The performance test utilized in the current study has previously been reported to be highly reliable for trained male rowers (18) and, although small improvements in reliability occurred during the three familiarization trials in the current study, there was little within-individual variation during the experimental trials. This finding shows that simulated 2,000 m rowing performance is highly reliable in both well-trained male (18) and female rowers, and highlights the need to include several familiarization trials in studies examining nutritional ergogenics to ensure consistent baseline performances and minimize the risk of type II errors. In addition to familiarizing our competitive athletes with the performance test, we also enforced dietary and training control 1–3 days prior to an experiment and restricted caffeine intake for 72 hours. Accordingly, we are confident that the magnitude of improvement produced by both caffeine doses would provide a worthwhile performance enhancement for competitive female rowers (14).

The −4 s and −6 s improvements in 2,000-m rowing time after caffeine ingestion were largely the result of different pacing strategies. During the first half of the 2,000-m row, subjects were −2 and −4 s faster after the low and high caffeine doses compared to placebo. Hagerman (12) has previously reported that the first 500 m of a 2,000-m race is the fastest, the middle two 500 m splits slower than the first by 3–4 s, and the last 500 m the second fastest, but still 1–2 s slower than the first quarter. It might be expected that if a rower went out too fast in the first 500 m of a race, they
would be unable to hold this pace for the entire duration and may actually end up producing a slower overall time than if they had adopted a more conservative pacing strategy. However, in the current study the rowers were able to retain the performance gains made in the first half of the row and produce the fastest overall times after caffeine ingestion.

The only other investigation to examine the effects of caffeine ingestion on short term, high-intensity exercise in females was conducted 25 years ago by Perkins and Williams (17). These workers reported that the ingestion of either 4, 7, or 10 mg·kg\(^{-1}\) caffeine 30 min prior to exercise resulted in no improvement in cycle time to exhaustion lasting ~300 s in their untrained subjects. However, highly trained athletes have genetic endowment, training history, and training programs that differ from the untrained or sub-elite athletes, and a treatment is likely to produce different effects on performance in these groups (14).

It is commonly accepted that the major mechanism by which caffeine improves endurance performance is by the “sparing” of intramuscular glycogen stores, the so called “metabolic theory.” Caffeine ingested before exercise stimulates lipolysis, enhances rates of fat oxidation (as estimated from respiratory gas exchange measurements), and decreases glycogenolysis (for review, see 20). However, performance in events lasting up to 1 hour are unlikely to be limited by muscle glycogen availability (13). In the present study the low dose of caffeine resulted in an elevation in the concentration of plasma FFA (Figure 3) and a substantial reduction in RER during the submaximal steady-state row compared to the placebo (Table 1). However, RER values after the high caffeine dose were similar to those observed with the placebo (Table 1). Such a finding is difficult to explain, particularly as caffeine induced a dose-response effect with regard to the elevation of plasma FFA concentrations (Figure 3). However, increased RER values with caffeine ingestion have been observed previously. Butts and Crowell (5) reported an elevated RER after a 5 mg·kg\(^{-1}\) caffeine dose compared with decaffeinated coffee in 15 “active” females (\(\text{VO}_{2\text{max}}\sim 50 \text{ ml·kg}^{-1}·\text{min}^{-1}\)) during moderate intensity cycle exercise and suggested that caffeine induced an acceleration of muscle glycogenolysis. Of interest in the current study was the observation that even during the maximal 2,000-m time trial, RER values were persistently lower with the 6 mg·kg\(^{-1}\) caffeine dose (0.98) compared to either the higher dose (1.04) or placebo (1.05). This corroborates the pattern of substrate utilization observed during the submaximal work bout (Table 1).

In the present investigation, caffeine ingestion tended to elevate blood glucose and blood lactate concentrations both before and after exercise. Others have also reported this phenomenon (2, 7–9, 15), which has been attributed to a caffeine-induced increase in epinephrine and a resultant increase in oxygen-independent metabolism (7, 8).

Caffeine is known to be a powerful central nervous system (CNS) stimulant (16), affecting medullary respiratory centers (22) and resulting in an increased rates of ventilation (21). In the current investigation, we found elevated rates of ventilation with the higher (9 mg·kg\(^{-1}\)) caffeine dose during both submaximal (Table 1) and maximal (Table 2) rowing. A number of studies have reported a decrease in the perception of effort at the same workload after caffeine compared to placebo ingestion (4, 10) and increases in work output at the same exertional level (6). In the present study, there was no difference in the rower’s RPE after caffeine ingestion during either the submaximal steady-state row or after the all-out 2,000-m time trials. Although this does not preclude an effect of caffeine on neuron excitability or
central neurotransmitter function (23), it does suggest that the mechanism of action of caffeine and the associated performance enhancement was possibly on skeletal muscle itself. However, we are unable to substantiate this hypothesis from the present data. While some of caffeine’s ergogenic effects are most likely manifested through effects on the CNS, it is almost impossible to quantify how much of caffeine’s ability to improve performance is due to “central” or “peripheral” effects (20).

Finally, we observed a dose-response increase in post-exercise urinary caffeine concentration after caffeine ingestion. Unfortunately, due to technical problems associated with the analyses of our samples, we were only able to obtain pre and post values for 4 of our 8 subjects. Nevertheless, there was a 66% increase in urinary caffeine concentration with the high dose compared with the moderate dose of caffeine. Interestingly, neither dose resulted in a post-exercise urinary caffeine concentration above the 12 μg · ml⁻¹ limit permitted by the International Olympic Committee. In a recent review, Graham and McLean (11) have noted that women may be “less responsive” than males to caffeine ingestion but that this does not necessarily mean a lesser ergogenic effect. In the present investigation, a post-study questionnaire revealed that 6 of the 8 subjects regularly consumed caffeine or caffeine containing products (2–3 servings of coffee and/or caffeinated beverages per day). Clearly there is a need for more research involving male and female subjects who are closely matched for level of training, nutritional status, and so on, in order to determine if the ergogenic and pharmokinetic effects of caffeine are different between genders.

In conclusion, this is the first study to investigate the effect of caffeine ingestion on short term endurance performance using competitive female athletes. Our results indicate that caffeine ingestion (6–9 mg · kg⁻¹) produced an enhancement of 2,000-m rowing performance in these subjects under well controlled laboratory conditions. Such a performance gain is likely to be worthwhile to the female competitive athlete. The enhanced performance with caffeine ingestion was largely the result of a different pacing strategy: these competitive rowers were able to retain the significant time advantage accrued early in the row and produce the fastest overall times after caffeine ingestion. The mechanism(s) underlying the performance enhancement with caffeine ingestion was not likely mediated by metabolic factors but is likely to reside in subtle changes in neuromuscular recruitment and/or a direct effect of caffeine on skeletal muscle.

References


