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Research article

Caffeine Exerts Neither Ergogenic nor Hypoalgesic Effects on Sprint Interval Exercise with Intensive Exercise-Induced Muscle Pain

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Abstract
Sprint interval exercise can cause transient, intense exercise-induced pain (EIP) during and several minutes after the activity. A hypoalgesic strategy for high-intensity exercise, such as sprint interval exercise, against EIP is necessary to maintain exercise habituation and improve training quality/exercise performance. Preexercise caffeine supplementation, a well-known ergogenic strategy, may improve sprint performance and alleviate EIP as the hypoalgesic strategy. However, whether preexercise caffeine supplementation exhibits both the ergogenic effect on sprint interval performance and the hypoalgesic effect on intensive EIP during and several minutes after high intensity sprint interval exercise remains unknown, and thus we investigated to clarify those points. In this double-blind, randomized, crossover trial, sixteen male collegiate athletes performed 3 sets of 30-sec all-out Wingate pedaling exercises at 2-min intervals. Participants ingested 6 $mg \cdot kg^{-1}$ caffeine or placebo via capsules at 60 min prior to exercise. Quadriceps EIP was measured using a visual analogue scale during and up to 20 min after exercise. The results showed that caffeine did not significantly affect peak or mean power during sprint interval exercise (peak power: $P = 0.196$, $\eta_p^2 = 0.11$, mean power: $P = 0.157$, $\eta_p^2 = 0.13$; interaction). No significant interactions were also found for quadriceps EIP during ($P = 0.686$, $\eta_p^2 = 0.03$) and immediately after exercise ($P = 0.112$, $\eta_p^2 = 0.12$), nor for changes in physiological responses (blood lactate and ammonia concentrations) and caffeine-induced side effects (all $P > 0.05$). In conclusion, caffeine had no ergogenic or hypoalgesic effects on sprint interval exercise with intensive EIP.

Key words: Exercise-induced pain, EIP, ergogenic aid, repeated sprint.

Introduction

Exercise causes acute pain during or several minutes after exercise – this pain is known as exercise-induced pain (EIP; Cook et al., 1997; Dannecker and Koltyn, 2014). The EIP is different from delayed-onset muscle soreness (DOMS), which occurs approximately 24 - 72 h after exercise (Cheung et al., 2003). Runners and cyclists at the recreational to elite level often experience EIP (Kress and Statler, 2007; McCormick et al., 2018). In the scientific literature, EIP has primarily been discussed in the context of resistance exercise or endurance cycling (Dannecker and Koltyn, 2014; Stevens et al., 2018). However, several previous studies reported that EIP is caused by sprint interval exercise (Foster et al., 2014; Kizzi et al., 2016; Monks et al., 2017; Wender et al., 2019). Sprint interval training is widely applied to improve aerobic/anaerobic performance and promote health due to its impact on musculoskeletal and cardiovascular adaptations for athletes (Almquist et al., 2021; Kim et al., 2011) and general populations (Sloth et al., 2013; Whyte et al., 2010). In addition, intensive EIP, nearly approaching "worst pain ever" in the pain scale, has been experimentally and/or empirically shown during and even several minutes after sprint exercise (specifically sprint interval exercise)(Aono et al., 2013), which can be naturally assumed given that this modality of exercise is performed with all-out effort (Almquist et al., 2021; Kim et al., 2011; Sloth et al., 2013; Whyte et al., 2010) and the magnitude of EIP increases with the exercise intensity (Cook et al., 1997). Intensive EIP has a negative affective component (Venhorst et al., 2018) and impairs muscle activation/strength needed to produce higher exercise performance (Norbury et al., 2022). Furthermore, increasing pain associated with exercise can be a barrier to exercise adherence (Jack et al., 2010), suggesting that intensive EIP may potentially lower adherence to sprint interval training. To date, establishing hypoalgesic strategies for high-intensity exercise remains a challenge that needs to be addressed to maintain exercise habituation and improve training quality/exercise performance (Wender et al., 2023).

One such proposed hypoalgesic strategy is the widely consumed ergogenic aid, caffeine (1, 3, 7-trimethylxanthine). Caffeine has been observed to acutely improve performance in both single- and multiple-sprint exercise (Grgic, 2018; Kizzi et al., 2016; Matsumura et al., 2023b; Schneiker et al., 2006). Interestingly, caffeine has been documented to decrease EIP during endurance pedaling exercise (Gliottoni and Motl, 2008; Gliottoni et al., 2009; Motl et al., 2003; 2006; O'Connor et al., 2004) and resistance exercise (Astorino et al., 2011; Duncan et al., 2013; Souza et al., 2019). Although the mechanism of the hypoalgesic effect of caffeine related to exercise is not fully understood, the effect is concepted to be based on adenosine receptor antagonism. Adenosine has been documented as the cause of pain, including muscle pain, by examining with adenosine infusion (Sawynok, 1998). Specifically, activation of the adenosine A_2 receptor causes an increase in pain, but caffeine is expected to reduce muscle pain, including EIP, by non-selectively blocking the adenosine receptor (Davis and Green, 2009; Sawynok, 1998).

On the other hand, few studies have paid attention to the hypoalgesic effect of caffeine on sprint interval exercise, despite the potential concern that higher levels of EIP may occur when conducting this modality of exercise. Kizzi et al. (2016) examined the effect of caffeine on pain

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perception during sprint interval exercise. The observed results led the researchers to conclude that caffeine may reduce pain during sprint interval exercise and improve sprint performance. However, this previous finding did not reflect normal training/competition because they demonstrated the effect of caffeine under glycogen depleted conditions, which attenuated exercise performance. Basically, despite increased exercise performance, higher exercise intensity is documented to lead to higher levels of EIP (Cook et al., 1997). Thus, the improvement in sprint performance may counteract the hypoalgesic effect of caffeine on EIP during or immediately after sprint interval exercise. Overall, it remains unclear whether caffeine confers both ergogenic and hypoalgesic effects even in the face of intensive EIP caused by high-performance sprint interval exercise. Clearing this viewpoint may provide more robust evidence for the hypoalgesic effects of caffeine on EIP with sprint interval exercise. Therefore, the purpose of this study was to investigate the effect of caffeine supplementation on athletic performance in sprint interval exercise and EIP during and several minutes after sprint interval exercise. We hypothesized that caffeine would improve sprint performance as well as reduce EIP both during and after sprint interval exercise.

Methods

Participants

To accurately describe localized muscle pain associated with high-intensity exercise, sixteen well-trained male collegiate athletes who often experience EIP during training and competition (sixteen long sprinters; mean ± SD; age: 20 ± 1 years, height: 175.5 ± 3.9 cm, weight: 66.0 ± 4.3 kg, habitual caffeine intake: 226 ± 168 mg per day) were recruited for this study. Habitual caffeine intake was estimated using a questionnaire based on a previous study (Bühler et al., 2014), and caffeine content was recorded using product websites and a list of typical caffeine content in beverages/foods (Paluska, 2003). All athletes were eligible for the following inclusion criteria: at least 2 h per day and 5 days per week of training habituation (including resistance training), no traumatic injury that would make it difficult to perform sprint exercise, no psychological or cardiovascular disease, no smoking status, and no allergy to caffeine or the prescribed meal. This study adhered to the tenets of the *Declaration of Helsinki* and was approved by the Ethics Committee for Human Experiments at Ritsumeikan University (BKC-LSMH-2023-081). Written informed consent was obtained from all participants.

Experimental design

This study used a randomized, double-blind, counterbalanced, crossover design. Participants ingested 6 mg·kg^{-1} anhydrous caffeine (Pure Caffeine; Myprotein, Manchester, UK) or a placebo (maltitol; Placeplus; Placebo Seiyaku, Shiga, Japan) via the same gelatin capsules (HF Capsules #0, Matsuya, Osaka, Japan) at 60 min before exercise in each caffeine/placebo condition. This dose of caffeine and timing is commonly utilized as an ergogenic aid (Guest et al., 2021). An independent researcher randomly assigned the order of the conditions, and blinding was concealed until all data were acquired. Participants performed sprint interval exercise in the laboratory at a controlled room temperature (22.2 ± 1.1 °C) and reported EIP during and up to 20 min after exercise (see "Quadriceps pain" in Figure 1).

Participants were instructed not to ingest caffeine or alcohol from 12 AM on the day before the experimental day and to fast from 11 PM on the day before the trials (Matsumura et al., 2023a; 2023b). Furthermore, participants took pictures of all meals on the day before the trials to ensure that the meal content remained consistent across the two conditions. These experiments were performed at the same time for participants from 8 AM to 11 AM with at least a 72-h interval as a washout period and were programmed taking into account participants' daily training cycle to ensure as much consistency as possible between conditions. We did not conduct the familiarization session in this study because participants were already familiar with the exercise modalities used in this study. Notably, there was no effect of trial order on exercise performance or physiological/psychological responses between the first and second experiments (all $P > 0.05$).

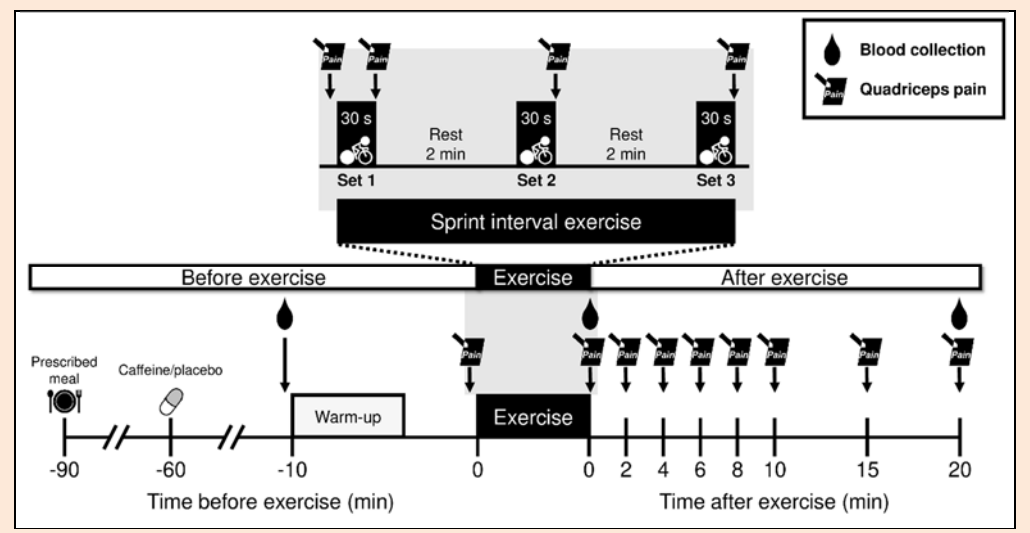

Figure 1. Experimental procedures.

Experimental procedures and data acquisition

The experimental procedure under one set of conditions is shown in Figure 1. Participants visited the laboratory 90 min before exercise, after which they consumed a prescribed meal consisting of a jelly and a nutrition bar (a total of 380 kcal), and rested until they started warm-up. During this rest period, participants ingested caffeine or placebo capsule 60 min before exercise. The saddle height of the ergometer was adjusted to the height at which the participants could comfortably pedal fully before warm-up in the first experiment. The height was recorded and kept consistent between the two conditions. Participants started the warm-up for 10 min before exercise. The warm-up exercises were standardized: 5 min of cycling with 1.0 kp of load at 100 rpm and 5 sec of all-out sprint cycling with 7.5% body weight of load. The rest between the warm-up and sprint interval exercise was set at 3 min.

For the sprint interval exercise, participants performed 3 sets of 30-sec sprint exercises separated by 2 min of rest between sets using a cycle ergometer (Power Max VIII; Konami, Tokyo, Japan). The exercise load was set at 7.5% of body weight (Ferragut et al., 2024; Bar-Or, 1987) and standardized between the two conditions. Participants were verbally encouraged during all sets of sprint exercises to maximize their sprint performance. Peak power and mean power were obtained at each sprint set. During the rest period between sets, participants rested passively on a bed next to the ergometer.

EIP was assessed as perceived pain in the quadriceps, which are the primary muscles involved in cycling exercise (Ericson et al., 1986), using the 100-mm visual analogue scale (VAS). Participants marked the magnitude of EIP on the VAS, with 0 and 100 mm representing "no pain" and "the worst pain ever", respectively. Participants were fully instructed prior to the exercise on how to rate their perceived pain using the VAS. For the time point of EIP during the exercise phase, participants evaluated EIP before sprints (Pre) and immediately after each set. For the postexercise assessment (i.e., the recovery phase) of EIP, after Set 3 of the sprint interval exercise, participants recorded their level of quadriceps pain every 2 min until 10 min postexercise, as well as at 15 and 20 min postexercise.

Blood samples were collected from the fingertip before warm-up (preexercise) and immediately (0 min postexercise) and 20 min after exercise (20 min postexercise) to determine blood lactate and ammonia concentrations. Both blood ammonia and lactate concentrations were measured immediately using a blood ammonia meter (PocketChem BA PA-4140; Arkray, Kyoto, Japan) and a lactate analyzer (Lactate Pro 2; Arkray, Kyoto, Japan), respectively.

Participants reported their perception of caffeine-induced side effects 20 min and 24 h after exercise. Participants were asked yes/no questions with their perception based on a previous study (Pallarés et al., 2013) on the following content: muscle soreness (24 h only), increased urine output (volume and/or frequency), tachycardia and heart palpitations (compared to the same situation in usual daily life), anxiety or nervousness, headache, gastrointestinal problems, insomnia (24 h only), increased vigor/activeness, and perception of performance improvement (20 min only). We excluded "muscle soreness", which was the original content of the previous study (Pallarés et al., 2013), 20 min after exercise because participants reported EIP using the VAS at the same time. Participants were also asked to confirm the effectiveness of blinding before warm-up and 20 min after exercise by indicating the belief which supplement (caffeine, placebo, or uncertain) they had taken in each trial (Saunders et al., 2017).

Statistical analyses

All data are expressed as the mean ± SD (if normally distributed) or the median (IQR) (if not normally distributed) after the Shapiro–Wilk test. The significance level was set at $P < 0.05$. All statistical analyses were performed using IBM SPSS software (version 29; Armonk, NY, US).

The sample size estimated with G*Power (Faul et al., 2007) showed a statistical power of 0.80 with 16 participants, based on the results of sprint performance or perceived pain in Kizzi et al. (2016).

Changes in sprint performance, perceived pain during exercise or recovery phases, and physiological responses (blood lactate and ammonia concentrations) were assessed using two-way (condition × time) repeated measures analysis of variance (ANOVA). If a significant interaction was detected, specific differences were identified by *post hoc* comparisons using a paired *t* test or the Wilcoxon signed-rank test (with a Bonferroni correction where necessary). We applied aligned rank transforms (Wobbrock et al., 2011) to compare the change in perceived pain during exercise or recovery phases using ANOVA because these data showed nonnormally distributions. The decreases in peak and mean power from Set 1 to 3 were compared between using paired *t* test. Peak pain during exercise (from Pre to Set 3) and recovery phases (from 0 to 20 min postexercise) was also compared between conditions using the Wilcoxon signed-rank test. In addition, the effect size and 95% confidence interval (CI) were calculated using the pooled SD or *Z* score to determine the magnitude of the difference in sprint performance and EIP between conditions, respectively. Hedge's *g* effect size was interpreted as very small ($g < 0.2$), small ($0.2 \leq g < 0.5$), medium ($0.5 \leq g < 0.8$), or large ($0.8 \leq g$; Sawilowsky, 2009), and the *r* effect size was interpreted as small ($0.1 \leq r < 0.3$), medium ($0.3 \leq r < 0.5$), or large ($0.5 \leq r$; Cohen, 1992). The incidence of side effects from caffeine ingestion and the number of participants who correctly identified the conditions were compared between conditions at each time point with McNemar's test. Bang's Blinding Index (Bang et al., 2004) was also calculated to evaluate the effectiveness of blinding at each condition before and 20 min after exercise. This index shows values ranging from -1 (completely opposite guessing) to 1 (completely correct guessing), and 0 means an ideal blinding. As shown in the Experimental design, the trial order effects were confirmed by comparing the above results between the first and second experiments using the same statistical methods used for comparisons between conditions.

Results

All included participants ($n = 16$) completed two condi-

tions of sprint interval exercise. However, blood lactate concentration data for one participant and blood ammonia concentration data for four participants were not available due to kit error.

Sprint performance

There were significant main effects of time on both peak power ($P < 0.001$, $\eta_p^2 = 0.84$; Figure 2A) and mean power ($P < 0.001$, $\eta_p^2 = 0.91$; Figure 2B). No significant main effect of condition (peak power: $P = 0.296$, $\eta_p^2 = 0.07$; mean power: $P = 0.627$, $\eta_p^2 = 0.02$) or interaction between time and condition (peak power: $P = 0.196$, $\eta_p^2 = 0.11$; mean power: $P = 0.157$, $\eta_p^2 = 0.13$) on peak power (Figure 2A) or mean power (Figure 2B) was found. The decreases in peak power (placebo: -184 ± 99 W, caffeine: -213 ± 87 W; $P = 0.171$, $g = -0.29$ [95% CI: -0.74 - 0.15]; Figure 2C) and mean power (placebo: -166 ± 58 W, caffeine: -184 ± 63 W; $P = 0.143$, $g = -0.28$ [95% CI: -0.69 - 0.12]; Figure 2D) from Set 1 to Set 3 did not change between conditions.

EIP

During exercise, a significant main effect of time was observed in EIP ($P < 0.01$, $\eta_p^2 = 0.87$), whereas neither significant main effect of condition ($P = 0.383$, $\eta_p^2 = 0.05$) nor interaction ($P = 0.686$, $\eta_p^2 = 0.03$) were identified (Figure 3). Peak pain during exercise did not change significantly between conditions (placebo: 94 [76–99] mm, caffeine: 88 [81-97] mm; $P = 0.394$, $r = -0.21$).

Although a significant main effect of time ($P < 0.001$, $\eta_p^2 = 0.87$) was identified in EIP at the recovery phase, there were no significant main effect of condition ($P = 0.105$, $\eta_p^2 = 0.17$) and interaction ($P = 0.112$, $\eta_p^2 = 0.12$; Figure 4). No difference in peak pain was observed between conditions (placebo: 100 [91-100] mm, caffeine: 99 [90-100] mm; $P = 0.767$, $r = -0.07$).

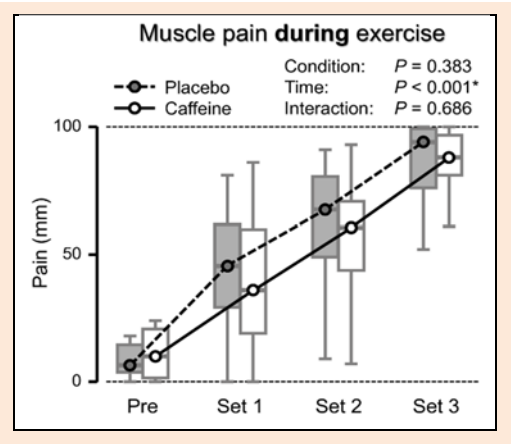

Figure 3. The effects of caffeine on exercise-induced pain in the quadriceps during exercise. Asterisk indicates statistical significance by two-way ANOVA. The values are presented as the medians (IQRs) ($n = 16$).

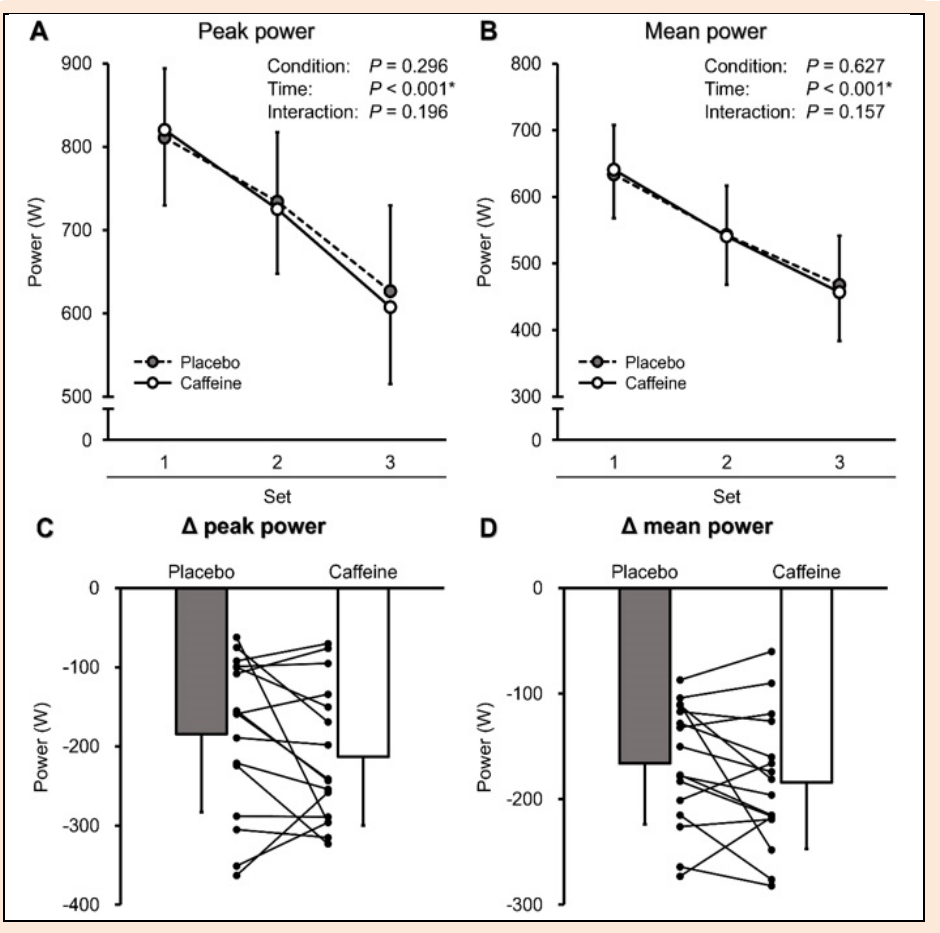

Figure 2. The effects of caffeine on peak power (A) and mean power (B) during the sprint interval exercise and the changes in peak power (C) and mean power (D) from Set 1 to Set 3. Asterisk indicates statistical significance by two-way ANOVA. The values are presented as the means ± SDs ($n = 16$) for each condition. Each participant's result is presented in the plot (black circles) in Figure 2C and D.

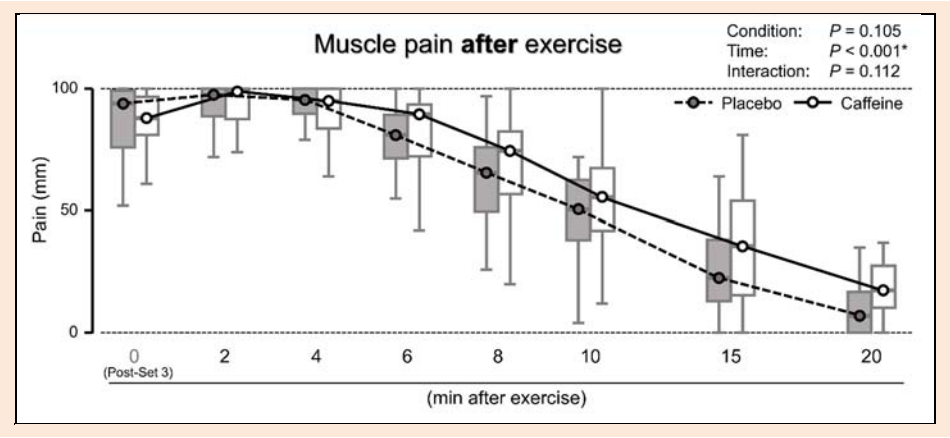

Figure 4. The effects of caffeine on exercise-induced pain in the quadriceps after exercise. Asterisk indicates statistical significance by two-way ANOVA. The values are presented as the medians (IQRs) (n = 16).

Table 1. Blood lactate (n = 15) and ammonia (n = 12) concentrations under the placebo and caffeine conditions before (Preexercise) and immediately (0 min) or 20 min after exercise (Postexercise).

	Preexercise	Postexercise	
		0 min	20 min
	Lactate (mmol·L^{-1})		
Placebo	2.2 ± 0.4	18.2 ± 3.4	14.5 ± 3.0
Caffeine	2.3 ± 0.5	18.2 ± 2.9	14.5 ± 2.5
	Ammonia (μg·dL^{-1})		
Placebo	78 ± 39	228 ± 55	182 ± 46
Caffeine	62 ± 28	232 ± 57	160 ± 57

The values are presented as the means ± SDs.

Physiological responses

A significant main effect for time was shown for both blood lactate (η_p^2 = 0.97) and ammonia (η_p^2 = 0.87) concentrations (both P < 0.001). However, there were no significant main effects of condition (lactate: P = 1.000, η_p^2 = 0.00; ammonia: P = 0.293, η_p^2 = 0.10) or interaction (lactate: P = 0.970, η_p^2 = 0.001; ammonia: P = 0.274, η_p^2 = 0.11) on either blood lactate or ammonia concentrations (Table 1).

Side effects

There was no significant difference in the prevalence of any side effects at 20 min or 24 h after exercise (all P > 0.05, Table 2).

Effectiveness of blinding

Bang's Blinding Index were -0.13 for the placebo condition and -0.06 for the caffeine condition before exercise as well as 0.31 for the placebo condition and 0.19 for the caffeine condition after exercise. The number of participants who correctly recognized the condition did not change significantly between conditions either before (Placebo: 19%, Caffeine: 25%; P = 1.000) or after exercise (Placebo: 44%, Caffeine: 31%; P = 0.687).

Table 2. The percentage of participants who reported side effects 20 min and 24 h after exercise (n = 16).

	Placebo		Caffeine		P value	
	20 min	24 h	20 min	24 h	20 min	24 h
Muscle soreness	–	13	–	6	–	1.000
Increased urine output	25	6	31	13	1.000	1.000
Tachycardia and heart palpitations	25	0	19	0	1.000	1.000
Anxiety or nervousness	13	0	13	0	1.000	1.000
Headache	19	6	19	0	1.000	1.000
Gastrointestinal problems	6	0	0	6	1.000	1.000
Insomnia	–	6	–	0	–	1.000
Increased vigor/activeness	13	6	38	6	0.125	1.000
Perception of performance improvement	25	–	25	–	1.000	–

P values show the results of comparisons between conditions using McNemar's test for each item at each time point.

Discussion

This study aimed to examine the ergogenic and hypoalgesic effects of caffeine supplementation associated with all-out sprint interval exercise with intensive EIP. The results showed neither ergogenic nor hypoalgesic effects of caffeine on sprint interval exercise. This study is the first to show the effect of caffeine on EIP not only during but also after exercise. It is important to note that there were no significant differences in the effectiveness of blinding between conditions or in any of the indices between the first and second experiments. Therefore, the results of this study would have less experimental concern for psychological confounding, such as the effectiveness of blinding and/or trial order effect (e.g., psychological barrier for the all-out sprint and familiarization for EIP), when performing sprint interval exercises and reporting EIP.

In the present study, intensive EIP was observed at the end of the exercise and a few minutes after exercise, regardless of caffeine ingestion. In contrast to our present study, several previous studies have demonstrated the hypoalgesic effect of caffeine during exercise. Specifically, Kizzi et al. (2016) indicated that 6 mg·kg^{-1} caffeine decreased EIP during sprint interval exercise. However, this

previous study revealed the hypoalgesic effect of caffeine during sprint interval exercise under glycogen depletion, which also decreased sprint performance compared to that under adequate glycogen condition (Kizzi et al., 2016). In endurance cycling, caffeine also alleviates EIP only during moderate-intensity exercise but not during high-intensity exercise (Black et al., 2015). Therefore, caffeine would decrease relatively mild EIP when the relatively lower power output was exerted (e.g., glycogen depletion) but not when the higher power output with intensive EIP was performed during sprint exercise, which might dampen the physiological relevance of caffeine on EIP.

Neither peak nor mean power during exercise changed between conditions, suggesting that caffeine did not act as an ergogenic aid in this study. However, this result contrasts with a previous meta-analysis (Grgic, 2018), which reported that caffeine improved performance during a 30-sec single Wingate exercise, thus indicating that higher sprint performance should be observed at least during the first set in the present study. A recent study reported that caffeine increased peak power only during the first session of four 30-sec sprint intervals, although the difference was not significant (Ferragut et al., 2024). On the other hand, in contrast to previous findings showing ergogenic effects of caffeine on sprint interval performance (Kizzi et al., 2016; Schneiker et al., 2006), caffeine has been shown to decrease sprint performance in the final set of four 30-sec sprints (Greer et al., 1998). Lee et al. (2012) suggested that the ergogenic effect of caffeine on repeated sprinting may be dependent on the length of the rest period, i.e., caffeine might be more effective with a longer rest period. This might be because of peripheral factors to cause a reduction in sprint performance rather than the action of caffeine. For example, the simultaneous increase in protons, ATP, and lactate in muscle would synergistically induce pain sensation (Pollak et al., 2014), as would other pronociceptive factors besides adenosine. The ergogenic effect of caffeine may be revealed if the interval between sprints is long enough to recover from the exhaustion induced by peripheral factors that cause a decline in sprint performance. Specifically, a higher resting ratio may be required, such as 1:12.5 (for the longer interval condition in Lee et al. 2012), rather than 1:4 (in the present study) or 1:8 (in Greer et al. [1998]) exercise:rest ratios. Furthermore, no changes were detected in EIP during exercise (Figure 3) or in the prevalence of caffeine-induced side effects (Table 2). This implies that these potential adverse effects of caffeine per se do not appear to substantially offset its potential ergogenic effects. Overall, more considerations are warranted to clarify the ergogenic effects of caffeine on sprint interval exercise.

Notably, caffeine did not significantly alter either peak or mean power during exercise (Figure 2), nor did it alter EIP during (Figure 3) and immediately after exercise (Figure 4), contrary to the expected hypoalgesic effect. If caffeine had a hypoalgesic effect, one would expect to see lower EIP at the same level of athletic performance (Gliottoni and Motl, 2008; Gliottoni et al., 2009; Motl et al., 2003; 2006) or the same level of EIP at a higher level of athletic performance (Astorino et al., 2011), considering the association between exercise intensity and EIP (Cook et al., 1997). In this regard, our findings that showed no changes in sprint performance and perceived pain during/after exercise in response to caffeine ingestion suggest that the ergogenic and hypoalgesic effects of preexercise caffeine ingestion on sprint interval exercise are not compatible.

One might argue that the participants in the present study did not experience the effects of caffeine because they consumed caffeine habitually (226 ± 168 mg per day). However, the recent meta-analysis revealed that habitual caffeine intake was not related to the ergogenic effects of caffeine (Carvalho et al., 2022). In fact, the previous study in sprinters of similar status, including caffeine habituation (205 ± 162 mg per day), resulted in an improvement in sprint performance independent of their daily caffeine habituation with 6 $mg \cdot kg^{-1}$ caffeine supplementation (Matsumura et al., 2023b). Therefore, habitual caffeine intake would not be responsible for the results of the present study.

As a limitation of this study, the localized muscle properties in the quadriceps (e.g., pH, proton, or lactate concentration) that may relate to EIP were not examined. Notably, both blood lactate and ammonia concentrations did not change between conditions (Table 1). At the very least, these results are consistent with the lack of change in sprint performance and EIP between conditions, but further clarification is needed. Another limitation is that we did not measure the plasma caffeine concentration. However, the plasma caffeine concentration is apparently elevated after 6 $mg \cdot kg^{-1}$ of capsulated caffeine (Matsumura et al., 2023a), using the same protocol as in the present study, and typically reaches a peak at 60 min and is maintained for several hours after caffeine ingestion (Guest et al., 2021). Thus, it is reasonable to assume that caffeine would circulate sufficiently throughout the exercise and postexercise recovery periods until 20 min after exercise. Finally, it is unclear whether the results of the present study are due to the genetic factors that may influence the ergogenic effects of caffeine (Guest et al., 2021).

Conclusion

Caffeine did not improve sprint performance during sprint interval exercise with intensive EIP. In addition, caffeine did not alleviate EIP during and immediately after sprint interval exercise. These results suggest that caffeine does not exert ergogenic or hypoalgesic effects in the context of intensive EIP induced by high-performance sprint interval exercise.

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Key points

- Caffeine supplementation does not improve sprint interval exercise consisting of 30-sec all-out Wingate pedaling exercises at 2-min intervals.
- Caffeine also does not relieve the exercise-induced pain during and immediately after the sprint interval exercise.
- Caffeine would not be compatible for both ergogenic and hypoalgesic effects in sprint interval exercise with intensive EIP.

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