

## The Effect of Caffeine as an Ergogenic Aid in Anaerobic Exercise

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The study examined caffeine (5 mg/kg body weight) vs. placebo during anaerobic exercise. Eighteen male athletes ( $24.1 \pm 5.8$  yr; BMI  $26.4 \pm 2.2$  kg/m<sup>2</sup>) 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 were observed between treatments for the leg press and average power, minimum power, and power drop (Wingate test). There was a significant treatment main effect found for postexercise glucose and insulin concentrations; higher concentrations were found in the caffeine trial. A significant interaction effect (treatment and time) was found for cortisol and glucose concentrations; both increased with caffeine and decreased with placebo. Postexercise systolic blood pressure was significantly higher during the caffeine trial. No differences were 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 attained during the Wingate test in competitive athletes.

**Keywords:** hormonal responses, exercise metabolites, physiological responses, RPE

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. It is also present in some widely consumed over-the-counter medications such as 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 its prohibited list and added it to its monitoring program during competition (WADA,

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2007b). WADA monitors substances like caffeine during competition to detect patterns of misuse in sport (WADA, 2007a). The National Collegiate Athletic Association still considers a urinary level of  $>15 \mu\text{g/ml}$  after competition illegal (National Collegiate Athletic Association, 2007). The WADA antidoping code has been accepted by many sport organizations including the International Olympic Committee. It takes about 13 mg per kilogram body weight (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 might be improved by ingesting as little as 150–250 mg of caffeine (~1–2 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 improves performance over a wide range of exercise protocols, particularly prolonged and exhaustive exercises (Bell & McClellan, 2003; Bell & McClellan, 2002; Graham & Spriet; Pasmán, 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).

The evidence for caffeine as an ergogenic aid for short-term, high-intensity athletic performance, however, 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, Chanal, & Prefaut, 1991; Collomp et al., 1992; Denadai & Denadai, 1998; Doherty, Smith, Davison, & Hughes, 2002; Doherty, Smith, Hughes, & Davison, 2004; Greer, McLean, & Graham, 1998; Jackman, Wendling, Friars, & Graham, 1996; Jacobs, Pasternak, & Bell, 2003; Jacobson, Weber, Claypool, & Hunt, 1992; Paton, Hopkins, & Vollebregt, 2001; Wiles et al., 1992). For example, caffeine ingestion (2–9 mg/kg BW) during short-term, high-intensity protocols (Bruce et al.; Doherty et al., 2002, 2004; Jackman et al.; Wiles et al.), sprint protocols (Anselme et al.; Collomp et al., 1992), and strength and power exercise (Jacobson, 1992) significantly improved exercise performance. In other studies, caffeine ingestion (~3.6–7 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.), sprint protocols (Collomp et al., 1992; Paton et al.; Williams, Signorile, Barnes, & Henrich, 1988), and strength and power exercise (Jacobs et al.) did not improve performance. The mixed results might be a result of 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 who regularly consumer caffeine might respond differently to caffeine in a research study than nonusers 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 might help us understand 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 and serum cortisol), exercise metabolites (serum free fatty acids [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 ingesting a shake with a 5.0-mg/kg BW dose of caffeine would not alter exercise performance (leg press, chest press, and 30-s Wingate test), hormonal responses, exercise metabolites, physiological responses, and RPE compared with 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 19 highly trained competitive male athletes, 18–40 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 at Arizona State University, and the procedures were conducted in accordance with their guidelines.

## Participants

Participants were eligible if they were male, 18–40 years of age, and participated in  $\geq 12$  hr/week of programmed physical activity. They were currently participating in a comprehensive training program including strength, movement, and endurance activities for 2–4 hr/day.

Participants reported to Athletes' Performance on two occasions separated by 1 week for exercise testing. They fasted for 8–12 hr and abstained from caffeinated products for 48 hr. So that we could assess their historical caffeine intake, participants completed the Arizona Food Frequency Questionnaire (AFFQ; University of Arizona, Tucson, AZ; Martinez et al., 1999). Each provided a urine sample at baseline to detect caffeine. HR and BP were also monitored. An 8-hr fasting blood sample was taken by venipuncture to assess serum cortisol, serum insulin, serum 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; carbohydrate [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 and 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 fluid oz of orange juice, 2 cups of Cheerios, 8 fluid oz of 1% milk, two pieces of toast, 2 tablespoons of peanut butter, and one banana (~917 kcal; 14% protein, 62% CHO, 24% fat). Researchers noted the amount of food and drink consumed by each participant. A midpoint HR and BP were measured 30 min after the participants drank the shake. Sixty minutes after consuming the shake, participants performed three exercise tests with 60 s of rest between tests. This scenario is similar to how an athlete would use caffeine in competition or practice.

Before the exercise tests, the participants completed a 10-min warm-up consisting of both dynamic and static stretches. The exercise tests consisted of a leg press, chest press, and 30-s Wingate test. The order of the leg press and chest press was determined at random; the Wingate test was always the final test. Participants completed the leg and chest press on Keiser exercise equipment (model A420, Fresno, CA), which uses pneumatic resistance during the extension phase. Participants were asked to push maximally with each repetition until they could no longer perform a full extension. The number of repetitions completed and total weight lifted (pounds  $\times$  number of repetitions) were recorded after each test. The Wingate test was completed on a Peak Bike by Monark (Ergomic model 894E, Vansbro, Sweden). Participants cycled with no resistance until they reached their perceived maximal speed. At this time, the predetermined load (7.5% BW) was dropped and the test continued at maximal effort for 30 s. The peak power, average power, minimum power, and power drop were calculated during the Wingate test using Monark's anaerobic test software (version 1.0). Peak power was the highest power output achieved during any 5-s interval. Average power was the average power output over the 30-s test. Minimum power was the lowest power output achieved during any 5-s interval. Power drop was the difference between the peak power and minimum power.

Participants estimated perceived effort after each exercise test using Borg's RPE (scale from 6 to 20). Researchers measured HR and BP immediately after participants completed the Wingate test, and then a postexercise blood sample was drawn to assess for serum cortisol, serum insulin, serum FFA, plasma lactate, and plasma glucose concentrations. Participants also provided a postexercise urine sample to detect the presence of caffeine.

## **Anthropometric Measures**

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

## **Blood Analysis**

A sample of 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 min later. The second blood draw was collected immediately after the final exercise test (postexercise).

After the blood samples were collected, they were immediately placed on ice and centrifuged within 30 min 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, postexercise).

Plasma glucose concentrations were run in duplicate at Arizona State University using an enzymatic kit from Sigma Diagnostics (St. Louis, MO). The intra-assay variation was 2.2%, and the interassay variation was 4.4%. Serum insulin concentrations were run in duplicate at Arizona State University by radioimmunoassay using the ImmuChem insulin <sup>125</sup>I RIA assay kit. The intra-assay variation was 3.7%, and the interassay variation was 10.8%. Serum cortisol was determined by radioimmunoassay using the ImmuChem cortisol <sup>125</sup>I RIA assay kit (MP Biomedicals Diagnostics Division, Orangeburg, NY). The intra-assay variation was 3.0%, and the interassay 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 postexercise) 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 so we could determine their usual eating patterns over the past few months. The AFFQs were analyzed by the NCS OpScan 5 (Al-Bassam International, 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 on postexercise 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), we determined that 20 participants would be recruited. Only 19 research participants completed the study, however.

## Statistical Analysis

We assessed the data for normality by examining histograms depicting the distributions, normal probability plots, and the Kolmogorov–Smirnov statistic (Altman & Bland, 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). Means and standard deviations presented in the result tables are the untransformed values;

the  $p$  values reflect the results using the transformed data. A nonparametric test was used to analyze RPE, systolic blood pressure (SBP), and diastolic blood pressure (DBP).

Descriptive statistics (age, height, weight, and body-mass index) were expressed as  $M \pm SD$ . 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 HR. Bonferroni's post hoc test was used to determine differences between the preexercise, midpoint, and postexercise HR values. Wilcoxon's signed rank test determined the independent effects of the treatment on RPE, as well as treatment and time effects on BP. All values were expressed as  $M \pm SD$ , and results were considered significant at the .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; 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, 18 participants were included in the final analysis (Table 1). Most participants were not regular caffeine users; typical dietary caffeine intake was  $40.8 \pm 51.0$  mg/day according to the AFFQ. Thirteen participants consumed  $\leq 50$  mg/day (caffeine

**Table 1** Characteristics of Study Participants ( $N = 18$ )

Variable	Value
Age (y), $M \pm SD$	$24.1 \pm 5.8$
Height (cm), $M \pm SD$	$183 \pm 7$
Weight (kg), $M \pm SD$	$88 \pm 9$
Body-mass index ( $\text{kg}/\text{m}^2$ ), $M \pm SD$	$26.4 \pm 2.2$
Typical dietary caffeine intake <sup>a</sup> (mg), $M \pm SD$	$40.8 \pm 51.0$
Caffeine $\leq 50$ mg/d <sup>a</sup> , $n$	13
Caffeine $> 50$ mg/d <sup>a</sup> , $n$	5
Treatment shake	
caffeine dose (mg/kg)	5
carbohydrate dose (g/kg)	0.125
Sport	
baseball, $n$	10
ultimate fighter, $n$	2
hockey, $n$	1
triathlete, $n$	1
football, $n$	1
gymnast, $n$	1
basketball, $n$	1
tennis, $n$	1

<sup>a</sup>Data obtained from Arizona Food Frequency Questionnaire.

naive), and 5 participants were low to moderate users, consuming >50 mg/day (68–179 mg/day). All participants consumed less than the average daily amount of caffeine in the U.S. population (193 mg/day; Frary, Johnson, & Wang, 2005).

## Urinary Caffeine

All participants' urine tested negative for the presence of caffeine at study entry. They also tested negative for caffeine postexercise during the placebo trial. During the caffeine trial, participants tested positive for caffeine postexercise.

## 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 than for 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 in the caffeine trial than in 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 than during 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 than during 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 in the caffeine trial than in the placebo trial, but 72% obtained a greater average power in the caffeine trial than in the placebo trial.

## Hormonal Responses and Exercise Metabolites

Fasting and postexercise 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

**Table 2** Exercise Performance of Study Participants ( $N = 18$ ),  $M \pm SD$

Variable	Caffeine	Placebo	<i>p</i>
Chest press, total weight lifted (lb $\times$ reps.)	5,254 $\pm$ 1,202	4,442 $\pm$ 1,164	.027*
Leg press, total weight lifted (lb $\times$ reps.) <sup>a</sup>	15,146 $\pm$ 5,590	13,884 $\pm$ 5,193	.091
Wingate peak power (W/kg body weight)	8.88 $\pm$ 0.77	8.43 $\pm$ 0.83	.024*
Wingate average power (W/kg body weight)	7.02 $\pm$ 0.65	6.82 $\pm$ 0.64	.110
Wingate minimum power (W/kg body weight)	5.48 $\pm$ 0.71	5.23 $\pm$ 0.75	.152
Wingate power drop (W/kg body weight)	3.30 $\pm$ 0.92	3.47 $\pm$ 0.97	.562

Note. Data were analyzed using a paired *t* test.

<sup>a</sup>Data were transformed using the inverse square root (1 over the square root of the variable) before statistical analysis;  $M \pm SD$  data are presented untransformed.

\*Significantly different between treatments ( $p < .05$ ).

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, postexercise plasma lactate, serum cortisol, plasma glucose, and serum insulin concentrations were all significantly higher during the caffeine than the placebo treatment (lactate: caffeine =  $14.4 \pm 4.0$ , placebo =  $12.1 \pm 2.9$  mmol/L; cortisol: caffeine =  $635 \pm 166$ , placebo =  $524 \pm 138$  nmol/L; glucose: caffeine =  $5.6 \pm 0.9$ , placebo =  $4.9 \pm 0.7$  mmol/L; insulin: caffeine =  $264 \pm 111$ , placebo =  $208 \pm 97$  pmol/L;  $p < .05$ ). Postexercise serum FFA were not significantly different between treatments (caffeine =  $0.17 \pm 0.07$ , placebo =  $0.15 \pm 0.05$  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. Postexercise blood concentrations significantly increased for serum insulin ( $p < .05$ ) and plasma lactate ( $p < .05$ ; Table 3). They significantly decreased for serum FFA with 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 postexercise caffeine trial but a decrease in the postexercise 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).

**Table 3 Effect of Caffeine on Fasting and Postexercise Blood Concentrations Between Two Treatments ( $N = 18$ ),  $M \pm SD$**

Variable	Normal values <sup>a</sup>	Caffeine		Placebo	
		Fasting	Postexercise	Fasting	Postexercise
Lactate <sup>b,c</sup> (mmol/L)	0.6–2.2	$1.6 \pm 1.1^*$	$14.4 \pm 4.0$	$2.1 \pm 1.4^*$	$12.1 \pm 2.9$
Cortisol (nmol/L)	138–635	$579 \pm 138\ddagger$	$635 \pm 166\ddagger$	$635 \pm 110\ddagger$	$524 \pm 138\ddagger$
Free fatty acids (mmol/L)	0.07–0.88	$0.44 \pm 0.20^*$	$0.17 \pm 0.07$	$0.38 \pm 0.17^*$	$0.15 \pm 0.05$
Glucose (mmol/L)	3.9–6.4	$5.2 \pm 0.4\ddagger$	$5.6 \pm 0.9\ddagger\ddagger$	$5.3 \pm 0.6\ddagger$	$4.9 \pm 0.7\ddagger$
Insulin <sup>c</sup> (pmol/L)	42–181	$118 \pm 49^*$	$264 \pm 111\ddagger$	$118 \pm 49^*$	$208 \pm 97$

Note. Data were analyzed using repeated-measures analysis of variance and Bonferroni's post hoc test.

<sup>a</sup>Normal values from Pagana and Pagana (2002). <sup>b</sup>Lactate was not run for 1 participant ( $N = 17$ ) because of a laboratory error. <sup>c</sup>Data were transformed using the inverse ( $1/\text{variable}$ ) before statistical analysis;  $M \pm SD$  data are presented untransformed.

\*Significant time difference within the same treatment (time effect;  $p < .01$ ). †Significant treatment difference for given time period (treatment effect;  $p < .05$ ). ‡Significant difference for the interaction (treatment and time;  $p < .05$ ).

## Heart Rate and Blood Pressure

A two-way repeated-measures ANOVA and Bonferroni's post hoc test were used to examine differences in HR by time and treatment and their interaction. Postexercise values for HR were significantly higher than both the fasting and midpoint measurements ( $p < .05$ ; Table 4). No differences in HR were seen for the treatment main effect or the interaction of treatment and time.

Because the SBP and DBP data were not normally distributed, we used Wilcoxon's signed rank test to test for differences. Postexercise values for SBP were significantly higher than both the fasting and midpoint measurements ( $p < .05$ ; Table 4). Postexercise values for DBP were significantly lower than both the fasting and midpoint measurements ( $p < .05$ ; Table 4). Midpoint DBP was significantly higher than the fasting DBP measurement in the caffeine treatment ( $p < .05$ ; Table 4). Postexercise SBP was significantly higher with the caffeine treatment than with the placebo treatment ( $p < .05$ ; Table 4). Midpoint DBP was significantly higher with the caffeine treatment than with 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 nonresponders. The responders ( $n = 5$ ) had a  $>10\%$  increase in SBP at the midpoint reading during the caffeine trial. The increase in SBP at the midpoint reading was  $\leq 10\%$  for the nonresponders ( $n = 13$ ). Four of the five responders reported daily caffeine intakes  $<50$  mg/day. Forty percent of the responders and 77% of the nonresponders lifted more total weight on the chest press during the caffeine trial. One hundred percent of the responders and 69% of the nonresponders 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 nonresponders obtained a greater average power during the caffeine trial.

## RPE

Because RPE data were not normally distributed, we used Wilcoxon's signed rank test for the analysis. No differences were observed for RPE between treatments in

**Table 4 Caffeine's Effect on Heart Rate and Blood Pressure Between Time and Two Treatments,  $M \pm SD$**

Variable	Caffeine			Placebo		
	Pre	Mid	Post	Pre	Mid	Post
SBP (mm Hg)	118 $\pm$ 8	121 $\pm$ 8	176 $\pm$ 18*‡	118 $\pm$ 10	120 $\pm$ 8	168 $\pm$ 22*
DBP (mm Hg)	72 $\pm$ 6	77 $\pm$ 5†‡	64 $\pm$ 12*	73 $\pm$ 6	72 $\pm$ 6	63 $\pm$ 11*
Heart rate (beats/min)	68 $\pm$ 9	65 $\pm$ 8	157 $\pm$ 18*	66 $\pm$ 9	69 $\pm$ 9	150 $\pm$ 23*

*Note.* Blood-pressure data were analyzed using Wilcoxon's signed rank test. Heart-rate data were analyzed using repeated-measures analysis of variance and Bonferroni's post hoc test. No significant difference was observed for the interaction (treatment and time). Pre = fasting; Mid = 30 min posttreatment; Post = postexercise; SBP = systolic blood pressure; DBP = diastolic blood pressure.

\*Significant difference from Pre and Mid by time (time effect;  $p < .05$ ). †Significant difference from Pre by time (time effect;  $p < .05$ ). ‡Significant difference between treatments at the same time (treatment effect;  $p < .05$ ).

any of the exercises (chest press: caffeine =  $19 \pm 1$ , placebo =  $20 \pm 1$ ; leg press: caffeine =  $19 \pm 1$ , placebo =  $19 \pm 1$ ; Wingate test: caffeine =  $19 \pm 2$ , placebo =  $19 \pm 1$ ).

## 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 the participants consumed less than the U.S. population's average intake of caffeine of 193 mg/day (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 muscle endurance used primarily with 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 1 participant vomited at the end of the testing. These feelings might have negatively affected their performance.

Unfortunately, our statistical tests were also underpowered (leg press, .161). Thus, if more participants were included, significant results might have been obtained. Nonetheless, 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 with the placebo trial. The participants lifted slightly more weight, however, in the leg press and bench press during the first set of three supersets after caffeine ingestion. Jacobs et al. included recreational athletes instead of highly trained athletes, which we used in our study. In addition, Jacobs et al. administered a lower dose of caffeine (4 mg/kg BW) 90 min before testing instead of the 5-mg/kg BW dose 60 min before testing in our study. The dose of caffeine used, the timing of the dose, and the fitness level of the participants might 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. It could be, however, 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 -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. Other studies, however, found no significant differences in performance with recreational athletes using 3.5–4 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 might be small and difficult to measure in short-term, high-intensity exercise performance. In addition, studies have used a variety of exercise protocols, caffeine doses, and research participants, thus making it difficult to compare results.

A Wingate test was used to measure anaerobic power output. Our results indicate 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, but 72% of the participants obtained a greater average power during the caffeine trial than during 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, but Bell et al. and Collomp et al. observed nonsignificant improvements. Greer et al. also found nonsignificant improvements in the first three of four Wingate tests. The dose of caffeine was 5 mg/kg BW in two of studies (Bell et al., 2001; Collomp et al., 1991) and 6 mg/kg BW in the other (Greer et al.). Greer et al. found a significant negative effect of caffeine on peak power in the last of four repeated Wingate tests. Conflicting results might be related to the training level of the participants. In the current study, highly trained athletes were evaluated, whereas in the other three Wingate studies, untrained or recreational participants were examined. Thus, caffeine might 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 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, and 5 consumed 68–179 mg/day. We might have found different results in exercise performance if they had been high caffeine users.

## Hormonal Responses

In our study, postexercise 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 than in the placebo trial. These results agree with those of Collomp et al. (2002). In our study, similar to Collomp et al., the research participants ate a preexercise meal before 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. The decrease, however, might have resulted from elevated plasma epinephrine concentrations. Petrie et al. (2004) also showed that acute caffeine ingestion significantly reduced insulin sensitivity in obese, nondiabetic males. The protocol used in those studies, however, 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 a result of increased blood glucose concentrations.

In the current study, a significant interaction (treatment by 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 agree with those of 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 might be responsible for the increase seen in glucose concentrations. There was a significant decrease in postexercise FFA concentrations in both treatments, however, possibly because of an increase in postexercise insulin concentrations. Availability of glucose and FFA is not the limiting factor in short-term, high-intensity exercise protocols.

## Exercise Metabolites

In our study, the participants ate a meal 45 min before testing. In addition, the shake 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 current study, postexercise glucose concentrations were significantly higher in the caffeine trial than in the placebo trial. There was also a treatment main effect, with higher plasma glucose concentrations during the caffeine trial than during the placebo trial. Bell et al. (2001) also observed significantly higher postexercise glucose concentrations with caffeine ingestion than in a placebo trial, but postexercise concentrations were higher than preexercise concentrations with both treatments. Denadai and Denadai (1998) also observed a slight increase in postexercise glucose concentrations with caffeine ingestion and a slight decrease in postexercise glucose concentrations with a 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 postexercise glucose concentrations, with the caffeine trial tending to be higher than the placebo trial. Caffeine ingestion might 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 Krebs's cycle or aerobic metabolism (Balady et al., 2000). Caffeine might inhibit lactate clearance. In the current study, plasma lactate concentrations were significantly higher postexercise in both trials than fasting concentrations. These results are consistent with the literature (Bell et al., 2001, 1998; Collomp et al., 2002, 1991, 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 postexercise plasma lactate concentrations during high-intensity cycling protocols (Denadai & Denadai; Jackman et al., 1996) or the Wingate test (Greer et al.). Others, however, have observed that postexercise lactate concentrations are significantly increased with caffeine compared with a placebo trial during high-intensity cycling protocols (Anselme et al., 1992; Bell et al., 2001, 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, postexercise 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 postexercise FFA concentrations were significantly higher with caffeine ingestion. Denadai and Denadai also reported no significant differences in postexercise 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. An increase in FFA oxidation and glucose sparing is not as important, however, 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). One study, however, reported an increase in resting HR with acute caffeine ingestion (Robertson et al., 1978). Most researchers have also reported that caffeine might increase postexercise HR, but not significantly when compared with placebo (Bell et al., 2001; Bond et al.; Denadai & Denadai; O'Connor, Molt, Broglio, & Ely, 2004). Our data are in agreement with most 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 (midpoint) and postexercise SBP were significantly increased during the caffeine trial compared with 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 postexercise (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 might exert an effect on the central nervous system (CNS) by acting as a potent adenosine antagonist, thus blocking adenosine receptors or interacting with dopamine receptors (Ribeiro, Sebastiao, & de Mendonca, 2003). Adenosine inhibits the release of most excitatory neurotransmitters in the brain, particularly dopamine, and might reduce dopamine synthesis (Ribeiro et al.). Because adenosine receptors are abundant in many areas of the brain, caffeine might inhibit adenosine from blocking neuronal transmission and thus alter perception of pain, sympathetic activity, motor recruitment, and fatigue. In this capacity, caffeine might also increase time to exhaustion. Because caffeine is a dopamine agonist, it could possibly reduce RPE. In our study, however, we did not see a reduction in RPE with caffeine.

Adenosine receptors are abundant in skeletal muscles. In anaerobic activity, the ergogenic effect of caffeine might result from the antagonism of adenosine receptors. Svenningsson, Nomikos, Ongini, and Fredholm (1997) found that caffeine doses as low as 7.5 mg/kg BW might cause an increase in motor activity in rats primarily through the antagonism of adenosine receptors. Highly trained athletes might have more muscle mass than recreational athletes and might respond to the ergogenic effect of caffeine differently (via the adenosine receptors) than recreational athletes. Thus, caffeine might 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 might also be associated with CNS stimulation (increased motor recruitment or masking of fatigue). In tests using maximal voluntary contraction, the level of muscle activation increases after caffeine ingestion (Kalmar & Cafarelli, 1999; Meyers & Cafarelli, 2005; O'Connor et al., 2004; Williams, Barns, & Gadberrry, 1987), suggesting that caffeine might influence sarcoplasmic handling of calcium. These results have only been significant, however, 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 might have affected the participants' responses to the caffeine. The training that the participants undertook during the weeks before the study might have been quite different. A crossover design was used to help control for this variability, however. Furthermore, at the time of study recruitment, the participants were training at the same facility. Thus, a similar training protocol was performed during the 2 days before each testing day. Caffeine might 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 before the start of exercise. Information

on the effects of caffeine before exercise would have been useful to include in the study design. In addition, we did not measure plasma caffeine concentrations. In our study, 13 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 increased upper body muscle endurance during the chest press and increased peak power during the Wingate test. Caffeine and a meal increased postexercise concentrations of plasma glucose, serum insulin, and serum cortisol during high-intensity exercise. Multiple mechanisms might be responsible for performance enhancement in different types of exercise (aerobic vs. 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 might influence the CNS. The participants did not report any differences in RPE, however, between the caffeine and placebo trials. The participants in our study were caffeine naïve or very low caffeine consumers, which might have contributed to the results. Regular consumers of higher doses of caffeine might exhibit more benefits of caffeine as an ergogenic aid. Thus, research is needed that compares the effect of caffeine in caffeine-naïve 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-naïve 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.

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