INTRODUCTION

Ergogenic effects of caffeine have been well established in aerobic (26, 28, 30, 31) and anaerobic (4, 14, 20, 34, 36) exercise. A meta-analysis by Doherty and Smith (16) indicated caffeine ingestion prior to exercise improved performance by as much as 12% during endurance, graded, and short-term exercise tests. Although various dosages have been studied, oral consumption of anhydrous caffeine 1 hr prior to exercise has been shown effective at 6 mg kg-1 body mass (6, 13).
Mechanisms by which caffeine may exhibit ergogenic effects continue to be explored. A competitive blockade of adenosine A1 and/or A2 receptors has been proposed (24, 51, 55). Such a blockade in the central nervous system may lead to a decreased perception of pain during exercise (7, 14, 18, 45) resulting in greater effort, improved performance, and altered perceptual responses (RPE). Further, previous research has shown caffeine may alter RPE during exercise. RPE may be lower at a given workload following caffeine ingestion (6), or an effect can be demonstrated when RPE estimations are similar between trials when caffeine (vs placebo) results in completion of significantly more work (11, 37).

Traditionally, RPE may be estimated by a participant during activity reflecting subjective feelings of exertion or as a method for prescription and regulation of intensity (i.e. RPE production) (1). RPE-based prescriptions have been validated for laboratory settings using the estimation-production paradigm (10, 19, 49, 50). With this approach, individuals estimate RPE during an incremental exercise trial and in subsequent training, effort is adjusted to self-regulate intensity producing a target RPE (9, 22, 38). Although inconsistencies exist, RPE generally is effective for prescribing and regulating intensity (23, 32, 33, 42, 52). Using the estimation-production paradigm, measurement of VO2, lactate [La], heart rate (HR) or other objective physiological variables is not essential.

The influence of caffeine on RPE based intensity regulation (i.e. estimation-production paradigm) is not well understood, as effort sense during exercise remains a complex phenomenon (18). Cole et al (11) examined the effects of a 6 mg·kg⁻¹ caffeine dose on the self-regulation of exercise intensity using three progressive RPE prescriptions. Participants performed a continuous 30 min cycling session subdivided into three 10 min bouts at prescribed RPE values (9, 12, and 15). Compared to placebo, caffeine resulted in significantly greater total work for the combined 30 min exercise (277.8 ± 26.1 kJ vs 246.7 ± 21.5 kJ). Interestingly, workloads within levels of RPE prescription were not significantly different following caffeine ingestion. The potential influence of caffeine ingestion on self-selected workloads during RPE production trials is unclear. The current study examined effects of 6 mg·kg⁻¹ caffeine on self-selected workload and associated physiological responses following RPE based intensity prescription.

METHODS

Participants
Male cyclists and runners were recruited (n = 9) as participants. Prior to data collection, participants completed a health screening questionnaire (PAR-Q) and a written, informed consent. Research was conducted in compliance with the guidelines and policies of the local Institutional Review Board for protection of human participants. During the initial lab session, descriptive data including age (yr), height (cm), and mass (kg) were collected using a standard stadiometer (Detecto, Webb City, MO) and a calibrated Tanita EWB-800 scale (Tokyo, Japan). Body fat percentage was estimated via 3-skinfold-site method (47) (chest, abdomen, thigh) using Lange calipers (Cambridge, MA). A daily caffeine exposure questionnaire was administered to assess typical ingestion. Participants reported to the lab for a total of three sessions. Session 1
included a maximal exertion test (VO2Peak) on a cycle ergometer (Monark 828E, Varberg, Sweden) and was separated from Session 2 by ≥ 48 hr. Sessions 2 and 3, caffeine (CAF) and placebo (PLA) sessions, were randomized, double-blind, and counterbalanced to control for ordering and separated by ≥ 24 hr. Time of testing for CAF and PLA was replicated for each trial within participants to limit effect of circadian rhythms on dependent measures. Participants were instructed to abstain from heavy exercise and caffeine use for ≥ 12 hr prior to each testing session and instructed to record and replicate meals for the night before and day of testing for production trials.

Protocol
Prior to all testing, participants were fitted with an appropriately-sized air cushion mask (VacuMed, Ventura, CA) and HR monitor transmitter (T31 Transmitter, Polar Electro, Stamford, Kempele, Finland) before exercise. Metabolic data (VO2, VCO2, RER and VE) were assessed from expired air via an indirect open circuit spirometry system (Vacu-Med, Ventura, CA) equipped with TurboFit software (TurboFit, VacuMed, Ventura, CA). The system was calibrated prior to all metabolic testing using a gas of known concentration (16% O2, 4% CO2). Ventilatory measurement was calibrated using a 3-L syringe (Hans Rudolph, Kansas City, MO). A 1-10 RPE scale (50) was displayed in view of participants at all times.

The Monark cycle ergometer was adjusted to individual preference for seat height and handlebar positioning. Participants were instructed to maintain 70 rev min-1 throughout and began the exercise test at 35 Watts (W) for 3 min as a warm-up. Following the warm-up, resistance increased 70 W every 2 min until volitional exhaustion or failure of participant to maintain pedaling frequency for 10 s when provided verbal encouragement. Overall RPE (RPE-O) was estimated by participants during the last 10 s of each minute. Metabolic data (20 s means) were reported during the last 10 s of each minute.

Caffeine and placebo (maltodextrin) capsules matched for appearance were administered in sealed containers prior to each trial. Ingestion of capsules at the appropriate time (1 hr prior to testing) was verified verbally upon arrival. During exercise trials, two capillary blood samples were taken from the fingertip and immediately analyzed for serum lactate concentrations [La] (1500 Sport Lactate Analyzer, Yellow Springs Instruments, Yellow Springs, OH). The mean of the two samples recorded at each time point was used for analysis. Prior to each trial, the analyzer was calibrated using 5 mmol L-1 standard and checked for linearity with 15 mmol L-1 standard as per manufacturer instructions.

During production trials, participants were instructed to maintain a pedaling frequency of 70 rev min-1 with cadence displayed digitally on the ergometer. Throughout exercise, participants were allowed to adjust the resistance on the ergometer; however, the readout display was concealed so that participants had no knowledge of the selected workload or PO. Participants began with a 3 min warm-up at 35 W. Immediately following the warm-up, participants were allotted 3 min to adjust the resistance as needed to produce an RPE of 4 (RPE4) based on overall feelings of exertion. Following the 3 min production period, PO was recorded (time: 0 min) and participants completed 20 min of cycling. Participants were only allowed to change the
resistance every 5 min, but were instructed to maintain the prescribed RPE. After cycling 20 min, participants dismounted the ergometer and recovered passively for 10 min. Following the 10 min recovery, participants estimated session RPE (S-RPE) and repeated the above protocol at a prescribed RPE of 7 (RPE7) based on overall feelings of exertion. At the completion of the RPE7 production trial, participants again recovered passively for 10 min following exercise termination and provided a S-RPE. Blood samples were collected and analyzed for [La] as described above at 5, 10, 15, and 20 min for RPE4 and RPE7. Corresponding metabolic data (60 s means) were also recorded at 5, 10, 15, and 20 min. HR was recorded continuously throughout each 20 min cycling trial (POLAR Team2 Pro, Polar Electro Oy, Kempele, Finland) and reported as 5 min means corresponding to [La] and metabolic data at 5, 10, 15 and 20 min. PO was recorded at 0, 5, 10, and 15 min with recording of physiological variables 5 min later per time point. This permitted a physiological steady state to be achieved which corresponded with the acute PO selection.

Statistical Analysis
All data were analyzed via SPSS software (Version 20). A series of repeated measures ANOVAs were used to assess differences with a principle focus on differences between treatments (CAF vs PLA). A 2 (trial) x 4 (time pt) repeated measures ANOVA was used for PO, HR, [La], VO2, RER, and VE with a principle focus on between trial differences at each time point. Tukey’s LSD (1 tailed p-values) was used as a follow-up test between trials. Paired sample t-tests were used to analyze differences in S-RPE for RPE4 and RPE7. Results were considered significant at p ≤ 0.05. Based on the results of a similar study by Cole et al (11), a power analysis indicated that a sample size of 13 participants would be sufficient.

RESULTS
Mean and standard deviation for VO2, HR, [La], RER, and VE between CAF and PLA are displayed in Table 2 (RPE4) and Table 3 (RPE7). A significant main effect for trial for VO2 was found for RPE7 (p = 0.01). Follow-up tests showed significantly greater values for CAF at 5, 10, 15 and 20 min. The main effect for VO2 for RPE4 approached significance (p = 0.08). Follow-up tests showed significant differences at 5, 15 and 20 min. Main effect for trial for HR was significant for RPE7 (p = 0.03), but HR during RPE4 did not reach significance (p = 0.18). Follow-up tests showed HR during RPE7 was significantly greater for CAF at 10, 15 and 20 min, while during RPE4 significant differences were found at 15 and 20 min. HR data for one participant during RPE4 trials were excluded due to equipment failure (n = 8). [La] was significantly greater for CAF vs PLA during both RPE4 (trial main effect, p = 0.03) and RPE7 (trial main effect, p = 0.03). [La] for CAF was significantly greater for RPE4 at 5 and 20 min, while values for RPE7 were significant at 10, 15 and 20 min and approached significance at 5 min (p = 0.057). For RPE4, VE approached significance (trial main effect, p = 0.07) with significance at 15 and 20 min. Within RPE7 trials, VE was significantly greater for CAF vs PLA (trial main effect, p = 0.03) with follow-up tests showing significance at 5, 10, 15, and 20 min. RER was not significantly different between treatments for either RPE4 (p = 0.68) or RPE7 (p = 0.64). There were no significant differences found for S-RPE for RPE4 (CAF = 4.0 ± 0.5, PLA = 3.7 ± 0.5, p = 0.20) or RPE7 (CAF = 7.1 ± 0.3, PLA = 6.9 ± 0.6, p = 0.35).
Table 1. Descriptive characteristics for participants.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>28.2</td>
<td>6.5</td>
</tr>
<tr>
<td>Ht (cm)</td>
<td>176.6</td>
<td>5.6</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>74.3</td>
<td>7.2</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>9.5</td>
<td>3.2</td>
</tr>
<tr>
<td>VO₂ peak (mL kg⁻¹ min⁻¹)</td>
<td>55.4</td>
<td>6.3</td>
</tr>
<tr>
<td>Mean daily caffeine consumption (mg)</td>
<td>257</td>
<td>174</td>
</tr>
</tbody>
</table>

Follow-up tests showed significantly greater power selection at each time point for CAF for RPE4 (Figure 1) and RPE7 (Figure 2).

Table 2. Physiological responses for CAF vs. PLA for RPE4 over time (Mean ±SD).

<table>
<thead>
<tr>
<th>Trial</th>
<th>5 min</th>
<th>10 min</th>
<th>15 min</th>
<th>20 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂ (mL kg⁻¹ min⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAF</td>
<td>30.0 ± 5.0*</td>
<td>31.0 ± 4.7†</td>
<td>31.6 ± 4.5*</td>
<td>32.4 ± 5.2*</td>
</tr>
<tr>
<td>PLA</td>
<td>25.1 ± 3.7</td>
<td>26.7 ± 5.8</td>
<td>27.6 ± 5.5</td>
<td>27.8 ± 6.3</td>
</tr>
<tr>
<td>HR (b min⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAF</td>
<td>132 ± 16</td>
<td>137 ± 18</td>
<td>141 ± 18†</td>
<td>144 ± 20*</td>
</tr>
<tr>
<td>PLA</td>
<td>127 ± 13</td>
<td>128 ± 14</td>
<td>131 ± 14</td>
<td>132 ± 15</td>
</tr>
<tr>
<td>[La] (mmol L⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAF</td>
<td>2.44 ± 0.54*</td>
<td>2.16 ± 0.97</td>
<td>2.30 ± 1.23†</td>
<td>2.39 ± 1.31*</td>
</tr>
<tr>
<td>PLA</td>
<td>1.78 ± 0.70</td>
<td>1.70 ± 1.24</td>
<td>1.67 ± 1.25</td>
<td>1.75 ± 1.30</td>
</tr>
<tr>
<td>Vₑ (L min⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAF</td>
<td>43.8 ± 6.0</td>
<td>45.9 ± 6.8†</td>
<td>47.36 ± 5.7*</td>
<td>48.78 ± 7.8*</td>
</tr>
<tr>
<td>PLA</td>
<td>39.2 ± 7.4</td>
<td>39.7 ± 7.5</td>
<td>41.03 ± 7.9</td>
<td>42.61 ± 8.8</td>
</tr>
<tr>
<td>RER</td>
<td>0.88 ± 0.06</td>
<td>0.87 ± 0.05</td>
<td>0.89 ± 0.05</td>
<td>0.88 ± 0.05</td>
</tr>
</tbody>
</table>

* p ≤ 0.05 CAF vs PLA. † p = 0.09 CAF vs PLA.
Table 3. Physiological responses for CAF vs. PLA for RPE7 over time (Mean ± SD).

<table>
<thead>
<tr>
<th>Trial</th>
<th>5 min</th>
<th>10 min</th>
<th>15 min</th>
<th>20 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂ (mL·kg⁻¹·min⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAF</td>
<td>35.3 ± 5.2*</td>
<td>37.5 ± 5.3*</td>
<td>39.1 ± 5.6*</td>
<td>39.6 ± 4.6*</td>
</tr>
<tr>
<td>PLA</td>
<td>29.8 ± 5.5</td>
<td>32.0 ± 4.9</td>
<td>33.9 ± 6.6</td>
<td>33.4 ± 7.7</td>
</tr>
<tr>
<td>HR (b·min⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAF</td>
<td>145 ± 18†</td>
<td>154 ± 17*</td>
<td>160 ± 17*</td>
<td>164 ± 17*</td>
</tr>
<tr>
<td>PLA</td>
<td>136 ± 19</td>
<td>140 ± 20</td>
<td>146 ± 20</td>
<td>150 ± 21</td>
</tr>
<tr>
<td>[La] (mmol·L⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAF</td>
<td>2.55 ± 1.3**</td>
<td>2.90 ± 1.50*</td>
<td>3.46 ± 1.64*</td>
<td>3.96 ± 1.66*</td>
</tr>
<tr>
<td>PLA</td>
<td>1.99 ± 1.4</td>
<td>2.02 ± 1.60</td>
<td>2.41 ± 1.71</td>
<td>2.45 ± 1.44</td>
</tr>
<tr>
<td>Vₑ (L·min⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAF</td>
<td>56.5 ± 12.3*</td>
<td>59.7 ± 9.4*</td>
<td>65.4 ± 9.7*</td>
<td>68.6 ± 10.4*</td>
</tr>
<tr>
<td>PLA</td>
<td>45.1 ± 8.1</td>
<td>48.8 ± 8.6</td>
<td>54.0 ± 12.0</td>
<td>54.6 ± 12.6</td>
</tr>
<tr>
<td>RER</td>
<td>0.91 ± 0.07</td>
<td>0.93 ± 0.04</td>
<td>0.95 ± 0.05</td>
<td>0.95 ± 0.05</td>
</tr>
</tbody>
</table>

* p ≤ 0.05 CAF vs PLA. ** p = 0.57 CAF vs PLA. † p = 0.09 CAF vs PLA.

DISCUSSION

Previous research indicates that caffeine attenuates estimated RPE, potentially associated with improved exercise performance (2, 16, 46). RPE may be mitigated when exercising at a given workload compared to placebo (7, 17, 41) or RPE may be similar (vs placebo) when greater amounts of work are performed (15, 37, 43). Both paradigms reflect an effect of caffeine on RPE. In regards to training, prescribing exercise utilizing RPE (RPE production) is well accepted (1, 39). Advantages include convenience due to a decreased need for assessing objective measures such as HR, [La] or VO₂. It is unclear if the mitigating effects of a moderate dose of caffeine persist when exercise intensity is prescribed and regulated using RPE. Therefore, this study examined effects of 6 mg·kg⁻¹ caffeine (vs placebo) on workload selection and associated physiological responses following exercise intensity prescriptions of RPE 4 and 7.

The main finding of the current study was that the ingestion of caffeine resulted in the self-selection of significantly higher workloads across time during lower (RPE4) (Figure 1) and higher (RPE7) (Figure 2) intensity production trials. A similar study by Cole et al (11) reported caffeine did not result in a significant increase in mean work output during RPE production (RPE: 9, 12, and 15), but resulted in significantly higher accumulated work. It was suggested that the increase in work output following caffeine ingestion was not dependent upon the RPE level. In contrast, current results suggest caffeine significantly increased self-selected PO during both RPE4 (CAF: 130.0 ± 22.6 W, PLA: 111.6 ± 25.5 W) and RPE7 (CAF: 164.8 ± 37.0 W, PLA: 142.7 ± 40.9 W). Although both studies showed a significant increase in total work, discrepancy between studies may be attributable to the protocol used for RPE prescription. Cole et al. (11) utilized a 30 min cycling exercise protocol consisting of three progressive levels of RPE (10 min each). The current study used an introductory 3 min production period, during this period participants were able to closely ensure their individual workload pertaining to each RPE level (4 and 7) prior to a 20 min cycling trial. This may be a source of the discrepancy since individual PO was selected to the target RPE before data collection commenced. Therefore, for RPE production during cycling exercise preceded by caffeine ingestion, the production period duration and protocol may impact acute outcomes.
Caffeine is known to limit the binding of endogenous adenosine to adenosine receptors reducing adenosine’s natural suppression of arousal and inhibitory modulation of neuronal excitability (24, 51). Therefore, it is plausible that caffeine acted as an analgesic reducing afferent feedback of exercise-associated pain and discomfort during production trials in the current study, resulting in selection of greater workloads. That is, responses observed for PO appear to have been due to the requirement of a greater stimulus (i.e. higher workload) to yield the prescribed RPE.

In the current study, participants were blinded to acute PO during production trials and intensity was regulated by gauging overall subjective feelings throughout twenty minute cycling bouts. During performance based paradigms (i.e. time-trials), caffeine has increased self-selected intensities and improved exercise performance as participants were aware of the performance based nature of the test (8, 12, 15, 40). Contrary to the nature of those studies, the current paradigm did not guide participants with any performance-based incentives; instead participants were instructed to maintain the prescribed intensity (RPE 4 and 7) as in a daily bout of exercise training. Consistent selection of higher workloads may increase total workload performed during daily exercise, which could result in greater total caloric expenditure during exercise sessions and enhanced intensity-dependent training adaptations.

Duncan et al. (21) reported that caffeine ingestion elevated subjective measures of participant readiness to invest both mental and physical effort after performing fatiguing tests and prior to exercise performance tasks. If this occurred in the current study, a caffeine-induced elevation in the motivational state of participants may have altered subjective feelings of intensity (18). In fact, 6 out of 9 participants selected a higher initial PO following caffeine compared to placebo ingestion. This may be an important consideration when caffeine consumption precedes RPE-regulated exercise training. However, current participants were not asked to identify CAF and PLA treatments and no attempt was made to directly assess motivational status. Regardless, ingestion of caffeine resulted in ergogenic benefits plausibly attributable to an alteration in RPE-workload congruence. Future research should more directly examine potential effects of caffeine on motivational status and performance during perceptually-regulated training.

Table 2 shows consistently higher VO2, HR, and VE, values for CAF compared to PLA during RPE4. Failure to achieve overall statistical significance for these variables may have been due to the relatively low intensity associated with RPE4. At a lower intensity, there may be a relatively low volume of pain, discomfort and change in other factors believed to mediate RPE. That is, if altered pain is the mechanism of action for caffeine, it is reasonable that low intensity exercise (vs higher intensity) presents a weaker paradigm in which to observe a convincing effect. A liberal approach to conducting follow-up tests was taken in the interest of identifying an effect, if it exists. More specifically, follow-up tests were conducted at specific time points for physiological variables when main effects approached significance because PO values were significant. More research is warranted as follow-up analyses revealed significantly greater VO2 values for CAF compared to PLA at 5, 15 and 20 min (Table 2).
Robertson et al (48) contended that HR may not be an important mediator of exertional perception, while ventilatory drive stands to be a potent and consciously monitored physiological mediator for respiratory-metabolic perceptual signals during dynamic exercise. Exertional perceptions during exercise may also be influenced by peripheral afferent input (48). In contention, Marcora et al (44) expressed that afferent feedback from locomotor and respiratory muscles lacked a significant contribution to RPE. Current results show significantly greater HR and VE (RPE7) for CAF vs PLA, while greater workloads were selected to maintain the prescribed RPE (Table 3). Therefore, HR and VE as physiological mediators of exertional perception may have been suppressed due to the effects of CAF, while peripheral afferent feedback may have been masked as CAF prompted the requirement of greater workloads during exercise to produce the prescribed RPE (Table 3). It is understood that RPE is not mediated by any single physiological or psychological variable and in the current study contributions from independent mediators could not be discerned. However, it would seem that during low intensity exercise following caffeine ingestion, peripheral afferent input from exercising muscles may serve as a more important mediator of RPE due to the lack of response from HR, VE, and VO2. Further, mediators of RPE appear to be less distinguished during a moderate (vs higher) exercise intensity. More work is warranted to extend the knowledge in this regard.

It was hypothesized that any significant increases in self-selected PO would subsequently elicit a response in [La]. Overall [La] during RPE4 was significantly greater for CAF vs PLA (Table 2), which concurs with other studies investigating the ergogenic effects of caffeine (5, 6, 40, 54). However, selection of greater workloads for CAF at RPE4 was only associated with significantly higher [La] for CAF vs PLA at 5 and 20 min (Table 2). [La] measurements for RPE7 (Table 3) were significantly greater for CAF compared to PLA with follow-up analyses showing significance at 10, 15 and 20 min, while approaching significance at 5 min (p = 0.057). In comparison to the lower intensity (RPE4), CAF manifested a greater effect at the higher intensity (RPE7) as [La] remained significantly elevated following the selection of higher power outputs. Future research should attempt to examine whether caffeine acted peripherally or centrally to affect metabolic changes or muscle recruitment.

While caffeine’s influence on free fatty acids (FFA) mobilization have been debated, the observed RER values were not expressive of a turbulent shift to fat utilization during either RPE4 (Table 2) or RPE7 (Table 3) trials. Previous research has shown caffeine to function as an ergogenic aid by extending time to fatigue with benefits attributed to enhanced FFA concentration (3, 37, 53). However, enhanced performance has been observed in absence of increased FFA (27, 29, 35) indicating benefits of caffeine do not solely rest on altered substrate availability. In the current paradigm, RER values were either similar or lower for CAF versus PLA (Tables 2 and 3) even though PO was greater for CAF. This suggests a possible increased reliance on fat utilization, however, direct measures of blood FFA were not assessed.

S-RPE involves subjective estimation of effort pertaining to the global difficulty of an entire exercise session. S-RPE estimation following exercise serves as a model for monitoring training responses and preventing the possible adverse effects of overtraining (25). Caffeine ingestion
has been shown to attenuate S-RPE even when total work volume is equated between caffeine and placebo trials (41). Current results revealed no significant difference for S-RPE (CAF vs PLA) for RPE4 (Table 2) or RPE7 (Table 3). Different from the constant-load exercise protocol with equated total work volume in Killen et al (41), during the current study RPE remained constant between CAF and PL conditions yet selection of PO was greater for CAF (vs PLA). Therefore, the similar S-RPE (CAF vs PLA) observed with concurrent higher PO indicates caffeine also altered S-RPE. A systematic increase would have been expected for S-RPE yet this was not observed. Consequently, current results confirm those of Killen et al. (41) that caffeine alters S-RPE responses.

A practical difference in PO was assumed to be a mean difference ≥ 20 W. Using this criterion, 3 participants were identified as non-responders and 2 as negative responders for RPE4. For RPE7, 3 participants were identified as non-responders. This observation supports the notion that individual responses vary. Following the removal of non-responders, the relative intensity based on %VO2Peak for each individual was calculated. Figure 3 and Figure 4 display calculated data (Mean ± SD) of responders for achieved percent relative VO2Peak (%VO2Peak) compared between CAF and PLA for RPE4 and RPE7, respectively. At RPE4, responders exercised at a mean %VO2Peak at least 15% greater during CAF than PLA. At RPE7, mean %VO2Peak for responders was at least 13% greater (CAF vs PLA). Assessment of individual results is important to ascertain the true impact of caffeine. From a statistical standpoint, those who fail to respond for whatever reason will alter the overall aggregate results and thus attenuate the magnitude of the impact for those who do respond. Further work is needed to determine the precise reasons for variation in responses among individuals.

**Figure. 3** Mean relative % VO2Peak CAF vs PLA over time for RPE4 for responders only (n = 4).

**Figure 4.** Mean relative % VO2Peak CAF vs PLA over time for RPE7 for responders only (n = 6).
RPE production is a validated and useful method for prescribing exercise that does not require monitoring of physiological variables such as HR. The results of this study show that a moderate dose of caffeine ingested 1 hr prior to exercise may alter an individual’s perception of effort sequentially increasing total work during cycling exercise. Based on the current RPE-production paradigm, individuals ingesting caffeine prior to exercise may benefit from increased total work performed without a noticeable increase in perception of effort. Future research should more directly examine potential effects of caffeine on motivational status and performance during perceptually-regulated training. Although individual variation exists, it should be noted that RPE-based exercise prescription may be affected by caffeine consumption.

Current results indicate caffeine ingestion (6 mg kg-1) resulted in an increase in PO selection during RPE-based exercise prescription. Associated physiological variables also changed concomitant with elevated PO. While direct mechanisms associated with the observed differences are not clear, results have implications with regard to RPE-based exercise prescriptions. Although the RPE production paradigm is useful, efficacy following the ingestion of an ergogenic aid such as caffeine is not well understood. Understanding the link between perceptual responses and caffeine is important due to the increasing utilization of caffeine as an ergogenic aid prior to or during exercise. Notable variations in responses among participants highlight the importance of assessing individual responses to specific ergogenic aids.

REFERENCES


