Interaction of muscle glycogen availability and nutrition on cell signalling and myofibrillar protein synthesis following resistance exercise

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Introduction: Training with low muscle glycogen concentration can augment adaptation responses to endurance training but the effect of glycogen availability on anabolic signalling and protein synthesis in skeletal muscle following resistance exercise (REX) is unknown.

Aim: To determine the effect of muscle glycogen availability and post-exercise nutrition on anabolic signalling and myofibrillar protein synthesis (MPS) during the early recovery period following REX.

Methods: Sixteen men (22.7 ± 0.9 years; BMI = 23.9 ± 0.5 kg/m^2, values are mean ± SEM) were randomly assigned to nutrient or placebo groups (n=8/group). After 48 h diet and activity control, subjects reported to the laboratory the evening before an experimental trial and performed a glycogen-depletion protocol consisting of one-leg cycling to fatigue (LOW), while the other leg rested (NORM). After exercise, subjects consumed a low carbohydrate (CHO) meal. After an overnight fast, a primed, constant infusion of L-[ring-13C6] phenylalanine was commenced and then subjects completed 8 sets of 5 unilateral leg press repetitions at 80% one repetition maximum. Immediately after REX and 2 h later subjects consumed a 500 mL bolus of a protein/CHO beverage (20 g whey + 40 g maltodextrin) or placebo. Muscle biopsies from both legs (*vastus lateralis*) were taken at rest and at 1 and 4 h after REX.

Results: The depletion protocol generated divergent muscle glycogen concentrations that were higher in the NORM than LOW leg in both nutrient and placebo groups ($P < 0.05$). Muscle glycogen in LOW increased between 1 and 4 h post-exercise in the nutrient (~84 mmol·kgdw^{-1}, $P = 0.009$) but not placebo group. Phosphorylation of mTORSer2448 increased above rest at 1 and 4 h (~8–18 fold) in NORM and from rest to 1 h (~11 fold) in LOW in the nutrient group, but was only elevated from rest at 1 and 4 h post-exercise (~2-4 fold) in LOW with placebo. There were no differences between NORM and LOW legs at any time point. Nutrient ingestion stimulated a greater rise in MPS compared to placebo in both NORM (nutrient *vs* rest: 0.070 ± 0.008 *vs* 0.045 ± 0.007 %h^{-1}, $P < .05$) and LOW legs (0.068 ± 0.006 *vs* 0.049 ± 0.006 %h^{-1}, $P < .05$) during the 1-4 h recovery period, but were not different between NORM and LOW within nutrient or placebo groups.

Conclusion: Our results indicate that commencing high-intensity REX with reduced muscle glycogen availability does not suppress anabolic signalling and subsequent rates of MPS, at least in the initial 4 h post-exercise recovery period. However, it remains plausible that the affects of undertaking REX in a low glycogen state may be manifest through muscle protein breakdown.