THE EFFECT OF CAFFEINE AS AN ERGOGENIC AID IN ANAEROBIC EXERCISE

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Abstract

Study examined caffeine [5 mg/kg body weight (BW)] versus placebo during anaerobic exercise. Eighteen male athletes (24.1 ± 5.8 y; BMI, 26.4 ± 2.2 kg/m²) completed a leg press, chest press, and Wingate test. During the caffeine trial, more total weight was lifted with the chest press and a greater peak-power was obtained during the Wingate test. No differences observed between treatments for the leg press and average power, minimum power, and power drop (Wingate test). A significant treatment main effect found for post-exercise glucose and insulin concentrations; higher concentrations found with the caffeine trial. A significant interaction effect (treatment and time) found for cortisol and glucose concentrations; both increased with caffeine and decreased with placebo. Post-exercise systolic blood pressure was significantly greater during the caffeine trial. No differences found between treatments for serum free fatty acid concentrations, plasma lactate concentrations, serum cortisol concentrations, heart rate, and rating of perceived exertion. Thus, a moderate dose of caffeine resulted in more total weight lifted for the chest press and a greater peak-power obtained during the Wingate test in competitive athletes.
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Introduction

Caffeine, one of the most widespread and socially acceptable drugs consumed around the world, is commonly ingested by people who participate in sports in an attempt to enhance performance (Desbrow & Leveritt, 2006; Ellender & Linder, 2005; Graham, 2001; Juhn, 2003; Keisler, & Armsey, 2006; Maughan, King, & Lea, 2004). Caffeine is found in a variety of foods and beverages, including coffee, chocolate, tea, cola drinks, and energy drinks. Caffeine is also present in some widely consumed over-the-counter medications, like analgesics and diuretics. Caffeine is most commonly consumed in the form of coffee, tea, and soda. Energy drinks, a relatively new category of beverages, contain caffeine in amounts that exceed those in soft drinks and are similar to the concentrations found in coffee (Mandel, 2002).

In high doses, caffeine is a monitored or restricted drug for competitive athletes. The World Anti-Doping Agency (WADA) recently removed caffeine from the Prohibited List and added it to the Monitoring Program during competition (World Anti-Doping Agency, 2007b). WADA monitors substances like caffeine during competition in order to detect patterns of misuse in sport (World Anti-Doping Agency, 2007a). The National Collegiate Athletic Association (NCAA) still considers a urinary level of > 15 micrograms per milliliter (µg/mL) following competition to be illegal (National Collegiate Athletic Association, 2007). The WADA anti-doping code has been accepted by many sport organizations including the International Olympic Committee. It takes about 13 milligrams per kilogram body weight (mg/kg BW) of caffeine (about 700 mg caffeine or 8
cups of brewed coffee) to reach the maximum allowable urinary concentration (Ellender & Linder, 2005; Graham, 2001).

Research has suggested that exercise performance may be improved with as little as 150 to 250 mg of ingested caffeine (~1 to 2 cups of coffee) (Anselme, Collomp, Mercier, Ahmaidi, & Prefaut, 1992; Collomp, Ahmaidi, Chatard, Audran, & Prefaut, 1992; Graham & Spriet, 1995; Wiles, Bird, Hopkins, & Riley, 1992). Many athletes use caffeine as an ergogenic aid in hopes of enhancing performance (Ellender & Linder, 2005; Graham, 2001; Juhn, 2003; Keisler & Armsey, 2006; Maughan et al., 2004). Evidence from the research literature suggests that caffeine enhances endurance and provides an improvement in performance over a wide range of exercise protocols, particularly prolonged and exhaustive exercises (Bell & McClellan, 2003; Bell & McClellan, 2002; Graham & Spriet, 1995; Pasman, vanBaak, Jeukendrup, & deHaan, 1995; Trice & Haymes, 1995). It has also been suggested that caffeine reduces fatigue, improves concentration, and enhances mental alertness (Lieberman, Tharion, Shukitt-Hale, Speckman, & Tulley, 2002; Magill et al., 2003; van Duinen, Lorist, & Zijdewind, 2005).

However, the evidence for caffeine as an ergogenic aid for short-term, high intensity athletic performance is mixed (Anselme et al., 1992; Bell, Jacobs, & Ellerington, 2001; Bell, Jacobs, & Zamecnik, 1998; Bruce et al., 2000; Collomp et al., 2002; Collomp, Ahmaidi, Audran, and Prefaut, 1991; Collomp et al., 1992; Denadai & Denadai, 1998; Doherty, Smith, Davison, & Hughes, 2002; Doherty, Smith, Hughes, & Davison, 2004; Greer et. al, 1998; Jackman, Wendling, Friars, & Graham, 1996; Jacobs, Pasternak, & Bell, 2003; Jacobson, Weber, Claypool, & Hunt, 1992;
Paton, Hopkins, & Vollebregt, 2001; Wiles, Bird, Hopkins, & Riley, 1992). For example, caffeine ingestion (2 to 9 mg/kg BW) during short-term, high-intensity protocols (Bruce et al., 2000; Doherty et al., 2002; Doherty et al., 2004; Jackman et al., 1996; Wiles et al., 1992), sprint protocols (Anselme et al., 1992; Collomp et al., 1992), and strength and power exercise (Jacobson et al., 1992) significantly improved exercise performance. In other studies, caffeine ingestion (~3.6 to 7 mg/kg BW) during short-term, high-intensity cycling protocols (Bell et al., 1998; Collomp et al., 2002), Wingate tests (Bell et al., 2001; Collomp et al., 1991; Greer et al., 1998), sprint protocols (Collomp et al., 1992; Paton et al., 2001; Williams, Signorile, Barnes, & Henrich, 1988), and strength and power exercise (Jacobs et al., 2003) did not improve performance. Mixed results may be seen due to training status of research participants, habitual use of caffeine by research participants, dose of caffeine used in the studies, and performance outcome measures.

Because of the widespread use of caffeine by athletes who engage in both aerobic and anaerobic activities, further research is needed to determine its efficacy as an ergogenic aid. Athletes that are regular consumers of caffeine may respond differently to caffeine in a research study than non-users of caffeine. Unfortunately, most previous studies have not examined typical caffeine intake of the research participants. Few studies have examined the impact of caffeine on biochemical markers during high-intensity exercise, such as the Wingate test. This information may aid in understanding the mechanisms by which caffeine exerts an ergogenic action on anaerobic exercise performance. Heart rate (HR) and blood pressure (BP) should be monitored for the safety of the participants and to help determine any negative side
effects. In addition, few studies have examined the effect of caffeine on short-term, high-intensity exercise or athletic performance in highly trained athletes. Thus, studies are needed that take a comprehensive approach including biochemical and physiological factors, fitness, and diet.

The primary purpose of this study was to examine the effects of a moderate dose of caffeine (5 mg/kg BW) on anaerobic exercise performance, hormonal responses (serum insulin, serum cortisol), exercise metabolites (serum FFA, plasma lactate, and plasma glucose), physiological responses (BP and HR), and rating of perceived exertion (RPE) in highly trained male athletes. Our primary null hypothesis was that the ingestion of a shake with a 5.0 mg/kg BW dose of caffeine would not alter exercise performance (leg press, chest press, 30-second Wingate test), hormonal responses, exercise metabolites, physiological responses, and RPE compared to a placebo in male athletes during anaerobic exercise.

Methodology

This randomized, double-blind, crossover study examined the impact of a moderate dose of caffeine (5 mg/kg BW) during anaerobic exercise in nineteen highly trained, competitive male athletes, 18 to 40 years of age.

Recruitment flyers were posted at Athletes’ Performance, a facility that trains elite and professional athletes. This study was approved by the Human Subjects Institutional Review Board (IRB) at Arizona State University and the procedures were conducted in accordance with these guidelines.

Participants
Participants were eligible if they fit the following inclusion criteria: male, between 18 and 40 years of age, and participated in ≥ 12 h/wk of programmed physical activity. Participants were currently participating in a comprehensive training program, including strength, movement, and endurance activities, for two to four hours per day.

Participants reported to Athletes’ Performance on two occasions separated by one week for exercise testing. Participants fasted for 8 to 12 hours and abstained from caffeinated products for 48 hours. To assess their historical caffeine intake, participants completed the Arizona Food Frequency Questionnaire (AFFQ) (University of Arizona, Tucson, AZ) (Martinez et al., 1999). They provided a urine sample at baseline to detect the presence of caffeine. Heart rate and blood pressure were also monitored. An 8-hour fasting blood sample was taken by venipuncture for assessment of serum cortisol, serum insulin, serum free fatty acids (FFA), plasma lactate, and plasma glucose concentrations.

Immediately after the blood draw, participants consumed a commercial shake either with caffeine (caffeine, 5.0 mg/kg BW; CHO, 0.125 g/kg BW) or without caffeine (placebo; CHO, 0.125 g/kg BW). Other ingredients in the shake included sucrose, natural and artificial flavors, pectin, mannitol, citric acid, sucralose, malic acid, Food & Drug and Cosmetics (FD&C) yellow #5, silicon dioxide, and FD&C blue #1. Fifteen minutes after ingesting the shake, participants ate a breakfast consisting of 8 fl oz of orange juice, 2 cups of Cheerios®, 8 fl oz of 1% milk, 2 pieces of toast, 2 T of peanut butter, and 1 banana (~917 kcal; 14% protein, 62% CHO, 24% fat). Researchers noted the amount of breakfast consumed by each participant. A mid-point heart rate and blood pressure were measured 30 minutes after the participants drank the shake. Sixty
minutes after consuming the shake, participants performed three exercise tests with 60 seconds of rest between each test. This scenario is similar to how an athlete would use caffeine in competition or practice.

Prior to the exercise tests, the participants completed a ten minute warm-up consisting of both dynamic and static stretches. The exercise tests consisted of a leg press, chest press, and 30-second Wingate test. The order of the leg press and chest press was determined at random; the Wingate test was always the final exercise test. Participants completed the leg and chest press on Keiser exercise equipment (model A420, Fresno, CA), which utilizes pneumatic resistance during the extension phase. Participants were asked to push maximally with each repetition until a full extension could no longer be performed. The number of repetitions completed and total weight lifted (pounds x number of repetitions) were recorded after each test. The Wingate test was completed on a Peak Bike by Monark (Ergomedic model 894E, Vansbro, Sweden). Participants cycled with no resistance until they reached their perceived maximal speed. At this time, the pre-determined load (7.5% body weight) was dropped and the test continued at maximal effort for 30 seconds. The peak power, average power, minimum power, and power drop were calculated during the Wingate test using the Monark Anaerobic Test Software (version 1.0). Peak power was the highest power output achieved during any five second interval. Average power was the average power output over the 30-second test. Minimum power was the lowest power output achieved during any five second interval. Power drop was the difference between the peak power and minimum power.
Participants estimated perceived effort after each exercise test using Borg's Rating of Perceived Exertion (RPE) (scale between 6 and 20). Researchers measured HR and BP immediately after research participants completed the Wingate test, and then a post-exercise blood sample was drawn to assess for serum cortisol, serum insulin, serum free fatty acids (FFA), plasma lactate, and plasma glucose concentrations. Participants also provided a post-exercise urine sample to detect for the presence of caffeine.

*Anthropometric Measures*

Height was measured (without shoes) to the nearest cm using a stadiometer with each participant's back against the wall. Each participant’s weight was measured to the nearest kg using a digital platform scale (Avadia model 758, Webb City, MO).

*Blood Analysis*

Approximately 20 mL of blood was taken twice by venipuncture by a trained phlebotomist at each visit. The first blood draw (fasting) was collected at arrival, and participants performed three exercise tests 60 minutes later. The second blood draw was collected immediately after the final exercise test (post-exercise). After the blood samples were collected, they were immediately placed on ice and centrifuged within 30 minutes at 4°C. The blood was separated and samples stored at -45°C until analysis. Concentrations of serum cortisol, serum insulin, serum FFA, plasma lactate, and plasma glucose were determined for each participant during each treatment (caffeine, placebo) and time (fasting, post-exercise).

Plasma glucose concentrations were run in duplicate at ASU using an enzymatic kit from Sigma Diagnostics (St. Louis, MO). The intra-assay variation was 2.2%, and the
inter-assay variation was 4.4%. Serum insulin concentrations were run in duplicate at ASU by radioimmunoassay using the ImmuChem Insulin $^{125}$I RIA assay kit (MP Biomedicals Diagnostics Division, Orangeburg, NY). The intra-assay variation was 3.7%, and the inter-assay variation was 10.8%. Serum cortisol was determined by radioimmunoassay using the ImmuChem Cortisol $^{125}$I RIA assay kit (MP Biomedicals Diagnostics Division, Orangeburg, NY). The intra-assay variation was 3.0%, and the inter-assay variation was 12.5%. Serum FFA and plasma lactate were analyzed by Sonora Quest Laboratories (Phoenix, AZ). Serum FFA concentrations were determined by an enzymatic assay using spectrophotometry, and plasma lactate concentrations were determined using a photometric assay.

Urine Analysis

Participants provided approximately 100 mL of urine twice (fasting and post-exercise) at each study visit. Urine was analyzed for the presence or absence of caffeine by Sonora Quest Laboratories (Phoenix, AZ) using thin layer chromatography.

Dietary Intake

Participants completed the AFFQ to determine their usual eating patterns over the past few months. The AFFQs were analyzed by the NCS OpScan 5 [Al-Bassam International Company (ABI) Al-Khobar, Saudi Arabia]. Usual caffeine intake was assessed for each research participant.

Power Calculations

Power calculations were completed to determine the participant sample size. Based upon post-exercise plasma glucose concentrations (Doherty et al., 2002; Greer et al., 1998), blood lactate concentrations (Collomp et al., 1992; Macintosh & Wright,
1995), serum FFA concentrations (Trice & Haymes, 1995), and plasma insulin concentrations (Graham, Helge, MacLean, Kiens, & Richter, 2000), it was determined that 20 participants would be recruited. However, only 19 research participants completed the study.

Statistical Analysis

The data were assessed for normality by examining histograms depicting the distributions, normal probability plots, and the Kolmogorov-Smirnov statistic (Altman, 1995). Tests for normality were met for all variables except serum insulin, plasma lactate, leg press (total weight), RPE, and blood pressure. Serum insulin and plasma lactate values were transformed using the inverse, and leg press data were transformed using the inverse square root (Bland & Altman, 1996). Mean and standard deviations presented in the result tables are the untransformed values; the p-values reflect the results using the transformed data. A non-parametric test was used for analysis of RPE, systolic blood pressure (SBP), and diastolic blood pressure (DBP).

Descriptive statistics [age, height, weight, body mass index (BMI)] were expressed as mean ± standard deviation. Paired t-tests determined the independent effects of caffeine versus no caffeine on exercise performance during the bench press, leg press, and Wingate test. A repeated-measures analysis of variance (ANOVA) determined the independent effects of caffeine versus no caffeine on plasma glucose, serum insulin, serum FFA, plasma lactate, serum cortisol concentrations, and heart rate. The Bonferroni post-hoc test was used to determine differences between the pre-exercise, mid-point, and post-exercise heart rate values. The Wilcoxon Signed Rank test determined the independent effects of the treatment on RPE as well as treatment
and time effects on blood pressure. All values were expressed as mean ± standard deviation, and results were found to be significant at the 0.05 level. Statistical analyses were completed using the Statistical Package for Social Sciences (SPSS) (version 14.0, 2005, Chicago, IL).

Results

Nineteen participants completed this study; however, one participant was excluded from the analysis because his blood markers (elevated glucose and insulin) suggested that he was not in a fasted state at baseline during the caffeine trial. Therefore, eighteen participants were included in the final analysis (Table 1). Most participants were not regular users of caffeine; typical dietary caffeine intake was 40.8 ± 51.0 mg/day using the AFFQ. Thirteen participants consumed ≤ 50 mg/day (caffeine naive), and five participants were low to moderate users, consuming > 50 mg/day (between 68 and 179 mg/day). All participants consumed less than the average daily use of caffeine in the United States population (193 mg/day) (Frary, Johnson, & Wang, 2005).

**Urinary Caffeine**

All participants tested negative in the urine for the presence of caffeine at study entry. Participants also tested negative for caffeine post-exercise during the placebo trial. During the caffeine trial, participants tested positive for caffeine post-exercise.

**Exercise Performance**

Performance on the exercise tests was analyzed using paired t-tests. Total weight lifted in the chest press was significantly greater for the caffeine trial compared to the placebo trial (p < .05) (Table 2), with 67% of the participants showing improvement
in the caffeine trial. Although total weight lifted in the leg press was not significantly higher for the caffeine trial compared to the placebo trial \((p = .09)\) (Table 2), 78% of the participants showed improvement in the caffeine trial. During the Wingate test, peak power was significantly greater during the caffeine trial compared to the placebo trial \((p < .05)\) (Table 2), with 78% of the participants showing improvement in the caffeine trial. Although mean scores for average power and minimum power were greater during the caffeine trial, the results were not statistically significant (Table 2). Power drop was less during the caffeine trial compared to the placebo trial; however, the results were not statistically significant (Table 2). Furthermore, only 50% of the participants had a higher minimum power and less of a power drop with the caffeine trial compared to the placebo trial. However, 72% of the participants obtained a greater average power with the caffeine trial compared to the placebo trial.

**Hormonal Responses and Exercise Metabolites**

Fasting and post-exercise blood samples were obtained from each participant and analyzed for concentrations of serum cortisol, serum insulin, serum FFA, plasma lactate, and plasma glucose. All fasting blood samples for each participant were within normal limits. Using paired t-tests, no differences in fasting concentrations of the blood markers were observed between the caffeine and placebo treatments. Using paired t-tests, post-exercise plasma lactate, serum cortisol, plasma glucose, and serum insulin concentrations were all significantly higher during the caffeine than the placebo treatment \((\text{lactate: caffeine } = 14.4 \pm 4.0, \text{ placebo } = 12.1 \pm 2.9 \text{ mmol/L}; \text{ cortisol: caffeine } = 635 \pm 166, \text{ placebo } = 524 \pm 138 \text{ nmol/L}; \text{ glucose: caffeine } = 5.6 \pm 0.9, \text{ placebo } = 4.9 \pm 0.7 \text{ mmol/L}; \text{ insulin: caffeine } = 264 \pm 111, \text{ placebo } = 208 \pm 97 \text{ pmol/L}; p < .05)\). Post-
exercise serum FFA were not significantly different between treatments (caffeine = 0.17 ± 0.07, placebo = 0.15 ± 0.05 mmol/L).

Using two-way repeated-measures ANOVA, blood markers were analyzed to test for differences in time, treatment, and their interaction. The time main effect was significant for serum insulin, plasma lactate, and serum FFA. Post-exercise blood concentrations significantly increased for serum insulin (p < .05) and plasma lactate (p < .05) (Table 3). Post-exercise blood concentrations significantly decreased for serum FFA for both treatments (p < .05) (Table 3).

The treatment main effect was significant for serum insulin and plasma glucose. Concentrations for serum insulin and plasma glucose concentrations were significantly higher during the caffeine treatment than the placebo treatment (p < .05) (Table 3). In addition, a significant interaction (treatment by time) was observed for serum cortisol and plasma glucose concentrations (p < .05) with an increase in the post-exercise caffeine trial but a decrease in the post-exercise placebo trial (Table 3). There were no significant treatment main effects for serum cortisol, serum FFA, and plasma lactate (Table 3). There were no significant interaction effects (treatment by time) for plasma lactate, serum insulin, and serum FFA (Table 3).

**Heart Rate and Blood Pressure**

A two-way repeated-measures ANOVA and Bonferroni post hoc test examined differences in HR by time, treatment, and their interaction. Post-exercise values for HR were significantly higher than both the fasting and mid-point measurements (p < .05) (Table 4). No differences in heart rate were seen for the treatment main effect or the interaction of treatment and time.
Because the SBP and DBP data were not normally distributed, the Wilcoxon Signed Rank Test was used to test for differences. Post-exercise values for SBP were significantly higher than both the fasting and mid-point measurements (p < .05) (Table 4). Post-exercise values for DBP were significantly lower than both the fasting and mid-point measurements (p < .05) (Table 4). Mid-point DBP was significantly higher than the fasting DBP measurement within the caffeine treatment (p < .05) (Table 4). Post-exercise SBP was significantly higher with the caffeine treatment compared to the placebo treatment (p < .05) (Table 4). Mid-point DBP was significantly higher with the caffeine treatment compared to the placebo treatment (p < .05) (Table 4). No significant differences in blood pressure were seen for the interaction of treatment and time.

We separated our research participants into two different groups: responders and non-responders. The responders (n=5) had > 10% increase in systolic BP at the mid-point reading during the caffeine trial. The increase in systolic BP at the mid-point reading was ≤ 10% for the non-responders (n=13). Four of the five responders reported daily caffeine intakes < 50 mg/day. Forty percent of the responders and 77% of the non-responders lifted more total weight on the chest press during the caffeine trial. One-hundred percent of the responders and 69% of the non-responders obtained a greater peak power and lifted more total weight on the leg press during the caffeine trial. One-hundred percent of the responders and 61% of the non-responders obtained a greater average power during the caffeine trial.

**Rating of Perceived Exertion**

Because RPE data were not normally distributed, the Wilcoxon Signed Rank Test was used for the analysis. No differences were observed for RPE between
treatments in any of the exercises (chest press: caffeine = 19 ± 1, placebo = 20 ± 1; leg press: caffeine = 19 ± 1, placebo = 19 ± 1; Wingate test: caffeine = 19 ± 2, placebo = 19 ± 1).

Discussion

This study examined the effect of a moderate dose of caffeine (5 mg/kg BW) on anaerobic exercise performance, hormonal responses (serum cortisol, serum insulin), exercise metabolites (serum FFA, plasma lactate, and plasma glucose), physiological responses (HR and BP), and RPE in highly trained, competitive male athletes. In our study, all of the participants consumed less than the US population’s average intake of caffeine of 193 mg/d (Frary et al., 2005). Thus, the participants were caffeine naive or very low caffeine consumers.

Exercise Performance

Performing a chest press and leg press to fatigue is a measure of muscular endurance used primarily during the anaerobic system. In our study, caffeine ingestion significantly improved performance for the chest press but not the leg press. The leg press uses larger muscle groups than the chest press. Thus, if caffeine acts directly on the skeletal muscle or increases muscle fiber recruitment, one would expect a greater effect of caffeine in the leg press. The chest press and leg press were administered in a random order to control for fatigue.

In our study, the participants were caffeine naive or very low caffeine consumers. Some participants reported feeling shaky or jittery and one participant vomited at the end of the testing. These feelings may have negatively impacted their performance.
Unfortunately, our statistical tests were also underpowered (leg press, 0.161). Thus, if more participants were included, significant results may have been obtained. However, 67% of the participants lifted more total weight during the caffeine trial for the leg press and chest press.

Jacobs et al. (2003) also examined the impact of caffeine on exercise performance during the leg press and bench press. In this study, no significant differences were observed with the caffeine trial compared to the placebo trial. However, the participants lifted slightly more weight in the leg press and bench press during the first set of three super-sets after caffeine ingestion. Jacobs et al. included recreational athletes instead of the highly trained athletes used in our study. Also, Jacobs et al. administered a lower dose of caffeine (4 mg/kg BW) 90 minutes prior to testing instead of the 5 mg/kg BW dose 60 minutes prior to testing in our study. The dose of caffeine used, the timing of the dose, and the fitness level of the participants may explain the differences in the results. Elite athletes generally have more muscle mass than recreational athletes. If caffeine acts directly on muscle fibers, the effect of caffeine would be greater in individuals with a larger muscle mass. However, it could be that a larger dose of caffeine is needed to get an ergogenic effect for short-term, high-intensity exercise.

Collomp et al. (1992) compared the effects of caffeine in trained and untrained swimmers and found a significantly greater swimming velocity using 4.3 mg/kg BW of caffeine during 100-m swimming sprints in the trained participants only. Using a 7 mg/kg BW dose of caffeine with elite football athletes, Jacobson et al. (1992) observed a significantly greater peak torque during leg extension and leg flexion exercises after
caffeine ingestion. Anselme et al. (1992) found a significantly greater maximal anaerobic power using 3.5 mg/kg BW of caffeine during high intensity cycling with recreational athletes. However, other studies found no significant differences in performance with recreational athletes using 3.5 to 4 mg/kg BW during a 10-km run (Bell & McClellan, 2002), cycle ergometer graded exercise test (Dodd, Brooks, Powers, & Tulley, 1991), and high intensity cycling (Collomp et al., 2002). The potential to improve may be small and difficult to measure in short-term, high-intensity exercise performance. In addition, studies have used a variety of exercise protocols, doses of caffeine, and research participants, thus making it difficult to compare results.

A Wingate test was used to measure anaerobic power output. Our results indicated that caffeine ingestion resulted in a significantly greater peak power. An increase in peak power is important for competitions involving sprints or power lifting. Average power, minimum power, and power drop were not significantly different between treatments. However, 72% of the participants obtained a greater average power during the caffeine trial compared to the placebo trial. Bell et al. (2001), Collomp et al. (1991), and Greer et al. (1998) found no significant differences in exercise performance on the Wingate test with caffeine ingestion. However, non-significant improvements with caffeine ingestion were observed by Bell et al. and Collomp et al. Greer et al. also found non-significant improvements in the first three of four Wingate tests. The dose of caffeine was 5 mg/kg BW in the first two studies (Bell et al., 2001; Collomp et al., 1991) and 6 mg/kg BW in the third study (Greer et al., 1998). Greer et al. found a significant negative effect from caffeine for peak power in the last of four repeated Wingate tests. Conflicting results may be related to the training level of the
participants. In the present study, highly trained athletes were evaluated, whereas in the other three Wingate studies, untrained or recreational participants were examined. Thus, caffeine may be ineffective for improving anaerobic performance during the Wingate test in recreational trained or untrained participants.

The participants in our study were all relatively low users of caffeine, consuming less than the average daily intake of caffeine in the United States. Attwood, Higgs, and Terry (2007) found that the sensitivity of caffeine consumers to the performance-enhancing effects of caffeine is related to habitual intake. They found that higher caffeine consumers performed better on cognitive tests after a dose of caffeine than moderate consumers of caffeine. Thirteen of our participants consumed less than 50 mg/day of caffeine while five participants consumed between 68 and 179 mg/day. Because our research participants were low users of caffeine, we might have found different results in exercise performance if they had been high caffeine users.

**Hormonal Responses**

In our study, post-exercise insulin concentrations were significantly increased, which would be expected after eating a meal. There was also a treatment main effect during the caffeine trial with higher insulin concentrations compared to the placebo trial. These results are in agreement with Collomp et al. (2002). In our study, similar to Collomp et al., the research participants ate a pre-exercise meal prior to testing. During competition or practice, an athlete would use caffeine in addition to consuming a meal or snack.

Keijzers, De Galan, Tack, and Smits (2002) observed that an acute caffeine infusion decreased insulin sensitivity (~15%) in healthy humans. However, the decrease
may have resulted from elevated plasma epinephrine concentrations. Petrie et al. (2004) also showed that acute caffeine ingestion significantly reduced insulin sensitivity in obese, non-diabetic males. However, the protocol used in these studies did not include measuring the impact of caffeine on insulin sensitivity during exercise. The additional increase in insulin concentrations observed after caffeine ingestion in our study is most likely due to increased blood glucose concentrations.

In the present study, a significant interaction (treatment/time) effect of caffeine was observed in serum cortisol concentrations after caffeine ingestion. Cortisol concentrations increased during the caffeine trial and decreased during the placebo trial. These results are in agreement with Laurent et al. (2000) and MacIntosh and Wright (1995). Cortisol is known to elevate blood glucose and FAA to ensure an adequate fuel supply. In our study, we saw an increase in cortisol concentrations after caffeine ingestion which may be responsible for the increase seen in glucose concentrations. However, there was a significant decrease in post-exercise FFA concentrations in both treatments possibly due to an increase in post-exercise insulin concentrations. Glucose and FFA availability is not the limiting factor in short-term, high-intensity exercise protocols.

**Exercise Metabolites**

In our study, the participants ate a meal forty-five minutes prior to testing. In addition, the shake also contained CHO. Glucose is needed for anaerobic glycolysis during short-term, high-intensity exercise bouts. Other studies have reported an increase in blood glucose with caffeine ingestion (Graham et al., 2000; Graham & Spriet, 1995; Trice & Haymes, 1995). In the present study, post-exercise glucose
concentrations were significantly higher in the caffeine trial compared to the placebo trial. There was also a treatment main effect with higher plasma glucose concentrations during the caffeine trial compared to the placebo trial. Bell et al. (2001) also observed significantly higher post-exercise glucose concentrations with caffeine ingestion compared to the placebo trial, but post-exercise concentrations were higher than pre-exercise concentrations with both treatments. Denadai and Denadai (1998) also observed a slight increase in post-exercise glucose concentrations with caffeine ingestion and a slight decrease in post-exercise glucose concentrations with the placebo trial, but no significant differences were observed between trials. Bell et al. (1998), Collomp et al. (2002), and Greer et al. (1998) observed slight increases in post-exercise glucose concentrations with the caffeine trial tending to be higher than the placebo trial. The caffeine ingestion may lead to a reduction in glucose uptake (via a caffeine-induced reduction in insulin sensitivity) or an increase in gluconeogenesis resulting in elevated blood glucose concentration.

During anaerobic exercise, lactate is formed during the metabolism of glucose instead of proceeding to the Kreb’s cycle or aerobic metabolism (Balady et al., 2000). Caffeine may possibly inhibit lactate clearance. In the present study, plasma lactate concentrations were significantly higher post-exercise in both trials compared to fasting concentrations. These results are consistent with the literature (Bell et al., 2001; Bell et al., 1998; Collomp et al., 2002; Collomp et al., 1991; Collomp et al., 1992; Denadai & Denadai, 1998; Greer et al., 1998). No significant treatment main effect was observed with plasma lactate concentrations. Some studies report no differences with caffeine in post-exercise plasma lactate concentrations during high-intensity cycling protocols.
(Denadai & Denadai, 1998; Jackman et al., 1996) or the Wingate test (Greer et al., 1998). However, other research studies have observed that post-exercise lactate concentrations are significantly increased with caffeine compared to a placebo trial during high-intensity cycling protocols (Anselme et al., 1992; Bell et al., 2001; Bell et al., 1998; Collomp et al., 2002) and the Wingate test (Bell et al., 2001; Collomp et al., 1991).

Fat oxidation does not play as big a role in short-term, high-intensity exercise protocols. In our study, post-exercise FFA concentrations were significantly reduced during both treatments. These results are similar to other short-term, high-intensity exercise studies (Bell et al., 1998; Denadai & Denadai, 1998). Bell et al. showed a significant decrease in FFA concentrations after exercise with both treatments. In that study, the post-exercise FFA concentrations were significantly higher with caffeine ingestion. Denadai and Denadai also reported no significant differences in post-exercise FFA concentrations between treatments during cycling above anaerobic threshold. These results do not support the theory that caffeine increases FFA oxidation, thereby sparing glucose concentrations in these conditions. However, an increase in FFA oxidation and glucose sparing is not as important in anaerobic exercise performance.

**Physiological Responses**

Previous studies have examined physiological responses to caffeine. Most researchers found no effect of acute caffeine ingestion on resting HR (Arciero, Gardner, Benowitz, & Poehlman, 1998; Bond et al., 1987; Bruce et al., 2000; Denadai & Denadai, 1998). However, one study reported an increase in resting HR with acute caffeine ingestion (Robertson et al., 1978). Most researchers have also reported that caffeine
may increase post-exercise HR but not significantly when compared to a placebo trial (Bell et al., 2001; Bond et al., 1987; Denadai & Denadai, 1998; O’Connor, Molt, Broglio, & Ely, 2004). Our data are in agreement with the majority of the research. Our results showed that resting HR was not significantly different between trials. In addition, HR was significantly increased after exercise in both trials but no significant differences were seen in HR between trials.

Fewer studies have examined the impact of caffeine on BP response to exercise. In our study, resting DBP (mid-point) and post-exercise SBP were significantly increased during the caffeine trial compared to the placebo trial. The research literature has documented that regular caffeine intake increases BP (Hartley, Lovallo, & Whitsett, 2004; Noordzij et al., 2005). Other research has reported no significant differences in resting (Arciero et al., 1998; Karatzis et al., 2005) or post-exercise (O’Connor et al., 2004) SBP and DBP after caffeine ingestion. Furthermore, we did not see any trends in sympathetic nervous system response to caffeine and exercise performance.

Potential Mechanisms

Caffeine may exert an effect on the central nervous system (CNS) by acting as a potent adenosine antagonist, thus, blocking adenosine receptors and/or interacting with dopamine receptors (Ribeiro, Sebastiao, & de Mendonca, 2003). Adenosine inhibits the release of most brain excitatory neurotransmitters, particularly dopamine, and may reduce dopamine synthesis (Ribeiro et al., 2003). Because adenosine receptors are abundant in many areas of the brain, caffeine may inhibit adenosine from blocking neuronal transmission and thus alter perception of pain, sympathetic activity, motor
recruitment, and fatigue. In this capacity, caffeine may also increase time to exhaustion. Because caffeine is a dopamine agonist, it could possibly reduce RPE. However, in our study we did not see a reduction in RPE with the caffeine.

Adenosine receptors are abundant in skeletal muscles. In anaerobic activity, the ergogenic effect of caffeine may be due to the antagonism of adenosine receptors. Svenningsson, Nomikos, Ongini, and Fredholm (1997) found that caffeine doses as low as 7.5 mg/kg BW may cause an increase in motor activity in rats primarily through the antagonism of adenosine receptors. Highly trained athletes may have more muscle mass than recreational athletes and may respond to the ergogenic effect of caffeine differently (via the adenosine receptors) than recreational athletes. Thus, caffeine may have a direct effect on muscle that is independent of substrate metabolism. Future studies should examine the impact of caffeine using different muscle groups.

Performance enhancement in anaerobic exercise may also be associated with CNS stimulation (increased motor recruitment and/or masking of fatigue). In tests using maximal voluntary contraction (MVC), the level of muscle activation increases after caffeine ingestion (Kalmar & Cafarelli, 1999; Meyers & Cafarelli, 2005; O’Connor et al., 2004; Williams, Barns, & Gadberry, 1987), suggesting that caffeine may influence sarcoplasmic handling of calcium. However, these results have only been significant using low frequencies, which would primarily affect endurance type activities.

Limitations

In our study, factors such as rest, prior exercise, and over-the-counter medications may have affected the participants’ responses to the caffeine. The training that the participants undertook during the weeks prior to the study may have been quite
different. However, a cross-over design was used to help control for this variability. Furthermore, at the time of study recruitment, the participants were training at the same facility. Thus, a similar training protocol was performed during the two days prior to each testing day. Caffeine may have produced a sympathetic nervous system response exhibited by an increase in HR or BP in some participants and no effect in other participants. We did not ask the participants which treatment they thought they received or how they felt after each treatment. Unfortunately, we did not take a blood sample after the meal just prior to the start of exercise. Information on the effects of caffeine before exercise would have been useful to include in the study design. In addition, plasma caffeine concentrations were not measured. In our study, thirteen of the participants were caffeine naïve. We might have found different results in exercise performance if our research participants had been high caffeine consumers.

Conclusion

Caffeine ingestion combined with a meal significantly enhanced increased upper body muscular endurance during the chest press and increased peak power during the Wingate test. Caffeine and a meal increased post-exercise concentrations of plasma glucose, serum insulin, and serum cortisol during high intensity exercise. Multiple mechanisms may be responsible for performance enhancement in different types of exercise (aerobic versus anaerobic). In anaerobic exercise, the mechanisms for improvement are likely to be independent of any effect of caffeine on fat mobilization. During anaerobic exercise, caffeine may influence the CNS. However, the participants did not report any differences in RPE between the caffeine and placebo trials. The participants in our study were caffeine naïve or very low caffeine consumers, which may
have contributed to the results. Regular consumers of higher doses of caffeine might exhibit more benefits with caffeine as an ergogenic aid. Thus, research is needed that compares the effect of caffeine in caffeine naive versus habituated caffeine consumers in anaerobic exercise performance. Future studies should include different muscle groups, trained versus untrained participants, and habituated caffeine consumers versus caffeine naive participants. Because of the widespread use of caffeine by athletes, more research is needed examining the impact and safety of this ergogenic aid on exercise performance.
References


<table>
<thead>
<tr>
<th>Variable</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>24.1 ± 5.8</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>183 ± 7</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>88 ± 9</td>
</tr>
<tr>
<td>BMI(^b) (kg/m(^2))</td>
<td>26.4 ± 2.2</td>
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<tr>
<td>Typical dietary caffeine intake(^c) (mg)</td>
<td>40.8 ± 51.0</td>
</tr>
<tr>
<td>Caffeine ≤ 50 mg/d(^c) (n)</td>
<td>13</td>
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<tr>
<td>Caffeine &gt; 50 mg/d(^c) (n)</td>
<td>5</td>
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<tr>
<td>Treatment shake</td>
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<tr>
<td>Caffeine dose (mg/kg)</td>
<td>5</td>
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<tr>
<td>CHO(^d) dose (g/kg)</td>
<td>0.125</td>
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<td>Sport</td>
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<td>Baseball (n)</td>
<td>10</td>
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<tr>
<td>Ultimate Fighter (n)</td>
<td>2</td>
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<tr>
<td>Hockey (n)</td>
<td>1</td>
</tr>
<tr>
<td>Triathlete (n)</td>
<td>1</td>
</tr>
<tr>
<td>Football (n)</td>
<td>1</td>
</tr>
<tr>
<td>Gymnast (n)</td>
<td>1</td>
</tr>
<tr>
<td>Basketball (n)</td>
<td>1</td>
</tr>
<tr>
<td>Tennis (n)</td>
<td>1</td>
</tr>
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</table>

Note. \(^a\)Data expressed as mean ± standard deviation. \(^b\)BMI = body mass index. \(^c\)Data obtained from Arizona Food Frequency Questionnaire (AFFQ). \(^d\)CHO = carbohydrate.
Table 2. Exercise performance for study participants (n = 18).^a

<table>
<thead>
<tr>
<th>Variable</th>
<th>Caffeine</th>
<th>Placebo</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest Press TWL(^b) (lb)</td>
<td>5254 ± 1202</td>
<td>4442 ± 1164</td>
<td>.027*</td>
</tr>
<tr>
<td>Leg Press TWL(^b,c) (lb)</td>
<td>15146 ± 5590</td>
<td>13884 ± 5193</td>
<td>.091</td>
</tr>
<tr>
<td>Wingate PP(^d) (W/kg)(^e)</td>
<td>8.88 ± 0.77</td>
<td>8.43 ± 0.83</td>
<td>.024*</td>
</tr>
<tr>
<td>Wingate AveP(^f) (W/kg)(^e)</td>
<td>7.02 ± 0.65</td>
<td>6.82 ± 0.64</td>
<td>.110</td>
</tr>
<tr>
<td>Wingate MinP(^g) (W/kg)(^e)</td>
<td>5.48 ± 0.71</td>
<td>5.23 ± 0.75</td>
<td>.152</td>
</tr>
<tr>
<td>Wingate Pdrop(^h) (W/kg)(^e)</td>
<td>3.30 ± 0.92</td>
<td>3.47 ± 0.97</td>
<td>.562</td>
</tr>
</tbody>
</table>

Note. \(^a\)Data were analyzed using paired t-test; values are mean ± standard deviation. \(^b\)TWL = total weight lifted (lbs x reps). \(^c\)Data transformed using the inverse square root (1/variable) prior to statistical analysis; mean ± standard deviation data are presented untransformed. \(^d\)PP = peak power. \(^e\)W/kg = watts per kilogram of body weight. \(^f\)AveP = average power. \(^g\)MinP = minimum power. \(^h\)Pdrop = power drop.

\(^*\)Significantly different between treatments (p < .05)
Table 3. Effect of caffeine on fasting and post-exercise blood concentrations between two treatments for study participants (n = 18).a

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal Valuesb</th>
<th>Fasting</th>
<th>Post Exercise</th>
<th>Fasting</th>
<th>Post Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactate&lt;sup&gt;c,d&lt;/sup&gt; (mmol/L)</td>
<td>0.6-2.2</td>
<td>1.6 ± 1.1&lt;sup&gt;f&lt;/sup&gt;</td>
<td>14.4 ± 4.0</td>
<td>2.1 ± 1.4&lt;sup&gt;f&lt;/sup&gt;</td>
<td>12.1 ± 2.9</td>
</tr>
<tr>
<td>Cortisol (nmol/L)</td>
<td>138-635</td>
<td>579 ± 138&lt;sup&gt;h&lt;/sup&gt;</td>
<td>635 ± 166&lt;sup&gt;h&lt;/sup&gt;</td>
<td>635 ± 110&lt;sup&gt;h&lt;/sup&gt;</td>
<td>524 ± 138&lt;sup&gt;h&lt;/sup&gt;</td>
</tr>
<tr>
<td>FFA&lt;sup&gt;e&lt;/sup&gt; (mmol/L)</td>
<td>0.07-0.88</td>
<td>0.44 ± 0.20&lt;sup&gt;f&lt;/sup&gt;</td>
<td>0.17 ± 0.07</td>
<td>0.38 ± 0.17&lt;sup&gt;f&lt;/sup&gt;</td>
<td>0.15 ± 0.05</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>3.9-6.4</td>
<td>5.2 ± 0.4&lt;sup&gt;h&lt;/sup&gt;</td>
<td>5.6 ± 0.9&lt;sup&gt;h&lt;/sup&gt;</td>
<td>5.3 ± 0.6&lt;sup&gt;h&lt;/sup&gt;</td>
<td>4.9 ± 0.7&lt;sup&gt;h&lt;/sup&gt;</td>
</tr>
<tr>
<td>Insulin&lt;sup&gt;d&lt;/sup&gt; (pmol/L)</td>
<td>42-181</td>
<td>118 ± 49&lt;sup&gt;f&lt;/sup&gt;</td>
<td>264 ± 111&lt;sup&gt;g&lt;/sup&gt;</td>
<td>118 ± 49&lt;sup&gt;f&lt;/sup&gt;</td>
<td>208 ± 97</td>
</tr>
</tbody>
</table>

Note. aData were analyzed using repeated measures analysis of variance (ANOVA) + Bonferroni post hoc test; values are mean ± standard deviation. bNormal values from Pagana and Pagana (2002). cLactate was not run for 1 participant (n = 17) due to lab error. dData transformed using the inverse (1/variable) prior to statistical analysis; mean ± standard deviation data are presented untransformed. eFFA = free fatty acids. fSignificant time difference within the same treatment (time effect; p < .01). gSignificant treatment difference for given time period (treatment effect; p < .05). hSignificant difference for the interaction (treatment and time; p < .05).
Table 4. The effect of caffeine on heart rate and blood pressure between time and two treatments.a

<table>
<thead>
<tr>
<th>Variable</th>
<th>Caffeine</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preb</td>
<td>Midc</td>
</tr>
<tr>
<td>SBPe (mm Hg)</td>
<td>118 ± 8</td>
<td>121 ± 8</td>
</tr>
<tr>
<td>DBPf (mm Hg)</td>
<td>72 ± 6</td>
<td>77 ± 5 i,j</td>
</tr>
<tr>
<td>HRg (beats per minute)</td>
<td>68 ± 9</td>
<td>65 ± 8</td>
</tr>
</tbody>
</table>

Note. aBlood pressure data were analyzed using Wilcoxon Signed Rank Test; heart rate data were analyzed using repeated measures analysis of variance (ANOVA) + Bonferroni post hoc test; no significant difference was observed for the interaction (treatment and time); values are mean ± standard deviation. bPre = Fasting. cMid = 30 min Post-Treatment. dPost = Post-Exercise. eSBP = systolic blood pressure. fDBP = diastolic blood pressure. gHR = heart rate. hSignificant difference from Pre and Mid by time (time effect; p < .05). iSignificant difference to Pre by time (time effect; p < .05). jSignificant difference between treatments at the same time (treatment effect; p < .05).