Depressd resting myofibrillar protein synthesis induced by short-term energy deficit is up-regulated by resistance exercise and post-exercise protein ingestion

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Dietary energy deficit (ED) induced body weight loss is accompanied by fat free mass (FFM) loss. Skeletal muscle is the main component of FFM and its reduction can impair metabolic health and locomotion. Resistance exercise (REX) and increased daily protein intake have shown to diminish the ED induced loss of FFM, but specifically how skeletal muscle protein synthesis is regulated is currently unknown. Therefore, our objective was to investigate the acute skeletal muscle anabolic response to REX and post-exercise protein ingestion during ED.

Young resistance-trained men (n = 8) and women (n = 7) intracellular protein signalling and myofibrillar protein synthesis (MPS) response was studied at rest during energy balance [EB: 45 kcal·(kg FFM·d)$^{-1}$] and after 5d of ED [30 kcal·(kg FFM·d)$^{-1}$] as well as in ED after REX and with the ingestion of whey protein (15 and 30 g). Resting MPS was diminished ~27% in ED compared to EB ($p < 0.001$), and REX in ED up-regulated MPS to values not different from those of resting EB. MPS increased in ED compared to resting EB by ~16 and ~34% ($p < 0.02$) with post-exercise ingestion of 15 and 30 g of protein respectively. Phosphorylation of p70 S6Kthr389 was increased from EB in ED combined with REX and protein intake (~2-7 fold; $p < 0.05$) but not after REX alone.

The current results indicate that 5 days of dietary ED reduce resting MPS, a single bout of resistance exercise in ED restores MPS to EB values and post-exercise protein ingestion further increases MPS above resting EB in a dose-dependent manner. In conclusion, the combination of resistance exercise and protein intake can rescue the deleterious effect of ED on skeletal muscle protein synthesis.