Effect of acute caffeine ingestion on EPOC after intense resistance training

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Aim. This study investigated the effect of acute caffeine (CAF) intake on postexercise oxygen consumption (EPOC) after intense resistance training.

Methods. Fourteen strength-trained men (mean±SD age and mass = 23.1±4.2 yr and 83.4±13.2 kg, respectively) who were caffeine users initially completed one-repetition maximum testing (1-RM) of four exercises: bench press, leg press, lat row, and shoulder press. On each of two days separated by one week, they completed four sets of each exercise to fatigue at 70% of 1-RM, which was preceded by ingestion of CAF (6 mg/kg) or placebo. Pre-exercise, indirect calorimetry was used to assess energy expenditure for 35 min; this was repeated for 75 min postexercise while subjects remained seated in a quiet lab. Two-way analysis of variance with repeated measures was used to examine differences in gas exchange variables across time and treatment.

Results. Results revealed that EPOC was significantly higher (P<0.05) with CAF (267±41 L) compared to placebo (228±38 L). With CAF ingestion, oxygen uptake was significantly higher (P<0.05) from 10 min pre-exercise to 70 min postexercise. Respiratory exchange ratio was significantly different (P<0.05) with CAF versus placebo. Caffeine intake increased total energy expenditure by 15% (P<0.05), but the additional calories burned was minimal (∼27 kcal).

Conclusion. Caffeine ingestion in individuals regularly completing rigorous resistance training significantly increases EPOC and energy expenditure pre-and post-exercise, yet the magnitude of this effect is relatively small.

KEY WORDS: Muscle contraction - Respiratory system - Energy metabolism - Oxygen consumption.

Obesity is of epidemic proportions in the United States as well as across the globe. Data report that 33.3% of men and 35.3% of women are obese, and approximately 65% of adults are overweight. This condition increases risk of various chronic diseases including coronary artery disease, hypertension, type II diabetes, and cancer, amongst others. The annual health care costs of obesity are alarming, as Finkelstein et al. reported that they approach $92.6 billion.

One potential strategy to prevent weight gain is acute ingestion of caffeine (CAF), which has been shown to enhance resting metabolic rate in a variety of individuals. Early data revealed that ingestion of 8 mg/kg caffeine or coffee increased metabolic rate in normal weight and obese persons, similar to data with a 100 mg CAF dose. In trained men, a higher metabolic rate and fat oxidation in response to a 4 mg/kg dose of caffeine were revealed compared to
untrained men. Furthermore, the amount of energy expended post-exercise, termed the post-exercise oxygen consumption (EPOC), is also enhanced with caffeine ingestion. In untrained women,^{5} 5 and 10 mg/kg doses of CAF enhanced (P<0.05) oxygen consumption (VO₂) and fat mobilization during a 1 h recovery following 90 min of submaximal exercise. EPOC is also elevated in response to resistance training, especially when the intensity is high.^{10} This prolonged energy expenditure is due to the perturbation that exists after intense exercise, such as glycogen and phosphocreatine resynthesis^{11} and proton and lactate removal.^{12} Data from several studies^{10,13,14} reveal an EPOC duration of at least 1 h after intense resistance training. In a study^{13} in which strength-trained men completed four circuits of bench press, barbell squat, and power clean at loads equivalent to 75% 1-RM, VO₂ was higher (P<0.05) postexercise compared to baseline, and remained elevated for 38 h after exercise, leading to an additional 773 kcal burned over a two-day period. This value is substantially higher than values previously reported in men^{10} and women^{14} completing resistance training.

An additional explanation for the EPOC after resistance training is increased lipid utilization. Melby et al.^{10} reported significantly lower RER, reflecting greater fat oxidation, on the morning after resistance exercise compared to the morning before. Similar findings were revealed in other investigations^{13,14} in which subjects completed intense resistance training. The extraoxygen needed to degrade lipids in the mitochondrion explains this increase in VO₂ postexercise.^{13} In persons seeking to lose weight, enhanced fat oxidation and metabolic rate in response to intense exercise are desirable, especially since it has been reported^{15} that overweight individuals may have a lesser ability to oxidize lipid.

To our knowledge, no study has examined the combined effect of caffeine intake and resistance training on EPOC and substrate use in response to rigorous resistance training. It is hypothesized that compared to placebo, caffeine will significantly enhance magnitude of EPOC and lipid utilization. Potential improvements in energy expenditure mediated by pre-exercise caffeine ingestion combined with resistance training may enhance individuals' control of body weight.

Materials and methods

Study population

Resistance-trained men (N.=14) ranging in age from 18-30 yr were recruited to participate in the study. Mean age, height, body mass, and percent body fat were equal to 23.1±4.2 yr, 1.8±0.1 m, 83.4±13.2 kg, and 13.9±2.8%, respectively. They were currently completing total-body resistance training a minimum of 2 d/wk, and had been training for 7.5±4.4 yr (range=2-5-18 yr). All subjects were regular caffeine consumers, with an average daily intake equal to 218.2±105.0 mg/d (range=120-500 mg/d). None were taking any medications or supplements that would affect the outcomes of the study. They initially filled out a health-history questionnaire and provided written informed consent before participating in the study. All experimental procedures were approved by the University Institutional Review Board.

Pre-testing assessments

Subjects' height, weight, and percent body fat (%BF) were initially assessed. Percent body fat was measured using a sum of three skinfold (Ω3SKF) model.^{16} The primary investigator recorded all measurements at the abdomen, thigh, and chest following standardized procedures. Heart rate (Polar Electro, Woodbury, NY, USA) and blood pressure (Omron HealthCare Inc., Vernon Hills, IL, USA) were obtained by telemetry and manual sphygmomanometry, respectively, after the subjects sat down for approximately 5 min.

Monitoring of exercise status and dietary intake

Subjects completed 24 h dietary and exercise recalls before each trial, and were asked to follow the same diet on the day before each trial. Subjects were provided a list of items that contain caffeine (coffee, chocolate, soda, energy drinks, etc., as well as common over-the-counter medications), so that they could refrain from caffeine intake for 48 h before each visit. Before each trial, subjects did not complete any intense exercise in the 48 h before and fasted for 3 h.
1-RM testing

Subjects initially warmed up on a commercial stationary bike (Precor, 846e, Woodinville, WA, USA) for 5 min. Pre-exercise measurements of HR and BP were recorded at 4 min. One-repetition maximum (1-RM) testing of barbell bench press, 45° seated sled leg press, bilateral wide-grip lat pull down, and barbell shoulder press ensued on free weight equipment (Cybex, Medway, WA) according to previous methods.18 Two minutes rest was allotted between sets, and 1-RM was identified as the heaviest weight lifted once with proper form. Subjects were given verbal encouragement throughout the protocol. Testing was used to determine the load equivalent to 70-80% 1-RM, which was used in subsequent testing. This session took approximately 1 h, and was completed 1 week before subsequent trials. The day-to-day error of 1-RM assessment in our laboratory is approximately 2-4%.19

Treatment ingestion

Anhydrous pharmaceutical-grade caffeine or placebo (dimethyl cellulose) was provided to subjects in identical capsules, and was ingested with water 1 h before subsequent exercise trials. These were prepared by a pharmacist with no involvement in the study. Treatment order was assigned to subjects in a randomized, double-blind, crossover design. The caffeine dose was equal to 6 mg/kg, which has been shown to maximize blood levels of caffeine.20 Four out of 14 subjects (28%) were able to correctly identify the CAF treatment.

Resistance exercise protocol

After ingestion of CAF or placebo, subjects completed four sets of each exercise to failure at 70 (shoulder press and bench press) - 80% 1-RM (leg press and lat pull down); this bout lasted approximately 45-50 min. Two minutes of rest was allotted between sets. At the end of the resistance training protocol, excess post-exercise oxygen consumption (EPOC) was measured for 75 min. Subjects returned 1 week later and repeated the identical protocol at the same time of day after ingestion of the other treatment.

Assessment of baseline VO₂ and EPOC

Gas exchange data (VO₂, VCO₂, and RER) were obtained using a metabolic cart attached to a personal computer (ParvoMedics True One 2400, Sandy, UT, USA). Pre- and postexercise, data were obtained every 15 s and time-averaged every 5 min. This metabolic cart was recently validated21 against the Douglas bag method for VO₂ measurement. At baseline and before acquisition of postexercise measurements, the metabolic cart was calibrated to gases of known concentration (16% O₂ and 4% CO₂) as well as to room air (20.93% O₂ and 0.03% CO₂). A 3-liter syringe was used to calibrate flow. For 35 min pre-exercise and 75 min postexercise, subjects quietly sat in a chair in a climate-controlled laboratory (temperature=24-26 °C, relative humidity=30-50%) to allow continual assessment of energy expenditure. They were instructed to move as little as possible. They wore a respiratory facemask (Hans Rudolph #7400, Kansas City, MO, USA) covering the nose and mouth to prevent leakage of air. Heart rate was continually recorded via telemetry (Polar Electro, Woodbury, NY, USA). EPOC was assessed using the total area under the curve technique.22 Subjects walked from the fitness center to the laboratory, so 7.1±0.4 min elapsed between completion of exercise and initiation of EPOC assessment. EPOC was reported in units of liters, with the constant 1 L O₂=5 kcal subsequently used to calculate postexercise energy expenditure (Melby et al., 1995).10

Statistical analyses

Data are reported as mean±standard deviation and were analyzed using SPSS 16.0 (Chicago, IL, USA). A two-way ANOVA with repeated measures was used to examine differences in gas exchange variables (VO₂ and RER) and energy expenditure across time and treatment. A paired t-test was used to detect differences in magnitude of EPOC between treatments. If a significant F ratio was obtained, Tukey’s post hoc test was used to detect significant differences between means. Statistical significance was set at P<0.05.

Results

Testing was well-tolerated by all subjects, despite two participants reporting feelings of nausea and anxiety during exercise in the caffeine trial.
Oae-repetition maximum performance

Mean (± SD) 1-RM for the four exercises were equal to: bench press (105.2±5.2 kg), leg press (367.9±19.0 kg), bilateral row (162.1±6.1 kg), and shoulder press (77.1±3.5 kg). These values validate subjects’ status as resistance-trained, and place them in the 80th percentile for bench press and above the 90th percentile for leg press for young men (Institute for Aerobics Research, unpublished data, 1994).

Magnitude of EPOC

EPOC was significantly higher (P<0.05) with CAF (26.7±4.1 L) versus placebo (22.8±3.8 L). Figure 1 reveals changes in VO2 recorded for 75 min post-exercise in a 29 year-old male (mass=98.6 kg) in the CAF and placebo trials. VO2 was consistently higher during recovery with CAF versus placebo.

VO2

Figure 2 reveals alterations in VO2 before and after exercise across both treatments. There was a significant main effect of time (P<0.01) and treatment (P<0.05) as well as a significant (P<0.05) treatment X time interaction for VO2. At 15 min pre-exercise, VO2 was similar (P>0.05) between treatments: 0.30±0.05 L/min (95% confidence interval [CI]=0.27-0.33) in CAF and 0.28±0.06 (95% CI=0.25-0.32) in placebo. Oxygen uptake was consistently 10-20% higher (P<0.05) with CAF versus placebo from 10 min pre-exercise to 70 min postexercise. For example, postexercise VO2 was higher at 5 (0.51±0.12 L/min, 95% CI=0.44-0.58 vs. 0.47±0.11, 95% CI=0.41-0.53) and 30 min (0.39±0.08 L/min, 95% CI=0.34-0.44 vs. 0.32±0.06 L/min, 95% CI=0.28-0.35) with CAF. In the placebo trial, VO2 at 75 min was still higher (P<0.05) than baseline, although in the CAF treatment, VO2 at 75 min postexercise was similar (P>0.05) to resting values.

RER

There was a main effect of time (P<0.01) on RER. At 15 min pre-exercise, RER was equal to 0.87±0.06 (95% CI=0.84-0.91) with CAF, and 0.82±0.06 in placebo (95% CI=0.80-0.87). At 30 and 75 min postexercise, RER was equal to 0.70±0.05 (95% CI=0.67-0.74), 0.76±0.06 (95% CI=0.73-0.80), 0.72±0.05 (95% CI=0.69-0.75), and 0.77±0.04 (95% CI=0.74-0.79) in CAF and placebo, respectively (Figure 3). All pre-exercise RER values were different (P<0.05) than values recorded post-exercise. There was a significant interaction (P<0.05) effect, as RER was higher (P<0.05) at 20-5 min pre-exercise with CAF compared to placebo, yet was lower (P<0.05) at 35-40 and 55-65 min postexercise.
Table 1—Number of repetitions completed (mean ± SD) during intense resistance training after caffeine or placebo ingestion.

<table>
<thead>
<tr>
<th>Exercise</th>
<th>Caffeine</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bench press</strong> (mean ± SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Set 1</td>
<td>12.6±2.17a</td>
<td>12.07±3.52a</td>
</tr>
<tr>
<td>Set 2</td>
<td>8.64±1.22</td>
<td>8.21±2.04</td>
</tr>
<tr>
<td>Set 3</td>
<td>6.71±1.94</td>
<td>6.57±1.55</td>
</tr>
<tr>
<td>Set 4</td>
<td>5.50±1.56</td>
<td>5.42±1.09</td>
</tr>
<tr>
<td><strong>Leg press</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Set 1</td>
<td>15.71±6.88a,9</td>
<td>14.07±6.17a</td>
</tr>
<tr>
<td>Set 2</td>
<td>13.14±5.27a</td>
<td>11.64±3.48</td>
</tr>
<tr>
<td>Set 3</td>
<td>10.21±3.40a</td>
<td>10.50±2.77a</td>
</tr>
<tr>
<td>Set 4</td>
<td>9.79±3.02a</td>
<td>9.43±3.13a</td>
</tr>
<tr>
<td><strong>Lat row</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Set 1</td>
<td>9.50±2.38b</td>
<td>9.36±2.02b</td>
</tr>
<tr>
<td>Set 2</td>
<td>7.64±1.55</td>
<td>7.36±1.55</td>
</tr>
<tr>
<td>Set 3</td>
<td>6.29±1.38</td>
<td>5.79±1.19</td>
</tr>
<tr>
<td>Set 4</td>
<td>5.79±1.72</td>
<td>5.71±1.38</td>
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<tr>
<td><strong>Shoulder press</strong></td>
<td></td>
<td></td>
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<tr>
<td>Set 1</td>
<td>11.09±2.48a</td>
<td>10.21±2.67b</td>
</tr>
<tr>
<td>Set 2</td>
<td>8.14±2.25</td>
<td>8.07±2.30</td>
</tr>
<tr>
<td>Set 3</td>
<td>6.21±1.76</td>
<td>6.07±1.90</td>
</tr>
<tr>
<td>Set 4</td>
<td>5.93±2.13</td>
<td>5.36±1.60</td>
</tr>
</tbody>
</table>

Note: all means for that exercise significantly different (P<0.05) from each other; b: all means for that exercise significantly different (P<0.05) from each other with the exception of sets 3 and 4; *: P<0.05 versus placebo.

These data are revealed in Table 1. Participants completed approximately 1.5 more repetitions in set 1 and 2 of leg press with caffeine compared to placebo.

**Discussion**

The primary aim of this study was to examine the effect of acute caffeine intake on EPOC in strength-trained men who completed intense resistance training. Data revealed a significant elevation in EPOC and energy expenditure with ingestion of a 6 mg/kg dose of caffeine. In both treatments, VO2 remained elevated for more than 1 h after exercise, similar to previous findings. RER was also lower postexercise with acute caffeine intake.

To our knowledge, no study has elucidated changes in EPOC and substrate use after resistance training preceded by acute caffeine ingestion. Our data corroborate findings from early studies in which aerobic exercise was performed. In one study, untrained women walked for 90 min at 55% VO2max after ingestion of 5 mg/kg of caffeine or placebo. VO2, RER, and free fatty acid (FFA) concentration were higher (P<0.05) with CAF compared to placebo throughout the 1 h recovery. A similar experimental protocol was used in a subsequent study, although 5 and 10 mg/kg doses of caffeine were administered, and exercise was performed on the cycle ergometer. Compared to placebo, postexercise VO2, RER, and FFA levels were higher (P<0.05) after ingestion of both caffeine doses, although there was no dose-response effect. In both studies, an enhanced reliance on lipid utilization was identified as one factor explaining the elevation in postexercise VO2.

Across both treatments, VO2 was elevated at least 70 min after exercise (Figure 2). This EPOC duration is similar to that reported in other investigations. However, this EPOC duration is longer than that revealed in studies in which the intensity and/or volume of resistance training was lower, resulting in attenuated metabolic disturbance. For example, EPOC only persisted for 15-60 min when circuit training or multi-set strength training was completed. However, VO2 remained elevated for 1 h after circuit training in women, a discrepancy that may be due to differences in the specific technique used to determine EPOC as well as subjects' fitness level.

There is large variability in the literature regard-
ing the magnitude of EPOC after resistance training, ranging from 1.1 L-12.6 L. Much of this discrepancy is due to varied training intensities, an inability to continuously measure gas exchange data, as well as different techniques used to determine EPOC. In one study, women completed weight training at loads equal to 45-85% 1-RM, and EPOC ranged from 1.1 L (45%) to 2.3 L (85%). Subjects were also periodically disconnected from the gas exchange system post-exercise. In another study in which men and women completed light (50% 1-RM) or heavy (80-90% 1-RM) resistance training, EPOC was equal to approximately 10 L, eliciting an additional energy expenditure of 50 kcal, similar to other studies. The EPOC recorded in the present study (22.8-26.7 L) is higher than that revealed in previous studies, potentially due to a greater exercise intensity as well as training volume completed (22 409.5±3 773.2 kg in CAF and 21 185.7±4 655.4 kg in placebo, P>0.05). Subjects were instructed to exercise to fatigue on every set, which likely induced severe metabolic perturbation. Subsequent removal of protons and lactate, promotion of glycogen and phosphocreatine resynthesis, restoration of the bicarbonate pool, elevation in blood flow and muscle temperature, and increased fat oxidation all require oxygen, which may explain the substantial EPOC observed in our subjects. Furthermore, mean heart rate at 75 min post-exercise (74.8±14.0 b/min) was higher than values reported immediately pre-exercise (66.4±8.8 b/min).

A common finding in the literature is a lower RER after resistance training, indicative of increased fat oxidation. In men completing three sets of seven exercises at a load equal to 12-RM, RER was lower (P<0.05) after exercise compared to a control day of no exercise. In men completing 50 sets of resistance training, postexercise fat oxidation was significantly higher compared to a control day, although it was similar to that recorded after aerobic exercise. Similar findings were also revealed in young women completing various protocols including traditional resistance exercise and circuit training. In the present study, post-exercise RER ranged from 0.70-0.77, and was significantly lower versus pre-exercise (Figure 3). Caffeine intake elicited a small but significant decline (P<0.05) in RER post-exercise compared to placebo. Two likely mechanisms to explain this attenuated RER include CO2 retention to restore blood bicarbonate as well as a postexercise sparing of carbohydrate to support glycogen resynthesis, as this substrate is the predominant fuel degraded during heavy resistance training dependent upon nonoxidative metabolism.

**Limitations of the study**

This study had a few limitations. First, there was a delay of 7.1±0.4 min from the time that subjects completed their final repetition of weight training exercise to initiation of EPOC assessment. This more than likely underestimated the magnitude of EPOC, which is highest immediately after exercise. Yet, this duration was similar in all subjects across both treatments. The CAF dose (6 mg/kg) was larger than what would be typically ingested pre-exercise. Dulloo et al. revealed that a 100 mg dose of caffeine increased metabolic rate by approximately 3.4%, lower than that observed in the present study. Furthermore, caffeine ingestion significantly increased (P<0.05) the number of repetitions completed in sets 1 and 2 of the leg press compared to placebo (Table 1). The slightly greater weight lifted in the CAF trial may have enhanced EPOC above and beyond the effects of the drug, yet this effect on energy expenditure would be minimal. Lastly, the 16-set exercise regimen completed at loads equal to 70-80% 1-RM is not practical for the typical exerciser, and may be best suited to athletes and/or persons experienced with this exercise mode. Further research is needed to investigate the magnitude and
duration of EPOC, and any potential synergistic effect of caffeine, in response to more traditional resistance training programming, such as that recommended by the American College of Sports Medicine for healthy adults. A smaller EPOC would be expected in response to less intense exercise that does not elicit the magnitude of metabolic disturbance as seen in the present study.

Conclusions

Data showed that a 6 mg/kg dose of caffeine ingested before intense resistance training elicited a small but significant increase in EPOC and energy expenditure (+15%) compared to placebo. For greater than 1 h postexercise, VO2 was elevated, and RER was significantly reduced versus placebo. Nevertheless, the practical significance of this increase in energy expenditure is minimal, as approximately 130 days of resistance training at this volume would be needed to burn an additional pound of fat.

References