

Caffeine and sprinting performance: dose responses and efficacy

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Brief running head: Caffeine and sprinting

ABSTRACT

The aims of this study were to evaluate the effects of caffeine supplementation on sprint cycling performance and to determine if there was a dose-response effect. Using a randomised, double-blind, placebo-controlled design, 17 well-trained males (age, height, and body mass: 24 ± 6 years, 1.82 ± 0.06 m, and 82.2 ± 6.9 kg, respectively) completed seven maximal 10 s sprint trials on an electromagnetically-braked cycle ergometer. Apart from Trial 1 (familiarisation), all trials involved subjects ingesting a gelatine capsule containing either caffeine or placebo (maltodextrin) one hour prior to each sprint. To examine dose-response effects, caffeine doses of 2, 4, 6, 8, and 10 mg·kg body mass⁻¹ were used. There were no significant ($p \geq 0.05$) differences in baseline measures of plasma caffeine concentration prior to each trial (grand mean: 0.14 ± 0.28 µg·ml⁻¹). There was, however, a significant supplement \times time interaction ($p < 0.001$), with larger caffeine doses producing higher post-supplementation plasma caffeine levels. In comparison with placebo, caffeine had no significant effect on peak power ($p = 0.11$), mean power ($p = 0.55$), or time to peak power ($p = 0.17$). There was also no significant effect of supplementation on pre-trial blood lactate ($p = 0.58$), but there was a significant time effect ($p = 0.001$), with blood lactate reducing over the 50 minute post-supplementation rest period from 1.29 ± 0.36 mmol·L⁻¹ to 1.06 ± 0.33 mmol·L⁻¹. The results of this study show that caffeine supplementation has no effect on short-duration sprint cycling performance, irrespective of the dosage used.

Key words: Methylxanthine, Wingate, sprinting, ergogenic.

INTRODUCTION

Caffeine, a trimethylxanthine, is one of the most commonly consumed drugs in the world, with no apparent long-term adverse health effects (17). Despite being previously on the World Anti-Doping Agency (WADA) list of controlled substances ($> 12 \mu\text{g}\cdot\text{ml}^{-1}$ in urine was considered a doping offence), its removal in 2004 opened up the potential for athletes to exploit its ergogenic potential. Research into the effects of caffeine on athletic performance has been focused largely around endurance exercise, particularly since early research hypothesised a glycogen-sparing mode-of-action. Although evidence of a positive effect on endurance is considerable (5), the shift to a central nervous system mediated mode of action (via adenosine receptor antagonism) has led researchers to also consider the effects of caffeine on shorter and more intense exercise paradigms. Research into the effects of caffeine on speed-endurance events (60 – 180 s) and multiple sprint events tends to support a positive effect (8). Indeed, in a recent investigation from this laboratory, caffeine supplementation was found to increase sprint performance in the early stages of a 12×30 m multiple sprint test (10). Moreover, the magnitude of the improvement in fastest sprint time was evenly distributed across each 10 m split, suggesting potential benefits over longer sprint durations and single sprint events. In contrast, research into the effects of caffeine on single bouts of brief maximal work (primarily focused on 30 s Wingate anaerobic tests) generally, though not always (1,21), fails to show any performance effects (3,7,14,15,16,20,22). However, the possibility of pacing strategies influencing sprint durations of around 30 s, even in well-trained individuals, is a confounding factor (23). Indeed, several factors such as: poor sample sizes; use of untrained participants; failure to test resting plasma/urine caffeine

concentrations; inadequate placebos; and the use of fixed rather than relative caffeine doses, may explain why research into the effects of caffeine on high-intensity exercise has many discrepancies.

The primary aim of the present study was to address the aforementioned methodological limitations in order to investigate the effects of acute caffeine ingestion on brief (10 s) maximal sprint cycling performance. In addition, the study aimed to investigate whether, as with endurance performance, effects followed a dose-related response (4,13). Finally, several investigations have observed elevations in blood lactate following caffeine supplementation. If those elevations cannot be explained by a caffeine-induced increase in lactate production by the working muscles (12), then supplementation should lead to an increase in resting blood lactate concentrations. Therefore, a final aim of the present study was to investigate the effects of caffeine supplementation on resting blood lactate concentrations.

METHODS

Experimental Approach to the Problem

All subjects completed seven maximal 10 s sprint trials on an electromagnetically-braked cycle ergometer (Lode Excalibur Sport, Groningen, Holland), which was fitted with standard pedals, toe-clips, and straps, and interfaced with a computer to enable high-frequency logging of the flywheel angular velocity. Trial 1 was a familiarisation test to limit the effects of learning on the outcome of the experiment and involved no blood sampling. In the remaining trials, which were randomised and conducted in a double-

blind manner, subjects consumed a gelatine capsule containing either 2, 4, 6, 8, or 10 mg·kg⁻¹ of caffeine (My Protein, Manchester, UK), or the same volume (4 mg·kg⁻¹) and colour of placebo (maltodextrin: My Protein, Manchester, UK). All trials were completed at approximately the same time of day with a minimum of 48 hours between each. Subjects were instructed to maintain their normal diet throughout the testing period, to avoid food and drink in the hour before testing, and to avoid strenuous exercise 24 hours prior to each trial. Subjects were provided with a list of dietary sources of caffeine and asked to refrain from consuming these 48 hours prior to each trial. A questionnaire was used to establish typical daily caffeine consumption.

Subjects

Seventeen male strength & conditioning and sport science students volunteered for the study which was approved by St Mary's University College Ethics Committee. Prior to testing, subjects received written and verbal instructions regarding the nature of the investigation and completed a training history questionnaire, which indicated that all had been actively involved in sport for approximately 12 years. Times spent training and competing each week were reported as 8.8 ± 7.2 hours and 3.9 ± 4.2 hours, respectively. Prior to commencement of the study, all subjects completed a health-screening questionnaire and provided written informed consent. Means \pm standard deviation for age, height, body mass (bm), and body fat (%) of the subjects were: 24 ± 6 years, 1.82 ± 0.06 m, 82.2 ± 6.9 kg, and $13.5 \pm 3.4\%$, respectively.

Procedures

All testing was conducted in a laboratory which was thermostatically controlled at 18°C. On arrival at the testing facility, and after approximately five minutes of seated rest, resting blood samples (~ 5 ml) were drawn from a branch of the basilic vein and collected in plain siliconised tubes. At the same time, blood samples were obtained from the earlobe via capillary puncture for the evaluation of blood lactate. Subjects then consumed the supplement after which they rested for 50 minutes before the same blood sampling procedures were repeated. Venous blood samples were allowed to clot at room temperature before being centrifuged at 2000 rpm for 20 minutes, with subsequently decanted serum samples frozen at -20°C until analysed for caffeine and primary metabolite content using high-performance liquid chromatography (HPLC). Capillary blood samples were analysed for lactate content using an automated analyser (Biosen C-Line, EKF Diagnostic, Ebendorfer Chaussee 3, Germany). The analyser was calibrated before all trials in accordance with the manufacturer's instructions.

After blood sampling procedures were completed, subjects performed a standardised warm-up comprising 3.5 minutes of cycling at 120 W using a cadence of 80 rpm after which subjects were asked to indicate on a 20 cm visual analogue scale (ranging from 'not ready' to 'very ready') their perceived readiness to perform the forthcoming 10 s sprint. Subjects then performed two 5 s maximal practice sprints interspersed with 30 s of passive recovery, followed by another minute of cycling at 120 W (see Figure 1). The saddle height and handlebar position for each subject were determined before the first trial and remained constant for all subsequent trials. On

completion of the warm up and starting from a stationary position, subjects performed a 10 s maximal sprint against a torque factor of $0.7 \times \text{bm}$. Subjects were verbally encouraged to give maximal effort during all trials. After each trial, subjects completed a cool-down by cycling at 120 W for a minimum of two minutes.

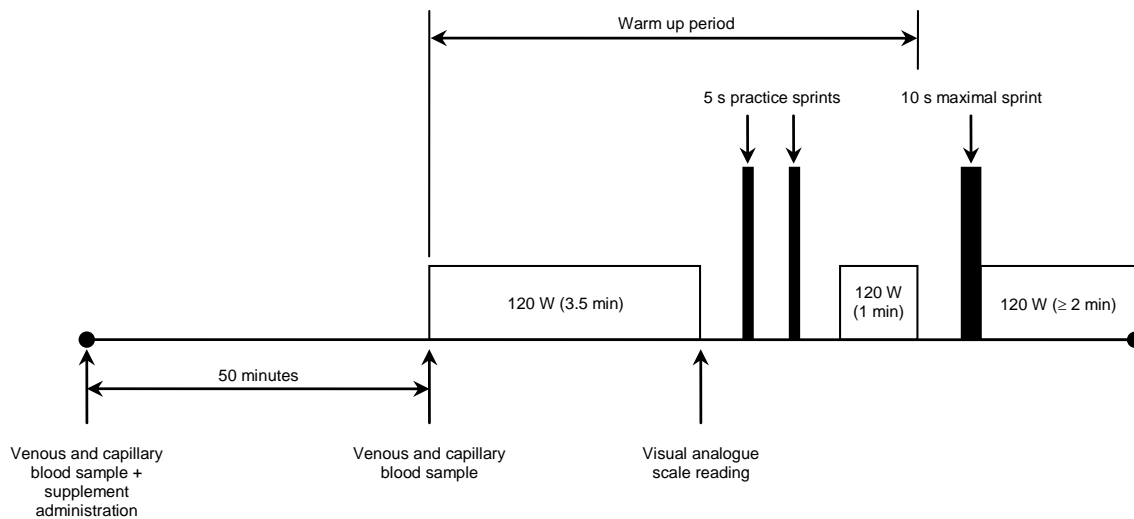


Figure 1. Schematic of the experimental protocol to investigate the effects of caffeine supplementation on 10 s sprint cycling performance.

Statistical Analyses

All data were analysed using the Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL). Measures of centrality and spread are presented as means \pm standard deviation. The effects of supplementation on serum caffeine concentrations and blood lactate were analysed using a two-way (time \times dose) ANOVA. The effects of caffeine dose on perceived readiness and on key sprint performance outcomes of peak power, mean power, and time to peak power were analysed using a one-way ANOVA. α was set at 0.05 for all comparisons and significant effects were followed up using Bonferroni-

adjusted *post hoc* analyses. The above analyses provided 95% confidence limits for all outcomes.

RESULTS

Analysis of the caffeine questionnaire data revealed that normal mean daily caffeine consumption of the subjects was 261 ± 224 mg (range: 10 – 677 mg). Analysis of the serum data (Table 1) revealed significant effects of time ($F_{(1,16)} = 297.64$, $p < 0.001$), dose ($F_{(2.7, 43.3)} = 32.89$, $p < 0.001$), and time \times dose ($F_{(2.7, 42.7)} = 33.48$, $p < 0.001$). *Post hoc* analyses showed that there were no significant differences in baseline serum caffeine concentrations obtained prior to each trial. In contrast, apart from placebo ($p = 0.299$), significant increases in serum caffeine concentrations were observed between baseline and post-supplementation samples ($p < 0.001$). The time \times dose interaction supported a dose-response effect of supplementation with higher doses producing higher serum caffeine concentrations; though not all *post hoc* comparisons were statistically significant (see Table 1). Blood lactate decreased significantly ($F_{(1,16)} = 18.32$, $p < 0.001$) from baseline to post-supplementation (mean change: $0.22 \text{ mmol}\cdot\text{L}^{-1}$; 95% likely range: 0.11 to $0.34 \text{ mmol}\cdot\text{L}^{-1}$); but there was no effect of caffeine dose ($F_{(5, 80)} = 0.76$, $p = 0.581$), and no time \times dose interaction ($F_{(5,80)} = 1.74$, $p = 0.135$) (Table 1). There was a significant effect of supplementation on perceived readiness ($F_{(5,80)} = 5.30$, $p < 0.001$), with placebo resulting in significantly lower scores than the 2, 8, and 10 mg caffeine doses (Table 1). Supplementation had no significant effect on sprint performance measures of peak power ($F_{(5,80)} = 1.88$, $p = 0.108$), mean power ($F_{(5,80)} = 0.80$, $p = 0.552$), or time to peak power ($F_{(3.63,58.15)} = 1.68$, $p = 0.172$) (see Table 2).

Table 1. The effects of various doses of caffeine supplementation on pre-sprint measures of serum caffeine concentration, blood lactate and perceived readiness. Values are means \pm standard deviation.

Caffeine dose (mg·kg bm ⁻¹)	Serum caffeine concentration ($\mu\text{g}\cdot\text{ml}^{-1}$)		Blood lactate ($\text{mmol}\cdot\text{L}^{-1}$)		Readiness score (%)
	Pre-supplementation	Post-supplementation	Pre-supplementation	Post-supplementation	
Placebo	0.20 \pm 0.32 ^a	0.19 \pm 0.30	1.25 \pm 0.36 ^f	1.04 \pm 0.39 ^g	46 \pm 19 ^j
2	0.09 \pm 0.16 ^a	3.68 \pm 2.01	1.20 \pm 0.30 ^f	1.05 \pm 0.33 ^g	67 \pm 15 ^h
4	0.19 \pm 0.36 ^a	9.06 \pm 3.72 ^b	1.28 \pm 0.39 ^f	1.03 \pm 0.32 ^g	65 \pm 19 ^{hi}
6	0.15 \pm 0.24 ^a	10.72 \pm 4.72 ^{bc}	1.23 \pm 0.34 ^f	1.14 \pm 0.33 ^g	63 \pm 23 ^{hi}
8	0.04 \pm 0.10 ^a	14.28 \pm 5.65 ^{cd}	1.42 \pm 0.39 ^f	1.14 \pm 0.35 ^g	74 \pm 13 ^h
10	0.20 \pm 0.39 ^a	17.6 \pm 8.14 ^d	1.34 \pm 0.41 ^f	0.98 \pm 0.30 ^g	70 \pm 21 ^h

Note: bm = body mass; mean values with the same superscript in each column are not significantly different ($p \geq 0.05$).

Table 2. The effects of caffeine supplementation on measures of maximal 10 s sprint cycling performance. Values are means \pm standard deviation.

Caffeine dose (mg·kg bm ⁻¹)	Peak power (W)	Mean power (W)	Time to peak power (s)
Placebo	1133 \pm 186	767 \pm 117	4.7 \pm 1.1
2	1133 \pm 200	769 \pm 113	4.6 \pm 1.2
4	1147 \pm 199	784 \pm 125	4.5 \pm 1.4
6	1111 \pm 204	772 \pm 115	4.3 \pm 1.3
8	1148 \pm 194	774 \pm 104	4.4 \pm 1.0
10	1141 \pm 195	776 \pm 105	4.2 \pm 1.0

Note: bm = body mass

DISCUSSION

The principal aim of this study was to examine the effects of caffeine supplementation on short-duration maximal-intensity sprint cycling. The main finding was that caffeine had no significant effect on any of the measures of sprinting performance. The absence of any significant effect of caffeine on short-duration (≤ 30 s) sprinting performance confirms a number of previous reports (3,7,14,15,16,20,22), but contrasts with others (1,21). Though the majority of studies support the absence of an effect of caffeine on sprinting performance, it is difficult to explain why this is not always the case. However, the fact that there was no dose-response effect of caffeine on performance suggests that between-protocol differences in dosing strategies are unlikely to be a confounding factor. Indeed, there are no common factors that explain the above

discrepancies, though a failure to measure resting serum or urine caffeine concentrations in several studies (1,7,14,15,16,22) is a concern.

One of the biggest problems when conducting research into responses to caffeine supplementation is that of blinding subjects to the treatment. Given the pronounced side effects of 'jitteriness' and 'nervousness' that generally accompany acute caffeine administration (19), it is often the case that subjects are able to determine when they have taken the supplement. Moreover, manifestation of those same side effects makes it difficult for researchers to also maintain a blind perspective. In the present study, differences in perceived readiness scores between caffeine and placebo trials suggests that subjects were aware of when they had taken caffeine. However, despite this, subjects seemed unable to identify differences between the various doses used. Moreover, the absence of any subsequent effect of caffeine on performance suggests that knowledge of the supplement did not influence their motivation to give a maximal effort. Whilst the same may not have been the case if the duration of the sprints had been extended (23), the absence of an effect of caffeine on most 30 s sprint trials (3,7,14,15,16), diminishes the possibility of a placebo effect (2) influencing the results.

A secondary aim of the present study was to examine the effects of caffeine on resting blood lactate concentration. If, as reported, caffeine has no effect on lactate release by the working muscles (6,12), then, since many studies have shown a caffeine-induced increase in blood lactate during constant-intensity exercise (11), an increase in blood lactate would be expected during resting conditions. However, the results of the

present study showed that regardless of the dosage used, caffeine had no significant effect on resting blood lactate; indeed, values significantly reduced across the 50 minute pre-trial resting period. Whilst it is possible that subtle differences in exercise intensity as a result of caffeine supplementation could explain many of the aforementioned elevations in blood lactate, more research is needed to clarify.

In summary, the results of this study suggest, in conjunction with a number of previous reports, that caffeine supplementation has no significant effect on short-duration (≤ 30 s) sprinting performance. Moreover, the absence of any effect on performance does not appear to be dose related. Given that caffeine appears to exert its effect via a central mechanism, and that the effects of caffeine on endurance exercise are well established, further research with rigorous methodological control is required to clarify the duration of exercise for which caffeine ceases to be ergogenic. Finally, if caffeine does indeed have no effect on sprinting performance, it is difficult to reconcile the positive effects of caffeine in some tests of repeated sprint performance, particularly since such effects have been observed from the start of each protocol (10,18).

PRACTICAL APPLICATIONS

The removal of caffeine from the WADA list of controlled substances in 2004 opened up opportunities for athletes to exploit its ergogenic effects. Nevertheless, although previous research supports positive effects in endurance exercise, the results of this study show that caffeine has no effect on short-duration sprinting performance, regardless of the dosage used.

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