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ORIGINAL ARTICLE

Effects of caffeine on session ratings of perceived exertion

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Abstract This study examined effects of caffeine on session ratings of perceived exertion (RPE) following 30 min constant-load cycling. Individuals ($n = 15$) of varying aerobic fitness completed a $\dot{V}O_2$ max trial and two 30 min cycling bouts (double-blind, counterbalanced) following ingestion of 6 mL/kg of caffeine or matched placebo. RPE overall, legs and breathing were estimated every 5 min and session RPE was estimated 30 min post-exercise using the OMNI pictorial scale. Session RPE for caffeine and placebo trails were compared using paired t test. Between-trial comparisons of HR, RPE overall, RPE legs and RPE breathing were analyzed using an independent 2 (trial) × 6 (time point) repeated measures analysis of variance (ANOVA) for each dependent variable. Caffeine resulted in a significantly lower session RPE ($p < 0.05$) for caffeine (6.1 ± 2.2) versus placebo (6.8 ± 2.1). Acute perceptual responses were significantly lower for caffeine for RPE overall (15, 20, 25, and 30 min), RPE breathing (15, 20, 25, and 30 min) and RPE legs (20 and 30 min). Survey responses post-exercise revealed greater feelings of nervousness, tremors, restlessness and stomach distress following caffeine versus placebo. Blunted acute RPE and survey responses suggest participants responded to caffeine ingestion. Caffeine decreased acute RPE during exercise which could partially account for lower session RPE responses. However, decreased session RPE could also reveal a latent analgesic affect of caffeine extending into recovery. Extending the understanding of session RPE could benefit coaches in avoiding overtraining when adjusting training programs.

Keywords Ergogenic aid · Performance · Perception

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Introduction

Caffeine is commonly used as an ergogenic aid by athletes (Graham 2001). It can be consumed in amounts that show ergogenic effects yet result in blood levels considerably lower than the acceptable limit of the International Olympic Committee (Graham 2001) and therefore continued use by athletes and recreational competitors is likely. Caffeine has been shown to enhance mental (Delbeke and Debackere 1984) and physical performance in a variety of exercise paradigms including aerobic (Jackman et al. 1996; Graham 2001; O'Rourke et al. 2007) and anaerobic performance (Goldstein et al. 2010) and muscular strength (Green et al. 2007b; Hudson et al. 2008; Warren et al. 2010). For in-depth reviews regarding performance effects of caffeine, the reader is referred to Warren et al. (2010), Davis and Green (2009), O'Rourke et al. (2007), and Graham (2001).

Borg (1962) developed the ratings of perceived exertion (RPE) scale (Borg 1962; Borg 1970) which is widely used for acute estimation of effort during exercise (ACSM

2010). Caffeine has been shown to suppress RPE during exercise (Doherty and Smith 2005; Hudson et al. 2008; Warren et al. 2010; Birnbaum and Herbst 2004; Jackman et al. 1996; Green et al. 2007b). Initially the ergogenic effects of caffeine were attributed to enhanced mobilization of free fatty acid and associated glycogen-sparing (McNaughton 1987). Caffeine is also viewed as an adenosine antagonist where it stimulates the central nervous system (McCall et al. 1982). Central nervous system function is altered by caffeine counteracting the repressor effects on arousal (Porkka-Heiskanen 1999) and neurotransmitter release (Okada et al. 1997). However, a potentially more viable explanation for the ergogenic effect of caffeine may be the analgesic effects which plausibly account for blunted RPE (effort) and pain during exercise. In brief, a standard exertional response occurs at a higher workload in individuals under the influence of caffeine (vs. placebo). Marcora (2009) suggests that afferent feedback from locomotor and respiratory muscles does not make a significant contribution to RPE. A blunted RPE reflects enhanced feelings of exertion potentially extending time to fatigue or resulting in self-selection of a higher exercise intensity (Birnbaum and Herbst 2004).

Previous studies regarding the influence of caffeine on perceptual responses have been limited to in-task or acute RPE (i.e. responses during exercise). In addition to other applications of RPE, Kilpatrick et al. (2009) suggested that predicted feelings of effort before exercise or post-exercise ratings may be an effective way to improve physical activity adoption and maintenance. While original applications of RPE were limited to intensity estimations during exercise, session RPE is obtained 20–30 min following termination of an exercise bout and is a subjective estimation of the difficulty of the entire exercise bout. This model was developed by Foster et al. (1995) as an instrument to detect overtraining in athletes. Various mediating factors have been identified with respect to session RPE including a hot (vs. cold) environment and also elevated blood lactate in response to interval (vs. constant-load) exercise (Green et al. 2007a). Session RPE also has been more closely linked to exercise intensity than duration (Green et al. 2009, 2010). While effects of caffeine on acute RPE are well established (Jackman et al. 1996; Birnbaum and Herbst 2004; Doherty and Smith 2005; Green et al. 2007b; Hudson et al. 2008; Warren et al. 2010), no studies to date have examined the potential influence of caffeine on session RPE. Determining the effects of caffeine ingestion on SRPE may allow greater understanding of how session RPE may be altered which could aid coaches using this perceptual model to monitor athletes in an effort to avoid overtraining. The purpose of the current study was to determine the effects of caffeine ingestion (6 mg/kg body weight) on session RPE. Based on previous studies reflecting a blunting of acute RPE, it was hypothesized that session RPE would also be blunted following caffeine ingestion.

Methods

Participants

Fifteen individuals (males: $n = 10$, females: $n = 5$) of varying aerobic fitness volunteered as participants. All procedures were approved by the local review board for the protection of human subjects and each participant signed a written informed consent outlining study requirements before initiation of data collection. Participants reported to the lab with instructions to be well rested ($\geq$24 h with no heavy physical activity) and well hydrated. Each participant received two 473 mL bottles of water: one to be consumed between dinner and bed the night prior to the trial and the other within an hour of reporting to the lab. Water was administered in attempt to ensure participants reported adequately hydrated in both trials. The caffeine and placebo capsules were administered in sealed containers prior to each trial. Placebo capsules contained maltodextrin and were identical in appearance with the caffeine capsules. Additionally, participants consumed the exact number of capsules for each trial. They were also instructed to avoid alcohol and caffeine (excluding treatment) 4 days (Fisher 1986) prior to each testing session. A calibrated beam scale and stadiometer (Detecto, Webb City, MO, USA) was used to record height (cm) and body mass (kg). Skinfold measurements (Lange, Cambridge, MD, USA) were taken at three sites (males: chest, abdomen, thigh; females: tricep, suprailiac, thigh) and used to estimate body fat percentage (Pollock et al. 1980). Each participant completed a survey to determine the amount of caffeine consumed over an average of five days. This information was also used to identify habitual caffeine users.

$\dot{V}O_2$ max trials

After anthropometric measurements were collected, participants completed a maximal exertion test on a Monark cycle ergometer (Monark 874E, Varberg, Sweden). The protocol consisted of a warm up pedaling at 0 W while maintaining 60 rev/min for 3 min through visual feedback. The digital dial displaying rev/min was in participants view at all times. Thereafter, power was increased by 50 W every 2 min for females and 75 W every 2 min until the participant reached volitional exhaustion or could no longer maintain 60 rev/min. Metabolic data ($\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$, and RER) were measured using a Vacumed Vista

mini-cpx metabolic measurement system (Vacumetrics, Ventura, CA, USA) with integrated software (TurboFit, Vacu-med, Ventura, CA, USA). The vacumed system collected data in a breath by breath manner. Software integrated with the metabolic system was set to report a 20 s average value for all metabolic data with an update every 10 s. Prior to each trial, the system was calibrated using a gas of known concentration (4.01 % CO_2, 15.98 % O_2) with ventilatory measurement calibrated using a 3-L syringe (Hans Rudolph, Kansas City, MO, USA). Heart rate response was assessed using a Polar monitor (T31 Transmitter, Polar Electro, Kempele, Finland). An Omni Pictorial 0-10 RPE scale (Robertson et al. 2004) was displayed in view of participants at all times. Overall RPE was estimated by participants during the last 10 s of each minute. Criteria for achievement of $\dot{V}O_2$ max included meeting at least two of the following: a HR $\geq$85 % age-predicted maximum, RER value $\geq$1.1, and a $\dot{V}O_2$ plateau as the workload increased (ACSM 2010).

Caffeine versus placebo trials

After completion of the maximal test, participants reported to the lab on two separate occasions to perform constant-load cycling. One session followed caffeine ingestion and one session followed ingestion of a matched placebo. Caffeine and placebo were counterbalanced to control for ordering and completed within 4–7 days with the investigators and participant blind to the pre-trial treatment. Each session consisted of a 5 min warm-up at 50 W, a 30 min trial at the workload determined to elicit approximately 75 % $\dot{V}O_2$ max, and a 5 min cool-down at 50 W. Cadence was maintained at 60 rev/min. Power output was calculated and recorded using software designed for the cycle ergometer (Monark 874E, Vansbro, Sweden). Heart rate (T31 Transmitter, Polar Electro, Kempele, Finland) was recorded from a Polar monitor the last 10 s of every 5 min along with acute RPE (overall, legs, breathing) using the Omni pictorial scale which was in full view of participants at all times. For these in-task RPE participants responded to the question "How hard do you feel you are working?" HR response was blinded to participants throughout the trial to control for potential influence on RPE. Each participant ingested gelatin capsules containing either caffeine (6 mg/kg body wt) or a matched placebo with 473 mL water 1 h prior to reporting to the lab. Participants verified water and gelatin capsules had been consumed by signing a consent form prior to each trial. Following exercise, participants remained in the laboratory (seated recovery) for 20 min after which they estimated session RPE using the same 0-10 pictorial scale with participants responding to the question "How was your workout?" Upon completion of each trial (caffeine and placebo), a questionnaire using a ten-point Likert scale was administered (Hudson et al. 2008). For each question a response of zero indicated the symptom was "not at all experienced" with ten indicating the symptom was "extremely" experienced. The questionnaire was used to determine if the participant experienced any adverse symptoms (fatigue, elevated mood, nervousness, restlessness, tremors, stomach distress) and to what degree the symptoms had been experienced as a result of caffeine consumption.

Statistical analysis

Means and standard deviations for descriptive characteristics of participants were calculated. Session RPE, mean HR and mean power output (caffeine vs. placebo) were compared using paired t tests. Separate 2 (trial) $\times$ 6 (time point) repeated measures ANOVAs were used for between trial comparisons of HR and RPE overall, RPE legs and RPE breathing. When necessary, a Bonferroni post hoc procedure was used for follow-up comparisons. Subjective responses from the post-trial questionnaire were compared using a paired t test for each dependent measure. Results were considered significant at $p < 0.05$.

Table 1 Descriptive characteristics for participants ($n = 15$)

Variable	Mean	SD
Age (years)	24.5	4.6
Height (cm)	172.0	11.3
Body mass (kg)	74.0	12.7
Body fat (%)	16.9	5.9
$\dot{V}O_2$ max (mL/kg/min)	43.4	10.8

Values are means and standard deviations

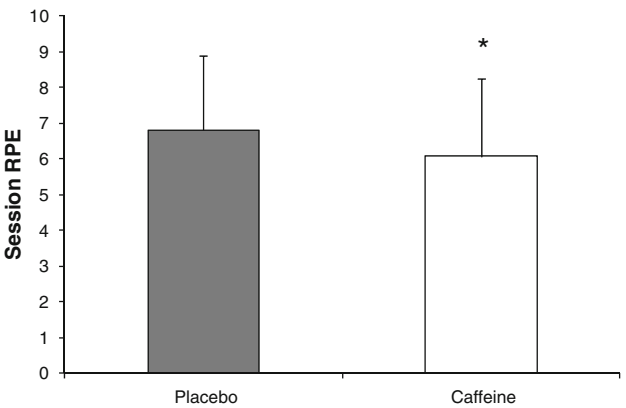

Fig. 1 Mean ± SD session ratings of perceived exertion between trials. *$p < 0.05$; caffeine versus placebo

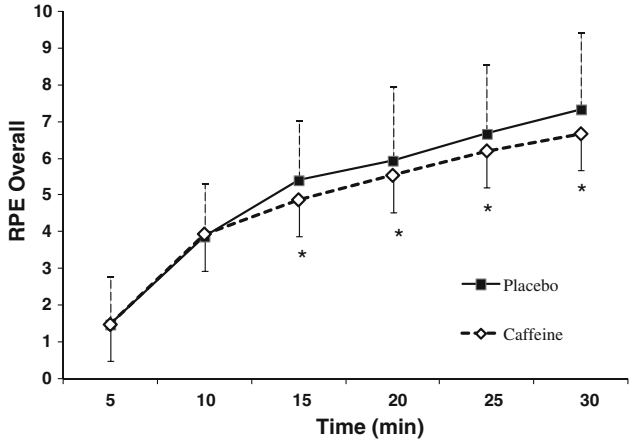

Fig. 2 Mean ± SD RPE overall between trials. *$p < 0.05$; caffeine versus placebo

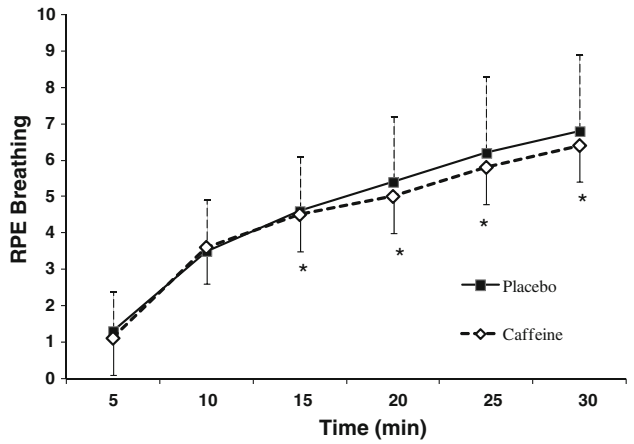

Fig. 3 Mean ± SD RPE breathing between trials. *$p < 0.05$; caffeine versus placebo

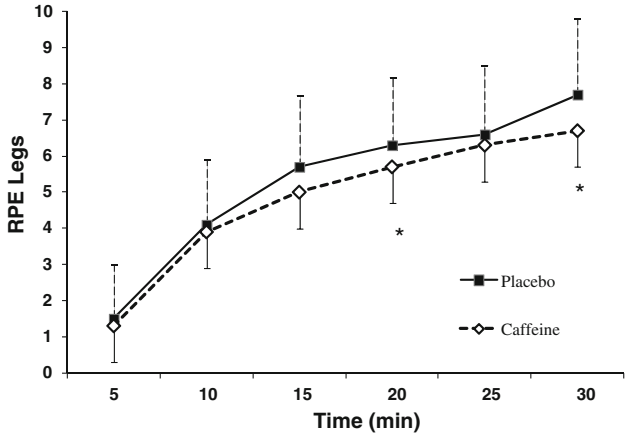

Fig. 4 Mean ± SD RPE legs between trials. *$p < 0.05$; caffeine versus placebo

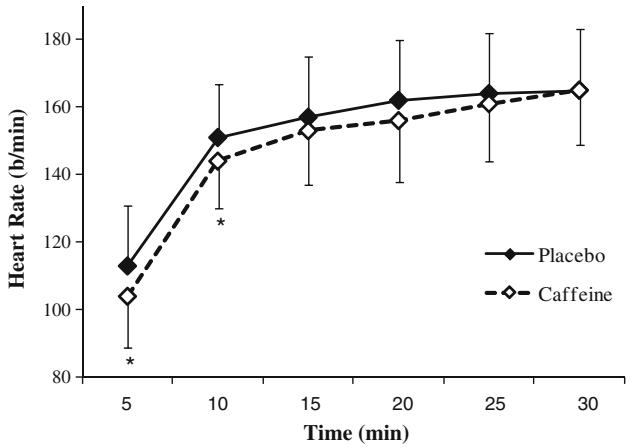

Fig. 5 HR (b/min) between trials. *$p < 0.05$; caffeine versus placebo

Results

Descriptive data for subjects are presented in Table 1. Session RPE was significantly lower for caffeine versus placebo (Fig. 1). There were main effects for caffeine versus placebo for RPE overall, RPE breathing and RPE legs. For RPE overall, caffeine was significantly lower than placebo at 15, 20, 25, and 30 min (Fig. 2). RPE breathing was significantly lower for caffeine at 15, 20, 25, and 30 min (Fig. 3). RPE legs were significantly lower for caffeine at 20 and 30 min (Fig. 4). HR response was significantly lower for caffeine at 5 and 10 min (Fig. 5). There was no significant difference in mean power output between caffeine (129.5 ± 45.5 rev/min) and placebo (135.9 ± 44.6 rev/min). Regarding subjective responses on the post-exercise survey, feelings of fatigue were significantly lower for caffeine (Fig. 6) while nervousness, restlessness, stomach distress, and tremors were significantly higher for caffeine (Fig. 6). Subjective responses for 'elevated mood' between caffeine and placebo trials approached but did not reach a priori level of significance for caffeine at $p = 0.11$.

Discussion

Determining the effects of caffeine ingestion on session RPE would help optimize use of this perceptual model to monitor athletes and help coaches adjust training loads in an effort to monitor athletes in training who consume caffeine. Previously, Birnbaum and Herbst (2004) and Jackman et al. (1996) reported a significant decrease of acute RPE during endurance exercise following caffeine ingestion. Further, Jackman et al. (1996) and Denadai and Denadai (1998) found a significant improvement in endurance performance following caffeine versus placebo ingestion. Many studies have reported a significant decrease in RPE following

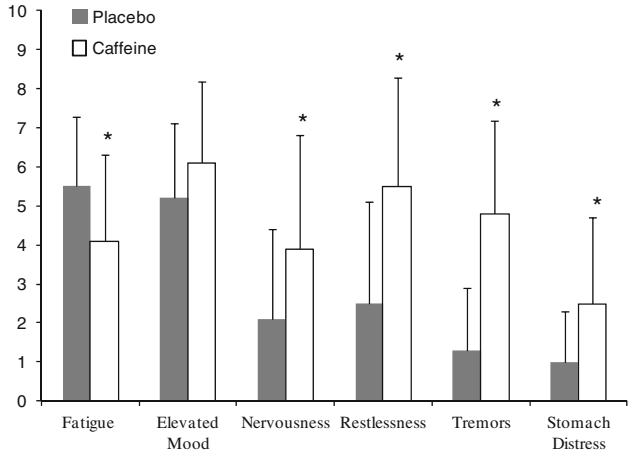

Fig. 6 Post-exercise questionnaire responses. Zero indicates no feeling of the described symptom, 10 indicates extreme feeling of described symptom. *$p < 0.05$; caffeine versus placebo

caffeine consumption (Jackman et al. 1996; Birnbaum and Herbst 2004; Doherty and Smith 2005; Green et al. 2007b; Hudson et al. 2008; Warren et al. 2010). However, no study to date has examined the potential influence of pre-exercise caffeine ingestion on session RPE. The current study examined this using a double-blind within-subjects design.

Current results indicate caffeine ingestion prior to exercise significantly reduced session RPE (Fig. 1). Mean values for session RPE were approximately 1 unit lower for caffeine versus placebo. Similar to previous studies, acute RPE (RPE overall, RPE legs, RPE breathing) were also significantly lower following caffeine ingestion (Figs. 2, 3, 4) even though workload was clamped and no significant difference was found between trials for total work. Mean HR responses at 5 and 10 min were significantly lower; however, the magnitude of the differences in b/min was negligible (Fig. 5). Further, HR at other time points was not significantly different suggesting work was significantly equated between trials helping to isolate the potential influence of caffeine on session RPE. Current research agrees with previous studies showing 6 mg/kg body weight caffeine reduced feelings of exertion during exercise (Jackman et al. 1996; Green et al. 2007b; Hudson et al. 2008). This is the first study to show caffeine blunts subjective estimations of global exertion (session RPE) when measures are taken 30 min post-exercise. These results indicate caffeine consumption should be considered when interpreting session RPE responses to monitor training. Based on session RPE it is plausible that caffeine may result in the physiological load being underestimated compared to a similar volume of work completed in absence of caffeine ingestion.

Doherty and Smith (2005) found caffeine ingestion (vs. placebo) resulted in lower RPE for constant-load exercise but found no difference for RPE at the conclusion of exhaustive exercise. In that study, even though RPE at the termination of the exhaustive exercise test was unchanged it was suggested that participants have a greater capacity to tolerate pain and fatigue resulting in an increased power output or time to exhaustion (Doherty and Smith 2005). Similar to Jackman et al. (1996), Denadai and Denadai (1998), and Birnbaum and Herbst (2004), current results show acute RPE was blunted by caffeine (Figs. 2, 3, 4). This is important as it can be concluded participants were responders to caffeine. Hudson et al. (2008) and Doherty et al. (2004) point out the importance of reviewing individualized responses when evaluating ergogenic aids because of the variation of interindividual responses. It is possible to overlook effects experienced by individuals if only group results are reported (Hudson et al. 2008). Because caffeine blunted acute RPE (Figs. 2, 3, 4), it can be concluded that, if caffeine influences session RPE, it would be detected in the current study; however, it is still important to examine individual responses. We defined a responder (to caffeine) as a participant displaying a 1 unit or greater difference between session RPE for placebo versus caffeine. In the current study, 53 % of participants were responders to caffeine. Forty-seven percent of participants were non-responders to caffeine (less than a 1 unit difference between trials). To apply a statistical analysis to differentiated groups, a two-tailed paired *t* test was conducted within each group (responders, non-responders) to compare session RPE between caffeine and placebo trials. Results showed a significantly greater mean session RPE for caffeine (6.9 ± 2.0) versus placebo (5.4 ± 1.8) for responders with no significant difference between trials for non-responders (caffeine 6.8 ± 2.4 vs. placebo 6.7 ± 2.3). Establishing groups based on presence of a response enhances the potential that a significant difference would be noted. However, providing an analysis offers greater precision than simply reporting of percentages of participants who achieved a pre-determined criteria for being a responder. Observations of raw data indicate responders showing a difference in acute RPE estimations seemed to also respond with respect to session RPE. Non-responders having near identical session RPE estimations also had similar acute ratings between trials. These results correspond well with previous studies showing that individuals respond differently to caffeine (Hudson et al. 2008). It is important also to note that aggregate analyses showed approximately a 1 unit difference between trials for session RPE (Fig. 1). Analysis of differentiated groups showed a 1.5 unit lower session RPE for caffeine for responders. Removing non-responders from the analysis magnified the response due to caffeine consumption. Therefore, caffeine has the potential to alter session RPE to a greater degree than shown by the analysis of group data in which mean values were diluted somewhat by inclusion of non-responders.

Identifying responders is important but it is also important to have a valid assessment of how they will be affected. Removal of non-responders from the analysis provides a more precise and thorough answer to the question regarding the potential influence of caffeine on session RPE.

Previous studies have indicated acute RPE is associated with session RPE (Foster et al. 2001; Green et al. 2009; Kilpatrick et al. 2009). In the current study, acute RPE was reduced when caffeine was consumed prior to the trial. The lower acute responses could have contributed to a reduction of session RPE. Lowered feelings of effort, even in light of equivalent workload, throughout the caffeine trial may have lead to an overall feeling of reduced effort due to caffeine consumption. That is, if session RPE is influenced by acute ratings, the blunted acute ratings could partially account for lower session RPE. One mechanism by which caffeine enhances performance is by decreasing perceived exertion in identical constant-load trials. Caffeine may also enhance performance by countering the inhibitory affect of adenosine on central nervous system function (McCall et al. 1982). Marcora (2009) suggests a significant contribution is not made to RPE from locomotor and afferent feedback from respiratory muscles. It is equally plausible the same mechanism altering acute RPE functions for session RPE. The same volume of work was perceived as less taxing which is reflected in subjective estimations, even in the post-exercise period.

In addition to the blunted acute RPE, subjective survey data also reveal participants were responders to caffeine further verifying that if a difference existed for session RPE it should have been detected in the current study. The post-exercise survey responses indicate significant changes were observed in subjective feelings following caffeine consumption. Participants reported feeling significantly more nervous and restless while also reporting more tremors and stomach distress (Fig. 6). Hudson et al. (2008) found similar results with participants reporting significant increases in restlessness, tremors, and stomach distress. These findings also suggest participants fulfilled treatment requirements and that caffeine was absorbed at least in responders. When asked in the survey, participants accurately reported (100 %) they felt they had ingested caffeine. While this could have influenced results, participants were not fully aware of the principle question in the study and intentional down-regulation of session RPE due to awareness of caffeine consumption is only speculative. Doherty and Smith (2005) suggested caffeine ingestion leads to a greater capacity to endure discomfort and has been shown to reduce the awareness of fatigue. It is plausible such effects carry over into the recovery period following exercise and result in lower session RPE.

Summary

In summary, current results suggest caffeine significantly reduces session RPE following cycling trials equated for total work volume. The current study provides evidence that caffeine has an erogenic effect during clamped constant-load exercise similar to studies such as Doherty and Smith (2005) and Birnbaum and Herbst (2004). More specifically, subjects reported lower acute RPE after consuming caffeine. In addition to lower acute RPE, session RPE taken 30 min post-exercise was also significantly lower. Also in accordance with previous studies, caffeine appears to produce inter individual differences with respect to perceptual estimations (responders vs. non-responders) during exercise. With session RPE being used to monitor overtraining, coaches as well as self-coached athletes, using session RPE should be more conservative with training prescription when caffeine is consumed prior to training or adjust the session RPE estimate to account for blunted responses. Multiple mechanisms may account for mitigated session RPE. However, regardless of the mechanism, because session RPE is altered from ingesting caffeine this should be considered when using a perceptual measure to monitor athletes to avoid overtraining. Further research is warranted regarding factors potentially mediating session RPE.

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