

Interaction of muscle glycogen status and nutrient supplementation on skeletal muscle adaptation following resistance exercise

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Background: The insulin/insulin-like growth factor (IGF) signalling pathway has been implicated in regulating exercise- and nutrient-induced adaptation in skeletal muscle given its putative capacity to direct diverse cell processes such as glucose transport, glycogen resynthesis, and protein synthesis (Coffey & Hawley, 2007). A downstream target within this pathway proximal to translation initiation is the ribosomal protein S6 (rpS6) which, when phosphorylated and activated, can enhance the translation of mRNAs that encode for ribosomal proteins and elongation factors that may represent a rate limiting step for protein synthesis (Ruvinsky & Meyuhas, 2006). The energy status of skeletal muscle has been shown to alter signal transduction networks that would be expected to effect translation and protein turnover (Atherton *et al.*, 2005). Muscle glycogen is the primary substrate for high intensity exercise and changes in metabolism with low muscle glycogen concentration may have detrimental effects on anabolic responses in skeletal muscle (Coffey *et al.*, 2009). Interestingly, while low glycogen may augment the adaptation response to a bout of endurance exercise the effect of decreased glycogen concentration on anabolic signalling and protein synthesis in muscle following resistance exercise is unknown.

Purpose: The aim of this study was to determine the effect of muscle glycogen content and nutrient supplementation on anabolic signalling in skeletal muscle during the early recovery period following resistance training (RT).

Methods: The evening before an experimental trial, eight male subjects (22.9 ± 0.9 years; BMI = 24.0 ± 0.8 kg/m²) performed a depletion protocol where one-leg undertook cycling exercise to exhaustion to lower muscle glycogen levels (Low) while the other leg rested and therefore maintained normal muscle glycogen (Norm). The following day subjects completed a unilateral bout of resistance exercise during which each leg performed 8 sets of 5 repetitions of leg press at 80% of the previously determined one repetition maximum. Following the completion of exercise subjects consumed a 500ml bolus protein/carbohydrate beverage (20 g whey protein + 40 g maltodextrin). Muscle biopsies were obtained from the *vastus lateralis* in both legs at rest and 1h after RT.

Results: The depletion protocol was effective in generating divergent muscle glycogen content that was higher in the control leg (Norm) than in the Low leg at rest (383 ± 43 vs. 184 ± 14 mmol/kg dry wt; $p < 0.05$) and remained different 1 h post-exercise (309 ± 41 vs. 135 ± 11 mmol/kg dry wt; $p < 0.05$). Nutrient ingestion resulted in significant increases in blood glucose and insulin ($p < 0.05$) during the early recovery period compared to resting levels. Ribosomal protein S6^{Ser235/6} phosphorylation increased significantly 1 h post exercise compared to rest ($p < 0.05$), however there were no differences between the Norm and Low legs at 1 h.

Conclusion: We show that despite significant disparity in muscle glycogen levels at rest and 1 h after resistance exercise, there were no differences in rpS6^{Ser235/6} phosphorylation between the Norm and Low glycogen legs. These results indicate that low muscle glycogen levels fail to suppress phosphorylation of a key component in skeletal muscle translation initiation following high-intensity resistance exercise when protein/carbohydrate supplementation is provided during recovery.

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