

Research article

Acute Maltodextrin Supplementation During Resistance Exercise

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Abstract

Most of the research investigating the ergogenic enhancing mechanisms of carbohydrate have been conducted using aerobic based exercise. Therefore, the purpose of this study was to investigate the effects of pre-exercise maltodextrin ingestion on resistance exercise performance, serum insulin, epinephrine, glucose, and muscle glycogen concentrations. In a double blind, cross over, repeated measures design, participants completed four sets to failure at 70% of 1-RM with 45s rest on the angled leg press with or without pre-exercise maltodextrin (2g/kg) after a 3hr fast. Serum glucose, epinephrine, and insulin were assessed at baseline, 30 min post-ingestion, immediately after, and 1hr post-exercise with or without carbohydrate supplementation. Muscle glycogen was assessed from biopsy specimens sampled from the vastus lateralis before supplementation, immediately after exercise, and 1hr post exercise under both conditions. There was no main effect of supplement on resistance exercise performance ($p = 0.18$). Muscle glycogen concentration decreased across time for both groups ($p < 0.001$). There was an interaction in serum glucose decreasing more during exercise in the carbohydrate condition ($p = 0.026$). An interaction occurred showing insulin decreased during exercise in the carbohydrate condition ($p = 0.003$). Also, there was a main effect of insulin being elevated with carbohydrate consumption ($p = 0.027$). Epinephrine was decreased across all time points after carbohydrate ingestion ($p = 0.023$). Carbohydrate supplementation before resistance exercise did not improve leg press performance to fatigue despite increased metabolic substrate availability. These results indicate that pre-exercise dietary carbohydrate will be utilized preferentially during exercise due to decreased epinephrine, decreased serum glucose, and increased insulin concentrations. However, the increases in glycolytic substrate availability will not increase exercise performance or glycogen content following 1hr of recovery.

Key words: Skeletal muscle, anaerobic exercise, hormones, glycogen.

Introduction

Glucose is stored in large quantities as glycogen in the liver and skeletal muscles. There have been numerous theories on what governs athletic performance and many studies have reported that endurance performance is limited with depleted or low glycogen levels in muscle (Lima-Silva et al., 2009; Coyle and Coggan, 1984; Hermansen et al., 1967). Decreased glycogen content (within the muscle) is linked to decreased contractile force which is detrimental to certain sport performances (Ørtenblad et al., 2011). During exercise, energetic demands increase substantially and the need for ATP increases to sustain muscle contractions. Glycogen serves as the main substrate for adenosine

triphosphate (ATP) synthesis during moderate- to high-intensity exercise (van Loon, et al., 2000; 2001). Theories attempting to explain mechanisms of fatigue involve glycogen depletion, concluding low metabolic carbohydrate fuel causes fatigue (Williams et al., 2013; Coyle and Coggan, 1984; Hermansen et al., 1967). Low glycogen levels have been linked to decreased sarcoplasmic reticulum Ca^{2+} release detrimentally altering muscle fiber contractility (Ørtenblad, et al., 2011; 2013). Individuals may have compromised contractile abilities if muscle glycogen levels are low.

Most sports teams aim to improve physical ability by utilizing some form of resistance training. The primary bioenergetic pathways involved with anaerobic exercise are intramuscular ATP hydrolysis, creatine phosphate (CrP)-ATP/phosphagen system, and anaerobic glycolysis primarily involving glycogen (Jacobs et al., 1982). Teams use multiple training sessions in a day to maximize adaptations and increase performance. Completing multiple training sessions in a 24-hour period requires glycogen replenishment to allow for continued exercise intensity in later training bouts. The amount of glycogenesis possible during recovery is directly related to the total availability of carbohydrates post-exercise (Bergstrom and Hultman, 1966; van Loon et al., 2000; Costill, 1991). Consequently, limited carbohydrate intake may decrease performance, training adaptations, and recovery.

Carbohydrate ingestion with aerobic-based exercise has been investigated over the past century (Harris et al., 2018). Studies have shown that carbohydrate consumption can increase time to exhaustion and delay fatigue in extended aerobic exercise (Coyle et al., 1985). In resistance exercise, positive ergogenic effects have been seen over longer durations of resistance exercise (>50min) with moderate loads and high volume, while negligible findings have been found when exercise is shorter (<40min), or loads are higher (Lambert et al., 1991; Haff et al., 2001; Haff and Stone, 2003; Jacobs et al., 1982; Kulik et al., 2008). This indicates that carbohydrates ergogenic aid may be more apparent when the exercise bout is more synonymous with aerobic exercise or once muscle glycogen levels have decreased appreciably. In addition, carbohydrate supplementation with resistance exercise may increase the ability to sustain intensity in subsequent training due to glycogen sparing and replenishment post exercise (Haff et al., 2000). While general carbohydrate intake guidelines for performance are available, the dose, timing, and mechanism for nutritional ergogenic aid is understudied and needs further investigation (Kerksick et al., 2017; Slater and Phillips, 2011; Tsintzas and Williams, 1998; Haff and

Stone, 2003). Hormonal responses to carbohydrate supplementation have practical applications for performance enhancement and recovery, dictating the amount of glycogen used during exercise and synthesized in recovery. Therefore, the purpose of this study was to investigate the effects of carbohydrate supplementation 30min before resistance training on exercise performance, serum glucose, epinephrine, insulin, and muscle glycogen before, immediately post-exercise, and 1hr post-exercise.

Methods

Ten healthy, recreationally resistance-trained men [regular (i.e. thrice weekly) for at least 1 year], who had a mean \pm SD body mass of 90 ± 18.2 kg, height 1.79 ± 0.06 m, age 21.6 ± 2.27 yr, 5.98 ± 1.55 leg press strength-to-body weight ratio, and body fat $21.3 \pm 8.1\%$, participated in this study. Only participants considered low risk for cardiovascular disease with no contraindications to exercise outlined by the American College of Sports Medicine, and who had not consumed any nutritional supplements (excluding multi-vitamins) one month prior to the study could participate. This study was approved by the Institutional Review Board for Human Subjects at Baylor University. Additionally, all experimental procedures involved in the study conformed to the ethical consideration of the Declaration of Helsinki.

Participants were familiarized to the study protocol via a verbal and written explanation outlining the study design and then read and signed a university-approved informed consent document. In addition, each participant was instructed to refrain from exercise for 48 hours before each testing session, eat a light, low carbohydrate meal 3 hours prior to reporting for each testing session, and record their dietary intake for two days (including the light meal the morning of testing) prior to each of the three testing sessions. Diets were not standardized but participants were asked not to change their dietary habits. The MyFitnessPal mobile application (Under Armor Inc., Baltimore, MD, USA) was used to determine the average daily macronutrient consumption of fat, carbohydrate, and protein. Participants completed a medical history questionnaire and underwent a general physical examination to determine whether they further met eligibility criteria.

At session 2, participants performed angled leg press one-repetition maximum (1-RM) tests with the National Strength and Conditioning Association (NSCA) recommendations (Moir, 2010). Foot placement was recorded and held constant over all testing conditions. A goniometer was used to establish 90° of knee flexion while positioned on the leg press machine and safety catches were adjusted just below this point for all tests to standardize the range of motion to 90° . The participants were instructed to lower the weight just above the catches before pressing the weight upward.

Participants warmed up with 10 repetitions at approximately 50% estimated 1-RM, rested 1 minute, and completed 5 repetitions at approximately 70% estimated 1-RM followed by 2 minutes rest. The weight was then

increased conservatively, and the participants attempted the first 1-RM. If the lift was successful, the participant rested for 2 minutes before attempting the next 1-RM. The 1-RM of each participant was compared to male 90% rank normative values of age specific strength-to-body weight ratios (2.27) (Hoffman, 2007). This substantiated that all participants were trained for at least a year as stated in the exercise questionnaire. Thirty minutes following the 1-RM test, participants completed 4 sets of maximal repetitions as outlined in the resistance exercise protocol.

During testing sessions 3 and 4, participants completed a warm-up set of 10 repetitions and 5 repetitions at 50% and 70% of the testing load, respectively. Each set was followed by two minutes of rest and then the exercise testing session began. Participants performed 4 sets of repetitions to volitional fatigue with 70% of the 1-RM on the angled leg press. A 45-second rest interval was provided between sets. When the subject was not able to perform another repetition in the set, study personnel assisted to help re-rack the weight safely. The total number of repetitions performed at each testing session was recorded.

At session 3 and 4, 30 minutes before resistance exercise, in a randomized, doubled-blind fashion, participants ingested either a placebo or a carbohydrate supplement. The 30min time point was selected in an attempt to maximize the bio-available glucose in the blood that could be used at the onset of exercise (Pannoni, 2011). The placebo consisted of a flavored, non-caloric beverage [(Crystal Light) Kraft Foods, Chicago, IL, USA] mixed with water and orally ingested. The carbohydrate supplement was maltodextrin (Carbo Gain, NOW Sports, Bloomington, IL, USA) at a dose of 2 g/kg body mass, mixed with Crystal Light in water, and orally ingested (Wax et al., 2012). Both supplements were of similar color, taste, volume, and consistency. A period of 7 to 10 days separated sessions 3 and 4 to allow for adequate supplement washout and muscle recovery.

Venous blood samples were obtained from an antecubital vein into 10 ml vacutainer tubes before supplement ingestion, immediately before resistance exercise (30 minutes following supplement ingestion), immediately post-exercise, and 1-hr post exercise. Blood samples stood at room temperature for 10 minutes and then centrifuged at 2500 rpm. The serum was removed and frozen at -80°C for later analysis.

Blood was drawn into microhematocrit tubes (in duplicate) by capillary action and sealed with a clay material. The microhematocrit tube was placed into the centrifuge, balanced, spun for 2 minutes, then removed and read on a hematocrit reader card. Normal ranges in adult males are between 42-52% and over 54% was considered dehydrated and rehydration was required before further testing (Pagana and Pagana, 2013).

Three percutaneous muscle biopsies (~30 mg each) were completed at visits 3 and 4 using fine needle aspiration upon entry, immediately post-exercise, and 1hr post-exercise. Biopsies were obtained from the middle portion of the vastus lateralis muscle of the dominant leg at the midpoint between the patella and the greater trochanter

of the femur at a depth between 1 and 2 cm. After the initial biopsy, remaining biopsies attempts were made to extract tissue from approximately the same location as the initial biopsy using the pre-biopsy scar, depth markings on the needle, and a successive puncture was made approximately 0.5 cm from medial to lateral. Adipose tissue was trimmed, and muscle specimens were immediately stored at -80°C for later analysis.

Serum glucose (Cayman Chemical, Ann Arbor, MI, USA) and muscle glycogen (BioVision, Milpitas, CA, USA) were assessed with colorimetric assays and absorbances read at wavelengths of 514 and 570 nm, respectively. Serum insulin (Cayman Chemical, Ann Arbor, MI, USA) and epinephrine (MyBioSource, San Diego, California, USA) concentrations were determined using a commercially-available enzyme-linked immunosorbent assay kits and absorbances were read at wavelengths of 414 nm and 450 nm, respectively. The absorbances of all serum variables were determined with a microplate reader (X-Mark, Bio-Rad, Hercules, CA, USA) and concentrations determined by linear regression against known standard curves using commercial software (Microplate Manager, Bio-Rad, Hercules, CA, USA). In addition, all samples were assayed in duplicate to ensure reliability of the assay results.

The statistical analysis for performance between supplement conditions was completed using a 2×4 [Condition (CHO, Placebo) \times Set (1,2,3,4)] factorial analyses of variance (ANOVA) with repeated measures for reps to fatigue. Statistical analyses for blood concentrations were performed by utilizing separate 2×4 [Condition (CHO, Placebo) \times Time (Pre, post-consumption, post-exercise, 1-hr post-exercise)] factorial ANOVA with repeated measures for serum glucose and insulin. The 2×4 [Condition (CHO, Placebo) \times Time (Pre, post-consumption, post-exercise, 1-hr post-exercise)] factorial ANOVA for epinephrine could not be run due to the test assumptions of normality being violated. The outliers ($> \pm 2\text{sd}$) were removed meeting the needed assumptions for the ANOVA; however, this created a statistical difference between baseline values in supplement conditions. To assess the differences in concentrations accounting for baseline differences between conditions, a 2×3 [Condition (CHO, Placebo) \times Baseline Change (Pre-Supplement, post-exercise, 1-hr post-exercise)] factorial ANOVA with repeated measures was completed. Muscle glycogen concentration analysis was performed by utilizing a 2×3 [Condition (CHO, Placebo) \times Time (Baseline, post-exercise, 1-hr post-exercise)] factorial ANOVA with repeated measures. If a significant

interaction was found, simple effects analysis was conducted to determine where the interaction occurred. If a significant interaction was present, analysis of main effects was conducted using the simple effects, pairwise comparisons with a Bonferroni adjustment to compare dependent variables within each independent variable condition. If no interaction was present, then normal pairwise comparisons with a Bonferroni adjustment were used to test main effects. Partial Eta squared (η^2), was used to estimate the proportion of variance in the dependent variable explained by the independent variable. Partial Eta squared effect sizes determined to be: weak = 0.17, medium = 0.24, strong = 0.51, very strong = 0.70. For all statistical analyses, an alpha level of 0.05 was adopted throughout.

Results

Macronutrient intake and hydration status recorded at each testing condition is displayed (Table 1). Results indicated that there was no difference for the intake of carbohydrate ($F = 1.051$, $p = 0.370$, $\eta^2 = 0.105$), fat ($F = 0.087$, $p = 0.917$, $\eta^2 = 0.01$), protein ($F = 0.576$, $p = 0.572$, $\eta^2 = 0.06$) or total kcals ($F = 0.48$, $p = 0.646$, $\eta^2 = 0.047$). No significant differences in body mass ($t = 0.429$, $p = 0.678$), body fat percentage ($t = 1.083$, $p = 0.307$), total body water ($t = -0.298$, $p = 0.773$), or packed cell volume ($t = 0.58$, $p = 0.576$) were detected over the two supplement testing visits.

Completed repetitions to fatigue for each condition and during each set are indicated in Table 2. No main effect for supplement on repetitions to fatigue during each set between supplement conditions was found ($F = 2.169$, $p = 0.175$, $\eta^2 = 0.194$). There was a main effect for the number of sets completed, however, showing as the number of sets increased the number of completed repetitions decreased ($F = 26.18$, $p < 0.001$, $\eta^2 = 0.933$). Pairwise comparisons indicated there was a significant difference between all set comparisons, except sets 3 and 4 ($p = 0.212$). There was not a significant interaction between set and supplement on repetitions to fatigue ($F = 0.337$, $p = 0.799$, $\eta^2 = 0.036$).

Table 2. Performance measures. Data are means (\pm SD).

Performance Measure	Placebo Visit	Carbohydrate Visit
Total Reps to Fatigue	53.8 (± 7.8)	51.8 (± 6.9)
1st Set to Fatigue	29.9 (± 5.9)	29.5 (± 4.9)
2nd Set to Fatigue	11.3 (± 1.7)	10.1 (± 3.3)
3rd Set to Fatigue	6.9 (± 2.7)	6.4 (± 2.1)
4th Set to Fatigue	5.7 (± 2.5)	5.8 (± 2.8)

Above shows the repetitions that were completed during each set for both supplement conditions. There was no overall difference in repetitions during each set between each condition.

Table 1. Dietary intake, body mass, and hydration. Data are means (\pm SD).

Macronutrient	Preliminary Visit	Placebo	Carbohydrate
Carbohydrate (g/kg)	3.3 (± 0.98)	2.8 (± 1.18)	2.7 (± 1.11)
Fat (g/kg)	1.3 (± 0.50)	1.1 (± 0.44)	1.08 (± 0.48)
Protein (g/kg)	2.1 (± 0.72)	1.5 (± 0.38)	1.3 (± 0.59)
Total kcal	2267 (± 463)	2404 (± 560)	2261 (± 514)
Body Mass	-	90.2 kg (± 17.5)	89.6 kg (± 18.7)
Body Fat Percentage	-	20.7 % (8.5)	20.6 % (± 8.27)
Total Body Water	-	49.1 kg (± 5.96)	49.3 kg (± 49.3)
Packed Cell Volume	-	47.8 % (± 2.44)	47.2 % (± 2.57)

There were no significant differences at each of the measured time points.

Table 3. Intramuscular glycogen (ug/mg) wet weight for both conditions. Data are means (\pm SD).

Condition	Pre-Exercise	Immediate Post	1hr Post
Placebo	3.22 (\pm 1.08)	2.07 (\pm 1.42)*	1.822 (\pm 0.73)**
Carbohydrate	2.41 (\pm 0.58)	1.60 (\pm 0.48)*	1.79 (\pm 0.61)**

* $p < 0.05$ between the pre-exercise and immediate post exercise time points** $p < 0.05$ between the pre-exercise and 1hr post time points.**Table 4. Serum glucose (mg/dl) at each time point in both conditions. Data are means (\pm SD).**

Condition	Pre-Supplement	Pre-Exercise	Immediate Post	1hr Post
Placebo	103.1 (\pm 7.3)	107.8 (\pm 6.6)	107.0 (\pm 1.31)	103.9 (\pm 3.8)
Carbohydrate	103.0 (\pm 3.9)	111.6 (\pm 14.1)*	98.4 (\pm 12.0)*	105.6 (\pm 7.7)

* $p < 0.05$ between the pre-exercise and immediate post exercise time points for the carbohydrate condition.**Table 5. Serum insulin (μ UI/ml) between each time point and condition. Data are means (\pm SD).**

Condition	Pre-Supplement	Pre-Exercise	Immediate Post	1hr Post
Placebo	1.67 (\pm 1.0)	1.77 (\pm .92)*	1.18 (\pm .81)**	1.71 (\pm .89)
Carbohydrate	1.45 (\pm .64)	9.54 (\pm 6.11)*	3.67 (\pm 1.49)**	6.84 (\pm 6.59)

* $p < 0.05$ between supplement conditions at the pre-exercise time point. ** $p < 0.05$ between supplement conditions at the immediate post time point.**Table 6. Serum epinephrine concentration (pg/ml) changes from baseline for each condition. Data are means (\pm SD).**

Condition	Post-Supplement	Immediate Post	1hr Post
Placebo	7.98 (\pm 10.3)	8.57 (\pm 12.39)*	7.76 (\pm 17.03)*
Carbohydrate	1.00 (\pm 9.56)	-6.04 (\pm 11.60)*	-7.46 (\pm 13.75)*

* $p < 0.05$ between supplement conditions at the immediate post and 1hr post exercise time points respectively

Intramuscular glycogen content for each time point and condition are indicated in Table 3. There was no main effect of supplement on glycogen content ($F = 2.847$, $p = 0.130$, $\eta^2 = 0.262$); however, there was a main effect for time ($F = 14.305$, $p < 0.001$, $\eta^2 = 0.64$). Pairwise comparisons showed a significant decrease between pre-exercise and immediately post-exercise ($p = 0.007$) and pre-exercise and 1hr post-exercise ($p = 0.004$), but no differences between immediately post-exercise and 1hr post-exercise ($p = 1.00$). There was no significant interaction between supplement and time ($F = 1.191$, $p = 0.330$, $\eta^2 = 0.130$).

Serum glucose concentrations for each time point and condition are indicated in Table 4 below. There was an interaction between supplement and time ($F = 5.324$, $p = 0.006$, $\eta^2 = 0.40$). Simple effects pairwise comparisons indicated that serum glucose was utilized more between pre-exercise and immediately post-exercise in the carbohydrate condition. No main supplementation effect for blood glucose was found ($F = 0.069$, $p = 0.799$, $\eta^2 = 0.01$). Pairwise comparisons also revealed there was not a main effect for time ($p > 0.05$).

Serum insulin concentration for each time point and condition are listed in Table 5. There was a significant interaction showing greater decreases in serum insulin between pre-exercise and immediate post exercise time points in the carbohydrate group ($F = 47.14$, $p = 0.003$, $\eta^2 = 0.479$). There was a main effect of carbohydrate supplementation increasing serum insulin content between conditions ($F = 7.72$, $p = 0.027$, $\eta^2 = 0.525$). Carbohydrate ingestion increased insulin pre-exercise ($p = 0.13$) and immediately post-exercise ($p = 0.006$). The simple effects pairwise comparisons indicated that there was not a main effect for time on insulin ($p > 0.05$).

Changes in serum epinephrine concentration relative to baseline for each time point and condition are listed

in the Table 6. There was a main effect of carbohydrate decreasing epinephrine concentration ($F = 7.924$, $p = 0.023$, $\eta^2 = 0.498$). There was not a main effect of time for epinephrine concentration ($F = 1.475$, $p = 0.258$, $\eta^2 = 0.156$). No interaction was found between supplement condition and time ($F = 1.94$, $p = 0.181$, $\eta^2 = 0.192$).

Discussion

It is accepted that carbohydrate ingestion increases aerobic work capacity and performance (Murray and Rosenbloom, 2018). Aerobic performance is limited by total glycogen content which can be depleted after an exhaustive aerobic bout (Murray and Rosenbloom, 2018). It has been proposed that when glycogen depletion is around 50% of initial levels, or reach concentrations lower than 75mmol/kg, muscle function may become impaired (Sherman and Wimer, 1991; Murray and Rosenbloom, 2018). Glycogen has been shown to be depleted by 20-40% following a single bout of resistance exercise (Koopman et al., 2006). Similar changes were seen in the current investigation where placebo and carbohydrate groups depleted approximately 34% and 28% of the initial glycogen content, respectively. Our data shows resistance exercise to fatigue may not be governed by the total availability of glycolytic substrate as seen in aerobic exercise. This may have been seen because habitual dietary carbohydrate intake was enough to sustain the workload or glycogen was not depleted enough for carbohydrate to have an ergogenic effect. The carbohydrate guidelines for athletes outlined by the Academy of Nutrition and Dietetics, Dietitians of Canada, and the American College of Sports Medicine are 3-5g/kg/d, 5-7g/kg/d, and 6-10g/kg/d for light, moderate, and high categories, respectively (Thomas et al., 2016). These intakes are thought to prevent the gradual depletion of glycogen over the course

of several bouts of training if adequate carbohydrate was not consumed, thereby maintaining athletic performance (Thomas et al., 2016). In the present study, analysis of participants' dietary intakes showed them to be consuming 2.9 g/kg/d of carbohydrate, close to the light consumption by the guidelines above. As a result, our participants' glycogen stores may have been full enough at baseline, negating any effect from additional carbohydrate during the brief exercise bout. Our results indicate that giving a carbohydrate supplement to an adequately carbohydrate-fed individual does not increase resistance exercise performance to fatigue in a similar manner that it may with endurance exercise to fatigue. Resistance exercise performance to fatigue on a brief single exercise may be predicated by other physiological and psychological factors that were not assessed in the present study. Moreover, carbohydrate supplementation may have a more substantial ergogenic role in delaying fatigue during extended durations of resistance exercise training (>50min) (Haff et al., 2003). Collectively this suggests carbohydrate supplementation may not be necessary for adequately fed individuals completed brief intense bouts of exercise to fatigue.

Non-statistically significant elevations in blood glucose during the pre-exercise time point indicated carbohydrate supplementation did not elevate serum glucose substantially within 30 minutes. This may have been caused by variations in glucose absorption by the gut, blood collection timing, subject variation, or the collective healthy participants' insulin responses to the glucose load (Pagana and Pagana, 2013; Guess et al., 2016). Our serum insulin concentrations increased similar to that of athletic and healthy populations during an oral glucose tolerance test (Guess et al., 2016). Additionally, serum insulin and glucose have a negative relationship with epinephrine concentrations, maintaining euglycemia during fed and fasted states (Penev et al., 2005). Our results show the planned timing of carbohydrate intake blunted epinephrine release during the onset of the resistance exercise bout. The timing of the carbohydrate supplement caused a decreased in epinephrine and an increase in insulin, creating a physiological state that appears to prepare an individual to utilize dietary glucose for exercise. This decrease in epinephrine concentration, however, did not lead to significant differences in glycogenolysis or differences in performance during exercise. Speculatively, a longer pre-exercise digestion period may cause increases in performance due to increased muscle glycogen concentration as seen in aerobic based exercise (Coyle et al., 1985; Hargreaves et al., 2003). If there is an ergogenic effect of pre-exercise carbohydrate consumption on short term resistance exercise performance, a longer digestion period may be required for discernable enhancements.

During recovery in the carbohydrate condition, insulin concentration rose while epinephrine concentration remained lower than baseline values. The elevated insulin concentrations indicate increased glucose influx into muscle during the recovery period fostering the potential for subsequent glycogen synthesis. Although not significant, the respective immediate post-exercise and 1hr post-exercise relative glycogen values for the placebo condition were 66% and 65% of the baseline values, while those of

the carbohydrate condition were 72% and 76%. Our results demonstrate possible continued glycogenolysis in the placebo condition, whereas the carbohydrate supplement ostensibly stimulated glycogen synthesis during recovery. Nevertheless, a longer post-exercise sampling time frame may have been necessary to observe significant increases in glycogen after exercise (Alghannam et al., 2005; Ivy, 2004).

Most of the research on pre-exercise carbohydrate consumption, glucose metabolism, and performance enhancement has been conducted on aerobic exercise (Coggan, 1991; Katz et al., 1991; Hearn et al., 2018). We are currently limited to viewing these hormonal and metabolic responses through the lens that has been provided by aerobic exercise studies. We used a single location within the vastus lateralis to assess glycogen concentration and as a result, this may not be reflective of glycogen metabolism throughout the entire muscle or in the other lower-extremity muscles involved in performing the leg press exercise. In addition, our results are limited by the sampling time points and may not reflect what possible benefits can be found with carbohydrate supplementation different exercise durations, dietary habits, or supplement timings. Despite these limitations, our observed alterations in insulin, glucose, glycogen, and epinephrine, in this experimental model, provide novel findings on timing and use of carbohydrate for performance and recovery following brief intense resistance exercise.

The current findings of this study provide data into a potential role that glucose supplementation has in resistance exercise performance and recovery. The hormonal changes that resulted from carbohydrate ingestion foster a greater potential for dietary glucose uptake during exercise and potential for a glycogen sparing effect to be seen over extended durations of exercise (Haff et al., 2000). However, this glycogen sparing effect and increases in performance were not seen in this brief single resistance exercise task. The dietary habits of our participants may have been sustaining their glycogen levels and the short duration of the overall exercise bout, alleviating the influence the additional carbohydrate may have had on performance (Lima-Silva et al., 2009; Ørtenblad et al., 2011; 2013; Thomas et al., 2016; Jacobs et al., 1982). While the glycogen values were not statistically different in the brief recovery sampling time frame, glycogenesis appears to be accelerated post-exercise with carbohydrate supplementation (Alghannam et al., 2005; Ivy, 2004). Even so, our results show large doses of carbohydrate before resistance exercise will not return glycogen concentrations to baseline values within one hour of the fatiguing resistance exercise. Timing of additional training sessions needs to be considered while attempting to allow complete metabolic recovery. If adequate recovery time cannot be allowed, alterations in volume and/or intensity for subsequent bouts of resistance exercise should be considered. Future carbohydrate supplementation research regarding resistance exercise performance and/or recovery, should investigate individuals on very light, light, moderate, and high carbohydrate dense diets for potential ergogenic effects during longer training bouts.

Conclusion

Carbohydrate supplementation before resistance exercise did not improve leg press performance to fatigue despite increased metabolic substrate availability. These results indicate that pre-exercise dietary carbohydrate will be utilized preferentially during exercise due to decreased epinephrine, decreased serum glucose, and increased insulin concentrations. However, the increases in glycolytic substrate availability will not increase exercise performance or glycogen content following 1hr of recovery.

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Key points

- Pre-exercise carbohydrate intake does not alter brief resistance exercise performance to fatigue when one is already consuming adequate dietary carbohydrate.
- The carbohydrate timing decreased in epinephrine while increasing insulin leading to higher dietary glucose utilization but not glycogen sparing during the brief exercise bout.
- Carbohydrate supplementation did not accelerate glycogen recovery within 1hr recovery.
- Longer time frames may be necessary to see glycogen concentration return to baseline (2-3hr).

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