

Caffeine lowers perceptual response and increases power output during high-intensity cycling

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The aim of this study was to determine the effects of caffeine ingestion on a 'preloaded' protocol that involved cycling for 2 min at a constant rate of 100% maximal power output immediately followed by a 1-min 'all-out' effort. Eleven male cyclists completed a ramp test to measure maximal power output. On two other occasions, the participants ingested caffeine (5 mg · kg⁻¹) or placebo in a randomized, double-blind procedure. All tests were conducted on the participants' own bicycles using a KingcycleTM test rig. Ratings of perceived exertion (RPE; 6–20 Borg scale) were lower in the caffeine trial by approximately 1 RPE point at 30, 60 and 120 s during the constant rate phase of the preloaded test ($P < 0.05$). The mean power output during the all-out effort was increased following caffeine ingestion compared with placebo (794 ± 164 vs 750 ± 163 W; $P = 0.05$). Blood lactate concentration 4, 5 and 6 min after exercise was also significantly higher by approximately 1 mmol · l⁻¹ in the caffeine trial ($P < 0.05$). These results suggest that high-intensity cycling performance can be increased following moderate caffeine ingestion and that this improvement may be related to a reduction in RPE and an elevation in blood lactate concentration.

Keywords: ergogenic aids, preloaded exercise, rating of perceived exertion, short-term high-intensity exercise.

Introduction

Over the last 25 years, caffeine has been one of the most widely studied ergogenic aids by sport scientists. During this time, there has been an almost unequivocal support for the beneficial effects of oral caffeine ingestion (3–9 mg · kg⁻¹) on prolonged submaximal exercise (Costill *et al.*, 1978; Graham and Spriet, 1991, 1995; Lindinger *et al.*, 1993; Graham *et al.*, 1998; Greer *et al.*, 2000; Bell and McLellan, 2002; Bell *et al.*, 2002). In addition, there is now growing evidence that caffeine can also improve performance in short-term high-intensity exercise (i.e. an exercise intensity requiring >100% $\dot{V}O_{2max}$) lasting from several seconds up to ~7 min (for reviews, see Spriet, 1995; and Graham, 2001).

While the caffeine literature on short-term high-intensity exercise is strongly supportive of an ergogenic effect, the mechanisms of action to explain this effect have remained elusive. One consistent outcome of

moderate caffeine ingestion during submaximal exercise is an attenuated rating of perceived exertion (RPE) (Costill *et al.*, 1978; MacIntosh and Wright, 1995; Cole *et al.*, 1996). Since a dampened perceptual response during exercise may be partly responsible for any ergogenic effect of caffeine (Spriet and Howlett, 2000), it is reasonable to extend the examination of RPE to investigations of short-term high-intensity exercise. Although measurement of RPE during short-term high-intensity exercise is a relatively novel concept, recent investigations have indicated that RPE during such exercise is just as reliable a measure as RPE obtained during submaximal exercise (Doherty *et al.*, 2001). Furthermore, Doherty *et al.* (2002) have shown that, following caffeine ingestion, there was a tendency for a dampened RPE during the first 2 min of constant load running exercise at 125% $\dot{V}O_{2max}$.

The short-term high-intensity exercise protocols used by researchers investigating caffeine can be grouped into three distinct categories: (1) constant rate tests continued to exhaustion (Doherty, 1998; Bell *et al.*, 2001; Doherty *et al.*, 2002); (2) 'all-out' single- or repeated-bout performance tests of either a fixed time

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(Collomp *et al.*, 1991; Anselme *et al.*, 1992; Greer *et al.*, 1998; Bell *et al.*, 2001) or fixed distance (Wiles *et al.*, 1992; Anderson *et al.*, 2000; Bruce *et al.*, 2000; Paton *et al.*, 2001); and (3) tests that combine a constant rate phase with an all-out performance test (Wiles *et al.*, 1992; Jackman *et al.*, 1996) – that is, a ‘preloaded’ protocol. The benefit of preloaded protocols is that the advantages of both constant rate and performance elements can be incorporated within a single protocol. That is, because of the requirement to complete a standardized constant rate phase during exercise capacity tests, comparisons between placebo and caffeine trials at matched points during the test (e.g. of oxygen consumption, blood lactate concentration, perceived exertion, etc.) are possible. This aspect of testing allows researchers to scrutinize any difference between treatments or interventions and can lead to an indication of underlying mechanisms of action (Doherty, *et al.*, 2003). On the other hand, the performance element of a test in which athletes set their own pace closely mimics the physiological and perceptual responses the athletes experience in competition and thus confers a large measure of ecological validity (Hickey *et al.*, 1992; Hopkins *et al.*, 1999).

The aim of the present study was to assess the effects of caffeine on a recently developed 3-min preloaded, short-term high-intensity cycling test (Doherty *et al.*, 2003) using trained cyclists. In particular, we wished to assess the effects of caffeine on perceived exertion in the constant rate phase (i.e. first 2 min of the 3-min test) and power output in the final minute of the test. We hypothesized that caffeine would lower the perceived exertion during the first 2 min of the 3-min test and increase the power output during the final 1 min of the test.

Methods

Participants

Based on recent reliability studies (Doherty *et al.*, 2001, 2003), we estimated that the smallest worthwhile effect (Hopkins, 2000) for the RPE scores (6–20 Borg scale) during the first 2 min of a 3-min bout of short-term high-intensity exercise, and the peak power output attained during the final minute of such exercise, would be 1 RPE point and 20 W, respectively. These effects, together with their respective standard errors of measurement, were used to estimate sample size for the present study (equation 1; Hopkins, 2000):

$$(8 \times \text{SEM})^2 / (d \times 2) \quad (1)$$

where SEM is the standard error of measurement and d is the smallest worthwhile effect. The sample size

estimates were less than 10; however, 11 trained male cyclists, recruited from local cycling clubs, volunteered to participate. Their mean ($\pm s$) age, height, body mass and season’s best 40-km performance were 33 ± 8 years, 1.80 ± 0.08 m, 76.8 ± 7.1 kg and $55:42 \pm 3:53$ min:s, respectively. The procedures used were approved by a departmental committee for ethics in research, and all participants provided written informed consent before testing. The participants were tested at the same time of day on three separate occasions over a 2-week period. Although the participants continued to train throughout the study, they were instructed to refrain from strenuous physical activity in the 24 h immediately before the tests. In addition, the participants were also asked to abstain from caffeine-containing medications, foods and drinks in the 24 h before testing and to present themselves at the laboratory in a 2-h post-absorptive state. A list of common caffeine medications, foods and drinks was shown to the participants to assist in this process (Bunker and McWilliams, 1989). This list was also used as the basis for estimating the participants’ daily caffeine consumption, which for the present participants amounted to 185 ± 90 mg caffeine per day (mean $\pm s$).

General procedures

All participants were tested on three separate occasions. The first visit involved determination of maximal minute power output. The final two visits required the participants to perform a standardized warm-up and a 3-min preloaded cycling test. Before the preloaded tests, the participants ingested caffeine and placebo in a fully randomized, double-blind, crossover manner. All tests were conducted on the participants’ own road bicycles using a KingcycleTM test rig (EDS Portaprompt, UK), with additional ‘sprint’ stability straps attached to the back wheel to prevent wheel slippage. All participants were habituated to regular testing and training on the ergometer. Calibration of the KingcycleTM was performed as previously outlined (Doherty *et al.*, 2003). Tyre pressure was standardized at 700 k Pa for each test. Power output (1 s sampling) was measured using SRMTM power cranks (4-strain gauge crankset; SRM, Julich, Welldorf, Germany), which were fitted to each bicycle and calibrated according to the manufacturer’s instructions before each test. This system has previously been shown to provide valid and reliable measures of submaximal and maximal power outputs (Balmer *et al.*, 2000; Doherty *et al.*, 2003). After their final trial, the participants were asked whether they could identify the caffeine trial.

Maximal ramp minute power output

A ramp test, designed to exhaust the participants within 10–12 min, was used to determine maximal power output (Balmer *et al.*, 2000). Starting power output was 193 ± 39 W, with a ramp rate of 20.5 ± 3.5 W. Visual feedback from a computer display using KingcycleTM software (version 5.5) enabled the participants to maintain the desired power output through self-selected gear ratio and pedal cadence. The participants ended the test voluntarily when they could no longer maintain the required power output. Maximal power output was subsequently recorded using the SRMTM power crank data and was defined as the highest average power recorded during any 60-s period of the test. Heart rate was recorded continuously throughout the test (Polar Vantage, Polar Electro, Kempele, Finland).

Caffeine and placebo ingestion

One hour before the preloaded test warm-up, the participants consumed one of two beverages: (1) $5 \text{ mg} \cdot \text{kg}^{-1}$ caffeine (Roche, Welwyn Garden City, UK) diluted in 200 ml of an artificially sweetened water drink (caffeine), or (2) 200 ml of artificially sweetened water drink (placebo). The participants drank the assigned beverage immediately and then relaxed in preparation for the warm-up.

Preloaded test warm-up

A continuous 12-min warm-up was performed before the preloaded test. This consisted of participants riding for 6 min at 70% and then 80% maximal power output. During the warm-up, heart rate was measured continuously, and RPE was recorded during the last minute of each 6 min.

Preloaded cycling test

The preloaded test was performed 10 min after the warm-up. For this test, each cyclist used the single gear ratio that was applied at the time of maximal power output. The participants began the test with a 'rolling' start of 50 W, adapted from the protocol of Davison *et al.* (2000a). Following a 3-s countdown, the participants were required to cycle at maximal power output for 2 min. Ratings of perceived exertion were recorded every 30 s during this constant rate phase of the test (Doherty *et al.*, 2002). Immediately after the 2-min constant phase, the participants were required to complete a final 1-min 'all-out' effort without pacing, during which as much distance as possible was to be covered. At this time, the SRM module and computer screen were covered to prevent

the participants from monitoring their performance. Peak power was defined as the highest mean power of any 5-s period during the 1-min all-out effort. Peak heart rate was considered to be the highest mean heart rate achieved in any 5-s period during the test. At the end of the test, the participants remained seated to enable post-exercise earlobe blood samples to be taken at 4, 5 and 6 min for subsequent analysis of blood lactate concentration.

Blood analysis

Arterialized capillary blood samples (20 μl) were taken from an earlobe puncture and immediately mixed with a lysing stabilizing agent in a safe-lock vial. This involved shaking the sealed vial for approximately 15 s. All samples were analysed within 15 min for the determination of blood lactate concentration (Biosen 5030, EKF Industrie, Elektronik, GmbH, Barleben, Germany). In a previous study from our laboratory, we demonstrated that this procedure is both valid and reliable (the coefficient of variation of test–retest data was 1.4%; Davison *et al.*, 2000b).

Ratings of perceived exertion

Participants reported RPE during the warm-up and the first 2 min of the preloaded test. To familiarize the participants with the RPE scale, they underwent perceptual 'anchoring' (Noble and Robertson, 1996) for the bottom of the perceptual rating (i.e. '7' on the 6–20 Borg scale; Borg, 1998). This required the participants to cycle at 50 W for 3 min before the warm-up and to adjust their perceptual response to '7'. The participants were also asked to cap the top end of the perceptual rating to the previously experienced sensation of the point of exhaustion during the maximal ramp test.

Data analyses

Paired *t*-tests were used to determine differences between the caffeine and placebo treatments for the combined final minute power output of the preloaded test. All other data were tested with two-way (treatment \times time of measurement) repeated analyses of variance (Bonferroni, *post-hoc*). The assumption of sphericity was checked by Mauchly's test of sphericity and, where violations occurred, the Greenhouse-Geisser correction was applied. Pearson product-moment correlation coefficients were used to determine the relationship between measured variables. Statistical significance was set at $P < 0.05$. All statistical procedures were performed using SPSS for Windows, Version 9.0 (SPSS, Inc., Chicago, IL).

The results are reported as the mean \pm standard deviation (s).

Results

Maximal ramp minute power output

Maximal power output was 381 ± 58 W, time to exhaustion was 671 ± 109 s and peak heart rate was 184 ± 6 beats \cdot min $^{-1}$.

Preloaded test warm-up

There were no differences ($P > 0.05$) between placebo and caffeine mean power output for either the initial 6 min (placebo *vs* caffeine: 234 ± 38 *vs* 236 ± 39 W) or the final 6 min (placebo *vs* caffeine: 272 ± 41 *vs* 274 ± 40 W) of the warm-up. Similarly, there were no differences in mean heart rate during these two periods (placebo *vs* caffeine: first 6 min, 146 ± 12 *vs* 141 ± 18 beats \cdot min $^{-1}$; last 6 min, 158 ± 13 *vs* 156 ± 21 beats \cdot min $^{-1}$; $P > 0.05$). In addition, there were no differences in RPE (placebo *vs* caffeine: first 6 min, 12.7 ± 1.6 *vs* 12.5 ± 1.3 RPE points; last 6 min, 15.2 ± 2.1 *vs* 14.6 ± 1.6 RPE points; $P > 0.05$).

Preloaded cycling test

There were no differences in the peak heart rates achieved in the two treatments (placebo *vs* caffeine: 182 ± 9 *vs* 185 ± 13 beats \cdot min $^{-1}$). Although there was a trend for peak power to be greater in the caffeine treatment than in the placebo treatment (placebo *vs* caffeine: 446 ± 87 *vs* 472 ± 109 W), these differences did not reach significance ($P > 0.05$). Similarly, although there was a tendency for the sequential 30-s power outputs (Fig. 1) to be enhanced in the caffeine treatment, these did not reach significance. However, the mean value of power output during the final 1-min performance stage was higher in the caffeine treatment than in the placebo treatment (placebo *vs* caffeine: 750 ± 163 *vs* 794 ± 164 W; $P = 0.05$). Ratings of perceived exertion were lower at 30, 60 and 120 s in the caffeine treatment than in the placebo treatment ($F = 0.02$; Fig. 2). In addition, blood lactate concentration was higher at 4, 5 and 6-min post-exercise in the caffeine treatment than in the placebo treatment (placebo *vs* caffeine: 11.5 ± 1.4 *vs* 13.6 ± 2.9 mmol \cdot l $^{-1}$; 12.3 ± 1.5 *vs* 13.3 ± 1.8 mmol \cdot l $^{-1}$; 12.3 ± 1.6 *vs* 13.3 ± 2.0 mmol \cdot l $^{-1}$, respectively). There was no relationship between the participants' daily caffeine consumption and the change in power output (i.e. the difference between placebo and caffeine power output) in the final 1-min performance stage ($r = 0.12$;

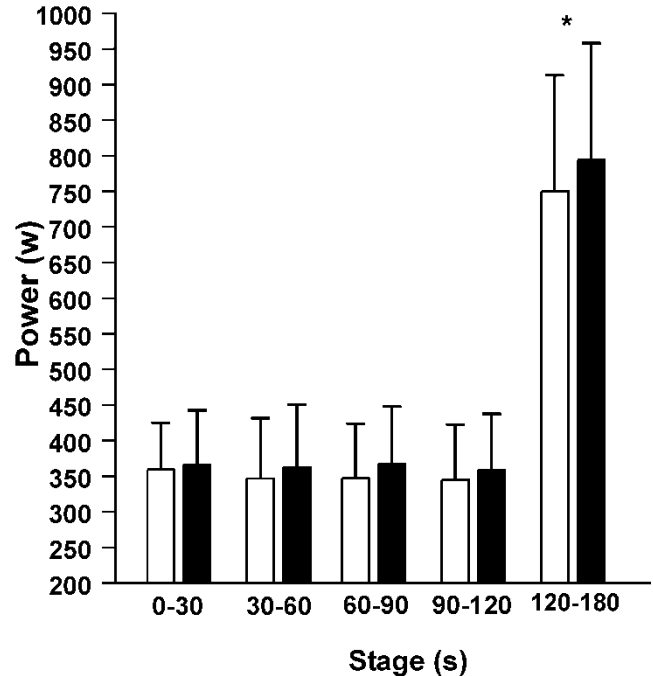


Fig. 1. Mean (\pm s) power output for the caffeine (■) and placebo (□) trials for the constant rate phase (0–120 s) and the performance phase (120–180 s) of the preloaded test. *Significant difference between treatments ($P = 0.05$).

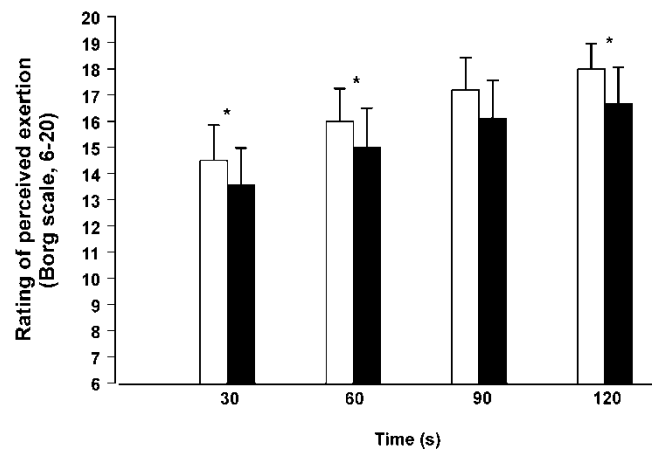


Fig. 2. Mean (\pm s) rating of perceived exertion scores at 30-s intervals during the constant rate phase of the preloaded test. ■, caffeine; □, placebo. * Significant difference between treatments ($P < 0.05$).

$P > 0.05$). Finally, 7 of the 11 participants correctly identified the caffeine trial.

Discussion

The main finding from this study was that the power output during the final 1 min of a 3-min preloaded test

protocol was increased by approximately 45 W (95% confidence interval, 0–88 w) after moderate caffeine ingestion ($P=0.05$). Having recently evaluated the reliability of the preloaded protocol in a group of similarly trained participants (where the typical error of measurement was 20 W; Doherty *et al.*, 2003), it is tempting to suggest that a 45-W improvement is 'worthwhile' (Hopkins, 2000). However, the wide confidence interval for the improvement is less than encouraging. In addition, because the test was designed as a research tool, *vis-à-vis* a constant rate phase and all-out effort, it is not typical of that performed by cyclists and it is difficult to determine if an improvement of 45 W is of 'practical' importance to any cycling event (Atkinson, 2003). In cycling, the relationship between power and speed is cubic, such that at high power outputs smaller changes in cycling speed result from any given change in power output (Martin *et al.*, 1998). Thus, more basic work to validate the present protocol for cyclists is required before issues relating to a given improvement can be addressed (Doherty *et al.*, 2003).

Notwithstanding the debate between statistical and practical significance, the findings of the study concur with several previous caffeine studies using short-term high-intensity exercise that have demonstrated an ergogenic effect (Collomp *et al.*, 1992; Wiles *et al.*, 1992; Jackman *et al.*, 1996). More recent research has shown that exercise to exhaustion during cycling and running at 125% $\dot{V}O_{2\max}$ lasting approximately 105–220 s, can be improved by as much as 8–15% following caffeine ingestion (Doherty, 1998; Bell *et al.*, 2001; Doherty *et al.*, 2002). Finally, Anderson *et al.* (2000) and Bruce *et al.* (2000) demonstrated that 2000-m rowing performance could be significantly improved in both competitive oarswomen and oarsmen.

A possible explanation for the enhanced power output in the present study was the reduction in RPE in the constant rate phase before the 1-min all-out effort (Fig. 2). Wiles *et al.* (1992) also found that caffeine could affect the perceptual response to short-term high-intensity exercise. These authors showed that immediate post-exercise (1500 m treadmill running) perceived exertion was the same in both caffeine and placebo trials, despite the fact that the caffeine treatment had produced a superior performance. More recently, Doherty *et al.* (2002) recorded RPE during short-term high-intensity exercise and found a reduction following caffeine ingestion after 90 s of treadmill running at a speed equivalent to 125% $\dot{V}O_{2\max}$, a finding that was implicated in the ergogenic effect of caffeine. In the present study, RPE was taken once every 30 s during the 2-min constant rate phase of the test (100% maximal power output). Participants' perceptual responses at 30, 60 and 120 s appeared to be blunted by approximately 1 RPE point (Fig. 2). Thus, during the

subsequent 1-min all-out effort (where a significant improvement in performance was demonstrated), the participants may have been able to maintain higher motor unit activation and/or were willing to better tolerate the discomfort associated with short-term high-intensity exercise.

Compared with the many endurance-based caffeine studies that have observed a reduction in RPE at the same standardized exercise intensity following caffeine ingestion (Costill *et al.*, 1978; MacIntosh and Wright, 1995; Cole *et al.*, 1996; Spriet and Howlett, 2000), this study provides further evidence that caffeine's ergogenic effect during short-term high-intensity exercise may also manifest itself via a dampened perceptual response.

Spriet and Howlett (2000) have suggested that the reduction in exercise RPE (or the increase in work accomplished at the same RPE) following caffeine ingestion could be caused by a decrease in the neuronal activation threshold of motorneurons and/or alterations in muscle contraction force. These changes, in turn, would result in an attenuation of muscle sensory processing that would reduce RPE because of more motor units being recruited for a given task, and because force for a given stimulus would be greater (Spriet and Howlett, 2000). Plaskett and Cafarelli (2001) also speculate that caffeine may modify not only afferent feedback but make alterations in feedforward information and/or in the central processing of either feedforward or feedback information. Finally, caffeine is a non-selective adenosine antagonist with established anti-nociceptive actions (Lieberman *et al.*, 1987; Ward *et al.*, 1991; Migliardi, *et al.*, 1994), and any explanation of caffeine's attenuation of the perceptual response to exercise should not discount its analgesic properties (Motl *et al.*, 2003).

In addition to central effects, the pervasive action of caffeine means that peripheral sites may also be implicated in any ergogenic explanation (Spriet, 1995; Graham, 2001). Bell *et al.* (2001) reported beneficial effects of caffeine during short-term high-intensity exercise and suggested that ATP production from anaerobic glycolysis is enhanced as a result of increased adrenaline following caffeine ingestion, a hypothesis first suggested by Collomp *et al.* (1991). In the present study, peak blood lactate was approximately $2 \text{ mmol} \cdot \text{l}^{-1}$ greater in the caffeine trial than in the placebo trial ($P < 0.05$). This elevation could reflect the increase in power output following caffeine ingestion and may have been due to increased anaerobic glycolysis as a result of increased adrenaline. However, while there is ample support in the literature on short-term high-intensity exercise for both an increase in the concentrations of adrenaline and blood lactate after caffeine ingestion (Collomp *et al.*, 1991, 1992; Anselme

et al., 1992; Bell et al., 2001; Doherty et al., 2002), direct muscle metabolite data suggest these findings may only be coincidental to any ergogenic effect (Jackman et al., 1996; Graham et al., 2000). In one study, caffeine elevated arterial lactate concentration during exercise, but muscle lactate concentration and release from the exercising leg were not altered (Graham et al., 2000).

Caffeine ingestion did not affect heart rate or RPE during the 12-min warm-up, and therefore these responses did not provide any foreknowledge of the subsequent enhancements that were made in the preloaded test. It is unclear why this was the case. One possibility is that the noted caffeine-induced effects during submaximal exercise (Costill et al., 1978; Graham and Spriet, 1991; Lindinger et al., 1993; Graham and Spriet, 1995; Graham et al., 1998; Greer et al., 2000; Bell and McLellan, 2002; Bell et al., 2002) require a longer duration than the 12-min warm-up in the present study.

In conclusion, this study has provided evidence that a moderate amount of oral caffeine can have an ergogenic effect during the last minute of a preloaded cycle test in trained participants. The improvement in performance may be related to a reduction in RPE during the constant rate phase and/or an increase in post-exercise blood lactate concentration. More basic research is required to establish the practical significance of the preloaded protocol and to distinguish between the central and peripheral effects of caffeine during short-term high-intensity exercise.

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