The Influence of Caffeine on Resistance Exercise Performance and Post-Exercise Blood Glucose Control

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ABSTRACT

The effects of oral caffeine (Caf) on resistance exercise performance and post-exercise glucose control were studied. Young adult males (n = 7) completed four experimental trials: two resting trials (Rest Caf and Rest Placebo (PL)) and two exercise (Ex) trials (Ex Caf and Ex PL). Caf (4 mg/kg of body weight) or PL was consumed 30 min prior to testing. An oral glucose tolerance test (75 min OGTT: 1 g/kg in 25% CHO solution) was conducted upon completion of exercise or at rest for resting trials. For Ex trials, four sets (6 reps/set) of knee extension and biceps curl (10-RM load) were completed. A fifth set for each lift was conducted to fatigue. Additional variables included blood pressure, grip strength, and blood lactate. Area under the curve (AUC) for glucose in Ex. Caf was 7.5%> than in Ex PL; Rest Caf was 7.3% > than Ex Caf; and Rest PL was 3.2% > than Ex PL. No significant difference between treatment or interactions for blood glucose (BG) or repetitions to fatigue were present. Blood Lactate (mmol/L) was significantly greater for the Ex Caf condition (7.9 vs. 5.8 in Ex PL condition). Rest Caf mean arterial pressure (MAP) tended to be higher than Rest PL ($p=0.053$), and Ex Caf MAP tended to be higher than Rest PL ($p=0.061$). Handgrip strength increased significantly from pre to post-exercise with and without caffeine for both treatments. Caffeine did not influence exercise performance; however, it was associated with a mild, non-significant reduction in OGTT control.

Keywords: glycemic control, oral glucose tolerance test, lactate, area under the curve;

INTRODUCTION

According to the National Diabetes Statistics Report (2017) there are 30.3 million (10.7%) people in the United States who have diabetes. Proper nutrition and regular exercise are the primary methods for prevention of diabetes (Eyre et al. 2004). Acute exercise has commonly been shown to transiently improve glucose regulation. Several studies have shown that a single bout of acute resistance exercise can acutely improve glycemic control in adults (Tong et al. 2017; Kido et al. 2016). Research studying the acute effects of resistance exercise on glycemic control is sparse, but generally these studies reveal an acute improvement in glycemic control following resistance exercise in both healthy controls and individuals with type 1 and type 2 diabetes mellites (Koopman et al. 2005; Reddy et al. 2019; Fenicchia et al. 2004)

Caffeine is arguably the most widely consumed drug in Western society. Caffeine is a central nervous system stimulant and is also commonly used as an ergogenic aid in sport. In the United States, for those aged 2 yr and above, the *per capita* consumption of caffeine averages 165 $mg \cdot day^{-1}$ and 85% of the population consumes one or more caffeinated beverages daily (Mitchell et al. 2014). Caffeine has been demonstrated to impede insulin-mediated glucose disposal (Bischof et al. 2001). Conversely, Tsuda (2015) found that caffeine and muscle contraction synergistically increase AMP-activated protein kinase activity (AMPK) and insulin-independent glucose transport; these effects were associated with less muscle fatigue in rat skeletal muscle. Therefore, there is considerable contradiction in the role that caffeine plays in glucose disposal.

Given the breadth of caffeine usage and the role that exercise can play in enhancing glycemic control, it is important to evaluate caffeine's influence on blood glucose regulation. Consequently, this study was designed to investigate the effects of acute caffeine consumption on resistance exercise performance and on post-exercise glycemic control in young adults. Findings could contribute to the expanding knowledge base related to exercise and nutrition for healthy individuals.

METHODS

The project methodology was approved by the University's Committee on the Protection of Human Research Subjects. This research was carried out fully in accordance with the ethical standards of the International Journal of Exercise Science (Navalta, 2019). Consent documents were read and signed by each study participant. Seven physically active young adult males (24.64±3.14 yrs; 78.26±16.60 kg; 177.03±10.3 cm; 12.70±3.7% fat;) completed all testing. Initially, subjects completed an orientation session during which 10-RM (10 repetition maximum) for knee extension and biceps curl lifts were determined using an isotonic seated barbell curl and a free weight leg extension machine. Demographic measures were also obtained, including body mass, height, body fat percentage, left and right-hand grip strength, resting blood pressure (BP) and heart rate (HR). Over four separate testing days (minimum of 48 h between each trial) subjects completed the experimental trials in a counter-balanced order at the same time of day to avoid diurnal variation. Prior to each trial, subjects were provided with capsules that contained caffeine (dosed at 4 mg/kg of body mass) or 1 gram of maltodextrin (placebo). Subjects were instructed to consume the assigned treatment 30 min prior to arrival at the scheduled testing time. Each trial employed a 75-min oral glucose tolerance test (conducted immediately following exercise for the exercise trials and immediately following baseline measurements for the resting trials). Trials consisted of 1) resting placebo trial (Rest PL); 2) resting caffeine trial (Rest Caf); 3) exercise placebo trial (Ex PL) and 4) exercise caffeine trial (Ex Caf).

For all trials, upon arriving to the laboratory, subjects sat quietly for 10-min after which time, baseline measurements were obtained. These included resting HR, BP (obtained using an automated blood pressure cuff), blood glucose (BG) and lactate (obtained from whole blood via fingertip collection), left- and right-hand grip strength (Lafayette Instruments, Lafayette IN), and hunger rating (using a 10 cm Likert scale where a rating of "zero" represented no hunger and a score of "ten" represented severe hunger). On the exercise trials, following baseline measures, the subject completed a 3-min warm up on a Monark 828e ergometer at 70 rpm at 1 kp of resistance. Upon completion of this, the participant completed one warm-up set (8 repetitions) using approximately 60% of the 10-RM load. Once experimental loads were placed, the subject alternated between lifts with 2-min recovery between sets. Exercise trials involved completion of four sets of 6 repetitions per set using the 10-RM load for the given lift. During the 5th set, subjects were encouraged to complete repetitions to failure (defined by inability to maintain a set cadence). Repetition cadence was controlled using a metronome set to 46 bpm. Immediately upon cessation of exercise, baseline measurements were taken prior to ingestion of the CHO solution.

Following baseline assessments on the resting control trials and upon completion of exercise on the active trials, subjects were supplied with a carbohydrate solution dosed at 1 g CHO/kg of body mass in a 25% solution. Once the solution was fully consumed, a 75 min oral glucose test was completed with serial fingertip samples obtained every 15 min (Bayer Contour, Bayer Healthcare LLC, Tarrytown NY), a validated and accurate blood glucose monitoring system (Halldorsdottir, 2013). Upon completion of the final sample time-point, baseline assessments were repeated for all conditions. Glucose area under the curve (arbitrary units: A.U) was determined as:

$$\text{Area} = [(\text{BG measure 1} + \text{BG measure 2})/2] \times 15$$

Where "15" represents the time (min) between samples. Each of six midpoint derivations (from seven sample points) was summed to derive the overall BG AUC for each test subject.

Trials were completed a minimum of 48 h apart and trial start time remained consistent amongst all participants. All trials were completed in the morning following an overnight fast. Subjects were instructed to refrain from exercise and caffeine for 36h and 12 h, respectively. Data were analyzed using repeated measures analysis of variance (SPSS, Chicago IL). Independent variables were treatment (resting placebo (Rest PL), caffeine resting control (Rest Caf), exercise placebo (Ex PL) and caffeine exercise control (Caf Ex) and time. Dependent variables included blood glucose, blood lactate, area under the curve (glucose), mean arterial pressure, Rating of Perceived Exertion, and repetitions to fatigue (during the 5th set).

RESULTS

Seven participants completed all testing under a fasted state. Repetitions to failure (Figure 1) were not found to be significantly different based on condition.

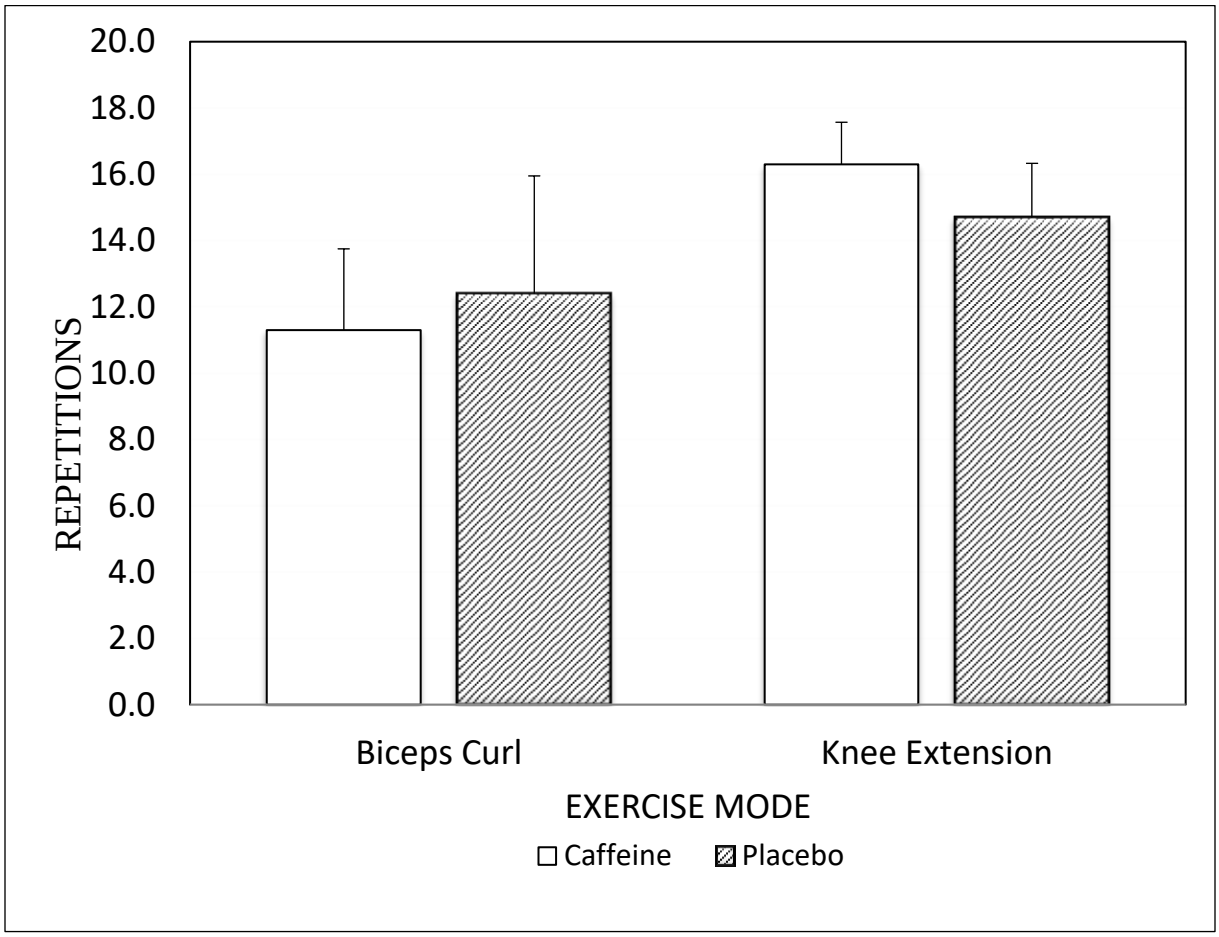

Figure 1. Repetitions to fatigue during the 5th set using a 10-RM load under caffeine and placebo conditions

Handgrip strength (right hand) was significantly increased (52.16±3.79 kg pre-ex vs. 53.89±3.93 kg post-ex) upon completion of exercise (p=0.032: time effect). Treatment did not affect this outcome. In addition, there was a tendency (p = 0.056) for the change in Ex Caf handgrip strength from baseline to end (2.42±1.13 kg) to be greater than the change in handgrip strength of the other conditions (Ex PL: -0.14±1.56; Rest Caf: -2.78±2.01; Rest PL: -4.14±1.99 kg). Blood lactate was significantly elevated by exercise ($p<0.05$) for both trials (time effect) and lactate was significantly greater post-exercise for the Ex Caf condition (Figure 2). MAP was significantly increased by the exercise trials (time effect) and was found

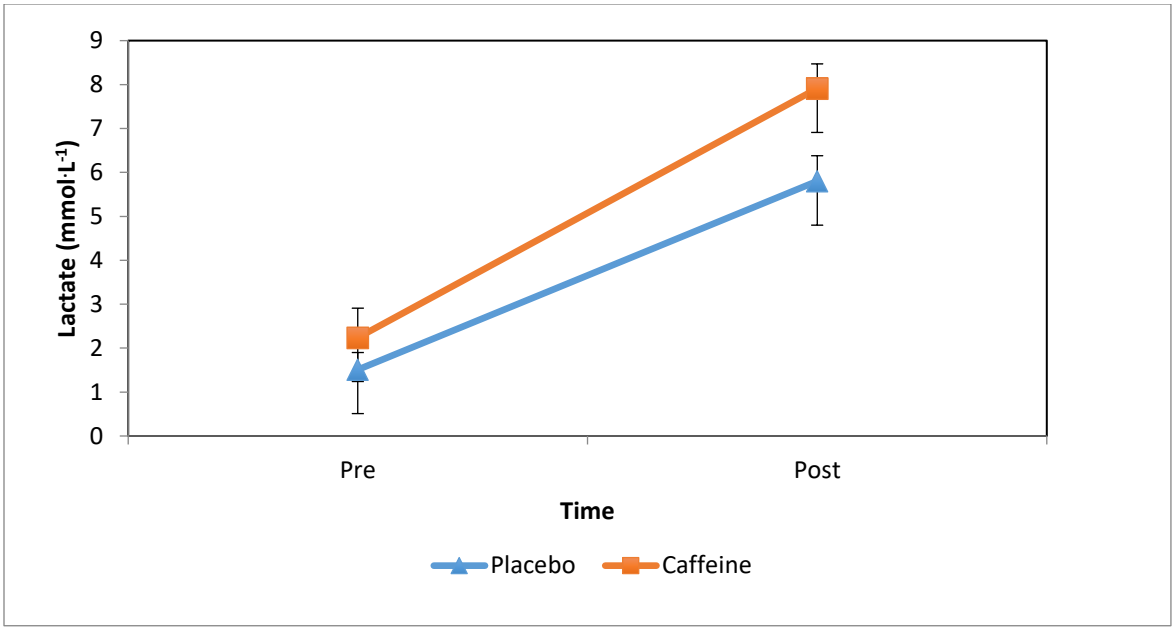

Figure 2. Blood lactate response prior to and following acute resistance exercise based on condition.

Table 1. Mean Arterial Pressure (mmHg) responses under testing conditions.

	Pre	Post-Exercise	End (post OGTT)
Rest Caf	96.81±2.22	N/A	89.87±0.96
Rest PL	88.41±1.67	N/A	86.67±2.16
Ex Caf	94.75±2.52	96.18±3.53	91.55±2.86
Ex PL	90.02±2.09	93.44±2.95	87.29±3.40

to be significantly lower by the end of the 75 min OGTT vs. the pre-exercise MAP (Table 1). There was a trend for higher MAP with the caffeine conditions (p=0.053). Blood glucose was not found to be different based on condition. However, significant time effects were present such that pre and post-exercise BG were significantly different from all time-points; BG 15 was significantly different from all time-points other than BG 75; BG 30 was significantly different from all time-points other than BG 45; and BG 60 was significantly different from all time-points (Figure 3). Area under the curve for BG was not different by condition (Figure 4).

DISCUSSION

Based on the results of the study, moderately dosed caffeine (4 $mg \cdot kg^{-1}$) was not found to significantly influence either work performance or post-exercise glycemic control. However, caffeine tended to increase the AUC for the resting caffeine and exercise caffeine conditions vs. the placebo conditions, albeit non-significantly (Ex PL AUC 7.5% smaller than Ex Caf and Rest PL 4.0% smaller than Rest Caf). These occurrences imply that caffeine may delay the rate of blood glucose disposal following exercise, however. In addition, blood lactate was significantly increased in the Ex Caf. condition compared to

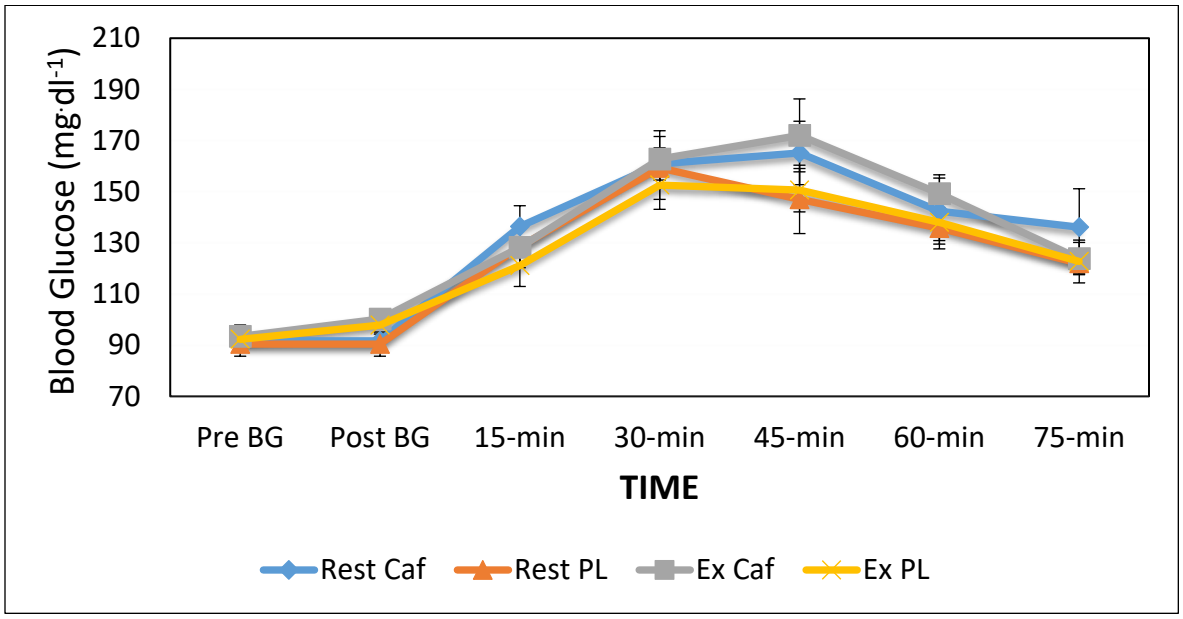

Figure 3. Blood glucose response during OGTT over time under resting (Rest Caf and Rest PL) and following resistance exercise (Ex Caf and Ex PL). See Results text for time effect differences. No treatment or time x treatment effects present.

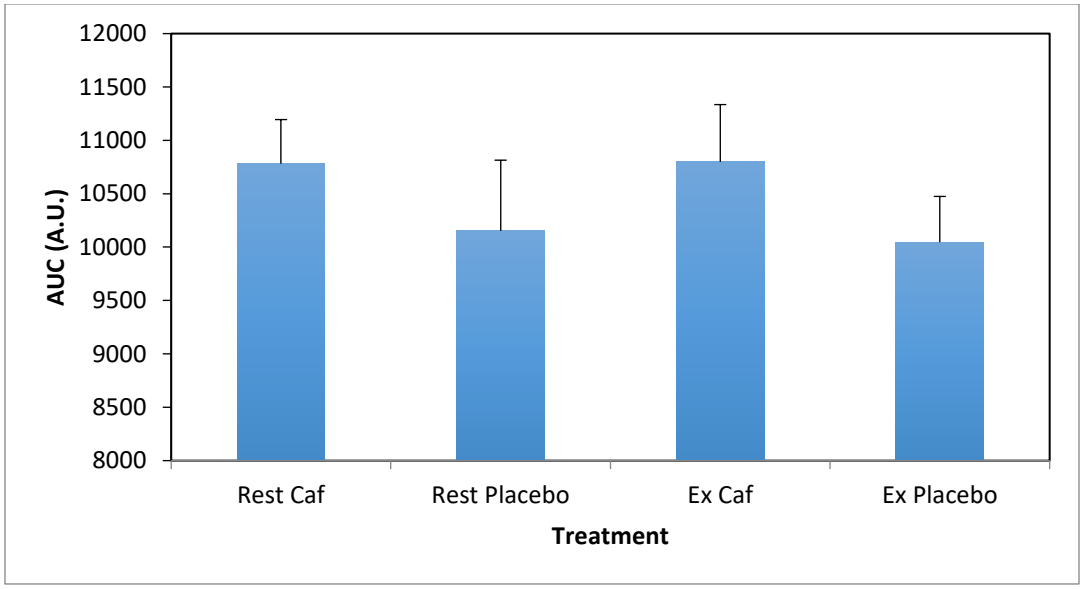

Figure 4. Glucose area under the curve response by condition. No differences between conditions.

the exercise placebo condition. Caffeine has been tied to an increased reliance upon anaerobic metabolism (Spriet et al. 1992; McNaughton et al. 2008) or a decrease in the clearance rate of blood lactate. Caffeine also tended to increase mean arterial pressure (MAP), consistent with findings of other studies (Casiglia et al. 1992; Bender et al. 1997; Sung et al. 1995).

It has been proposed that caffeine, through phosphodiesterase (PDE) inhibition (Vernikos and Harris 1968), causes an increase in cyclic adenosine monophosphate (cAMP) in the brain (Toccfondi et al. 1983; Katz and Greenough 1975) stimulating catecholamine release. An increase in catecholamine levels could, in turn, improve endurance exercise performance by delaying carbohydrate utilization and

preserving muscle glycogen stores (Van and Sarais 2000). Caffeine may promote a glycogen sparing effect as a result of the catecholamine effect on FFA mobilization and oxidation. Essig (1980) and Erickson (1987) both reported 30%–40% reductions in muscle glycogen use during cycling at 65%–70% of maximal oxygen uptake following caffeine ingestion compared with a placebo control. This metabolic advantage may confer a performance-enhancing benefit for persons with diabetes who typically have lower muscle (Shulman et al. 1990) and/or liver (Bischof et al. 2001) glycogen stores and for otherwise healthy individuals for whom glycogen depletion may be a limiting factor in exercise performance.

One concern, however, for T2D patients is that caffeine intake may limit the beneficial effects that exercise has on lowering blood glucose levels, as its use may promote skeletal muscle and hepatic insulin resistance. This detrimental effect on blood glucose homeostasis may also be the case for sedentary individuals with insulin resistance. Though, in the present study, caffeine intake was not found to significantly modify the glycemic response to an OGTT. Yet, AUCs for caffeine trials were 4-7.5% higher compared to the PL trials. Consequently, there is the chance that a larger sample size could provide more insight into a potential adverse effect on glucose homeostasis in healthy populations, which may address concerns for the latter.

Caffeine ingestion was not found to induce significant gains in repetitions completed for either the knee extension or the biceps curl. In contrast, a significant gain in handgrip force was observed post-exercise; though, treatment did not impact the magnitude of change. The mechanism for this change is not evident, but it may be related to increased sympathetic nervous system stimulation resulting from the exercise. Further, there was a tendency for the absolute change in handgrip strength from baseline to end to be greater during the Ex Caf condition. Astorino (2008) reported no significant strength enhancing effects with caffeine ingestion in a group of resistance trained men. However, recent work (Grgic and Mikulic 2017) found a significant (3%) increase in 1-RM squat strength following caffeine ingestion of 6 $mg \cdot kg^{-1}$. In addition, Goldstein (2010) reported a significant (2%) increase in 1 RM bench press in females following caffeine ingestion of 6 $mg \cdot kg^{-1}$. This is similar to previous research (Woolf et al. 2008; Beck et al. 2006) where results on chest press and Wingate peak power were enhanced following moderately dosed caffeine (5 $mg \cdot kg^{-1}$) and low dose (2.1-3.0 $mg \cdot kg^{-1}$) caffeine, respectively. Given the importance of strength and power in many sports, caffeine effects on these types of variables have been widely investigated. A recent study by Ali (2016) reported no significant effects on countermovement jump height after consumption of caffeine. In contrast, other investigators have shown acute significant ergogenic effects of caffeine on countermovement jump height and peak force (Bloms et al. 2016). Consequently, more work is needed in the area of caffeine and performance.

Grgic (2018) conducted a meta-analysis on the effects of caffeine intake on muscle strength and power. Through a subgroup analysis they indicated a significant increase in upper body, but not lower body strength following caffeine ingestion. Warren (2010) suggested that larger muscles, such as with the lower body, have a greater motor unit recruitment capability with caffeine intake than small muscles groups, such as in the upper body. Further, enhancement of RT performance through caffeine has been tied to motor unit recruitment, as well as reduced RPE values and the central effects of adenosine on transmission and arousal (pain perception as well).

Additional research is required to further substantiate effects of caffeine on resistance exercise performance and on post-exercise glucose regulation. Our primary finding was that lactate was significantly greater post-exercise for the exercise caffeine condition than placebo. Despite the statistical power limitations, caffeine does seem to elicit some influence over post-exercise glucose regulation by lowering the rate of glucose disposal. The influence of caffeine on resistance exercise and post-exercise glucose should be investigated further with a larger sample size, and this type of work should be

conducted in pre-diabetic and diabetic populations to weigh the impact of acute exercise on post-exercise glycemic control.

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