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Caffeine supplementation and multiple sprint running performance

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Running head

Caffeine and multiple sprints

ABSTRACT

Purpose: The aim of this study was to examine the effects of caffeine supplementation on multiple sprint running performance. **Methods:** Using a randomised double-blind research design, 21 physically active men ingested a gelatine capsule containing either caffeine (5 mg·kg body mass^{-1}) or placebo (maltodextrin) one hour before completing an indoor multiple sprint running trial (12 × 30 m; repeated at 35 s intervals). Venous blood samples were drawn to evaluate plasma caffeine and primary metabolite concentrations. Sprint times were recorded via twin-beam photocells and earlobe blood samples were drawn to evaluate pre and post-test lactate concentrations. Heart rate was monitored continuously throughout the tests with ratings of perceived exertion (RPE) recorded after every third sprint. **Results:** Relative to placebo, caffeine supplementation resulted in a 0.06 s (1.4%) reduction in fastest sprint time (95% likely range: 0.04 to 0.09 s), which corresponded with a 1.2% increase in fatigue (95% likely range: 0.3 to 2.2%). Caffeine supplementation also resulted in a 3.4 b·min^{-1} increase in mean heart rate (95% likely range: 0.1 to 6.6 b·min^{-1}), and elevations in pre (+ 0.7 mmol·L^{-1}; 95% likely range: 0.1 to 1.3 mmol·L^{-1}) and post-test (+ 1.8 mmol·L^{-1}; 95% likely range: 0.3 to 3.2 mmol·L^{-1}) blood lactate concentrations. In contrast, there was no significant effect of caffeine supplementation on RPE. **Conclusion:** Although the effect of recovery duration on caffeine-induced responses to multiple sprint work requires further investigation, the results of the present study show that caffeine has ergogenic properties with the potential to benefit performance in both single and multiple sprint sports.

Key words: METHYLXANTHINES, REPEATED SPRINT ABILITY, INTERMITTENT EXERCISE, ERGOGENIC, FATIGUE

INTRODUCTION

Caffeine (a trimethylxanthine) is the most commonly consumed drug in the world, with minimal associated health risks (20). Following oral administration, caffeine is rapidly absorbed from the stomach with typical experimental doses of 4 – 6 $mg \cdot kg^{-1}$ giving rise to peak plasma concentrations of approximately 6 – 8 $\mu g \cdot ml^{-1}$ (30 – 40 $\mu mol \cdot L^{-1}$) within one hour (2, 25). Whilst the benefits of caffeine on endurance performance are well established in both human and animal models (7, 9, 14, 22, 24), effects on short-term (≤ 60 s) bouts of maximal exercise are generally unclear (1, 5, 6, 8, 23, 37). Of the mechanisms purported to explain the beneficial effects of caffeine supplementation, recent findings support a central nervous system (CNS) response mediated by antagonism of adenosine receptors leading to increases in neurotransmitter release, motor unit firing rates, and dopaminergic transmission (16, 25). Although further research is required to clarify the relative contribution made by each of these processes to various activities and to confirm or refute the possibility of an intramuscular mechanism of action (29, 36), the removal of caffeine in 2004 from the World Anti Doping Agency's list of prohibited substances has opened up the opportunities for athletes to exploit its ergogenic properties.

One form of exercise which has received little attention regarding the effects of caffeine supplementation is that of repeated sprint performance, particularly the type often experienced in many field and court sports. Based on the results of several time-motion analyses, research into this type of activity has typically utilised repeated bouts (≤ 20) of brief (≤ 6 s) maximal work interspersed with relatively short (≤ 60 s) recovery periods to simulate competitive demands (17, 34). Although the combination of speed and endurance associated with this type of exercise suggests a potential benefit from caffeine supplementation, research to date shows equivocal

results. For instance, Paton *et al.* (30) reported negligible effects of caffeine ingestion on 10 × 20 m sprints repeated at 10 s intervals. However, findings were limited by the use of single-beam photocells (38) and a failure to measure resting and pre-exercise plasma caffeine concentrations. More recently, Schneiker *et al.* (33) and Stuart *et al.* (35) have examined the effects of caffeine on prolonged periods of high-intensity intermittent exercise designed to simulate the physical demands of field sports. Whilst the protocols were considerably longer (80 and 72 minutes, respectively) than a typical test of multiple sprint ability, the findings generally supported caffeine-induced enhancements in high-intensity performance and, in line with typical findings on endurance exercise, an apparent progressive reduction in fatigue. As in many previous investigations, results were somewhat limited by the use of relatively small sample sizes ($n \leq 10$). The aim of the present study was to address the aforementioned methodological limitations in order to investigate the effects of acute caffeine ingestion on physiological and performance responses to multiple sprint work.

METHODS

Subjects

21 male 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 13 years and that most (n = 19) regularly participated in some form of multiple sprint sport. Times spent training and competing each week were reported as 7.3 ± 4.3 hours and 3.8 ± 3.5 hours respectively. Prior to commencement, all subjects completed a health-screening questionnaire

and provided written informed consent. Means ± standard deviation for age, height, body mass, and body fat (13) of the subjects were: 21 ± 3 years, 177.4 ± 5.9 cm, 77.7 ± 13.5 kg, and 14.6 ± 5.3% respectively.

Experimental procedure

All subjects completed four trials of the multiple sprint test, which consisted of 12 × 30 m straight-line sprints repeated at 35 s intervals. Trial 1 was a familiarisation test to limit the effects of learning on the outcome of the experiment. Trial 2 was a baseline test to enable the effects of both caffeine and placebo to be evaluated. Trials 3 and 4 were the experimental trials which were randomised and conducted in a double-blind manner. All trials were completed at approximately the same time of day with a minimum of four days between trials. 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.

On arrival at the testing facility, with the exception of the familiarisation trial, resting blood samples (~ 5 ml) were drawn from a branch of the basilic vein and collected in plain siliconised tubes. In trials 3 and 4, subjects were subsequently 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: Starmax Nutrition, Hereford, UK) one hour prior to testing. After supplementation, subjects rested for 45 minutes before a second blood sample

was drawn. Blood samples were allowed to clot at room temperature after which they were 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). After Trial 4 subjects were asked if they could distinguish between the two supplements and if so to identify the basis of that decision. Nine subjects correctly identified the caffeine trial *post hoc*. Common explanatory characteristics were terms such as 'energised', 'boosted', and 'fidgety'.

All testing was conducted indoors on a synthetic running surface in an environment which was thermostatically controlled at 20°C. Prior to each multiple sprint test, subjects performed a standardised warm-up (approximately five-minutes) comprising 400 m of jogging (self-selected pace), a series of sprint drills (high-knees, heel-flicks, and walking lunges), and three practice sprints. Following the warm-up, subjects were given five minutes to stretch and prepare themselves for the test. Each sprint was initiated from a line 30 cm behind the start line (to prevent false triggering of the first timing gate) and times were recorded electronically via twin-beam photocells (Swift Performance Equipment, Lismore, Australia) placed at 10 m intervals. Alternate sprints were performed in the opposite direction thereby enabling subjects to maximise the available recovery time between sprints. Computer-generated audio signals provided a 5 s countdown to the start of each sprint and subjects were verbally encouraged to give maximal effort. The reliability of this multiple sprint protocol has recently been established (18). One minute before the start of each test and immediately afterwards, capillary blood samples were drawn from a hyperaemised earlobe for the analysis of blood lactate via a hand-held analyser (Lactate Pro: Arkray Inc., Kyoto, Japan). The reliability and validity of this equipment has

previously been reported (28, 31). The analyser was calibrated prior to each trial in accordance with the manufacturer's instructions. Heart rates were monitored continuously during each test using heart rate monitors (Polar S610: Polar Electro Oy, Kempele, Finland). Heart rate transmissions were synchronised with the audio signals to provide direct comparisons between trials. Individual ratings of perceived exertion were recorded after every third sprint using a 15-point scale (3). Fatigue during each test was calculated from 30 m sprint times using the percentage decrement calculation (15) as recommended by Glaister *et al*. (19):

Percentage decrement calculation

Fatigue = (100 × (total sprint time ÷ ideal sprint time)) – 100

Where:

Total sprint time = sum of sprint times from all sprints.

Ideal time = number of sprints × fastest sprint time.

Statistics

All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS for Windows, SPSS Inc., Chicago, IL). Measures of centrality and spread are presented as means ± standard deviation (SD). A two-way (supplement × sprint number) ANOVA with repeated measures on both factors was used to evaluate the effects of caffeine on multiple sprint performance. Analyses for measures of blood lactate and RPE were similar to those for measures of performance except that the factors of 'supplement' and 'sprint number' were replaced with those of 'trial' (3 levels) and 'time'. The effects of caffeine supplementation on plasma caffeine

(including primary metabolites of paraxanthine, theophyline, and theobromine), heart rate, and summary multiple sprint data (fastest time, mean time, and fatigue) were determined using one-way ANOVA with repeated measures. α was set at 0.05 for all analyses with Bonferroni adjustments for multiple comparisons. The possibility that the effects of caffeine supplementation on performance were influenced by habitual caffeine consumption was investigated by deriving correlations between estimated daily caffeine consumption and caffeine-induced changes (relative to placebo) in summary multiple sprint data. Correlation coefficients were interpreted in accordance with the following scale of magnitudes as devised by Cohen (4): $r < 0.1$ is trivial; $0.1 \leq r < 0.3$ is small; $0.3 \leq r < 0.5$ is moderate; $r \geq 0.5$ is large. The above analyses provided 95% confidence limits for all estimates.

RESULTS

Supplementation

The effects of supplementation on plasma caffeine and primary metabolite concentrations are presented in Table 1. Caffeine supplementation resulted in significant increases in plasma caffeine ($F_{(1.02,\ 20.31)} = 62.34$, $p < 0.001$), paraxanthine ($F_{(2.24,\ 44.80)} = 25.32$, $p < 0.001$), and theophylline ($F_{(2.70,\ 54.05)} = 9.48$, $p < 0.001$) concentrations. In contrast, caffeine supplementation had no significant effect on theobromine concentration ($F_{(1.98,\ 39.52)} = 1.18$, $p = 0.318$). *Post hoc* analyses revealed that significant differences were solely attributable to contrasts involving post-caffeine samples, supporting subject compliance with caffeine abstinence. Mean habitual caffeine consumption of the subjects was 88.4 ± 87.3 mg·d^{-1}. The relationship between habitual caffeine consumption and plasma caffeine response was small ($r = -0.192$, $p = 0.41$).

Multiple sprint performance

The effects of caffeine supplementation on multiple sprint performance are presented in Figure 1, with effects on summary performance measures presented in Table 2, and change scores relative to baseline presented in Table 3. Relative to placebo, caffeine resulted in a 0.06 ± 0.05 s reduction in fastest 30 m sprint time (95% likely range: 0.04 to 0.09 s) and a 1.2 ± 1.7% increase (5.9 ± 2.9% versus 4.6 ± 1.8%) in fatigue (95% likely range: 0.3 to 2.2%), which was reflected in a significant 'supplement' × 'sprint number' interaction ($F_{(5.3,\ 105.9)} = 4.66$, $p = 0.001$). In fact, all but one of the participants showed an improvement in fastest sprint time following caffeine ingestion. Supplementation also revealed evidence of a placebo effect, with mean sprint time of both caffeine and placebo trials being significantly faster than baseline (Table 3). Correlations between habitual caffeine consumption and caffeine-induced changes in fastest sprint time and fatigue (relative to placebo) were $r = –0.13$ ($p = 0.56$) and $r = 0.15$ ($p = 0.51$) respectively. There was however a large positive correlation between the improvement in fastest sprint time and the increase in fatigue ($r = 0.63$, $p = 0.002$).

Blood lactate

There was a significant increase in blood lactate over the course of each multiple sprint trial ($F_{(1,20)} = 265.76$, $p < 0.001$) and caffeine led to a significant 'supplement' ($F_{(2,40)} = 9.21$, $p = 0.001$), and 'supplement' × 'time' interaction ($F_{(2,40)} = 7.54$, $p = 0.002$) (Table 4).

Heart Rate

A significant main effect for trial ($F_{(2,20)} = 5.39$, $p = 0.009$) revealed that mean heart rate was significantly higher in the caffeine trial (174 ± 10 b·min^{-1}) than the placebo trial (170 ± 10 b·min^{-1})

(Figure 2). However, values were not significantly different from baseline (174 ± 10 b·min^{-1}). There was no significant difference in peak heart rate between any of the trials ($F_{(2,20)}$ = 2.94, p = 0.064).

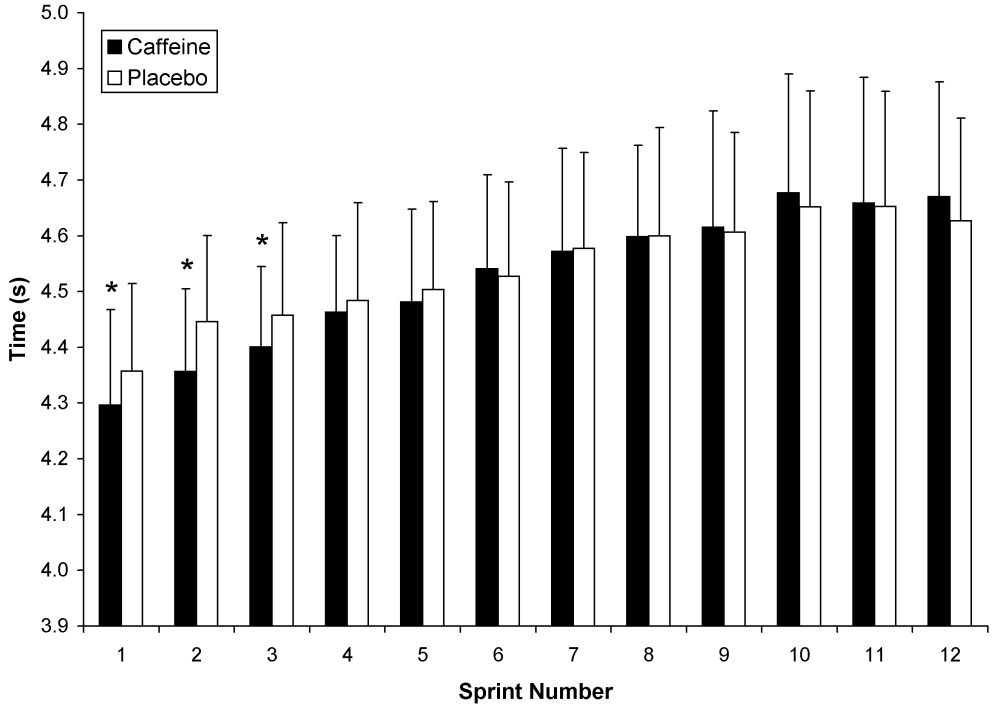

Figure 1. The effects of caffeine supplementation on multiple sprint running performance (12 × 30 m; repeated at 35 s intervals). Values are means; bars are standard deviations. * indicates significantly ($p < 0.05$) different from placebo.

RPE

RPE increased progressively over each trial ($F_{(1.25,25.03)}$ = 123.88, p < 0.001) (Figure 3). However, there was no effect of 'supplement' ($F_{(1.47,29.31)}$ = 0.25, p = 0.713) and no 'supplement' × 'time' interaction ($F_{(6,120)}$ = 0.66, p = 0.685).

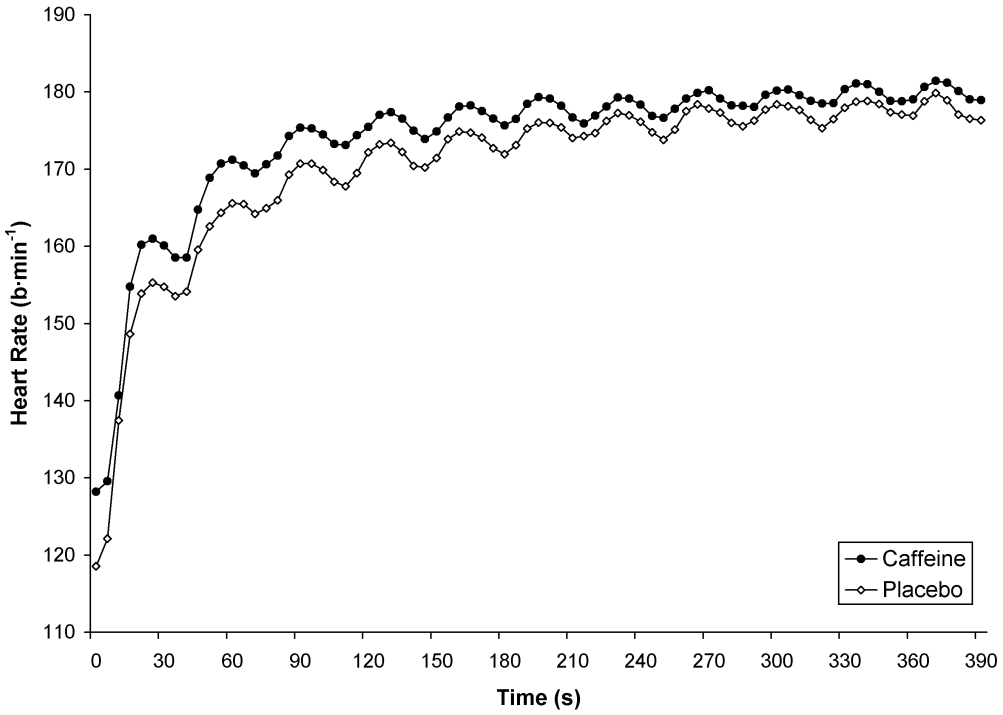

Figure 2. The effects of caffeine supplementation on heart rate during 12 × 30 m sprints; repeated at 35 s intervals. Values are means.

Table 1. Plasma caffeine concentrations before and after caffeine and placebo supplementation. Values are means ± standard deviation.

	Caffeine ($\mu g.ml^{-1}$)	Paraxanthine ($\mu g.ml^{-1}$)	Theophylline ($\mu g.ml^{-1}$)	Theobromine ($\mu g.ml^{-1}$)
Baseline	0.07 ± 0.06	0.06 ± 0.05	0.018 ± 0.009	0.51 ± 0.51
Pre Caffeine	0.18 ± 0.21	0.14 ± 0.19	0.026 ± 0.031	0.79 ± 1.12
Post Caffeine	*5.86 ± 3.32	*0.45 ± 0.26	*0.055 ± 0.034	0.70 ± 1.07
Pre Placebo	0.13 ± 0.22	0.10 ± 0.15	0.023 ± 0.026	0.56 ± 0.86
Post Placebo	0.11 ± 0.16	0.09 ± 0.12	0.024 ± 0.026	0.45 ± 0.70

* indicates significantly ($p < 0.05$) different from corresponding values at various time points

Table 2. The effects of caffeine supplementation on summary multiple sprint performance measures (12 × 30 m; repeated at 35 s intervals). Values are means ± standard deviation.

	Fastest sprint time (s)		Mean sprint time (s)	
Distance (m)	Caffeine	Placebo	Caffeine	Placebo
0 – 10	*1.78 ± 0.08	1.81 ± 0.05	1.89 ± 0.06	1.90 ± 0.06
0 – 20	*3.05 ± 0.12	3.10 ± 0.10	3.24 ± 0.10	3.25 ± 0.11
0 – 30	*4.28 ± 0.16	4.34 ± 0.14	4.53 ± 0.16	4.54 ± 0.16

* indicates significantly ($p < 0.05$) different from placebo.

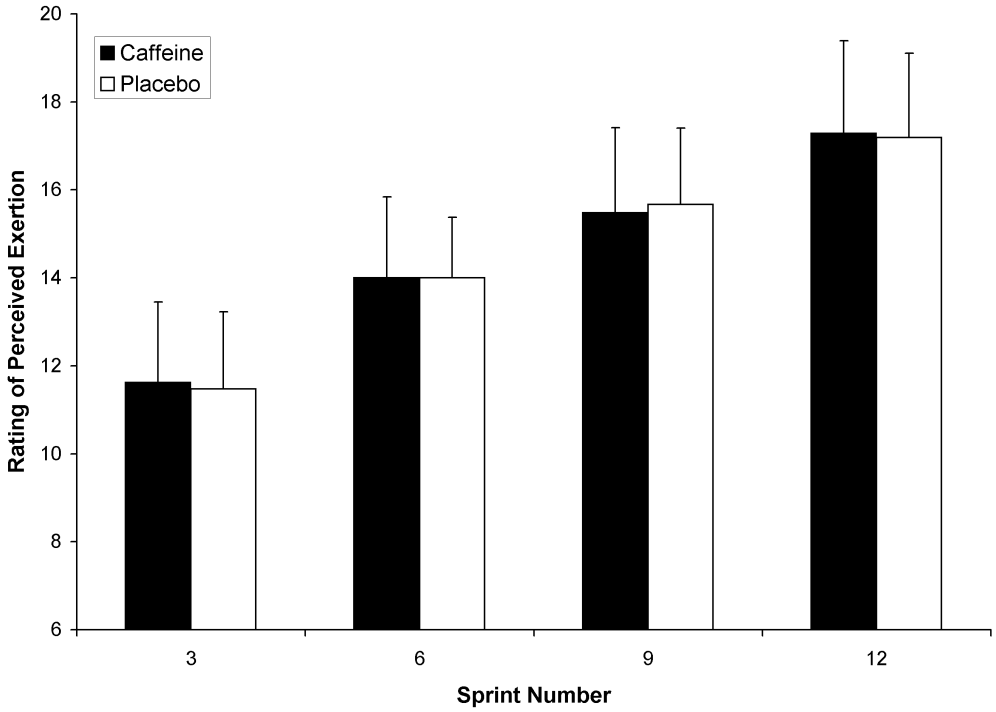

Figure 3. The effects of caffeine supplementation on ratings of perceived exertion during 12 × 30 m sprints; repeated at 35 s intervals. Values are means; bars are standard deviations.

Table 3. The effects of caffeine and placebo ingestion on summary multiple sprint performance measures (12 × 30 m; repeated at 35 s intervals) relative to baseline. Values are means ± standard deviations.

	Fastest sprint time (s)	Mean sprint time (s)	Fatigue (%)
Baseline – caffeine	0.08 ± 0.07	0.06 ± 0.06	–0.7 ± 1.9
95% CL	*0.04 to 0.12*	*0.02 to 0.09*	*–1.8 to 0.5*
Baseline – placebo	0.02 ± 0.07	0.04 ± 0.05	0.6 ± 1.4
95% CL	*–0.02 to 0.06*	*0.01 to 0.07*	*–0.2 to 1.4*

CL = Confidence limits

Table 4. The effects of caffeine supplementation on blood lactate prior to and following 12 × 30 m sprints; repeated at 35 s intervals. Values are means ± standard deviations.

	Blood Lactate (mmol·L^{-1})	
Occasion	Caffeine	Placebo
Pre-test	*3.9 ± 1.2	3.2 ± 1.2
Post-test	*12.9 ± 3.0	11.1 ± 3.0

* indicates significantly ($p < 0.05$) different from placebo.

DISCUSSION

The purpose of this investigation was to examine the effects of caffeine supplementation on a test of multiple sprint running performance designed to evaluate the performance capabilities of athletes involved in field and court sports. The enhancements in sprint performance observed in the initial stages of the test (including fastest sprint) corroborate previous reports of caffeine-induced improvements in short-term bouts of maximal exercise performance (1, 6, 26) and contrast with others reporting no effect (5, 8, 37). However, previous research is potentially flawed by a number of methodological limitations including: a failure to measure resting and pre-exercise caffeine concentrations (1, 5, 6), the use of relatively small sample sizes (generally $\leq$ 14) and the use of extremely intense exercise protocols (8, 23, 30). Indeed, Crowe *et al.* (8) reported that the intense nature of their protocol (2 × 60 s maximal cycle sprints; 3 minute recovery period) could have caused participants to adopt a pacing strategy. Despite the above limitations, most of these investigations showed a trend towards an enhancement of high-intensity performance with caffeine, and none reported a negative effect.

Although caffeine improved sprint performance in the initial stages of the test, the benefits were offset by an overall increase in fatigue. This pattern of response is similar to that observed in previous research. For example, Greer *et al*. (23) observed increases in peak power in the first of four 30 s cycle sprints (four minute rest periods) whilst the reverse was true of the last. Similarly, Paton *et al*. (30) reported a trend towards faster sprints in the early stages of a 10 × 20 m (repeated every 10 s) sprint running test and a corresponding progressive increase in fatigue. However, in both instances, effects were not statistically significant. Once again, problems with participant motivation (due to the extremely intense nature of these protocols) combined with the use of relatively small sample sizes are possible reasons for these discrepancies.

The mechanisms responsible for the caffeine-induced changes in performance observed in the present study are difficult to elucidate; particularly since current research supports a CNS effect mediated by antagonism of adenosine receptors as the most likely cause. However, plasma caffeine concentrations were consistent with those of previous research using similar caffeine doses and were of a magnitude sufficient to promote an effect via adenosine receptor antagonism (16). Whilst theophylline (in particular) and paraxanthine are also potent adenosine receptors antagonists, the concentrations achieved in the circulation as a result of caffeine metabolism are unlikely to have contributed to the effects observed in the present study.

Despite the improvements in performance, caffeine had no effect on RPE during the multiple sprint protocol. In effect, despite the fact that participants sprinted faster in the early stages of the test and showed a greater rate of fatigue, individual perceptions of exertion were the same as those of baseline and placebo. In previous research, caffeine has consistently been shown to alter perceptual responses either by allowing a greater amount of work to be performed at the same perception of effort, or by reducing the perception of effort for the same exercise intensity (11). Although perceptions of effort have been shown to be reliable during short-term (2 minutes) high-intensity (125% VO_{2max}) activity (12), their application in multiple sprint work is yet to be established. Nevertheless, the same suppressive effects of caffeine on perceptions of effort have been observed in a prolonged test (2 × 36 min halves of 18 × 4 s sprints interspersed with active recovery periods) of multiple sprint performance (33). Moreover, the fact that the progressive increases in RPE in the present study corresponded with progressive increases in sprint time and heart rate over the course of the multiple sprint tests suggests that the scale may indeed be valid for this type of activity.

In addition to its effects on performance, caffeine resulted in significant increases in blood lactate. This response has been observed in a number of studies using short-duration maximal-intensity exercise (1, 5, 6, 8, 23), although once again, a lack of rigour in protocol design may account for the observance of a trend towards, rather than a significant increase in some instances (6, 8, 23). Whilst the mechanisms to explain this effect remain unclear, the fact that blood lactate was elevated prior to, as well as following, the multiple sprint protocol adds credence to reports that caffeine-induced elevations in blood lactate are not the result of an increase in lactate efflux from the exercising muscles (21).

The one within-protocol anomaly observed in this investigation was that regarding the non-significant difference in mean heart rate between baseline and caffeine trials despite a significant effect being observed between caffeine and placebo. Whilst increases in heart rate could result from the small (but significant) increase in circulating levels of epinephrine that are reported to accompany caffeine supplementation (20), these two responses do not appear to be synonymous (21). Moreover, whilst caffeine-induced increases in heart rate have been observed in some endurance-based protocols (2, 32), the absence of corresponding baseline trials prevents comparisons with the present study. Overall, whilst the consensus appears to support no effect of caffeine on heart rate during exercise (27), the current findings support the need for further investigation.

Finally, the absence of any significant effects of caffeine habituation on the aforementioned performance responses are in line with previous research using graded exercise performance (10) and low frequency (20 Hz) stimulated contraction force (36), but contrast with others using

submaximal endurance performance (2). However, habitual caffeine consumption of the participants in the present study was considerably less than typical values reported for the general population (> 200 $mg \cdot d^{-1}$) (16) and clearly more research is required to establish the effects of caffeine habituation on subsequent caffeine responses. Nevertheless, in line with previous research (25), there was considerable variability in individual responses to caffeine, with those subjects showing the greatest improvement in fastest sprint time tending to display the greatest decrements in fatigue. In effect, it appears that the effects of caffeine on fatigue were not a direct result of supplementation *per se*, but rather were a consequence of the benefits afforded by caffeine on sprint performance in the early stages of the test. In fact, when recovery periods are lengthened, the benefits of caffeine on multiple sprint performance appear to be extended well beyond the first few sprints (33, 35).

In summary, tests of repeated sprint ability are becoming increasingly popular as a means of evaluating the performance capabilities of athletes involved in multiple sprint sports. Whilst the results of this investigation support a clear ergogenic effect of caffeine in this type of activity, further research is required to establish the mechanisms of this response. Moreover, whilst caffeine had a significant effect on performance in the early stages of the multiple sprint protocol, the precise effect of recovery duration on the time-course of that effect requires further investigation, particularly given the corresponding negative effect on fatigue. Overall, the results of this study suggest that caffeine has ergogenic properties with the potential to benefit performance in both single and multiple sprint sports.

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Conflict of Interest

The authors have no conflicts of interest that are relevant to the content of this article. The results of the present study do not constitute endorsement by ACSM.

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