Effects of Acute Caffeine Ingestion on Anaerobic Cycling Performance in Recreationally Active Men

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ABSTRACT

Cakir-Atabek, H. Effects of Acute Caffeine Ingestion on Anaerobic Cycling Performance in Recreationally Active Men. JEPonline 2017; 20(1):47-58. Caffeine increases aerobic performance, but research findings are mixed regarding anaerobic performance (AnP). Thus, the purpose of this study was to investigate the effects of acute caffeine ingestion on high-intensity cycling anaerobic exercise in 14 healthy and physically active non-habitual caffeine users (mean age: 20.93 ± 1.73 yrs). After the subjects were familiarized with the Wingate Anaerobic test (WAnT) intensity (on a separate date), placebo or caffeine treatments were performed. Both treatments were separated by 1 wk in a randomized, double-blind manner. One hr before the WAnT, the subjects ingested a drink mixture of water, lemonade, and Sprite as a placebo (PLA) or containing caffeine (CAF, 5 mg·kg⁻¹); both drinks were equal in calories. The WAnT was performed for 30 sec with the resistance set at 7.5% of the subject’s body mass. Peak power, mean power, and fatigue index were determined. A paired sample t-test was used to compare the AnP variables between treatments. Heart rate (HR), blood pressure (BP), blood lactate (LA), and glucose (GLU) levels were analyzed with a two-way repeated-measures ANOVA. The results showed no significant effect of caffeine on AnP (P>0.05). Increases in HR, BP, and blood LA levels were independent of treatments (P>0.05). However, blood GLU levels significantly differed between the treatments (P<0.05). The findings indicate that a single dose of CAF was insufficient to elicit an ergogenic effect on AnP in unaccustomed and non-habitual caffeine users.

Key Words: Ergogenic, Untrained, Non-Habitual Caffeine Users
INTRODUCTION

Caffeine is the most widely socially consumed food constituent in the world (22,28,39). Since caffeine was removed from the World Anti-Doping Agency’s prohibited substance list, it has become popular among competing athletes (6). Studies conducted in Canada (37) and the United States (15) indicate that 11 to 19-yr-olds use caffeine to improve their performance. Additionally, it has been reported that 59.9% of cyclist and 32.6% of track and field athletes in the UK consume caffeine to enhance their performance (8).

The main ergogenic mechanism of caffeine is antagonism of the adenosine receptors (18). Caffeine may also affect performance via both central and peripheral mechanisms by altering pain and effort perception (13), calcium kinetics in the sarcoplasmic reticulum (1,35), and sodium/potassium ATPase pump activity (29) among other potential mechanisms (18). However, the effectiveness of caffeine may depend on the dose, the timing of caffeine administration, the participants’ dietary status, and the users’ characteristics, such as a training experience and habitual use of caffeine, or a combination of any of these factors (4,10,27,40).

Although numerous studies have examined the effect of caffeine on many psychological, physiological, and bio-motoric variables that affect athletic performance such as mood state (34), pain and rate of perceived exertion (RPE) (3,6), simple reaction time (25,34), repeated sprint ability (24,32), agility (27), strength (3), and anaerobic performance (34,39,41), the effects of caffeine ingestion on sport performance remain questionable (40). Since the anaerobic performance is one of the basic motoric abilities, many studies have investigated the effects of caffeine on anaerobic performance. The findings are equivocal with some studies reporting enhanced performance (26,34,39,41) while others suggesting that caffeine does not provide significant advantage (9,17,20,27).

The highly individual variations in the caffeine metabolism response and the lack of sufficient evidence regarding caffeine as an ergogenic aid potentially attributable to testing of untrained subjects and habitual versus non-habitual caffeine users formed the basis of this research. Therefore, the purpose of this study was to investigate the effects of acute caffeine ingestion on a high-intensity cycling anaerobic exercise in healthy and physically active non-habitual caffeine users.

METHODS

Subjects
Fourteen healthy and physically active male university students participated in this study (age = 20.9 ± 1.7 yrs, height = 174.7 ± 4.8 cm, mass = 72.1 ± 6.6 kg, body fat = 14.8 ± 3.5%, body mass index = 23.7 ± 2.1 kg·m⁻²). The subjects were not engaged in a specific anaerobic or cycling training program, but were participating in general fitness and recreational activities that included running, squash, tennis, and football 4 to 5 d·wk⁻¹. Major exclusion criteria were smoking, lower limb injury, and disease. A list of foods and beverages containing caffeine was given to the subjects. They were told not to consume these products during the study. Additionally, the subjects were told not to use alcohol or any medicine during this period. These conditions were monitored with diet record forms that were completed during the 3 days before the treatments. At the start of the study, each subject was informed of the
experimental risks, and written consent forms were obtained. The study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki, and it was approved by the University Institutional Review Board. All subjects were informed of their right to withdraw from the study at any time without negative consequences.

**Procedures**

Each subject visited the laboratory three times. During the first visit, the subjects completed the questionnaire to determine the physical activity and smoking status, and height, mass, percent body fat, and body mass index were measured. Additionally, each subject was familiarized with the Wingate Anaerobic Test (WAnT) by performing the first 10 sec of the test. This was used to set the seat height and allow the subjects to become familiar with the Wingate test intensity (39). On visit 2 and 3, which were separated by 1 wk, one of the treatment drinks (placebo or caffeine) was administered in a cross-over, random and double-blind manner (Figure 1). To blind the study, a colleague (not included in the study) was responsible for randomizing and coding the trials and mixing the drinks. All subjects were instructed not to eat at least 2 hrs before the test.

The treatment drinks were prepared according to Turley et al. (38,39), Astorino et al. (3), and Wiles et al. (40) with minor modification. The placebo drink (PLA) was a mixture of 30 ml of water, 100 ml of Sprite (38,39), and 80 ml of lemonade (3,40). The caffeine treatment drink (CAF) consisted of 210 ml of the placebo with 5 mg·kg\(^{-1}\) of body mass of caffeine anhydrous dissolved in solution (Doga Medicament, Istanbul, Turkey). Both drinks were equal in calories. This dose of caffeine was chosen because it has been established to elicit an ergogenic effect, and it is routinely used in studies investigating caffeine’s impact on exercise performance (39,41).

The experiment started when the subjects ingested the treatment drinks (placebo or caffeine) 60 min before the test (32,33,34). A 1-hr period was chosen because research has determined that caffeine is quickly absorbed, with plasma levels reaching a maximum level within 1 hr of ingestion (19), and maximal cardiovascular effects (30) occur at that time. During the 1-hr period, the subjects stayed in the laboratory, spending time on computers reading the news, surfing in social networks, and etc. Hydration was allowed during this period. Sixty minutes after the consumption of the treatment drink (PLA or CAF), the WAnT was performed on a mechanical braked cycle ergometer (894 Ea, Peak Bike by Monark AB,
Subjects sat on the ergometer to allow adjustments to the ergometer to be made to ensure an optimal cycling position. The seat height was adjusted to each subject's satisfaction. Toe clips with straps were used to prevent the subjects’ feet from slipping off the pedals. The WAnT was conducted according to widely accepted recommendations for standardization (23). It was administered for 30 sec. Resistance was set at 7.5% of body mass. The WAnT session started with a standardized warm-up of 5 min of cycling at 50 to 60 rev·min\(^{-1}\) against no load. Following the warm-up, the subjects rested for 5 min. The subjects were encouraged to pedal as fast as they could prior to the application of resistance. Following the application of resistance, the subjects attempted to pedal at maximum speed throughout the remaining 30 sec. Strong verbal encouragement was provided by the investigator. Absolute and relative peak power (PP), absolute and relative mean power (MP), and minimum power were calculated automatically by the WAnT program via computer (Monark Exercise AB, Sweden) and recorded for further analysis. A fatigue index (FI) was calculated using the following equation (23):

\[
FI = \frac{\text{[(Peak Power - Minimum Power) / Peak Power]} \times 100}
\]

Heart rate (HR), systolic blood pressure, and diastolic blood pressure (SYS BP and DIA BP) were measured at the arm using a sphygmomanometer (Microlife BP A100, Switzerland) just before the consumption of the treatment drink after at least 10 min of rest in the sitting position. The measurements were repeated every 15 min during the 1 hr before the test (Pre T60min, Pre T45min, Pre T30min, Pre T15min, and Pre T0min) and every 15 min (for one hour) after the test (Post T0min, Post T15min, Post T30min, Post 45min, and Post 60min).

The blood samples (~20 µl) were obtained from an earlobe or fingertip (if the subject did not want to puncture the ear, the blood samples were obtained from the fingertip) just before the consumption of the treatment drink and every 15 min after the test (for 1 hr) (Pre T60min, Post T0min, Post T15min, Post T30min, Post 45min, and Post 60min). Blood lactate (LA) (mmol·L\(^{-1}\)) and glucose (GLU) (mg·dL\(^{-1}\)) were analyzed using the enzymatic-amperometric measuring method and administered with the aid of the Biosen C-line (EKF Diagnostic GmbH, Germany) technical device.

All subjects were instructed to maintain their normal dietary habits during the investigation. Also, each subject completed a diet record form for the 3 days before the treatments (PLA and CAF). The diet record forms were used to quantify the average intake of calories, protein, carbohydrate, and fat, which were analyzed using a computerized dietary assessment program (BEBİS, version 6.1, Turkey). Additionally, the subjects were given a list of foods and beverages containing caffeine. They were requested to abstain from consuming them for 48 hrs before the tests.

**Statistical Analyses**

All data are presented as the mean ± standard deviation (SD) in the text and in the Figures. The K-S test of normality demonstrated that the data were normally distributed. Then, paired sample \(t\)-tests were used to determine differences between CAF and PLA treatments for peak power, mean power, and fatigue index, as well as for the total intake of calories (kcal·day\(^{-1}\)) and macronutrients [carbohydrate (g), fat (g), and protein (g)]. Heart rate, SYS BP, and DIA BP, blood LA, and GLU were tested using a two-way (treatments x measurements) repeated measures ANOVA. The Greenhouse–Geisser correction was used
where sphericity was violated. The Bonferroni and the LSD methods were applied to determine pairwise differences. A power analysis was performed (%), effect sizes were calculated as the partial eta-squared ($\eta^2_p$), and these values were reported for significant findings. All statistical analyses were performed using the 18.0 version of SPSS (Statistical Package for the Social Sciences, SPSS Inc.) for Windows. The level of statistical significance was set at P<0.05.

RESULTS

No significant differences were observed for dietary variables (P>0.05; Table 1) between the two treatments (PLA vs. CAF). The results obtained from the dietary logs demonstrated that none of the subjects were habitual caffeine users. The subjects drank one or less cups of coffee per day (24). Anaerobic performance data are presented in Table 2. There were no significant differences between PLA and CAF treatments for the anaerobic performance data (P>0.05).

Table 1. Dietary Intakes Assessed During the 3 Days Before the PLA and CAF Trials.

<table>
<thead>
<tr>
<th>Variables</th>
<th>PLA</th>
<th>CAF</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calories (kcal·day^{-1})</td>
<td>1597.7 ± 449.4</td>
<td>1584.9 ± 449.4</td>
<td>0.93</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>196.8 ± 61.9</td>
<td>185.6 ± 74.3</td>
<td>0.63</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>58.1 ± 22.4</td>
<td>61.5 ± 17.6</td>
<td>0.67</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>68.4 ± 17.9</td>
<td>67.9 ± 15.1</td>
<td>0.94</td>
</tr>
</tbody>
</table>

Table 2. Anaerobic Performance Values Recorded for PLA and CAF Trials.

<table>
<thead>
<tr>
<th>Variables</th>
<th>PLA</th>
<th>CAF</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Load (kg)</td>
<td></td>
<td>5.4 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>Absolute PP (w)</td>
<td>952.4 ± 179.0</td>
<td>954.5 ± 145.6</td>
<td>0.92</td>
</tr>
<tr>
<td>Relative PP (w·kg^{-1})</td>
<td>13.3 ± 2.5</td>
<td>13.3 ± 1.8</td>
<td>0.96</td>
</tr>
<tr>
<td>Absolute MP (w)</td>
<td>516.7 ± 64.3</td>
<td>521.1 ± 65.9</td>
<td>0.51</td>
</tr>
<tr>
<td>Relative MP (w·kg^{-1})</td>
<td>7.2 ± 0.6</td>
<td>7.2 ± 0.6</td>
<td>0.55</td>
</tr>
<tr>
<td>Fatigue Index (%)</td>
<td>98.6 ± 17.9</td>
<td>103.8 ± 29.0</td>
<td>0.53</td>
</tr>
</tbody>
</table>

PP = peak power; MP = mean power.

The changes observed in HR, SYS-BP, and DIA-BP are shown in Figure 2, and the changes observed in blood LA and GLU are shown in Figure 3. The results indicated that HR significantly increased after the WAnT (F = 203.4; P<0.001; $\eta^2_p = 0.94$; 100%). The HR values measured after the WAnT (PostT0min, PostT15min, PostT30min, and PostT45min) were significantly higher compared to the pre-test HR values. This increment was independent of the treatments (F = 0.2; P = 0.70). There was no significant treatments x measurements interaction for HR (F = 1.6; P = 0.12).

The SYS-BP values significantly changed after the WAnT (F = 18.8; p<0.001; $\eta^2_p = 0.59$; 100%). The SYS BP value measured after the WAnT (PostT0min) was significantly higher compared to pre-test and post-test SYS BP values; whereas, post-test values (PostT15min,
PostT30min, and PostT60min) were significantly lower compared to pre-test SYS BP values (supporting information Figure 2B, ANOVA with LSD, P<0.01 and 0.05). This increment was not different between treatments (F = 0.3; P = 0.61). There was no significant treatments x measurements interaction for SYS BP (F = 0.8; P = 0.63).

The results indicated that there was a significant main effect of measurements for DIA BP (F = 4.4; P<0.01; \( \eta^2_p = 0.25; 91.6\% \)). As a result of the pairwise comparison, PostT0min DIA BP value was significantly higher compared to the PostT15min, PostT30min, PostT45min, and PostT60min values (supporting information Figure 2B, ANOVA with LSD, P<0.01 and 0.05). These changes were independent of treatments (F = 1.8; P = 0.21). However, the treatments x measurements interaction was significant for DIA BP values (F = 2.3; P = 0.02; \( \eta^2_p = 0.15; 89.3\% \)).

Figure 2. (A) Heart Rate (HR), (B) Diastolic (DIA BP), and Systolic Blood Pressure (SYS BP) Responses to PLA and CAF Treatments (mean ± SD). **P<0.01, denotes significantly different from all other (pre-test and post-test) values (ANOVA with Bonferroni); aP<0.01, denotes significantly different from all other test values - except PostT45min HR value (ANOVA with Bonferroni); bP<0.01, denotes significantly different from all other test values - except PostT30min HR value (ANOVA with Bonferroni); cP<0.05, denotes significantly different from all other test values - except PostT30min HR value (ANOVA with Bonferroni); dP<0.05, denotes significantly different from all pre-test values (ANOVA with LSD); eP<0.05, denotes significantly different from post-test values (ANOVA with LSD); fP<0.05, denotes significantly different from PreT45min and PreT0min values (ANOVA with LSD).
As illustrated in Figure 3A, blood LA values increased significantly after the WAnT (F = 192.7 P<0.001; \(\eta^2_p = 0.94; 100\%\)). This increment was not different between the treatments (F = 0.02; P = 0.88), furthermore, there was no significant treatments x measurements interaction for blood LA (F = 0.22; P = 0.85).

![Figure 3. (A) Blood Lactate (LA) and (B) Blood Glucose (GLU) Responses to PLA and CAF Treatments (mean ± SD). **P <0.01, denotes significantly different from all other test values; #P <0.01, denotes significantly different from all other test values - except PostT15min LA value; †P <0.01, denotes significantly different from all other test values - except PostT0min LA value; ‡P <0.05, denotes significant difference between treatments (PLA vs. CAF); αP <0.05, denotes significantly different from PostT30min and PostT45min GLU values; βP <0.05, denotes significantly different from PostT15min, PostT30min and PostT45min GLU values (all results: ANOVA with Bonferroni).](image)

The results indicate that the blood GLU levels significantly changed after the WAnT (F = 8.9; P<0.001; \(\eta^2_p = 0.41; 100\%\); supporting the information in Figure 3B, ANOVA with Bonferroni). The blood GLU levels significantly differed between the treatments (F = 24.9; P<0.001; \(\eta^2_p = 0.66; 99.6\%\)). The blood GLU levels at the PostT30min and the PostT45min measure time were significantly different between the treatments (P=0.03 and P<0.01, respectively). As a result of the pairwise comparison (for each treatment), significant differences were observed in PLA treatment (the PreT60min and the PostT0min GLU levels were significantly different
DISCUSSION

The results from the present study demonstrate that 5 mg·kg⁻¹ caffeine ingestion does not have an ergogenic effect on PP, MP, and FI during a 30-sec all-out Wingate performance test. These performance results are consistent with previously reported (5,9,20,21,27,34) findings, but are in contrast to findings by Kang et al. (26), Woolf et al. (41), and Turley et al. (39).

Kang et al. (26) reported a significant ergogenic effect of CAF by administering PLA, 2.5 mg·kg⁻¹, and 5 mg·kg⁻¹ caffeine to 7 professional cyclists and 7 students in which PP and MP were significantly increased in CAF versus PLA, with no difference between the two caffeine doses. Similarly, 1 hr after consuming 5 mg·kg⁻¹ of caffeine, highly trained competitive male athletes performed a leg press, chest press, and the WAnT. The authors reported that PP significantly increased in CAF compared to PLA (8.88 ± 0.77 vs. 8.43 ± 0.83 w·kg⁻¹, respectively) (41). Turley et al. (39) reported that caffeine ingestion of 5 mg·kg⁻¹ significantly increased the MP output (w) in 8- to 10-yr-old boys. Additionally, Souissi et al. (34) demonstrated that caffeine ingestion of 5 mg·kg⁻¹ significantly increased PP (w·kg⁻¹) and MP output (w·kg⁻¹) (P<0.001 and P<0.05, respectively) in young judokas athletes. Based on these findings, it has been suggested that caffeine could improve performance by increasing the release of Ca²⁺ from the sarcoplasmic reticulum in active muscle or by increasing the recruitment of muscle through stimulation of the central nervous system (21,31,32).

On the other hand, different results have been reported (9,17,20,27) with no improvement in 30 sec Wingate performance after caffeine ingestion. Greer et al. (21) investigated the effects of caffeine on anaerobic performance with electromyographic signals (EMG) and similar to the aforementioned studies, failed to find an ergogenic effect of caffeine ingestion. In agreement with the current study findings, Greer et al. (21) showed that a 5 mg·kg⁻¹ dose of caffeine did not impact power output (PP, MP, and FI%) during a 30-sec WAnT. Also, it was reported that caffeine did not impact the neuromuscular properties of the muscle, as indicated by similar integrated EMG scores between treatments (caffeine vs. placebo) in recreationally active college men.

We used the same dose of caffeine as previous studies that reported positive effects of caffeine ingestion (5 mg·kg⁻¹) (26,39,41), but we failed to detect a significant improvement in the subjects' Wingate performance after caffeine ingestion. We conclude that the amount of caffeine may have been insufficient in the present study. Furthermore, we administered the caffeine 60 min before the WAnT, which is a common protocol used by researchers because the peak blood concentration has been observed at 30 to 60 min after ingestion (27). The lack of measurement of the caffeine concentration in the blood or urine is one of the limitations of the current study. However, Souissi et al. (34) reported that caffeine ingestion of 5 mg·kg⁻¹ significantly increased the plasma caffeine concentration after 60 min and that the plasma caffeine was significantly higher after WAnT in comparison with the pre-WAnT value (i.e., 3.45 mg·L⁻¹ vs. 0.28 mg·L⁻¹). Additionally, it has been reported that the urinary caffeine concentrations, measured approximately 2.5 hrs following the ingestion of 6 mg·kg⁻¹ of
caffeine, ranged from 3.5 to 9.1 µg·mL⁻¹ (33), which shows the individual variations in the rate of metabolized caffeine by the liver. Even though it was administered at 5 mg·kg⁻¹ of body mass in the present study, it is plausible that the subjects have variable metabolic and performance-induced responses to a given dose of caffeine (21).

The results of the subjects’ dietary analysis verified that they maintained a consistent diet with no significant difference in the total intake of calories (kcal·day⁻¹) or macronutrients [carbohydrate (g), fat (g), and protein (g)] between the CAF and PLA treatments (Table 1). The results obtained from the dietary logs indicated that none of the subjects was a habitual caffeine user. It has been suggested that caffeine might not induce an ergogenic effect in habitual caffeine consumers because of improved tolerance to caffeine (36). In contrast to this proposal, Jordan et al. (24) reported that caffeine has the potential to enhance sprint performance (12 x 30 m sprint test with a 35-sec rest interval between sprints) in both caffeine-naïve and habitual caffeine users. It has been reported that caffeine may induce negative side-effects especially in non-habitual caffeine users, which may impair performance (28). Although none of the subjects involved in the current study reported such side-effect, this may have influenced the results.

The effectiveness of caffeine may depend on the users’ characteristics, such as a training experience. Woolf et al. (41) and Souissi et al. (34) reported significant increases in PP and MP values of WAnT in well-trained competitive athletes. As suggested by Collomp et al. (10), it is possible that the specific physiological adaptations present in highly trained anaerobic athletes, such as increased regulation of acid-base balance (i.e., intracellular buffering of H⁺), are necessary for caffeine to exert an ergonomic effect (18). In contrast, the subjects in the current study were recreationally active men, but were not engaged in a specific training program and, therefore, were physiologically unaccustomed to the strenuous anaerobic exercises. Consistent with the findings of the current study, Crowe et al. (11) demonstrated that caffeine ingestion of 6 mg·kg⁻¹ did not have an ergonomic effect on PP (w·kg⁻¹) and total work (J·kg⁻¹) done during two 60-sec maximal cycling bouts in physically active men. Because, untrained muscle lacks specific physiological and neural adaptation (vs. trained muscle), a lower percentage and absolute number of total fibers may be activated during work (2), which may explain the findings in the current study.

While some studies reported increased HR after caffeine supplementation (4,16), other studies have found no significant increase in HR (11,25,36). Crowe et al. (11) demonstrated that caffeine ingestion did not affect max HR in 2 x 60-sec maximal cycle test. Furthermore, Doherty et al. (14) reported that there were no significant differences in the peak HR achieved with PLA or CAF treatments (182 ± 9 vs. 185 ± 13). The results of the current study demonstrated that caffeine ingestion of 5 mg·kg⁻¹ did not have a main effect on HR (Figure 2A); the increases in HR values were independent of the treatments.

Crowe et al. (11) demonstrated that the caffeine ingestion did not affect the blood GLU concentration. Previously, it has been suggested that caffeine elevates the level of circulating epinephrine, causing increased carbohydrate metabolism, work output, and post-exercise lactate concentration (9,18,19). The current study’s findings showed that the blood GLU concentrations significantly differed between CAF and PLA treatments; where the blood GLU levels were higher in the CAF treatment. Although it has not been measured in the current study, well documented caffeine’s effects on epinephrine could explain this effect. It has been
shown that caffeine amplifies epinephrine (adrenaline) output from the adrenal medulla. Thus, it would seem possible that blood glucose would subsequently increase more with caffeine administration (12).

Many studies (7,11,17,20,33) have reported that blood LA concentrations are significantly increase during exercise with caffeine compared to placebo. However, in contrast to these studies, blood LA levels in the current study were not affected by caffeine ingestion. Many factors such as exercise protocol, blood sampling time, the subjects’ physical activity status and/or caffeine dose could explain this conflicting result. Also, it should be point out that the exercise protocol in the current study was not intermittent, which is different from the aforementioned studies. Furthermore, the subjects were not anaerobically trained and were not accustomed to the intense anaerobic exercises like the subjects involved in the earlier studies performed by Glaister et al. (17) and Schneiker et al. (33).

CONCLUSIONS

There are many factors that affect caffeine efficiency (e.g., special training experience, habits of caffeine consumption, and individual differences). Nevertheless, many athletes prefer to use caffeine to enhance sports performance because of the individual variability in caffeine efficiency. The results of the current study indicate that a single dose of caffeine consumption did not have an ergogenic effect on a single trial of anaerobic performance in recreationally physically active participants who were non-habitual caffeine users.

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