Caffeine and sprint cycling performance: effects of torque factor and sprint duration

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Abstract

Purpose: The aim of this study was to investigate the influence of torque factor and sprint duration on the effects of caffeine on sprint cycling performance. *Methods*: Using a counterbalanced, randomized, double-blind, placebo-controlled design*,* 13 men completed nine trials. In Trial 1, participants completed a series of 6 s sprints at increasing torque factors, to determine the torque factor, for each individual, which elicited the highest ($T_{OPTIMAL}$) peak power output (PPO). The remaining trials involved all combinations of torque factor (0.8 $N·m·kg^{-1}$ versus $T_{OPTIMAL}$), sprint duration (10 s versus 30 s), and supplementation (caffeine [5 $mg·kg^{-1}$] versus placebo). *Results*: There was a significant effect of torque factor on PPO, with higher values at $T_{OPTIMAL}$ (mean difference: 168 W; 95% likely range: 142 – 195 W). There was also a significant effect of sprint duration on PPO, with higher values in 10 s sprints (mean difference: 52 W; 95% likely range: 18 – 86 W). However, there was no effect of supplementation on PPO ($p = 0.056$). Nevertheless, there was a significant torque factor $\times$ sprint duration $\times$ supplement interaction ($p = 0.036$), with *post hoc* tests revealing that caffeine produced a higher PPO (mean difference: 76 W; 95% likely range: 19 – 133 W) when the sprint duration was 10 s and the torque factor was $T_{OPTIMAL}$. *Conclusions*: The results of this study show that when torque factor and sprint duration are optimized, to allow participants to express their highest PPO, there is a clear effect of caffeine on sprinting performance.

Key words: Sprinting, coffee, ergogenic, anaerobic exercise.

Introduction

Caffeine, is a ubiquitous socially acceptable drug which, since 2004, is no longer on the World Anti-Doping Agency Prohibited List.[1] Research into the effects of caffeine on athletic performance has been focused largely around endurance exercise, with benefits of up to 6% across various modes of exercise.[1] Although early research supported a glycogen-sparing mode of action, the key mechanism by which caffeine is now believed to enhance athletic performance is via the antagonism of adenosine receptors.[1,2] Given the abundance of adenosine receptors and their ability to elicit multiple responses, depending on which of the four receptor subtypes is activated, researchers have begun to consider the effects of caffeine on shorter and more intense exercise paradigms.

Most of the research into the effects of caffeine on sprinting performance has been performed using 30 s cycle ergometer sprints (3-9) usually with torque factors of $0.75 - 0.90$ N·m·kg^{-1}.[3-6,8,9] With the exception of Woolf et al.,[9] all have found no effect of caffeine on performance. Nevertheless, Anselme et al.[10] and Glaister et al.[11] observed a significant effect of caffeine on peak anaerobic power output as determined from a series of 6 s sprints at incrementally increasing torque factors. As such, it seems that the failure of previous research to observe an effect of caffeine on sprinting performance could be due to motivational issues associated with 30 s sprints, insufficient torque factors to allow participants to express their true peak power output (PPO), or a combination of the two. Indeed, there is evidence that individuals produce a lower PPO during 30 s sprints than 10 s sprints, most likely due to subconscious pacing strategies.[12] Moreover, the torque factor which produces the highest PPO is generally reported to be around $1.0 - 1.25$ N·m·kg^{-1}. [10,11,13,14] The aim of the present study therefore, was to investigate the influence of torque factor and sprint duration on the ergogenic effects of caffeine on sprint cycling performance.

Methods

Participants

Thirteen recreationally-active male Sport Science students were recruited for the study which was approved by St Mary's University Ethics Committee. The sample size was based on previous research which found a significant effect of caffeine on sprint cycling performance.[10,11] Means ± standard deviation for age, height, body mass, and body fat[15] of the participants were: 20 ± 2 years, 1.78 ± 0.06 m, 75.3 ± 7.6 kg, and 13.0 ± 4.5%, respectively. Prior to testing, participants 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 11 ± 5 years and that the amount of time spent training each week was 8 ± 5 hrs. Prior to commencement, all participants completed a health-screening questionnaire and provided written informed consent. Participants were instructed to maintain their normal diet throughout the testing period, to avoid food and drink in the hour before each trial, and to avoid strenuous exercise 24 hours prior to each trial. Participants were provided with a list of dietary sources of caffeine and asked to refrain from consuming these for 24 hours prior to each trial. A questionnaire was used to estimate typical daily caffeine consumption. All trials were completed at approximately the same time of day with a minimum of 48 hours between each.

Design

All participants completed nine trials on an electromagnetically-braked cycle ergometer (Lode Excalibur Sport, Groningen, Holland) in a laboratory which was thermostatically controlled at 19°C. The saddle height and handlebar position for each participant were determined at the start of the first trial and remained constant for all subsequent trials. All sprints were performed in a seated position using standard pedals fitted with toe clips and straps. In Trial 1 participants completed a series of maximal 6 s sprints, interspersed with 5-minute passive recovery periods, against increasing torque factors to establish the torque factor which produced the highest ($T_{OPTIMAL}$) peak power output (PPO). The experimental trials (trials 2 − 9) followed a counterbalanced, randomized, double-blind design in which torque factor (0.8 N·m·kg^{-1} versus $T_{OPTIMAL}$), sprint duration (10 s versus 30 s), and supplementation (caffeine versus placebo) were manipulated such that all possible combinations of conditions were experienced by each participant (Figure 1).

Procedures

To provide sufficient data to examine the peak power/torque relationship, and since previous research has suggested that $T_{OPTIMAL}$ occurs at around 1.00 − 1.25 N·m·kg^{-1}, [11,13,14] the torque factors for Trial 1 were: 0.4, 0.6, 0.8, 0.9, 0.95, 1.0, 1.05, 1.1, 1.15, 1.2, and 1.25 N·m·kg^{-1}. If peak power had not reached a clear asymptote by 1.25 N·m·kg^{-1}, further sprints were completed using 0.05 N·m·kg^{-1} increments, until peak power started to decline. Strong verbal encouragement was provided during every sprint of every trial and all sprints were performed from the same stationary starting position. 5 minutes after completion of the 6 s sprint series, a 30 s maximal sprint against a torque factor of 0.8 N·m·kg^{-1} was performed to familiarize participants with the demands of a maximal sprint of the type required during the experimental trials.[16]

In the experimental trials, after blood sampling and supplementation procedures were completed (see below), a standardized 3-minute warm-up was conducted during which participants cycled at 100 W using a cadence of 80 rpm. The warm up included two 5 s practice sprints at increasing intensity (~ 75% and 90% of maximum) to prepare participants for the subsequent experimental maximal sprint. 60 s after completion of the warm up, participants performed a maximal sprint for the required duration against the appropriate torque factor. After each trial, participants performed a cool-down by cycling at 100 W for a minimum of three minutes.

At the start of each trial, 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 lithium-heparin tubes (Vacutainer; Becton Dickinson, Oxford, United Kingdom). Apart from Trial 1, participants were administered subsequently a gelatine capsule containing 5 mg·kg bm^{-1} of caffeine (MyProtein, Manchester, UK) or placebo (maltodextrin: MyProtein, Manchester, UK). After supplementation, participants rested for 45 minutes, to allow blood caffeine concentrations to peak,[17] before a second blood sample was drawn. Blood samples were immediately centrifuged at 3000 rpm for 10 minutes, with subsequently decanted plasma samples frozen at -80°C until analysed for caffeine content using high-performance liquid chromatography (HPLC). Before analysis, plasma samples were thawed, transferred to a separating flask, and made up to 1 mL with HPLC grade water. Following the addition of an internal standard, samples underwent solvent extraction using a chloroform/IPA mix (85%/15%). Each sample was extracted twice and the organic phase was removed each time. The organic phases of each extract were subsequently combined, evaporated to dryness under nitrogen, and re-suspended in HPLC grade water. Analysis of caffeine content was carried out

by reverse phase HPLC using a C18 column (Zorbax Eclipse Plus, Agilent Technologies Ltd., Stockport, UK) with a mobile phase of 80%/20% water/methanol, a flow rate of 1.5 mL·min^{-1}, and UV detection at 274 nm.

Statistical Analyses

All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS for Windows, IBM SPSS Inc., Chicago, IL). Measures of centrality and spread are presented as means ± standard deviation. A one-way repeated measures ANOVA on PPO in the order in which the trials were conducted was used to evaluate the potential influence of learning or training effects on the results. Differences between trials in measures of PPO, time to PPO (TTPP), and cadence at PPO were evaluated using a three-way (torque factor × sprint duration × supplement) ANOVA. α was set at 0.05 for all analyses. Significant interactions were followed-up using *post hoc* tests with Bonferroni adjustments for multiple comparisons. The above analyses provided 95% confidence limits for all estimates.

Results

Mean daily habitual caffeine consumption of the participants was 152 ± 290 mg (median: 60 mg; range: 0 − 1000 mg). Subject compliance with caffeine restriction prior to each trial was confirmed by the fact that in all non-caffeine supplemented conditions, plasma caffeine concentrations were low (0.17 ± 0.20 µg·mL^{-1}), whereas values were high (5.31 ± 2.08 µg·mL^{-1}) following caffeine. There was no significant effect of trial order on measures of PPO ($F_{(7,84)}$ = 1.742; p = 0.11).

T$_{OPTIMAL}$ occurred at a torque factor of 1.19 ± 0.08 N·m·kg^{-1}. The effects of torque factor, sprint duration, and supplementation on measures of PPO, TTPP, and cadence at PPO are presented in Figures 2, 3, and 4, respectively. There was a significant effect of torque factor on PPO ($F_{(1,12)}$ = 188.3; p < 0.001), with higher values at T$_{OPTIMAL}$ (mean difference: 168 W; 95% likely range: 142 − 195 W). There was also a significant effect of sprint duration on PPO ($F_{(1,12)}$ = 11.4; p = 0.006), with values being higher in 10 s sprints (mean difference: 52 W; 95% likely range: 18 − 86 W). However, there was no effect of supplementation on PPO ($F_{(1,12)}$ = 4.5; p = 0.056). There were no significant interactions between torque factor × sprint duration ($F_{(1,12)}$ = 2.5; p = 0.143), torque factor × supplement ($F_{(1,12)}$ = 1.8; p = 0.206), or sprint duration × supplement ($F_{(1,12)}$ = 1.1; p = 0.322) on PPO. Nevertheless, there was a significant torque factor × sprint duration × supplement interaction ($F_{(1,12)}$ = 5.5; p = 0.036), with *post hoc* tests revealing that caffeine produced a significantly higher PPO (mean difference: 76 W; 95% likely range: 19 − 133 W) only when the sprint duration was 10 s and the torque factor was T$_{OPTIMAL}$.

There was a significant effect of torque factor on TTPP ($F_{(1,12)}$ = 20.5; p = 0.001), with shorter times achieved using T$_{OPTIMAL}$ (mean difference: 0.96 s; 95% likely range: 0.50 − 1.42 s). However, there were no significant effects of sprint duration ($F_{(1,12)}$ = 2.8; p = 0.118) or supplementation ($F_{(1,12)}$ = 4.5; p = 0.055) on TTPP. Moreover, there were no significant interactions between torque factor × sprint duration ($F_{(1,12)}$ = 0.2; p = 0.672), torque factor × supplement ($F_{(1,12)}$ = 3.4; p = 0.091), sprint duration × supplement ($F_{(1,12)}$ = 0.1; p = 0.816), or torque factor × sprint duration × supplement ($F_{(1,12)}$ = 0.2; p = 0.645) on TTPP.

There were significant effects of torque factor ($F_{(1,12)}$ = 59.3; p < 0.001) and sprint duration ($F_{(1,12)}$ = 27.5; p = 0.001) on cadence at PPO. *Post hoc* tests revealed that cadence was fastest with the standard (0.8 N·m·kg^{-1}) torque factor (mean difference: 21 rpm; 95% likely range: 15

– 27 rpm) and with the 10 s sprint duration (mean difference: 6 rpm; 95% likely range: 4 – 9 rpm). There was, however, no effect of supplementation on cadence at PPO ($F_{(1,12)} = 4.1$; $p = 0.065$), though there was a significant torque factor × supplement interaction ($F_{(1,12)} = 5.8$; $p = 0.033$); the latter reflecting a pattern suggestive of a dissipation of potential effects of supplementation on cadence at PPO as torque factor increased. There were no effects of torque factor × sprint duration ($F_{(1,12)} = 0.1$; $p = 0.769$), sprint duration × supplement ($F_{(1,12)} = 0.2$; $p = 0.675$), or torque factor × sprint duration × supplement ($F_{(1,12)} = 0.5$; $p = 0.475$) on cadence at PPO.

Discussion

The aim of this study was to investigate the effects of caffeine supplementation on sprint cycling performance and how those effects are influenced by torque factor and sprint duration. The main findings were that sprint performance was influenced by torque factor and sprint duration. Moreover, it was only when torque factor and sprint duration were optimized to allow participants to express their highest PPO that an effect of caffeine supplementation on sprint performance was realised.

The finding that PPO was influenced by sprint duration confirms previous reports that, despite strong verbal encouragement, participants appear to subconsciously adopt pacing strategies in 30 s sprints;[12] as reflected in lower cadences at PPO despite no significant effect of sprint duration on TTPP. Moreover, the results of the present study show that irrespective of the torque factor utilized, the adoption of a pacing strategy masks any effect of caffeine on PPO and, as such, is likely to explain why previous research into the effects of caffeine on 30 s sprints has failed to find an effect.[3-8] Indeed, the adoption of subconscious pacing strategies in 30 s sprints may also explain findings in other ergogenic aids such as creatine[18] and sodium bicarbonate.[19]

In contrast to the above, the effect of torque factor on PPO is more difficult to rationalise, but is likely related to neuromuscular constraints associated with high pedalling frequencies.[20,21] In sprint cycling, the cadence which optimizes PPO is reported to vary depending on fitness status and muscle fibre type, with optimal values from mathematical models of 120 rpm,[22] but with values ranging from 100 – 135 rpm in endurance and sprint/power athletes respectively.[20] The torque factor-cadence relationship follows a negative linear response pattern[23] with lower torque factors leading to higher cadences and vice versa, as reflected in the results of the present study. Given that the relationship between cadence and PPO follows an inverted-U response,[23] the faster cadences required to achieve PPO under the standard torque conditions may have resulted in suboptimal muscle coordination. Indeed, Samozino et al.[21] reported that cadences faster than optimal, for each individual, moved force production during each pedal stroke to less effective phases of each crank cycle (i.e. later in the downstroke and during the early phase of the upstroke). Moreover, the ability to synchronise muscle activity into a coordinated efficient movement pattern and to minimise the level of agonist-antagonist coactivation, both within and between limbs, is likely to be impaired at cadences faster than optimal.[20] In the present study, the absence of any effect of supplementation on PPO under the standard torque conditions suggests that caffeine does not influence the limitations associated with muscle coordination and, as such, provides an explanation as to why most previous studies using torque factors lower than optimal have failed to observe an effect of caffeine on sprint cycling performance.[3-6,8]

The reason why the present study observed an effect of caffeine on PPO, once methodological issues associated with sprint duration and torque were addressed, is most likely due to an increase in muscle force production during each crank cycle; particularly given the absence of any significant effect of supplementation on cadence at PPO. The importance of strength for PPO in sprint cycling is inferred from the fact that PPO is generally greater in men than in women and that it increases from childhood to adulthood; peaking at the end of the third decade.[20] Furthermore, strength training has been shown to improve sprint performance, at least in running.[24] Although research into the effect of caffeine on muscle strength is limited and generally inconclusive,[1] there is evidence from a meta-analysis that caffeine appears to increase maximal voluntary contraction strength of the knee extensor muscles.[25] Moreover, the knee extensors provide a large contribution (~ 34%) to overall mechanical energy production during cycling[26] and knee extensor strength is particularly important during the downstroke of each crank revolution.[20] Indeed, the fact that the above meta-analysis found no effect of caffeine on electrically-evoked measures of strength led the authors to suggest that the effect of caffeine on knee extensor strength was most likely attributable to a central nervous system (CNS) response;[25] particularly since only around 85 – 95% of knee extensor muscles are recruited during a maximal voluntary contraction.[27] The effects of caffeine on the CNS have been attributed to its ability to bind to adenosine receptors and thereby reduce the inhibitory effects of adenosine on neurotransmitter release and firing rates.[2] Nevertheless, the ubiquitous nature of adenosine receptors and the contrasting effects of the various receptor subtypes, means that a direct effect of caffeine on muscle function cannot, at present, be completely discounted.

One final point to consider regarding the results of the present study is their application outside of the laboratory setting. In contrast to fixed torque factors which may, as in the present study, force athletes to chase high cadences to try to maximize performance; in real-world environments, cyclists seek to optimize performance via appropriate gear selection. Moreover, when bicycles are fitted with fixed gears, as in track cycling, sprinters choose gear ratios which allow them to optimize power output.[23] As such, the results of the present study suggest that caffeine is likely to enhance sprint performance in those athletes.

Conclusion

The results of the present study confirm the significant effects that sprint duration and torque factor have on sprint cycling performance; the former most likely due to subconscious pacing issues associated with 30 s sprints, and the latter most likely due to impairments in motor control from the fast cadences associated with low torque factors. However, when torque factor and sprint duration are optimized to allow participants to express their highest PPO, there is a clear effect of caffeine on sprint cycling performance. Moreover, the benefits of caffeine on PPO appear to be due to improvements in muscular force during the downstroke of each crank cycle, and most likely from a CNS-stimulated increase in the contribution from the knee extensor muscles.

Practical Applications

For those wishing to evaluate sprint cycling performance in the laboratory, it is important to recognise that standard Wingate torque factors of around 0.8 N·m·kg⁻¹ fail to optimize peak power output. As such, it would be prudent to establish the force-velocity profile of an athlete before setting the load for a sprint performance test. Even then, peak power output in a 30 s

test is likely to be impaired by subconscious pacing strategies. However, when torque factor and sprint duration are optimized, as is often the case in real-world settings, caffeine supplementation of around 5 mg·kg bm^{-1}, taken approximately 45 minutes prior to exercise, has a clear positive effect on sprint cycling performance.
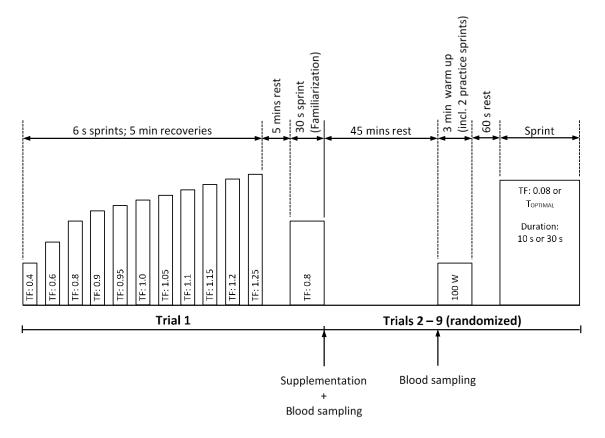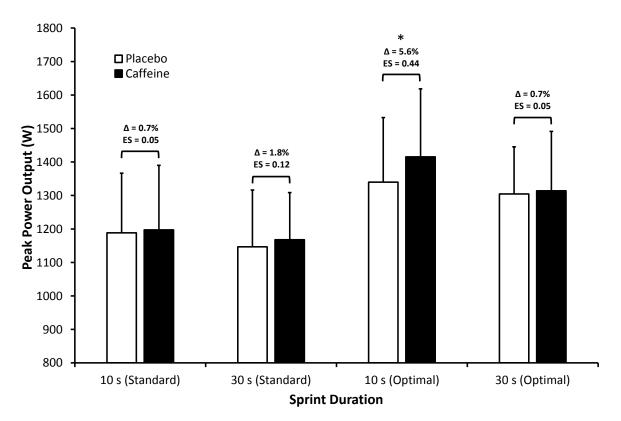
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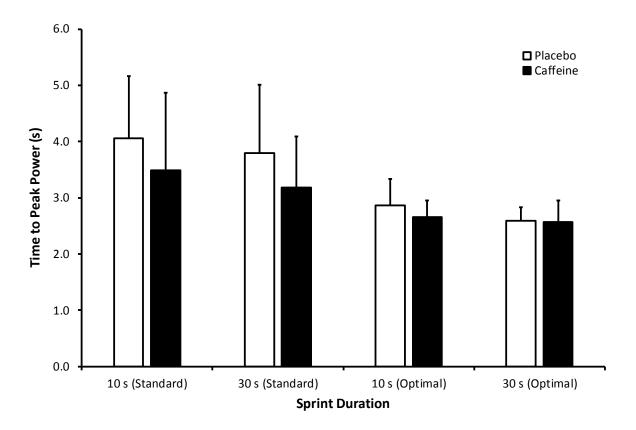
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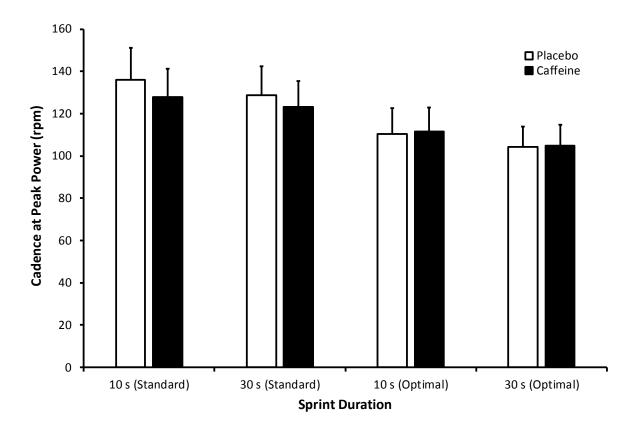
Figure 1. A schematic representation of the methods used in the study. TF = Torque factor in N·m·kg^{-1}; T$_{OPTIMAL}$ = the torque factor which produced the highest peak power output for each participant.

Figure 2. The effects of torque factor, sprint duration, and supplementation on peak power output during a maximal sprint on an electromagnetically-braked cycle ergometer. Values are means, bars are standard deviations. Note: Standard = standard torque factor of 0.8 N·m·kg⁻¹; Optimal = the torque factor required to optimize peak power output; *significantly different (*p* < 0.05) from placebo at the same torque factor and sprint duration; Δ = the percentage difference in peak power output between caffeine and placebo conditions; ES = the effect size for paired comparisons.

Figure 3. The effects of torque factor, sprint duration, and supplementation on time to peak power output during a maximal sprint on an electromagnetically-braked cycle ergometer. Values are means, bars are standard deviations. Note: Standard = standard torque factor of 0.8 $N \cdot m \cdot kg^{-1}$; Optimal = the torque factor required to optimize peak power output.

Figure 4. The effects of torque factor, sprint duration, and supplementation on cadence at peak power output during a maximal sprint on an electromagnetically-braked cycle ergometer. Values are means, bars are standard deviations. Note: Standard = standard torque factor of 0.8 N·m·kg^{-1}; Optimal = the torque factor required to optimize peak power output.