2020, Volume 3 (Issue 4): 20 OPEN ACCESS

Acute Bout of Resistance Training Reduces Glucagon-like Peptide 1 Concentration in Females

Original Research

Jeffrey E. Herrick, PhD[1], Sean M. Collins, PhD, CSCS-D[1]

[1]*University of Lynchburg, Lynchburg, Virginia, USA*

Abstract

Introduction: Resistance training (RT), is a common and recommended strategy for improving muscle mass and function that may modify appetite biomarkers. Glucagon-like peptide-1 (GLP-1) is a rapid acting appetite control hormone. The purpose of this study was to determine if RT effects GLP-1 in females independent of an exercise promoted calorie deficit.

Methods: Two randomized experimental beverages with differing calories were given prior to two RT days: 1) matched calories for RT (MC) and 2) no calories (NC). RT; 3 sets, 10 repetitions, 8 standard exercises at 70% 1-RM. Fasted blood was collected pre-beverage (PRE), 15-minutes post-beverage (POST), 5P, 15P, and 30P-minutes post-RT.

Results: Six females (age 21.8 ± 2.6 yrs) completed the study. Differences from PRE to POST, 5p, 15P, 30P were measured with ANOVA, and main effects analyzed with Cohen's d effect size (d). NC promoted a significant reduction in GLP-1 at 15P, 30P RT (GLP-1 PRE 3.25 ±2.5, 15P 2.42 ±2.6, 30P2.46 ±2.1 ng/dL, $p \leq 0.05$). There was no significant difference in GLP-1 in the matched calorie treatment (MC) at any time point in the study (PRE to POST, 5P, 15P & 30P).

Conclusions: Our research suggests that RT does not activate GLP-1 secretion following an acute bout of exercise independent of caloric expenditure. Rather, RT performed without pre-exercise calorie consumption reduced GLP-1 in physically active females.

Key Words: Resistance training, hormones, females.

Corresponding author: Jeffrey E. Herrick, PhD, herrick_je@lynchburg.edu

Published December 10, 2020

Introduction

Resistance training exercise is a common intervention for increasing skeletal muscle mass which is associated with a multitude of additional health and performance benefits [1]. In weight control programming resistance training is a common exercise to both increase acute and long-term skeletal muscle caloric expenditure in an attempt to positively effect an individual's energy balance status. However, the effectiveness of exercise prescriptions for weight control may be contraindicated if appetite biomarkers are increased following exercise; which may trigger the desire to eat and confound energy balance status. Understanding the mediating effects of resistance training on endocrine appetite control mechanisms is critical to support weight control programming.

The majority of exercise and appetite control mechanisms research is almost exclusively on continuous, steady-state endurance exercise with little to no research using resistance training. Continuous endurance training exercise is a high calorie expenditure activity and has been shown to reduce perceived appetite and hunger [2]. Indeed, in healthy males, endurance exercise was shown to decrease both the perception of hunger and actual ad libitum energy intake following exercise [3]. In addition, 60-minutes of moderate

intensity treadmill running similarly reduced the perceptions of hunger immediately following exercise without a promoted change in ad libitum energy intake once appetite returned in healthy males [4]. Exercise duration and/or total calorie expenditure may mediate appetite suppression, as a 60-minute bout of moderate intensity aerobic exercise promoted greater appetite suppression and post exercise caloric consumption when compared to multiple short high intensity bouts of maximal sprinting [5]. In contrast to continues endurance exercise, resistance training is typically shorter in duration and lower in acute energy expenditure then aerobic exercise. As such, it is plausible that the acute caloric dependent mechanisms of endurance exercise that suppress appetite may not exist in resistance training. Therefore, it is not clear if resistance training with shorter movement times and lower acute caloric expenditure mediates the fast-acting appetite signals.

Glucagon-Like Peptide 1 (GLP-1) is a potent episodic (fast-acting) satiety hormone that responds to both changes in gut volume and nutrient content and acts centrally to suppress appetite via the vagal reflex to the hunger centers of the brain [6–8]. Specifically, a drop in blood GLP-1 is associated with an increased appetite and a drive to eat. GLP-1 blood concentrations have been linked to decreased rate of gastric emptying and is associated with reduced food intake and prolonged feelings of satiation [7,8]. The rapid and robust response of GLP-1 secretion following endurance exercise suggests it may be a strong endocrine biomarker to understand an exercise appetite effect. GLP-1 secretion appears to be up-regulated during continuous endurance exercise independent of caloric ingestion suggesting that mechanisms other than gut nutrient contents may mediate GLP-1 during prolonged endurance type exercise. A relationship between short duration resistance training, energy intake status and GLP-1 concentration are not currently understood. Therefore, the purpose of this study was to investigate the direct independent effects of a resistance training session on short term post-exercise GLP-1 concentration using an acute energy balance model.

Methods

Two randomized blinded counterbalanced experimental resistance training trials were completed on different days with three to five days between the practice sessions and each experimental trial. The 400 milliliter experimental beverages consisted of meal replacement shake with calories estimated to the individual resistance training session from visit two which represents the acute energy balance treatment (HRM Foods, San Diego, CA) and 400 milliliter zero-caloric flavored drink energy deficit treatment (Crystal Light, Kraft Foods, Northfield, IL). Each participant consumed the experimental beverages 30-minutes before the start of the exercise session following an overnight fast.

Participants

Six physically active females were recruited to participate in this study and all participants did not engage in regular resistance training (Table 1). Eligible participants self-reported that they were free of cardiopulmonary, metabolic or respiratory diseases, binge eating disorders, non-smoker, and were not currently prescribed any medication for asthma, hypertension, gastrointestinal disorders, and/or neurological disorders. All subjects completed self-reported medical history questionnaire and informed consent prior to enrollment. Ethical approval for this study was granted by the university Institutional Review Board and each subject was informed of the risks and benefits of the investigation prior to signing and informed consent.

Variable	Mean ± Standard Deviation
Total Female Participants	6
Age (Years)	21.8 ±2.6
Height (cm)	171.2 ±5.6
Weight (kg)	57.4 ±2.1
BMI (kg/m^2)	20.6 ± 0.7
Total % Fat	26.9 ±2.1
Total Fat (kg)	14.5 ±1.5
Total Fat Free Mass (kg)	39.3 ±1.7

Table 1. Participant profiles and anthropometrics.

Procedures

Each participant visited the laboratory on four separate days (Figure 1). The first visit included determination of inclusion criteria and the informed consent process, in addition to baseline anthropometric assessment and determination of one-repetition maximum for each resistance training exercise. Visit two, each subject completed the resistance training protocol including three sets of ten repetitions at 75% of one repetition maximum (1RM). I addition, the resistance training exercise session caloric cost was determined sequence with a one to two-minute rest between. Total caloric cost of the exercise session was determined during Visit two via indirect calorimetry (Cosmed, K4B2, Cosmed Corp, Milan, Italy). Visits three and four were the randomized experimental trials which involved the consumption of the experimental beverages consisting of a caloric-matched (MC) and a caloric-deficit (NC) trial followed by a ~60-minute resistance training exercise session.

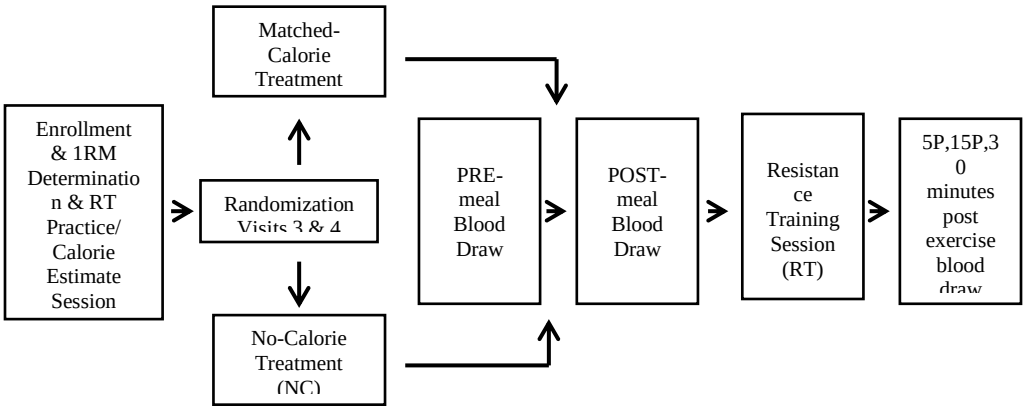

Figure 1. Experimental protocol work flow. GLP-1 and HVAS Measurement at Pre-beverage (PRE), 15-minute post-beverage (POST), and 5, 15, and 30-minutes post resistance training, (5P, 15P, 30P, respectively)

Anthropometric and Body Composition Assessment

Initial assessment involved measurement of height, weight, body mass index, using standard procedure (American College of Sports Medicine, 2013). Body composition was determined using dual –x-ray absorptiometry (DXA, GE iDXA, Milwaukee, WI) to determine fat mass, fat-free mass along with the regional and total fat percentages.

Resistance Training Exercise Protocol

One repetition maximum (1RM) was estimated using an indirect ten repetition maximum protocol to predict 1RM during visit one (Haff & Triplett, 2016). On visit two subjects completed one experimental protocol practice session of three sets of ten repetitions at 75% of the estimated 1RM for the chest press, bicep curl, triceps extension, lat pulldown, abdominal flexion, back extension, leg extension, leg curl, leg press, and hip abduction exercises to ensure capacity to complete experimental protocol (Cybex International, Medway, MA). A recovery of 30-60 seconds was provided between each resistance exercise set. Determination of total volume load for each resistance training exercise and for the total training session was calculated based off the estimated 1RM using standard procedures [9]. The volume load was calculated by multiplying number of sets by number of repetitions by total weight lifted (i.e. 3 sets x 10 repetitions x 30 kg = 900), volume load was used to compared work between the experimental trials.

Determination of Caloric Cost of Exercise Session

Resistance training oxygen consumption (VO_2) and subsequent caloric expenditure were determined as the total session time difference between resting VO_2 (3.5 $ml \bullet kg^{-1} \bullet min^{-1}$ used for all participants) times

total exercise time and the actual VO_2 each minute during the entire exercise session. Relative oxygen consumption values ($ml \bullet kg^{-1} \bullet min^{-1}$) were then converted to absolute oxygen consumption ($L \bullet min^{-1}$) and used to convert to calories per minute using standard procedures and summed to represent the calorie cost of the resistance training exercise [10].

Glucagon-Like Peptide-1 (GLP-1) Measurement

Collected blood samples were stored at -80◦ C and were batch analyzed for total concentration of glucagon-like peptid-1 (active) via standard enzyme-linked immunosorbent assay (ELISA) methods with pre-coated antibody plates (Linco Inc, St. Charles, Missouri) using an absorbance microplate reader with a 450 nM filter (iMark, Bio-Rad, Hercules, California). Whole blood was collected via venipuncture five times (Figure 1) during the experimental protocol: fasted prior to experimental meal, 15-minutes post experimental meal, and immediate-post exercise, 15-minutes post exercise, and 30-minutes post whole blood was processed and stored in a -80 ◦ C refrigerator for batch analysis.

Statistical Analysis

Statistical differences were measured within each treatment between pre-treatment GLP-1 and each subsequent time point using analysis of variance and Tukey HSD, significance was set as a $p \leq 0.05$. In addition, we assessed the magnitude of the effect of the independent variable (IV - calorie content consumed before exercise; NC and MC treatments) on the dependent variables (DV) of GLP-1 with Cohen's d effect size statistic, d . We compared the magnitude of effect of the fasted pre-beverage dependent variables GLP-1, to the GLP-1 at each time point following consumption of the beverage (POST) and at the 5, 15, and 30-minute post exercise time point, 5P, 15P, 30P respectively. Cohen's d effect size was interpreted as a small effect 0.0-0.2, medium effect 0.3-0.5, and a large effect as 0.6-2.0 [11].

Results

All enrolled subjects (N = 6) completed all four visits of the experimental design and were able to complete all the prescribed resistance training exercises on both the NC and MC visits. The total volume load of each resistance training session was not different between experimental days (Volume Load; NC 6852 ±1181 vs MC 6836 ±1197 weight·reps, $p = 0.99$). There was no significant difference 15-minutes after consuming the beverage (PRE vs POST) in the no-calorie (NC) treatment in GLP-1 ($p = 0.93$). GLP-1 decreased significantly at the 15P and 30P post exercise measurement ($p = 0.1$ and $p = 0.005$, respectively). There was no significant difference in GLP-1 in the matched calorie treatment (MC) at any time point in the study (PRE to POST, 5P, 15P & 30P). All GLP-1 descriptive statistics may be seen in Table 2.

	PRE	POST	5P	15P	30P
NC GLP-1 (ng/dL)	3.52 ±2.5	3.58 ±3.0	2.97 ±3.2	2.42 ±2.6*	2.46 ±2.1*
MC GLP-1 (ng/dL)	2.48 ±1.8	3.42 ±2.3	3.36 ±2.9	2.99 ±2.2	3.38 ±3.0

GLP-1-Glucagon like peptide-1, NC-no calorie treatment beverage, MC-matched calorie treatment
* Significant difference to PRE measurement, $p \leq 0.05$.

Table 2. Changes in GLP-1 between trials and over the experimental time points.

In addition to GLP-1 differences between the two treatment conditions we assessed overall effect of the treatments in GLP-1 and hunger using the Cohen's d statistic. The no calorie treatment (NC) had no effect on reducing GLP-1 concentration immediately following consumption of the experimental beverage (NC GLP-1; PRE 3.52 ± 2.5 vs POST 3.58 ± 3.0 ng/mL, $d = 0.010$). Following the NC exercise session there was moderate effect on reducing GLP-1 concentration at the 15P time point which was sustained at 30P when compared to PRE (15P $d = 0.20$; 30P $d = 0.22$). The matched calorie treatment (MC) produced a moderate effect on increasing GLP-1 concentration following consumption of the beverage (PRE vs POST), which was sustained throughout following the exercise session. (MC PRE GLP-1 2.48 ±1.88, POST 3.42 ±2.3, $d = 0.21$).

Discussion

The primary findings from our research indicates that an acute bout of resistance training (RT) does not independent of calorie expenditure effect GLP-1 secretion. Specifically, we report that RT decreased

GLP-1 when no calories (NC) were consumed prior to the acute exercise session, suggesting a calorie dependent response. This calorie dependent response was not observed when an exercise matched calories beverage (MC) was consumed prior to exercise. Further, our observed drops in GLP-1 concentration occurred at 15 and 30-minutes post exercise, which suggests there is a ~15-minute delay in the peripheral secretion of GLP-1 following an acute bout of resistance training.

The timing between meal consumption and sustained aerobic exercise is of great interest and has been shown to influence perceptions of hunger along with GLP-1 levels [12,13]. Many factors regarding the exercise dose may affect the physiological and perceptive level of appetite; such as exercise mode, total caloric load of the exercise session, exercise intensity, and exercise duration. For instance, the consumption of food calories prior to 50-minutes of constant aerobic exercise reduced perceptions of hunger to a greater extent in comparison to exercising in a fasted state in healthy young men, although appetite was suppressed in both states [12]. Additionally, 60-minutes of constant aerobic exercise performed in a fed state has also been shown to elevate levels of GLP-1 during and immediately following a bout of moderate intensity exercise [13]. Suggesting that aerobic exercise alone does not directly suppress GLP-1 secretion. Significantly lower feelings of hunger in men when an exercise-induced energy loss was counterbalanced with excess energy intake several days prior to the moderate intensity aerobic exercise [14]. In contrast, resistance exercise poses a far different disruption to energy homeostasis and the blood substrate concentration and does not activate similar caloric and or cholinergic mechanisms to suppress appetite.

Study limitations that must be considered when interpreting the results: The sample size of 6 was fairly homogeneous and may limit the generalizability of the findings beyond the profile of our participants. A larger and more diverse pool of participants in race, age, and training status may provide greater understanding of resistance training effect on GLP-1 secretion. In addition, our experimental design only collected blood samples up to 30-minutes post exercise. It is plausible the GLP-1 post exercise effect may extend beyond 30-minutes. Lastly, despite GLP-1 acting as a biomarker for appetite signaling, we did not specifically ask the participants to rate their appetite. Subjective reports of appetite may better provide a translatable exercise effect of resistance training on appetite.

In conclusion, we demonstrated that resistance exercise does not appear to independently activate GLP-1 outside of any caloric expenditure. Rather, resistance training appears to effect GLP-1 secretion through a caloric dependent mechanism. In addition, we demonstrated that consuming a caloric beverage that matched the estimated energy expenditure of a single bout of resistance training (acute energy balance) attenuated a drop of the rapid appetite suppressing hormone GLP-1 following a single bout of resistance training.

Media-Friendly Summary

Resistance training may prove to be an effective exercise intervention to promote gains in lean muscle mass and overall muscular strength. Resistance training may best be administered following consumption of a caloric drink that matches the estimated energy expenditure of the exercise session, which may limit the potential increase in appetite.

Conflict of Interest

The authors report no conflict of interest.

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