ORIGINAL ARTICLE

Caffeine Supplementation and Peak Anaerobic Power Output

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***Word count*:** 3496

***Abstract word count*:** 249

Abstract

The aim of this study was to investigate the effects of caffeine supplementation on peak anaerobic power output (W_{max}). Using a counterbalanced, randomised, double-blind, placebo-controlled design; 14 well-trained men completed three trials of a protocol consisting of a series of 6 s cycle ergometer sprints, separated by 5 min passive recovery periods. Sprints were performed at progressively increasing torque factors to determine the peak power/torque relationship and W_{max}. Apart from Trial 1 (familiarisation), participants ingested a capsule containing 5 mg·kg^{-1} of caffeine or placebo, one hour before each trial. The effects of caffeine on blood lactate were investigated using capillary samples taken after each sprint. The torque factor which produced W_{max} was not significantly different ($p \geq 0.05$) between the caffeine (1.15 ± 0.08 N·m·kg^{-1}) and placebo (1.13 ± 0.10 N·m·kg^{-1}) trials. There was, however, a significant effect ($p < 0.05$) of supplementation on W_{max}, with caffeine producing a higher value (1885 ± 303 W) than placebo (1835 ± 290 W). Analysis of the blood lactate data revealed a significant ($p < 0.05$) torque factor × supplement interaction with values being significantly higher from the sixth sprint (torque factor 1.0 N·m·kg^{-1}) onwards following caffeine supplementation. The results of this study confirm previous reports that caffeine supplementation significantly increases blood lactate and W_{max}. These findings may explain why the majority of previous studies, which have used fixed torque factors of around 0.75 N·m·kg^{-1} and thereby failing to elicit W_{max}, have failed to find an effect of caffeine on sprinting performance.

Keywords: *Wingate, sprinting, ergogenic aid, peak power*

Introduction

Caffeine, a trimethylxanthine, is one of the most commonly consumed drugs in the world, with no apparent long-term adverse health effects (Nawrot et al., 2003). 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 (Burke, 2008), the shift to a central nervous system mediated mode of action (via adenosine receptor antagonism), leading to increases in neurotransmitter release, motor unit firing rates, and pain suppression (Kalmar, 2005), has led researchers to consider also the effects of caffeine on shorter and more intense exercise paradigms.

Most of the research into the effects of caffeine on single bouts of brief (≤ 30 s) maximal exercise, predominantly using 30 s sprint cycling tests, shows no effect (Bell, Jacobs, & Elerington, 2001; Collomp, Ahmaidi, Audran, Chanal, & Prefaut, 1991; Glaister et al., 2012; Greer, Morales, & Coles, 2006; Hoffman et al., 2007; Lorino, Lloyd, Crixell, & Walker, 2006; Williams, Signorile, Barnes, & Henrich, 1988; Woolf, Bidwell, & Carlson, 2009). However, several review articles on the topic suggest that the evidence is inconclusive (Burke, 2008; Davis & Green, 2009; Graham, 2001; Magkos & Kavouras, 2005; Sökmen et al., 2008). This discrepancy tends to result from the fact that Anselme, Collomp, Mercier, Ahmaidi, and Prefaut (1992) found a significant effect of caffeine on maximal anaerobic power output (W_{max}), as derived from a series of maximal 6 s cycle sprint tests. However, the study by Anselme et al. (1992) has some limitations including: 1) the use of a mixed gender sample; 2) the use of a fixed (250 mg), rather than a body mass-relative caffeine dose; 3) the absence of serum caffeine analysis to confirm caffeine abstinence; and 4) the absence of a familiarisation trial. Moreover, although Anselme et al. (1992) found a significant effect of caffeine on W_{max}, the authors failed to report the effects of caffeine on the other sprints performed at various torque factors during the experiment. Instead, the authors chose to focus on the significant effects of caffeine on blood lactate; a response which the authors state that they monitored due to reports, at the time, that plasma lactate was correlated with plasma caffeine (Astrup et al., 1990). However, although Astrup et al. (1990) reported a significant positive correlation between caffeine dose and plasma lactate (r = 0.52),

the correlation between plasma caffeine concentration and plasma lactate was not significant (r = 0.29). Nevertheless, and despite a lack of corroborative research in sprint-based protocols, significant increases in blood lactate have often been observed during exercise following caffeine supplementation; however, in many instances, concomitant increases in performance make it difficult to determine the cause of the effect (refer to Davis & Green, 2009; Graham, 2001). The aim of the present study, therefore, was to repeat the study by Anselme et al. (1992), addressing the aforementioned issues, in an attempt to provide a clear answer as to whether caffeine has an effect on sprint cycling performance.

Methods

Participants

Fourteen male Strength & Conditioning and Sport Science students, regularly active in strenuous physical activity, volunteered for the study which was approved by St Mary's University Ethics Committee. 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 approximately 12 years. Times spent training and competing each week were reported as 8.1 ± 5.3 hours and 3.9 ± 3.1 hours respectively. Prior to commencement of the study, all participants completed a health-screening questionnaire and provided written informed consent. Means ± standard deviation for age, height, body mass, and body fat (Durnin & Womersley, 1974) of the participants were: 21 ± 3 years, 1.80 ± 0.05 m, 79.7 ± 10.5 kg, and 12.6 ± 3.2% respectively.

Experimental procedure

All participants completed three trials of a protocol consisting of a series of 6 s maximal sprints, separated by 5 min passive recovery periods, on an electromagnetically-braked cycle ergometer (Lode Excalibur Sport, Groningen, Holland). The saddle height and handlebar position for each subject were determined before the first trial and remained constant for all subsequent trials. All trials were

completed in an air-conditioned laboratory at a constant temperature of 19°C. Trials were completed at approximately the same time of day with a minimum of 48 hours between each. Participants 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. Participants were provided with a list of dietary sources of caffeine and asked to refrain from consuming these for 48 hours prior to each trial. A questionnaire was used to establish typical daily caffeine consumption.

Each sprint series was preceded by a 5 min warm up (at a cadence of 80 rpm and a power output of 120 W) followed by 5 mins of passive recovery. Trial 1 was used for familiarisation purposes and consisted of participants completing the first four sprints of the experimental trials. Trials 2 and 3 were the experimental trials which were counterbalanced and double-blind, and in which participants completed the sprint series at progressively increasing torque factors. One hour prior to each of the experimental trials, resting blood samples (~ 5 ml) were drawn from a branch of the basilic vein via venepuncture and collected in lithium-heparin tubes. Participants were then administered a gelatine capsule containing either 5 $mg \cdot kg^{-1}$ of caffeine (Sigma-Aldrich, Steinheim, Germany), or the same volume (4 $mg \cdot kg^{-1}$) and colour of placebo (maltodextrin: My Protein, Manchester, UK). After supplementation, participants rested for 45 mins before the same blood sampling procedure was repeated. Venous blood samples were centrifuged at 2000 rpm for 15 mins, 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 2 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 phase from both extracts 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 $mL \cdot min^{-1}$, and UV detection at 274 nm.

To provide sufficient data to determine the peak power/torque relationship, and since previous research has suggested that W_{max} occurs at around 1.00 – 1.25 N·m·kg^{-1} (Buśko, 2005; Winter et al., 1996), the torque factors used for the experimental trials were: 0.4, 0.6, 0.8, 0.9, 0.95, 1.0, 1.05, 1.1, 1.15, and 1.2 N·m·kg^{-1}. If peak power had not reached a clear asymptote by 1.2 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 and all sprints were performed from the same stationary starting position. At the end of each sprint series, participants completed a second sprint at 0.4 N·m·kg^{-1} as a check on the extent to which fatigue may have influenced performance during each trial. The effects of caffeine on blood lactate were investigated using 20 µl capillary samples taken immediately before the warm up, two minutes prior to each sprint series, and immediately after each sprint. 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.

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 ± standard deviation. W_{max} was determined as the highest peak power output achieved during each trial. The effects of supplementation on W_{max} were analysed using paired t-tests. A one-way ANOVA was used to evaluate the effects of supplementation on pre-test plasma caffeine concentrations. A two-way (supplement × torque factor) ANOVA with repeated measures on both factors was used to evaluate the effects of caffeine on peak power output, mean power output, and blood lactate for the 10 common sprints during each trial. A two-way (supplement × time) ANOVA was also used to evaluate the effects of supplementation on differences in peak power output between the first and last sprints of each trial and between pre-test measures of blood lactate. The possibility that the effects of caffeine supplementation on performance were influenced by habitual caffeine consumption was investigated by correlating estimated daily

caffeine consumption with caffeine-induced changes in W_{max} using a Pearson correlation. Correlation coefficients were interpreted in accordance with the following scale of magnitudes as devised by Cohen (1988): $r < 0.1$ is trivial; $0.1 \leq r < 0.3$ is small; $0.3 \leq r < 0.5$ is moderate; $r \geq 0.5$ is large. α was set at 0.05 for all analyses with Bonferroni adjustments for multiple comparisons. The above analyses provided 95% confidence limits for all estimates.

Results

Analysis of plasma caffeine data revealed a significant effect of supplementation ($F = 73.56$, $p < 0.001$), with *post hoc* analyses supporting subject compliance with the dietary restrictions such that in the non-caffeine conditions (pre-caffeine; and pre- and post-placebo) values were significantly lower (mean value: 0.16 ± 0.13 $\mu g \cdot ml^{-1}$) than they were following caffeine supplementation (4.20 ± 1.72 $\mu g \cdot ml^{-1}$). At the end of the investigation, participants were asked if they could identify the caffeine trial; six participants were correct in their assumption; the remainder being either incorrect ($n = 4$) or unsure ($n = 4$). Participants reported no adverse reactions to the supplements.

The effects of supplementation on peak and mean power output are presented in Figure 1, with the pattern of the power output responses for two of the common between-trial torque factors presented in Figure 2. There was a significant effect of torque factor on peak power output ($F = 54.59$, $p < 0.001$); but there was no effect of supplement ($F = 2.42$, $p = 0.144$), and no supplement × torque factor interaction ($F = 1.27$, $p = 0.262$). Similarly, with measures of mean power output; there was a significant effect of torque factor ($F = 51.83$, $p < 0.001$); but no effect of supplement ($F = 0.14$, $p = 0.716$), and no supplement × torque factor interaction ($F = 0.40$, $p = 0.934$). *Post hoc* analysis of the effect of torque factor revealed a levelling off of power output (peak and mean) at the higher torque factors, with no significant differences at torque factors ≥ 1.0 $N \cdot m \cdot kg^{-1}$. Analysis of peak power output in the first and last sprints of each trial, at the matched torque factor of 0.4 $N \cdot m \cdot kg^{-1}$, revealed no effect of supplement ($F = 0.61$, $p = 0.447$), time ($F = 1.92$, $p = 0.189$), or supplement × time ($F = 0.58$, $p = 0.460$).

The pattern of the power output response for the torque factors which elicited W_{max} is presented in Figure 2. The torque factor which produced W_{max} was not significantly different (p = 0.671) between the caffeine (1.15 ± 0.08 N·m·kg^{-1}) and placebo (1.13 ± 0.10 N·m·kg^{-1}) trials. There was, however, a significant effect (p = 0.028) of supplementation on W_{max}, with caffeine producing a higher value (1885 ± 303 W) than placebo (1835 ± 290 W). Analysis of caffeine questionnaire data revealed that mean daily caffeine consumption of the participants was 89 ± 95 mg (range: 0 – 283 mg). There was a small non-significant negative correlation between the amount of habitual caffeine consumption and the caffeine-induced change in W_{max} (r = -0.19; 95% likely range: -0.38 to 0.65).

Blood lactate concentration increased from rest (caffeine: 0.96 ± 0.33 versus placebo: 0.95 ± 0.27 mmol·L^{-1}) until two minutes before each trial (caffeine: 2.58 ± 0.97 versus placebo: 2.42 ± 1.11 mmol·L^{-1}) as a result of the warm up. However, there was no effect of supplement (F = 0.46, p = 0.509) and no supplement × time interaction (F = 0.31, p = 0.587) on pre-trial blood lactate concentration. There was, however, a significant effect of supplementation (F = 10.59, p = 0.006), torque factor (F = 34.45, p < 0.001), and supplement × torque factor (F = 4.40, p = 0.006) on blood lactate during the trials (Figure 3). *Post hoc tests* revealed significant differences between caffeine and placebo in the last five of the ten common trial torque factors.

Discussion

The aim of this study was to investigate the effects of acute caffeine supplementation on sprinting performance, particularly W_{max}. The main findings were that caffeine significantly increased W_{max} and caused significant elevations in blood lactate as each trial progressed. Although this study addressed some of the limitations of the study by Anselme et al. (1992), the key findings were the same.

Previous research into the effects of caffeine on sprint cycling performance has generally used traditional (Bar Or, 1981) fixed frictional torque factors of 0.75-0.80 N·m·kg^{-1} (Beck et al., 2006; Bell

et al., 2001; Collomp et al., 1991; Glaister et al., 2012; Lorino et al., 2006; Woolf, Bidwell, & Carlson, 2008). Apart from Woolf et al. (2008), all reported no effect on peak or mean power output. However, research suggests that muscle force-velocity relationships are optimized at higher torque factors (Buśko, 2005; Winter, Brown, Roberts, Brookes, & Swaine, 1996). Indeed, the torque factors which produced W_{max} in the present study are in line with previously reported optimization values of 1.00-1.25 $N \cdot m \cdot kg^{-1}$ (Buśko, 2005; Winter et al., 1996). Given that the results of the present study (and those of Anselme et al., 1992) showed no effect of caffeine at torque factors lower than those required to elicit W_{max}, it is possible that the lack of evidence supporting an effect of caffeine on sprinting performance in previous research could be due to the use of torque factors which fail to allow for the expression of W_{max}. Indeed, the pattern of the power output responses presented in Figure 2 shows how the effects of caffeine changes with increasing resistive torque. Then again, studies using torque factors of 0.9 (Greer et al., 2006), 1.1 (Williams et al., 1988) and 1.2 $N \cdot m \cdot kg^{-1}$ (Hoffman et al., 2007) have also found no effect of caffeine on sprint cycling performance. It may be that given individual variability in the torque factor required to elicit W_{max}, the use of fixed torque factors fails to allow for a significant effect of caffeine on sprinting performance to be identified. Alternatively, it may be that the lack of an effect of caffeine on sprinting performance in previous research is due to the duration of the protocols used. Most of the previous research has used 30 s Wingate tests to evaluate the effects of caffeine on performance (Beck et al., 2006; Bell et al., 2001; Collomp et al., 1991; Greer et al., 2006; Hoffman et al., 2007; Lorino et al., 2006; Woolf et al., 2008). However, peak power output is reported to be lower in 30 s versus 10 s Wingate tests even in highly-trained individuals; most likely as a result of subconscious pacing effects (Zajac, Jarzabek, & Waskiewicz, 1999). Given that fixed-torque protocols of 10 s (Glaister et al., 2012) and 15 s (Williams et al., 1988) have also failed to find an effect of caffeine on sprint cycling performance, it may be that the interplay between torque and sprint duration is responsible for the discrepant findings in previous studies.

The ergogenic effects of caffeine supplementation are currently considered to arise from the antagonism of adenosine receptors, leading to increases in neurotransmitter release, motor unit firing rates, and pain suppression (Davis & Green, 2009; Graham, 2001). Although it is difficult to identify

the reason why significant effects on peak power output were observed only at W_{max}, there was a trend towards an effect as the duration of the protocol increased (Figure 1). In effect, it seems likely that at low torque factors, where peak power output is determined largely by cycling velocity, the benefits of caffeine on neural drive may be outweighed by the inability of participants to maximise cycling cadence due to motor control issues. However, as resistive torque increases towards an optimal muscle force-velocity relationship, participants are able to meet the coordination demands of the task and the effects of caffeine on neural drive are then realised.

Despite the absence of significant effects of caffeine on peak (apart from W_{max}) and mean power output throughout the trials, there was, as with the results of Anselme et al. (1992), a significant effect of caffeine on blood lactate as the protocol progressed. Very few of the previous studies that have examined the effects of caffeine on sprinting performance have evaluated concomitant effects on blood lactate. Of those that have, Bell et al. (2001) and Collomp et al. (1991) reported significant increases in blood lactate despite finding no effects on performance, while Woolf et al. (2008) observed no effect on blood lactate, but a significant effect on performance. Similar contradictions exist in research into the effects of caffeine on blood lactate responses during longer bouts of exercise (refer to Davis & Green, 2009; Graham, 2001). In short, it is difficult to attribute caffeine-induced elevations in blood lactate solely to corresponding caffeine-induced increases in exercise performance. Indeed, even when caffeine-induced increases in exercise performance are accompanied by elevations in blood lactate, it is difficult to attribute those elevations to an increase in lactate release by the active muscles (Graham, Helge, MacLean, Kiens, & Richter, 2000).

Finally, despite considerable evidence to the contrary (refer to Davis & Green, 2009), there has been some suggestion in recent research of a habituation effect of caffeine (Sökmen et al., 2008; Woolf et al., 2008). Although there is evidence of an up-regulation of adenosine receptors as a result of regular caffeine intake (Holtzman, Mante, Minneman, 1991), the results of the present study add credence to reports which suggest that this has no effect on individual response to the supplement (Graham, 2001).

Conclusion

The results of this study confirm previous reports of a significant effect of caffeine supplementation on W_{max} and blood lactate. Although further research is required to confirm, the absence of significant effects of caffeine on sprint cycling in the majority of previous research is possibly due to the use of torque factors which fail to maximise peak power output combined with the use of protocols which are prone to pacing effects. Finally, although the results of the present study show that the effects of caffeine on sprinting performance are realised only under very specific conditions, the fact that skilled athletes in sprint cycling and running adopt strategies that serve similarly to optimise power output (Gardner, Martin, Martin, Barras, & Jenkins, 2007; Miller, Umberger, & Caldwell, 2012), supports a likely benefit of caffeine supplementation on sprinting performance outside of the laboratory environment.

Acknowledgements

The authors would like to thank all the athletes who participated in this investigation for their enthusiasm and dedication to the project. In addition, the authors would like to thank the School of Sport, Health, and Applied Sciences Research Fund (St. Mary's University) for funding the project.

Conflict of interest

The authors have no conflicts of interest that are relevant to the content of this article.

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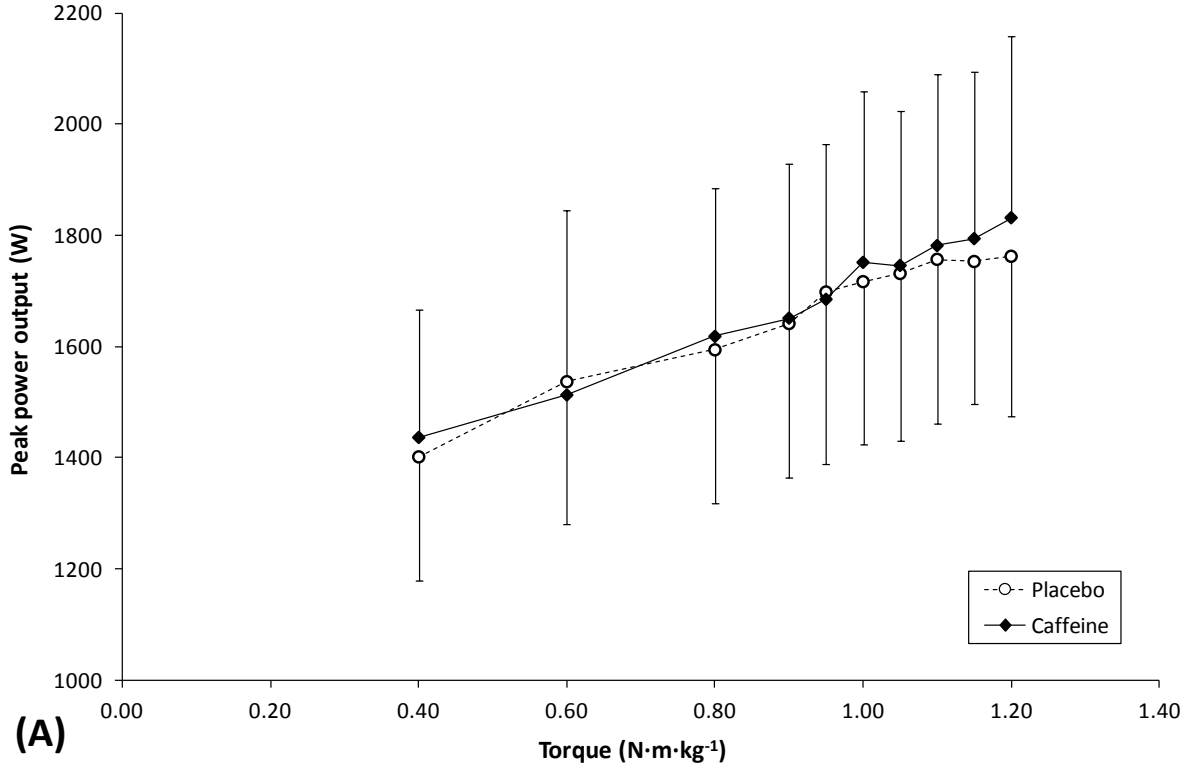

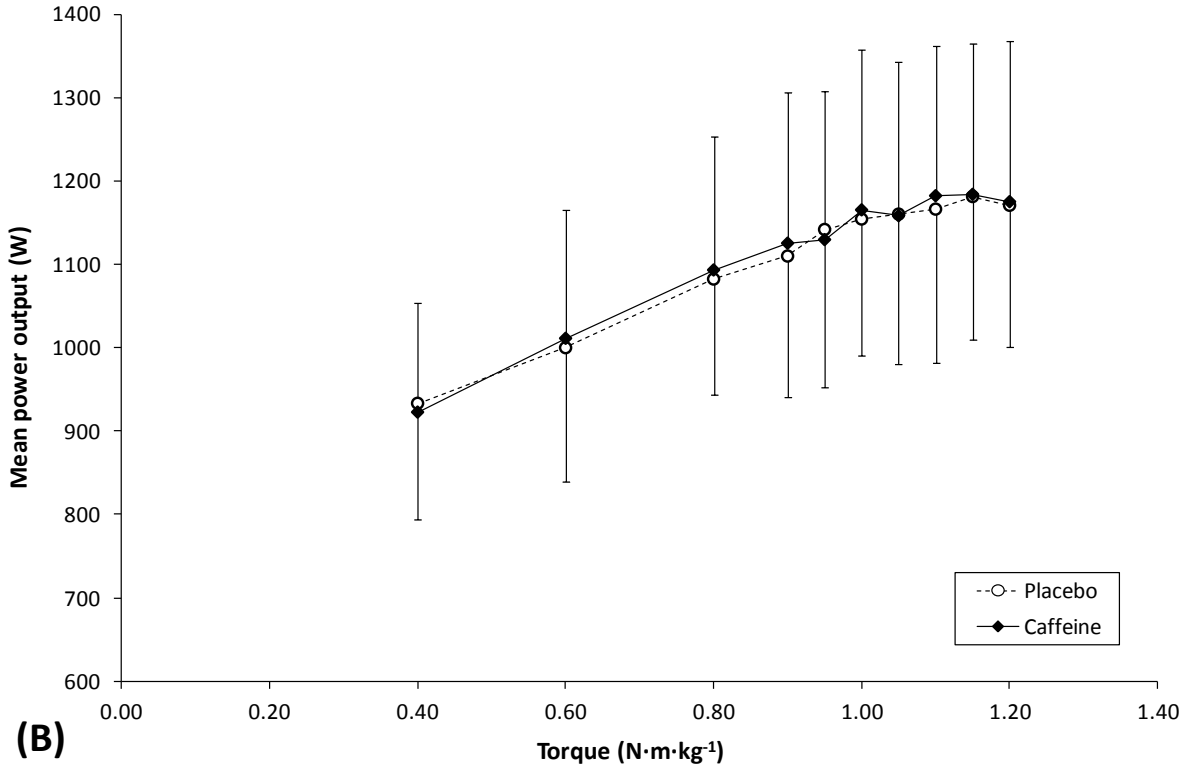

Figure 1. The effects of caffeine supplementation on A) peak and B) mean power output during a series of 6 s sprints (5 min recovery periods) using various torque factors. Values are means; bars are standard deviations.

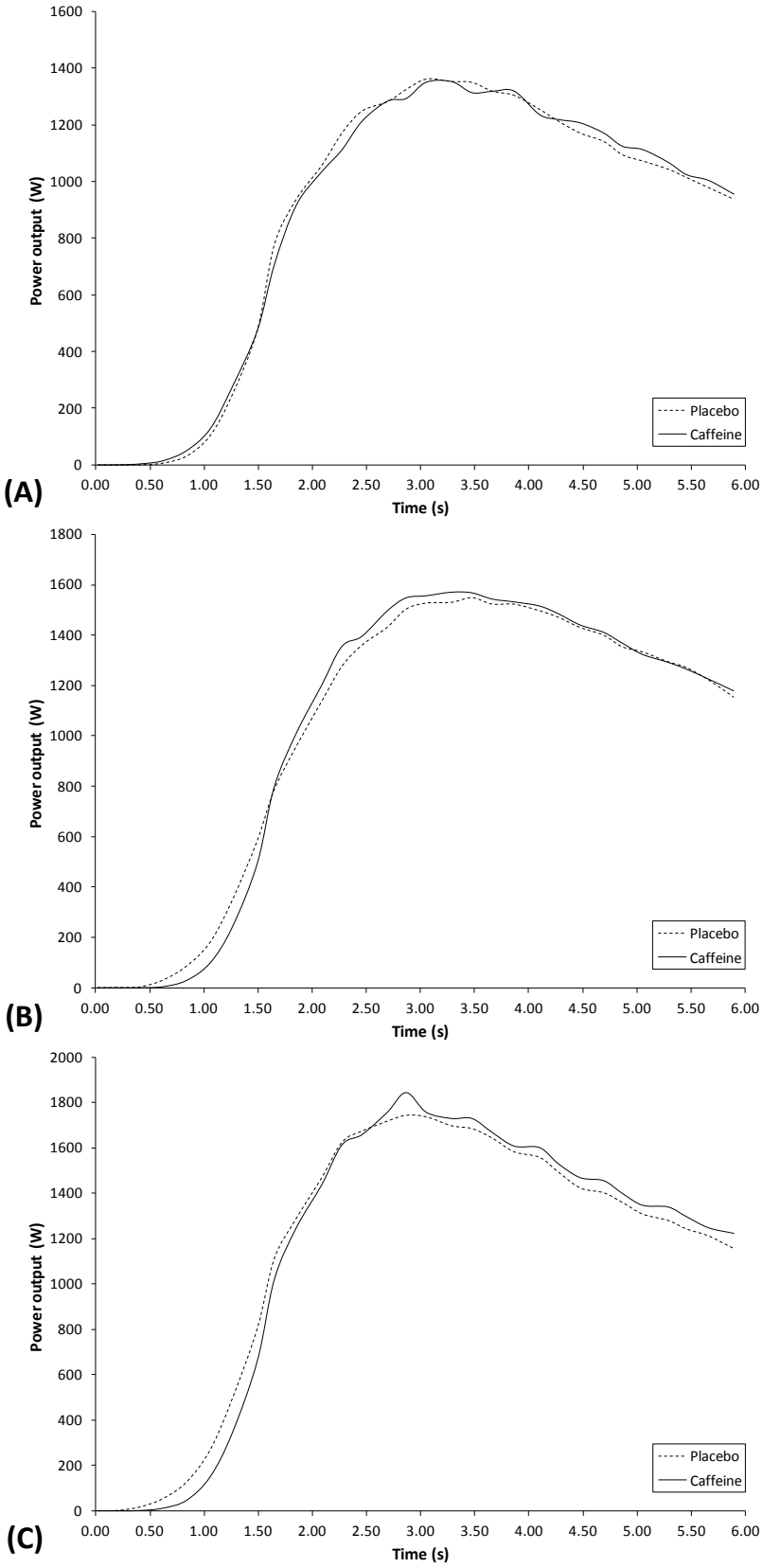

Figure 2. The effects of caffeine supplementation on the pattern of the power output response during a 6 s sprint at torque factors of: A) 0.4 $N \cdot m \cdot kg^{-1}$, B) 0.8 $N \cdot m \cdot kg^{-1}$, and C) the torque factor eliciting maximal anaerobic power output. Values are means.

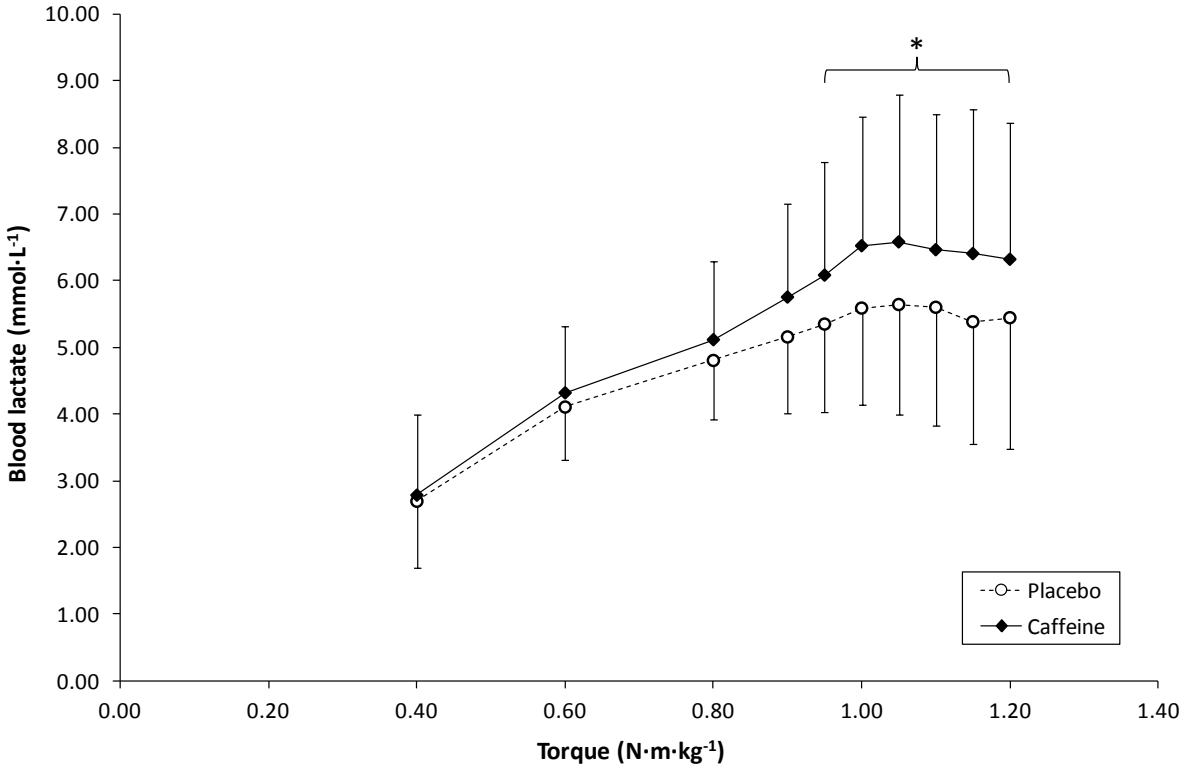

Figure 3. The effects of caffeine supplementation on blood lactate during a series of 6 s sprints (5 min recovery periods) using various torque factors. Values are means; bars are standard deviations. *significant ($p < 0.05$) difference between caffeine and placebo conditions.