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Research article

Caffeine attenuates acute growth hormone response to a single bout of resistance exercise

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Abstract
The purpose of this study was to investigate the effects of caffeine consume on substrate metabolism and acute hormonal responses to a single bout of resistance exercise (RE). Ten resistance-trained men participated in this study. All subjects performed one repetition maximum (1RM) test and then performed two protocols: caffeine (CAF, 6 mg·kg^{-1}) and control (CON) in counter balanced order. Subjects performed RE (8 exercises, 3 sets of 10 repetitions at 75% of 1RM) after caffeine or placebo ingestion one hour prior to RE. Blood samples collected prior to treatment ingestion (pre-60), immediately prior to RE (pre-exe), and 0, 15, 30 min post to RE (P0, P15, P30) for analysis of insulin, testosterone, cortisol, growth hormone, glucose, free fatty acid and lactic acid. Each experiment was separated by seven days. In this study, statistical analysis of a two-way analysis of variance (treatment by time) with repeated measures was applied. After ingesting caffeine, the concentrations of free fatty acid (pre-exe, P0, P15, P30) in CAF were significantly higher than CON ($p < 0.05$). Additionally, the responses of GH (P0, P15, P30) in CAF were significantly lower than CON ($p < 0.05$), whereas the concentrations of insulin, testosterone and cortisol were not different between CAF and CON ($p < 0.05$) after RE. The results of this study indicated that caffeine ingestion prior to RE might attenuate the response of GH. This effect might be caused by the elevation in blood FFA concentration at the beginning of RE.

Key words: Nutritional supplementation, growth hormone, free fatty acid, ergogenic aids.

Introduction

Caffeine is found in common substances, such as coffee, tea, energy drinks, alcoholic beverages, and chocolate, and is commonly consumed in most people's diets. Numerous studies have demonstrated that caffeine intake enhances endurance and improves performance, particularly prolonged, intermittent and exhaustive exercises (Bell and McClellan, 2002; 2003; Graham et al. 1995; Trice and Haymes, 1995). However, the ergogenic effects of caffeine ingestion during resistance exercise (RE) performance are controversial. Recently, research has indicated that caffeine-containing supplementation prior to RE significantly increase upper-body muscle strength (1RM) between caffeine and placebo groups, but no difference was seen in lower-body (Beck et al., 2006). Woolf et al. (2008) also confirmed that the amount of caffeine consumed (5 mg·kg^{-1}) could result in more total weight lifted (work load multiplied by repetitions) for the chest press (upper-body), whereas the results of another experiment suggest that caffeine ingestion (6 mg·kg^{-1}) prior to RE does not significantly alter muscle strength or endurance (bench press and leg press) (Astorino et al., 2008). Furthermore, several researches have demonstrated that caffeine ingestion (5 or 10 mg·kg^{-1}) reduces quadriceps muscle pain intensity ratings during moderate cycling exercise (60% VO_2max) (Gliottoni et al., 2009; Motl et al., 2006; O'Connor et al., 2004; Motl et al., 2003). Maridakis (2007) also found that caffeine significantly reduces muscle pain resulting from eccentric resistance exercise–induced, delayed-onset muscle soreness (DOMS). Consequently, caffeine ingestion prior to RE may enhance muscular performance and recovery from intense RE.

It is well known that RE elicits a milieu of acute physiological responses and chronic adaptations (Kraemer and Ratamess, 2005). These responses, including hormonal response that play a critical role in the cellular remodeling process of myofibrillar proteins and are closely involved with protein synthesis and degradation (Florini, 1987). A multitude of hormones that exert anabolic effects such as testosterone, growth hormone (GH) super family, insulin and insulin-like growth hormone-I regulate protein synthesis, whereas cortisol stimulates muscle degradation and inhibits protein synthesis, as a catabolic hormone (Kimball et al., 1988; Griggs et al., 1989; Fryburg et al., 1991). Recent research has indicated that caffeine ingestion (5 mg·kg^{-1}) prior to RE significantly elevates serum cortisol concentration (Woolf et al., 2008). Results of another study have reported a small elevation (21%±24%) in testosterone and a moderate elevation (52% ± 44%) in cortisol with high dose of caffeine ingestion (800 mg). However, caffeine effect on the testosterone:cortisol ratio slightly reduces (14%±21%). Although this study reported that a high dose of caffeine ingestion elevates testosterone secretion, this benefit is tempered by a concurrent elevation in cortisol (Beaven et al., 2008). Recently, Goto et al. (2005; 2007) demonstrated that higher serum free fatty acid (FFA) results from endurance and sprint exercise prior to RE, attenuating GH response. Conversely, the attenuate effects of FFA on testosterone and cortisol are not significant. Several previous studies have found negative correlation between FFA concentration and GH response (Nakagawa et al., 2002; Van Dam et al., 2000). However, researchers have well established that caffeine ingestion may raise serum FFA levels at rest before exercise via higher catecholamine response (Costill et al., 1978; Essig et al., 1980; Ivy et al., 1979). Although the results of

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these studies infer that caffeine ingestion prior to RE may produce negative effects on anabolic hormonal responses, previous studies have focused on exercise performance benefits. Therefore, little is known about the mechanisms of caffeine ingestion on the acute hormonal responses to RE.

The primary purpose of this study was to examine the effects of caffeine ingestion (6 mg·kg^{-1}) on acute hormonal response to RE (insulin, GH, total testosterone and cortisol) and the interaction with energy substrate concentrations (FFA and blood glucose). We hypothesized that caffeine ingestion prior to RE may cause elevated cortisol and attenuated GH secretion after RE.

Table 1. Subject demographic data (n=10).

Parameter	mean	SD
Age (year)	21.5	1.4
Height (m)	1.75	.05
Mass (kg)	73.5	8.5
Body fat (%)	15.9	3.5
Daily caffeine consumption (mg.d^{-1})	33.7	12.1

Methods

Participants

Ten healthy, resistance-trained, physical education male students volunteered to participate in this study. They each had at least six months experience in performing resistance exercise (at least three days per week in the six-month period before the study), and were familiar with the exercises and equipment used in this study. These subjects reported low daily caffeine consumption (i.e., <50 mg·d^{-1}) and no hypersensitivity to caffeine. Table 1 presents the demographic characteristics of the subjects. Before participating in this study, all subjects signed an informed consent form and completed a health and exercise history questionnaire. All subjects were judged healthy according to the American College of Sport Medicine Guidelines (ACSM, 1998).

Experimental design

To examine caffeine ingestion effect on the acute hormonal response to a single bout of RE, the current study used a randomized, doubled-blind, within subject crossover design. Subjects visited the laboratory on two occasions prior to the experimental treatments. During the first visit, subjects signed the informed consent form and completed the health and exercise history questionnaire, and their height, body mass and body composition were measured. During the second visit, the subject's 1RM was determined. Finally, all subjects were randomly assigned to caffeine (CAF) and control (CON) in a counter balanced order. Subjects performed the two sessions at least seven days apart, and each session was performed at the same time of day. In the CAF and CON treatments, participants reported to the lab at 7:00 A.M. and ate a standardized breakfast consisting of toast (0.5 g·kg^{-1}) (toast contains 52 g CHO, 10 g fat and 12 g protein per 100 g) and water (500 ml), then sat quietly for 180 min to minimize the effects of breakfast ingestion on hormonal responses. The volume of fluid ingestion (only ingested water 500 ml in breakfast) was restricted to control hydration state of subjects around RE. Caffeine (6 mg·kg^{-1}) or a placebo consisting of dimethyl cellulose was provided to participants in identical capsules for ingestion 60 min prior to RE (8 exercises, 3 sets of 10 repetitions at 75% of 1RM). Blood samples were drawn prior to treatment ingestion (pre-60), immediately prior to RE (pre-exe), and 0, 15, 30 min post to RE (P0, P15, P30). The Blood glucose was determined and serum was analyzed to measure the concentrations of lactic acid, free fatty acid, insulin, GH, total testosterone and cortisol. Figure 1 shows the experimental timeline.

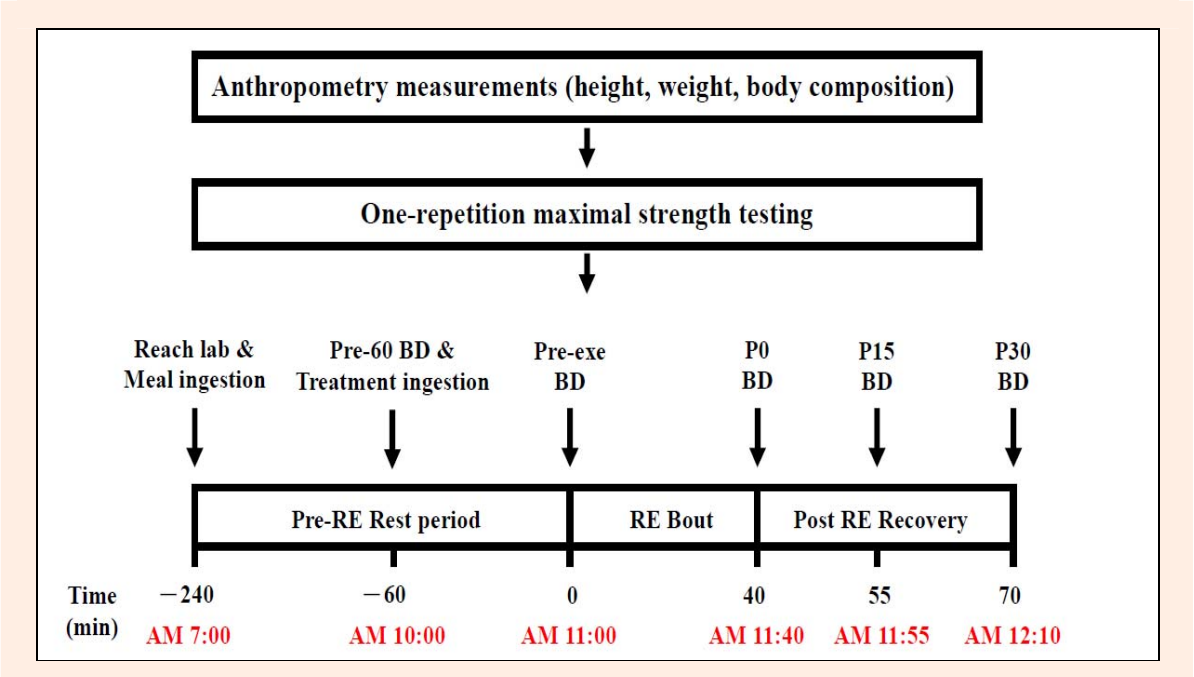

Figure 1. Acute resistance exercise bout protocol of treatment ingestion and blood sampling. BD, blood draw; pre-60, 60 min prior RE; pre-exe, immediately prior RE; P0, immediately post to RE; P15, 15min post to RE; P30, 30 min post to RE.

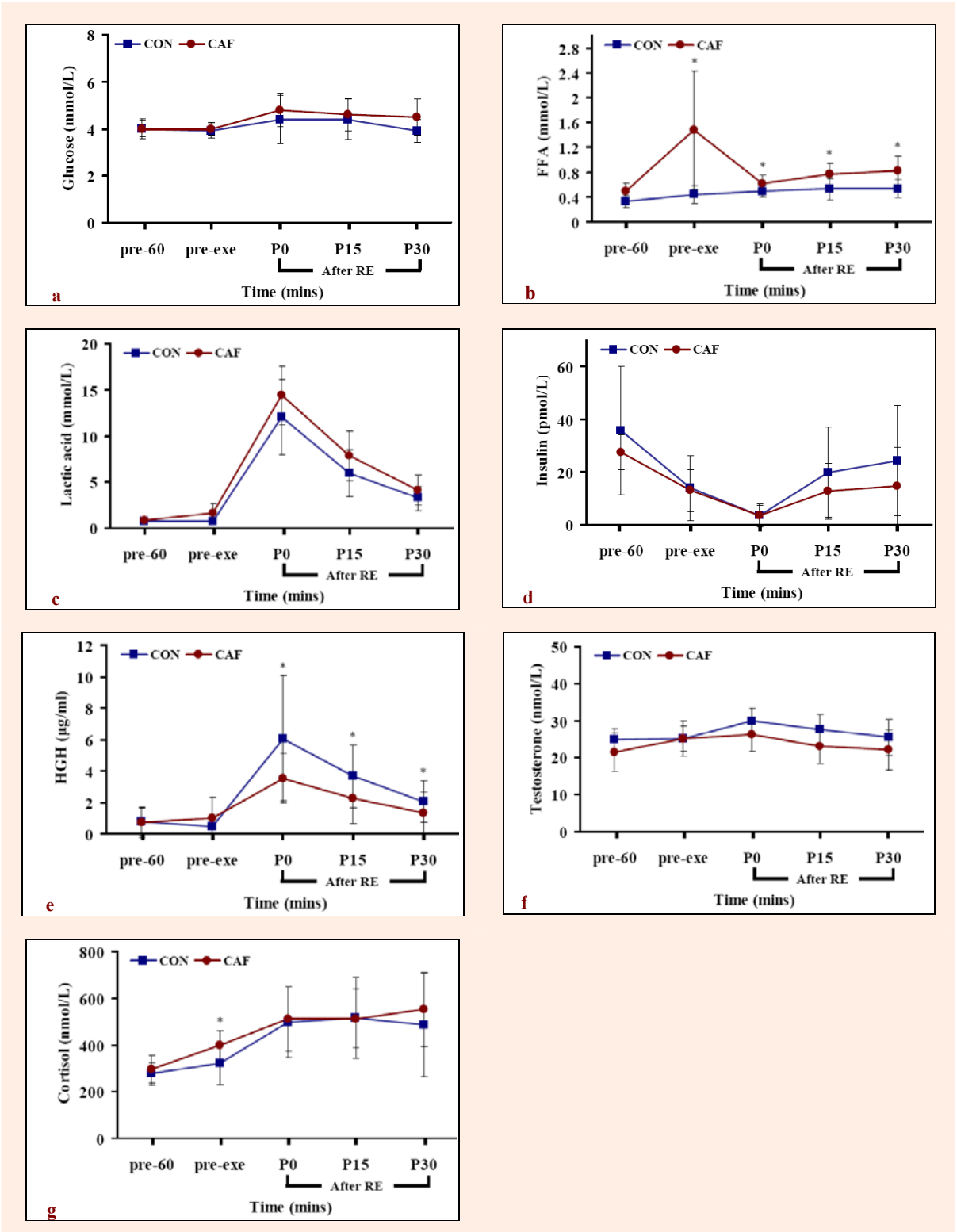

Figure 2. Changes in concentrations of a) blood glucose, b) serum free fatty acid (FFA), c) serum lactic acid, d) serum insulin , e) serum growth hormone (GH), f) serum testosterone and g) serum cortisol. Values are presented as mean ± standard deviation. * $p < 0.05$ between CON and CAF.

Exercise status and dietary control

All subjects were required to consume the same dinner and breakfast on the day prior to each trial. A dietary log for the preceding 24 h was collected to assess caffeine intake, and reminders were given to ensure dietary compliance. Participants also completed a short questionnaire at the conclusion of training to ascertain their perception of the caffeine dose ingested. Subjects were instructed to refrain from ingesting dietary caffeine such as coffee, tea, chocolate, caffeine-containing beverages and alcohol for the period of this experiment (about 3 weeks). Participants were also required to abstain from intense exercise in the 72 h before each protocol.

1-RM strength test

Before measuring 1RM, warm-up set of 5-10 repetitions at 50% of perceived maximum 1RM and stretching major muscle groups of the exercises were performed. After a 3-min rest period, the loads were set according to the perceived maximum 1RM of each subject. If the subject successfully completed the load more than two times during each trail, the tester increased the load, increasing the weight progressively until the subject failed at the given load. Three to five trials were conducted to determine 1RM. A 3-min rest was allocated between trails (Baechle and Earle, 2000).

Treatment ingestion

During the experimental session, caffeine (caffeine anhydrous, USB Corporation, Cleveland, OH, USA) or a placebo consisting of dimethyl cellulose was provided to participants in identical capsules for ingestion 60 min prior to RE. The caffeine dose was equal to 6 $mg \cdot kg^{-1}$ to raise the maximal blood level of caffeine (Graham and Spriet, 1995). Seven days later, subjects ingested the other treatment, and repeated the same resistance exercise protocol.

Resistance exercise protocol

The subjects completed a resistance exercise program that consisted of barbell arm curl, standing rowing, bench press, triceps press down, incline leg press, half squat, bent over rowing and leg extension exercises at 10 repetitions of 75% 1RM × 3 sets. The 1-minute rest between each set and the 2-min rest between each exercise were allowed for recovery. If the weight load became too heavy during the second or third sets, the subject was assisted during the last few repetitions. The resistance exercise session lasted approximately 50 min in the present study.

Blood sampling and analysis

After an overnight fast (12 hours), subjects arrived at the exercise physiology laboratory at 7:00 A.M. and ate a standardized breakfast immediately, then sat quietly for 180 min prior to the first blood collection. Venous blood was obtained from the antecubital forearm vein at resting (prior to consuming caffeine or placebo) immediately prior to RE (pre-exe), and 0, 15, 30 min post to RE (P0, P15, P30).

The blood samples were allowed to stand at room temperature and subsequently centrifuged at 3000 rpm for 10 min. These blood samples were kept frozen at -70 degrees until analysis. Blood glucose and lactic acid were determined by an enzymatic method (VITROS 5-1 FS, Ortho-Clinical Diagnostic Inc., Rochester, NY, USA), which with detection limits lower than 20 mg% and lower than 0.5 $mmol \cdot L^{-1}$, respectively. Serum GH, insulin, total testosterone and cortisol concentrations were determined by radioimmunoassay (RIA) (DPC, Diagnostic Products Corporation., LA, CA, USA) with detection limits lower than 1.3 $\mu IU \cdot ml^{-1}$, 0.01 $ng \cdot ml^{-1}$, 0.08 $ng \cdot ml^{-1}$ and 0.3 $\mu g \cdot dl^{-1}$, respectively. Serum FFA was determined by RIA (TOSHIBA CI8200, Toshiba Corporation, Otawara, Japan) with detection limits lower than 0 $mmol \cdot L^{-1}$. Analysis of glucose, lactic acid, insulin, GH, total testosterone, cortisol and FFA showed intra-assay coefficients of variation were 2%, 2%, 8%, 10%, 10%, 5% and 5%, respectively. Further, hemoglobin (XT-1800i, Sysmex UK Ltd, UK) and hematocrit (Microscopy) levels were measured to correct acute changes in plasma volume (Dill and Costill, 1974).

Statistical analysis

Descriptive data were generated for all variables and expressed as mean ± standard error of the mean. A 2 (treatments) × 5 (time) analysis of variance (ANOVA) with repeated measure was used to analyze hormonal and blood variable data. Following LSD post hoc tests were used to examine pairwise difference when significant F values were seen. Significant interactions were analyzed by simple main effects. Statistical power calculations ranged from 0.70 to 0.80 in the present study. Statistical significance in the present study was set at $p \leq 0.05$.

Results

Blood glucose

Figure 2a shows the blood glucose response before and after RE. A significant main effect was seen ($F = 7.1$, $\eta^2 = 0.44$, $p = 0.00$) with no significant interaction ($F = 1.8$, $\eta^2 = 0.17$, $p = 0.15$). In the CAF protocol, blood glucose concentrations after RE (P0, P15, P30) were significantly higher than resting value (pre-60) ($p < 0.05$), whereas those in the CON protocol were not. No difference was seen in resting and after RE concentrations between CON and CAF protocols for glucose.

Serum free fatty acid

Figure 2b shows the serum FFA response before and after RE. A significant main effect ($F = 9.8$, $\eta^2 = 0.52$, $p = 0.00$) and interaction ($F = 7.3$, $\eta^2 = 0.45$, $p = 0.00$) were seen. In the CAF protocol, serum FFA concentrations after CAF ingestion (pre-exe, P0, P15, P30) were significantly higher than resting value (pre-60) ($p < 0.05$), and the CON protocol also displayed mild elevation similar to that of the CAF protocol ($p < 0.05$). The FFA concentrations in the CAF protocol at pre-exe, P0, P15 and P30 were significantly higher than in the CON protocol ($p < 0.05$).

Serum lactic acid

Figure 2c shows the serum lactic acid response before and after RE. A significant main effect was seen ($F = 126.7$, $\eta^2 = 0.93$, $p = 0.00$) with no significant interaction ($F = 2.0$, $\eta^2 = 0.18$, $p = 0.11$). In the CAF protocol, serum lactic acid concentrations after RE (P0, P15, P30) were significantly higher than resting value (pre-60) ($p < 0.05$), and the CON treatment also displayed a similar trend to that of the CAF protocol ($p < 0.05$). Conversely, no significant difference was seen in resting and after RE concentrations between CON and CAF protocols for serum lactic acid.

Serum insulin

Figure 2d shows the serum insulin response before and after RE. A significant main effect was seen ($F = 13.1$, $\eta^2 = 0.59$, $p = 0.00$) with no significant interaction ($F = 0.7$, $\eta^2 = 0.07$, $p = 0.63$). In the CAF protocol, serum insulin

concentrations at pre-exe, P0 and P15 ($p < 0.05$) displayed a significant decrease relative to the resting value (pre-60), in the CON protocol also showed a significant decrease (pre-exe, P0) relative to the resting value (pre-60) ($p < 0.05$). In addition, no difference was seen in resting and after RE concentrations between CON and CAF protocols for insulin.

Serum GH

Figure 2e shows the serum GH response before and after RE. A significant main effect ($F = 17.4$, $\eta^2 = 0.66$, $p = 0.00$) and interaction ($F = 5.0$, $\eta^2 = 0.36$, $p = 0.003$) were seen. In the CAF protocol, serum GH concentrations after RE (P0, P15) ($p < 0.05$) were significantly higher than resting value (pre-60), and the CON treatment also displayed a similar trend to that of the CAF protocol ($p < 0.05$). Significant differences were seen after RE (P0, P15, P30) concentrations between CON and CAF protocols for GH ($p < 0.05$).

Serum testosterone

Figure 2f shows the serum testosterone response before and after RE. A significant main effect was seen ($F = 14.9$, $\eta^2 = 0.62$, $p = 0.00$) with no significant interaction ($F = 2.2$, $\eta^2 = 0.20$, $p = 0.08$). In the CAF protocol, serum testosterone concentration after RE (P0) was significantly higher than resting value (pre-60) ($p < 0.05$), and the CON protocol data also showed a significant elevation (P0, P15) ($p < 0.05$) relative to resting value (pre-60). Conversely, no significant difference was seen in resting and after RE concentrations between CON and CAF protocols for testosterone.

Serum cortisol

Figure 2g shows the serum cortisol response prior to and after RE. A significant main effect was seen ($F = 16.9$, $\eta^2 = 0.65$, $p = 0.00$) with no significant interaction ($F = 1.0$, $\eta^2 = 0.09$, $p = 0.45$). In the CAF protocol, serum cortisol concentrations at pre-exe, P0, P15 and P30 ($p < 0.05$) displayed a significant elevation relative to the resting value (pre-60), and the CON protocol data also showed a significant elevation (P0, P15, P30) ($p < 0.05$) relative to the resting value (pre-60). Additionally, a significant difference was evident immediately prior to RE (pre-exe) between CAF and CON protocols, whereas no significant differences were seen after RE (P0, P15, P30)

Discussion

The present investigation examined the influence of caffeine ingestion (6 $mg \cdot kg^{-1}$) on acute hormonal responses to a single bout of RE. This work hypothesized that caffeine ingestion prior to RE may elevate cortisol and attenuate GH secretion after RE. Primary findings from this investigation suggest that ingesting caffeine prior to RE results in significantly higher serum FFA concentration and a significant decrease in GH responses to RE. However, no significant difference was evident in other hormones (insulin, testosterone and cortisol) between CON and CAF protocols.

Findings show RE to significantly elevate human GH concentrations 15~30 minutes after RE (Kraemer et al., 1993). It appears that acute GH response to RE is highly influenced by total work (intensity, volume and rest intervals between sets), exercise selection and amount of muscle mass recruited of resistance exercise protocol (Kraemer and Ratamess, 2005). GH secretion from the hypothalamic-pituitary is regulated mainly by the GH-releasing hormone (GHRH) (Florini et al., 1996). The results of this study demonstrated a significant decrease in 22-kD GH concentrations after RE (P0, P15, P30), between CON and CAF protocols. 22-Kd GH is one form of the GH super family, but the concentrations of other variants are not examined in the present study. Although most studies focus on 22-Kd GH (Kraemer and Ratamess, 2005), a few investigations have suggested that other variants of GH appear responsive to RE (Hymer et al., 2001; Kraemer et al., 2003). Therefore, further research is obviously required to elucidate the impact of other GH variants to RE. A number of possible mechanisms exist for reduced acute GH secretion. A higher blood glucose level might suppress GH response to GHRH (Frystyk et al., 1997; Hjalmarsen et al., 1996; Nakagawa et al., 2002; Van Loon et al., 2003), however, caffeine ingestion prior to RE did not significantly elevate blood glucose concentrations of CAF in the present study. Several previous researches have reported that carbohydrate or amino acid ingestion around RE may raise insulin concentration, resulting in elevated GH after RE, thus insulin response was shown to affect GH response (Bird et al., 2006; Chandler et al., 1994; Kraemer et al., 1998; Volek, 2004), but no significant difference was seen for insulin response in the present study. Therefore, it is necessary that further studies identify the relation between caffeine ingestion and insulin response after RE. High correlations between blood lactic acid and GH response to exercise have been reported (Hakkinen and Pakarinen, 1993), but the acute blood lactic acid concentrations changes were similar between CAF and CON protocols. The present study also corrected hormonal responses post to RE for plasma volume changes, but acute GH concentrations after RE remained significantly decreased in CAF protocol, which does not appear to be a possible mechanism for GH response.

In addition, caffeine ingestion may elevate epinephrine levels to release FFA from adipose tissue cells at rest prior to exercise (Graham, 2001). The present study also reported that serum FFA concentrations significantly elevated before and after RE (pre-exe, P0, P15, P30). Several previous studies have indicated that higher FFA concentration may attenuate GH secretion (Blackard et al., 1971; Fineberg et al., 1972; Imaki et al., 1986). Lanzi et al. (1999) have also reported that higher circulating FFA levels exert negative feedback on the hypothalamus to attenuate GHRH secretion, resulting in the anterior pituitary decreasing the release of GH. The results of these studies have established a negative correlation between FFA concentration and GH secretion. A previous animal experiment has found that a high dose of caffeine infusion (30 and 50 $mg \cdot kg^{-1}$) stimulates the hypothalamus to release a growth hormone inhibiting hormone to suppress GH secretion (Spindel et al., 1980). Ratamess et al. (2007) have reported that consumed caffeine-containing energy drink (110 mg) prior to RE sig-

nificantly reduced acute GH secretions, but the FFA concentration was not examined. Furthermore, Goto et al. (2005; 2007) have also suggested that an acute rise in FFA concentrations prior to RE may attenuate acute GH secretions. Indeed, our data are in accord with the results of the previous studies that higher FFA levels may attenuate GH secretion. However, further investigation is needed to examine GHRH secretion and the growth inhibiting hormone to elucidate the relation between FFA concentration and GH response.

Testosterone has shown to elevate in response to RE and has been linked to strength and muscle mass gain (Kraemer and Ratamess, 2005). In the present investigation, serum testosterone concentrations of CAF and CON displayed a significant elevation relative to the resting value (pre-60). However, no significant difference was evident between CAF and CON protocols in our data. A previous animal experiment has found that a high dose of caffeine infusion (30 $mg \cdot kg^{-1}$ and 60 $mg \cdot kg^{-1}$) elevated plasma concentrations of testosterone (Pollard, 1988). Moreover, Beaven et al. (2008) also demonstrated a small elevation (21% ± 24%) in testosterone with a high dose of caffeine (800 mg) ingestion, whereas caffeine doses of ≥400 mg tended to cause a small decrease in testosterone after ingestion. Another investigation also indicated that consumed caffeine-containing energy drink (110 mg) prior to RE significantly reduced acute testosterone secretions (Ratamess et al., 2007). Caffeine dose ingestion in the present study was approximately 310~430 mg (6 $mg \cdot kg^{-1}$), accordingly, the variation in testosterone might not be enough to produce significant treatment effect. To our knowledge, few studies have examined the effect of caffeine on testosterone concentrations before and after RE, therefore, the detailed mechanism between caffeine ingestion and testosterone response to RE is not well understood. Previous study has shown that FFA attenuates testosterone secretions (Meikle et al., 1989), whereas the other has shown no significant effect (Murai et al. 1991). Our data also suggest that higher FFA levels do not attenuate testosterone secretions after RE.

Cortisol is a catabolic hormone, and may stimulate muscle protein degradation and inhibit protein synthesis in both type I and type II muscle fibers (Kazarian et al., 1983). Cortisol is known to elevate blood glucose and FFA to ensure an adequate fuel supply. Thus, variations of blood glucose and FFA concentrations around the time of RE may affect cortisol responses. Recent researches have reported that carbohydrate only or carbohydrate combined with amino acid supplement before and during RE significantly attenuated cortisol responses compared with a placebo (Bird et al., 2006; Tarpenning et al., 2003). However, others have suggested no significant effects (Bloomer et al., 2000; Kraemer, et al., 1998; Williams et al., 2002). Therefore, nutritional supplementation suppressing cortisol response is debatable. Woolf et al. (2008) displayed that caffeine ingestion (5 $mg \cdot kg^{-1}$) combined with the meal (~917 kcal; 14% protein, 62% CHO, 24% fat) prior to RE significantly elevated post-exercise cortisol concentrations, which might be responsible for the elevation in glucose concentrations. The result of another study also reported moderate elevation (52% ± 44%) in cortisol with high dose caffeine ingestion (800 mg) prior to RE, nevertheless, this study did not examine the concentrations of glucose and FFA (Beaven et al., 2008). The results of these studies infer that caffeine ingestion prior to RE may stimulate cortisol response. In our data, serum cortisol concentrations of CAF and CON displayed a significant elevation relative to the resting value (pre-60). Additionally, a significant difference evidenced immediately prior to RE (pre-exe) (rest period) between CAF and CON treatments. Recent research has also confirmed that caffeine ingestion might elevate cortisol at rest by stimulating the central nervous system (Lovallo et al., 2006), whereas no significant difference was observed after RE (P0, P15, P30) in our data. Moreover, no significant difference evidenced between CAF and CON protocols for blood glucose in the present study. Therefore, our study did not support that caffeine ingestion prior to RE may stimulate cortisol secretion after RE. These studies used a variety of experimental designs, exercise protocols and caffeine doses, making the results of these experiments difficult to compare with our study. Whether caffeine ingestion may stimulate cortisol response to RE remains unclear, thus this issue requires further investigation.

In this research, caffeine consumption of the subject, training state and hydration state might have affected the acute hormonal concentrations to caffeine. The participants in this study were very low caffeine consumers, which might have contributed to the results. Furthermore, we did not measure plasma caffeine and serum epinephrine concentrations, thus we could not estimate the effects of caffeine in subjects prior to RE. Subjects in the present study were department of physical education students, thus the physical activity that the subjects undertook during the weeks before the study might have been quite different, but a similar volume of physical activity was performed during the two testing weeks. A crossover design was applied to help control for this variability. This study also restrained subjects from performing RE and exhaustive exercise during the experiment period. The hydration state of subjects also might have affected the hormonal concentrations of this study. Although we did not take a urine sample prior to RE, the volume of fluid ingestion was restricted to control hydration state of the subjects around RE. We also measured hemoglobin and hematocrit to correct acute changes in plasma volume after RE. The results of this study indicate that caffeine ingestion might attenuate the acute response of GH. However, acute GH response may not directly affect muscle synthesis. We should observe long-term effects of caffeine ingestion and resistance training on hormonal responses in a further investigation.

Conclusion

In summary, the present study demonstrates that caffeine ingestion (6 $mg \cdot kg^{-1}$) one hour prior to RE reduces GH response after a single bout of RE. This effect might result from elevated FFA concentration of blood before and after RE. Although we did not examine GHRH and growth hormone inhibiting hormone responses in this study, we could not exclude that the negative feedback mechanism operates in the attenuation of GH response.

In addition, the current study findings show no significant treatment effect in insulin, testosterone and cortisol after RE. The relation between caffeine ingestion and hormonal response is not well-understand. This issue requires more investigations as to whether caffeine ingestion effects secretions of anabolic and catabolic hormone to RE.

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Key points

- Caffeine ingestion may attenuate the response of GH to a single bout of resistance exercise.
- The depression of GH response may be caused by the elevation in serum FFA concentration at the beginning of resistance exercise.
- Caffeine ingestion before resistance exercise may not alert the concentration of cortisol and testosterone.

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