

Original Research

The Effects of Acute Caffeine Supplementation on Repeated-Sprint Ability in Healthy Young Non-Athletes

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ABSTRACT

International Journal of Exercise Science 15(2): 846-860, 2022. The ergogenic effects of caffeine supplementation on repeated-sprint ability (RSA) have produced equivocal results. This study aimed to examine the effects of 200 mg of caffeine during repeated-sprint running on heart rate (HR), rating of perceived exertion (RPE), blood lactate (BLa) concentration, and sprint time (ST). Thirty-two individuals (males: *n* = 17, females: *n* = 15; age: 22 ± 1 years) participated in the study. The study followed a double-blind, randomized, placebo-controlled, crossover design, in which each participant ingested 200 mg of caffeine or placebo on separate visits 60 minutes prior to repeated-sprinting exercise. The repeated-sprint protocol consisted of three sets of six maximal-effort 30 meter sprints with 20 seconds and 5 minutes of active recovery in between sprints and sets, respectively. During each set, HR, RPE, BLa, and ST were recorded. Caffeine supplementation did not significantly (set 1: *p* = 0.535; set 2: $p = 0.602$; set 3: $p = 0.189$) impact HR during exercise. Similarly, RPE was not statistically ($p = 0.052$) altered between conditions during any of the sprint sets. The caffeine trials elicited greater BLa values after all three sets compared to the placebo trials (*p* < 0.001). Moreover, the caffeine trials demonstrated significantly reduced total STs during all sets compared to the placebo trials (*p* < 0.001). Thus, our findings suggested that 200 mg of caffeine supplementation elicited an increase in RSA in young, healthy non-athletes. These findings are accompanied by a blunted perceived exertion relative to an increase in exercise intensity during repeated-sprint exercise.

KEY WORDS: Repeated-sprint ability, running, anaerobic, dietary supplements, ergogenic aids

INTRODUCTION

Caffeine is one of the most widely used substances in the world, commonly ingested in coffee, tea, soda, energy drinks, and even over-the-counter medications due to its benefits of increasing mental and physical capabilities (5, 20). In the United States, 85% of the population consumes at least one caffeinated beverage a day, and the 90th percentile intake is 380 mg/day for all age groups combined (49). Additionally, many individuals ranging from recreational to elite athletic

populations supplement caffeine as an ergogenic aid to enhance exercise performance (26, 33, 40, 42). Around 70% of young adults use at least one nutritional supplement, and the most popular supplements include caffeinated energy drinks and caffeine pills (28, 36) making it a compelling area of research.

The term, "repeated-sprint ability" (RSA), has been proposed by sports science professionals to play an important factor in sport performance (59). Repeated-sprints is a form of high-intensity interval training (HIIT) that can produce physiological benefits through training (e.g., improvements in maximal oxygen consumption; production, resynthesis, and utilization of adenosine triphosphate; resynthesis of phosphocreatine; peak power output; exercise tolerance; fat oxidation; resting glycogen content; and vascular structure and function and reductions in glycogen diminishment rate and lactate accumulation) (30, 44). The combination of this mode of exercise training and the ergogenic effect of caffeine supplementation has the potential to augment exercise performance. Previous investigations have revealed that caffeine possesses the ability to enhance mental alertness and concentration and reduce fatigue (25, 46, 48). Moreover, caffeine has been demonstrated to improve exercise performance during prolonged and exhaustive bouts of exercise (11, 18, 19, 39). The proposed mechanisms that underlie the ergogenic effects of caffeine include blunted pain perception and perceived exertion via adenosine inhibition (21, 24, 51, 57), enhanced anaerobic metabolism consequent of increased epinephrine secretion (7, 16, 34), augmented motor unit recruitment (6, 9), improvements in muscle function through adjustments in K^+ and Ca^{2+} kinetics (2, 50), and/or elevated blood flow and muscle oxygenation through activation of endothelial nitric oxide synthase (56, 65). Although the prospective effects of caffeine on exercise performance have been extensively explored, most of the research focused primarily on endurance exercises and the evidence for caffeine supplementation on short-term high-intensity bouts of exercise (i.e., repeated-sprints) remains controversial (20).

There is conflicting evidence that acute caffeine supplementation elicits ergogenic effects on RSA with some studies demonstrating an improvement (13, 22, 31, 32, 45, 54, 55, 58, 61) and others demonstrating a negligible effect (4, 15, 27, 34, 53). Moreover, few studies pinpointed the effect of caffeine on repeated-sprint running performance. Given the widespread use of caffeine and the increasing involvement in recreational team sports and HIIT, further research is needed to determine the efficacy of caffeine on repeated-sprint running performance. In addition, most of the studies investigating the effects of caffeine on RSA focused on trained athletes and used a relative dosage. Many recreationally-trained individuals use supplements with an absolute dosage of caffeine (e.g., pre-workout, caffeinated energy drinks, and caffeine pills) as an ergogenic aid; therefore, the effect of an absolute dosage on the general population should be explored further. The purpose of this study is to establish the impact of 200 mg of caffeine on repeated-sprint running performance in young, healthy non-athletes. We hypothesized that 200 mg of caffeine would result in a significant ergogenic effect during repeated-sprints characterized by a reduction in total sprint time (ST). Additionally, we hypothesized that blood

lactate (BLa) concentrations would be elevated, and rating of perceived exertion (RPE) would be lowered during all three sprint sets in the caffeine trials compared to placebo.

METHODS

Participants

Thirty-five participants were recruited to participate in the study through verbal proposal to Kinesiology and Physical education classes at Northern Illinois University (DeKalb, IL) and flyers posted in various locations throughout the university. However, thirty-two individuals completed the study. Descriptive statistics for all subjects are provided in Table 1. The population recruited were men and women ranging from 18-30 years of age. Exclusion criteria included cardiovascular, respiratory, or neuromuscular limitations to exercise; hypertension (resting systolic arterial blood pressure (BP) >140/90 mmHg); bloodborne diseases; usage of prescription medications; or status as a collegiate-, professional-, or elite-level athlete. Sample size was estimated to detect a significant decrease in ST based on previous human studies examining the effects of caffeine supplementation on sprint performance (13, 31). This calculation indicated that at least 18 subjects were required to detect a statistically significant decrease in ST with caffeine supplementation (two sided α of 5%, power of 0.8). All procedures were approved by Northern Illinois University's Institutional Review Board and were compliant with the ethical standards of the International Journal of Exercise Science (52).

Table 1. Participant characteristics.

When noted, values are means ± SE.

Protocol

The participants reported to the laboratory on three different occasions within a 3–4-week period: one assessment visit and two experimental visits (Figure 1). Upon arrival to the assessment visit, participants underwent a comprehensive screening procedure that included informed consent, inclusion criteria, health history, caffeine usage, and blood health. Caffeine usage was screened by participants completing a survey. Participants answered the following: if they intake caffeine, forms of caffeine ingestion, daily and weekly consumption, time of day of caffeine intake, and how long they have been regularly consuming caffeine. Low caffeine usage was considered less than one caffeinated beverage per day. Moderate caffeine usage was 1-2 caffeinated beverages per day; heavy usage was more than three caffeinated beverages per day (1). Height and weight were then measured to the nearest centimeter and kilogram, respectively, and body mass index (BMI) was calculated. Arterial BP was measured in a seated position in a low-noise environment using a stethoscope and sphygmomanometer. The participants who met all inclusion criteria and qualified for participation in the study then completed a familiarization exercise trial. This consisted of one set of 6 x 30-meter sprints at a comfortable pace that was self-selected by the participant. After completing the familiarization trial, participants scheduled the two experimental visits, and the assessment session was concluded.

Figure 1. Flow diagram representing the study design. BP, blood pressure; HR, heart rate; RPE, rating of perceived exertion; BLa, blood lactate; ST, sprint time.

Subjects were assigned to a randomized, double-blind, placebo-controlled crossover design to orally receive caffeine (100% Pure Caffeine Anhydrous; NutraBio Labs, Middlesex, NJ; 200 mg) or a placebo (microcrystalline cellulose) in pill form 60 minutes before undergoing the repeatedsprint protocol. The dosage used in this investigation was in agreement with previous studies demonstrating an increase in exercise performance by ingesting as little as 150-250 mg of caffeine (3, 17, 33, 67). The administration time is consistent with the literature, as caffeine is completely absorbed within the stomach roughly 45 minutes after ingestion and has a half-life of about 3-4 hours (5). Participants labeled as low- to moderate-caffeine users during the assessment visit were asked to refrain from caffeine ingestion 48 hours prior to experimental visits. If a participant was classified as a heavy-caffeine user, they were asked to refrain from caffeine for two weeks prior to the second visit, in order to mitigate any withdrawal symptoms (35), and 48 hours prior to the third visit.

Participants were asked to maintain their normal dietary habits throughout the study and refrain from intense exercise and alcohol intake 24 and 12 hours, respectively, prior to each experimental visit. During experimental days, participants arrived at the laboratory and resting arterial BP was assessed using a stethoscope and sphygmomanometer. Following, participants ingested either the caffeine or placebo pill in front of a researcher. In the 60-minute period between supplementation and the start of the repeated-sprint protocol, subjects refrained from ingesting any food or drinking any fluids other than water. After the wait-period, resting arterial BP was re-assessed and baseline values of BLa was taken from the finger and was measured on a handheld analyzer (Nova Biomedical Lactate Plus; Nova Biomedical, Waltham, MA). A heart rate (HR) monitor (Polar H7; Polar Electro Inc., Lake Success, NY) was attached on the thorax and a baseline measurement was recorded. Additionally, baseline RPE was assessed using the Borg Scale (6-20 scale). The same techniques were utilized to measure BLa, HR, RPE during the repeated-sprint protocol.

After preparatory procedures and baseline measurements were completed, participants performed a warm-up consisting of 3 minutes of a light jog (>50% of maximum heart rate) followed by 3 minutes of dynamic stretching. The dynamic stretches were chosen by the participant and, if needed, the researchers helped coach the participant. The repeated-sprint protocol comprised of three sets of 6 x 30-meter sprints with 20 seconds of active recovery between the sprints. Previous reports utilizing a repeated-sprint running protocol after caffeine supplementation used sprint distances between 20-40 meters (47). The gymnasium floor was marked with floor tape from the start/finish line to another tape marker that was 15 meters apart. After completion of the warm-up protocol, the participant lined up at the start/finish line. On the researcher's command, the participant sprinted down to the second marker and sprinted back to the finish line after touching the second tape marker with their hand. One maximaleffort sprint down to the second marker and back to the start/finish line equated to 30 meters. Participants completed a total of six 30-meter sprints within a single set with 20 seconds of active recovery (light walk around the start/finish line at the participant's own pace) in between the sprints. Single ST and total ST were recorded. The same blinded researcher recorded sprint times with the same stopwatch throughout the duration of the study. Following each set, HR, RPE, and BLa were collected, and participants performed 5 minutes of active recovery (light walk around the gymnasium at the participant's own pace) before repeating this process two additional times for a total of three sets.

Statistical Analysis

The primary outcome of this investigation was ST, while all other variables were considered secondary. All data were recorded in Microsoft Excel (Microsoft Corporation, Redmond, WA) and later transferred and analyzed using SigmaPlot 14.5 (Systat Software, San Jose, CA) and SPSS Statistics 23 (SPSS, Inc., Chicago, IL). The results of each outcome variable were analyzed by a two-way repeated-measures ANOVA to determine any significant interactions or main effects. Post hoc analyses were conducted using the Student-Newman-Keuls test. The alpha level for significance was set at *p* < 0.05 for these tests. Cohen's *d* effect size (ES) was calculated as $ES = (M_1 - M_2)/s_{pooled}$ and was used to estimate the degree to which caffeine supplementation influenced each outcome variable. An ES of 0.2 or less was deemed small, 0.5 moderate, and 0.8 or greater was large. Data are presented as means ± SE.

RESULTS

The results of the two-way repeated-measures ANOVA for systolic BP indicated there was no significant interaction $[F(1,31) = 2.107, p = 0.157]$, but there were main effects for condition [*F*(1,31) = 6.526, *p* = 0.016] and time [*F*(1,31) = 6.102, *p* = 0.019]. Results indicated that the caffeine condition resulted in significant (*p* = 0.016, Cohen's *d* = 0.46) elevations in systolic BP at postsupplementation compared to placebo (Table 2). For diastolic BP, there was a significant Condition x Time interaction $[F(1,31) = 4.285, p = 0.047]$. Results indicated no significant (*p* = 0.450, Cohen's *d* = 0.13) difference in diastolic BP between conditions at pre-supplementation, but the caffeine condition resulted in significant (*p* = 0.003, Cohen's *d* = 0.60) elevations at postsupplementation compared to placebo (Table 2).

Table 2. Systolic and diastolic blood pressure at pre-supplementation and post-supplementation under caffeine and placebo conditions.

	Caffeine	Placebo
Systolic		
Pre-supplementation	$124 + 1$	123 ± 1
Post-supplementation	$127 \pm 1*$	124 ± 1
Diastolic		
Pre-supplementation	$78+2$	$79 + 1$
Post-supplementation	$82+1*$	$79 + 1$

Values are means \pm SE. *p < 0.05 vs. placebo.

The two-way repeated-measures ANOVA resulted in a significant Condition x Time interaction [*F*(3,93) = 18.950, *p* < 0.001] for BLa. The results revealed no statistical (*p* = 0.812, Cohen's *d* = 0.13) difference between conditions at rest but indicated that the caffeine condition resulted in greater exercising BLa at each sprint set (set 1: *p* < 0.001, Cohen's *d* = 0.87; set 2: *p* < 0.001, Cohen's *d* = 0.61; set 3: *p* < 0.001, Cohen's *d* = 0.73; Figure 2, *left*).

There was no significant Condition x Time interaction [*F*(2,62) = 1.766, *p* = 0.179] for total ST, but there were significant main effects for Condition $[F(1,31) = 36.839, p \le 0.001]$ and Time $[F(2,62)$ $= 5.806$, $p = 0.006$. The analyses indicated that the caffeine condition resulted in significantly reduced total ST during all three sprint sets compared to the placebo condition (set 1: *p* < 0.001, Cohen's *d* = 0.36; set 2: *p* < 0.001, Cohen's *d* = 0.35; set 3: *p* < 0.001, Cohen's *d* = 0.46; Figure 2, *right*).

There was a significant two-way (Condition x Time) interaction for HR [F(3,93) = 3.368, $p = 0.022$. The analyses revealed a significant (*p* = 0.016, Cohen's *d* = 0.27) decrease in HR at rest in the caffeine trials compared to placebo. However, exercising HR was not statistically different within each sprint set (set 1: *p* = 0.535, Cohen's *d* = 0.07; set 2: *p* = 0.602, Cohen's *d* = 0.07; set 3: *p* = 0.189, Cohen's *d* = 0.18) (Figure 3, *left*).

Figure 2. Group mean blood lactate concentrations (*left*) at baseline and after each sprint set under caffeine and control conditions. Group mean total sprint time (*right*) for each sprint set under caffeine and placebo conditions. **p* < 0.05 vs placebo.

For RPE, there was no significant Condition x Time interaction [*F*(3,93) = 1.602, *p* = 0.194] or main effect for condition [*F*(1,31) = 4.090, *p* = 0.052; set 1: Cohen's *d* = 0.18, set 2: Cohen's *d* = 0.26, set 3: Cohen's *d* = 0.26], but there was a main effect for time [*F*(3,93) = 292.810, *p* < 0.001]. These results indicate no significant difference in RPE between the caffeine and placebo trials at any of the time points (Figure 3, *right*).

Figure 3. Group mean heart rate (*left*) and rating of perceived exertion (*right)* at baseline and during each sprint set under caffeine and control conditions. **p* < 0.05 vs placebo.

DISCUSSION

Caffeine supplementation is extensively used to enhance exercise performance with a wide range of research conducted on improving aerobic and anaerobic performance, yet research investigating RSA is equivocal. The majority of studies have focused on athletic populations using a relative dose supplement and have concentrated on RSA that is non-running in nature. Caffeine utilization amongst non-athletic populations is widespread with an increasing surge

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being demonstrated in individuals who participate in HIIT activity and competitive recreational sports focusing on interval bouts of low and high intensity exercise. The purpose of this study was to investigate the effects of 200 mg of caffeine on RSA in healthy young adults and it was determined that caffeine supplementation ingested 60 minutes before exercise decreased time to completion for all three sprint sets.

Our findings, similar to other reports (13, 22, 31, 32, 45, 54, 55, 58, 61), demonstrated significantly reduced ST after caffeine supplementation. Carr et al. (13) concluded that 6 mg kg⁻¹ of caffeine 60 minutes prior significantly improved sprint running performance in team-sport athletes. Similarly, Pontifex et al. (55) determined that 6 mg $kg⁻¹$ of caffeine 60 minutes prior to repeatedsprints resulted in significantly lower total ST and lower fastest single ST in male athletes. Del Coso et al. (22) found that a lower dosage of 3 mg $kg⁻¹$ displayed a significant improvement in a repeated-sprint protocol in semi-professional soccer players. Further, Glaister et al. (31) demonstrated improvements in multiple sprint performance, including significant reductions in fastest ST, after 5 mg kg⁻¹ of caffeine in physically active men. Similar to Carr et al. (13), our findings demonstrated a decrease in total ST for all sets suggesting caffeine elicits ergogenic effects in a non-time dependent manner, but challenges Stein et al. (61) who displayed an improvement in only the second set of sprints.

The findings from our study challenge those reports revealing a negligible effect of caffeine on RSA (4, 15, 27, 34, 53). Paton et al. (53) exhibited an insignificant ergogenic effect on 6 mg kg⁻¹ of caffeine on repeated-sprint running ability in team-sport athletes. Similarly, Clarke et al. (15) found no improvement in repeated cycling performance with 3 mg $kg⁻¹$ of caffeine ingestion in untrained males. Interestingly, Ermolao et al. (27) investigated the effects of 300 mg of caffeine on recreationally-trained team-sport athletes and revealed no significant difference in RSA between the caffeine and placebo trials. The disparity among studies may be attributed to the varying levels of fitness among subjects, differing caffeine dependency among subjects, caffeine dosage, and/or exercise mode.

Our investigation revealed significant elevations in BLa values during all three sprint sets during the caffeine trials, supporting studies showing a significant elevation in exercising BLa after caffeine ingestion (13, 32, 58) and contradicting the ones that do not (34). This is noteworthy, as increased BLa concentrations are associated with increased anaerobic glycolysis utilization (64) and exercise intensity (8). Enhanced utilization of anaerobic glycolysis will, in turn, result in greater power output and improve RSA (10, 29). Moreover, RPE was unchanged between conditions during all three sprint sets. In normal conditions, we would expect RPE to increase in parallel with BLa since RPE increases with exercise intensity and BLa is sensitive to changes in exercise intensity (60, 62). However, one reason why we did not witness a significant difference in RPE may be because the participants were exercising at maximal efforts regardless of condition. Past reports have displayed substantial reductions in RPE with caffeine supplementation despite elevated performance (24) revealing an explanation for its ergogenic

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effects on exercise performance. Even though our RPE findings challenge the literature, we still witnessed a blunted RPE response relative to increases in BLa and exercise intensity during the caffeine trials. These two observations from our report suggest that caffeine supplementation induces greater anaerobic glycolysis utilization during repeated-sprints, while diminishing perceived exertion. This may be due to caffeine's ability to inhibit adenosine, a compound known to enhance pain perception (63), resulting in greater potential to endure greater exercise intensities and allow for improvements in RSA.

In the current study, both systolic and diastolic BP were significantly increased after 60 minutes of caffeine ingestion. This finding is in line with the literature that suggests that caffeine results in acute increases in BP (14, 43). According to James (38), acute elevations in BP from 5 to 15 mmHg systolic and 5 to 10 mmHg diastolic are representative in doses equivalent to those consumed in everyday life. On the other hand, baseline HR was decreased in the caffeine trials and the exercising HR was not statistically different between groups. Our observed exercising HR is in line with Goods et al. (32) but challenges Glaister et al. (31). Some possible factors that may have played a role in the study by Goods et al. are that the subjects were highly trained male athletes. Further, the amount of caffeine was at a relative value of 3 mg kg⁻¹. Glaister et al.

studied subjects who were in the average fitness level and used a relative dosage of 5 mg $kg⁻¹$. In our investigation, an absolute dose of 200 mg of caffeine was used. It may be possible that a higher dosage results in a significant increase in exercising heart rate during repeated sprints, suggesting a dose-response relationship.

An absolute dosage of caffeine instead of a relative dosage was employed herein to replicate practical usage in the regular recreationally-trained individual. Jagim et al. (37) reported that the average caffeine content in the top 100 commercially available multi-ingredient pre-workout supplements was approximately 250 mg. In a paper by Desbrow et al. (23), it was found that the average caffeine content per serving for the top-selling multi-ingredient pre-workout supplements in Australia ranged between 100-390 mg. Since caffeine is commonly ingested as an ergogenic aid in the form of multi-ingredient pre-workout supplements, our approach simulates practical real-world caffeine use in the general population. Our findings of a significant ergogenic effect using an absolute caffeine dosage agree with other reports that tested an absolute dosage of caffeine on sprint performance (17, 54, 66). Collomp et al. (17) evaluated the effects of 250 mg of caffeine on trained and untrained swimmers but only found a significant improvement in swimming velocity in the trained individuals. Comparably, Wellington et al. (66) assessed the impact of 300 mg of caffeine on repeat-high-intensity efforts in semiprofessional rugby players and observed a significant decrease in repeated-sprint time after caffeine supplementation. To our knowledge, our investigation is the first to report an ergogenic effect of an absolute caffeine dosage on RSA in young, healthy non-athletes.

The present investigation has its share of strengths and limitations. Strengths include a large sample size with a near-balanced number for sex of participants with a strict caffeine-restriction criteria, and the study employed a robust experimental design (i.e., double-blind, randomized, placebo-controlled, crossover trial). One limitation was that subject compliance to caffeine restriction during the washout period was based on instructions provided by the researchers, but no caffeine logs were completed. Similarly, normal dietary habits were maintained throughout the study, and intense exercise and alcohol intake were restricted before each experimental session based on instructions provided by the researchers; however, no food or activity logs were completed. Additionally, our investigation did not assess plasma caffeine concentrations or rate of caffeine metabolism, but it has been reported that doses of 6 and 9 mg \cdot kg-1 were equally effective in increasing power output during intense exercise (12). Moreover, research has demonstrated that caffeine pills increase plasma caffeine concentration and that they are the standard used for comparison of caffeinated products (41).

This investigation provides evidence that supplementing 200 mg of caffeine prior to exercise enhances RSA, determined by decreased total ST, in young, healthy non-athletes. This may be attributed to caffeine's ability to blunt elevations in perceived exertion despite increased exercise intensity measured by BLa concentrations. Since caffeine is one of the most widely used supplements in the world, it is imperative to understand how it impacts the general non-athletic population during high-intensity exercise. The findings from our investigation can help further recognize the ergogenic effects of caffeine on RSA in the young, healthy general population.

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