

Poster presentation

## The effects of BCAA and leucine supplementation and lower-body resistance exercise on the ERK 1/2 MAPK pathway signal transduction

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### Background

A randomized, double-blind, placebo-controlled study was performed to evaluate the effects of oral BCAA and leucine supplementation on the ERK1/2 MAP Kinase signal transduction pathway in conjunction with an acute bout of lower-body resistance exercise (RE).

### Methods

30 males (22.5 yrs; 81.1 kg) ingested either leucine (60 mg/kg/bw), BCAA (120 mg/kg/bw), or placebo. Supplementation was ingested at 3 time points (1/3 of the total dosage at each time point): 30 minutes prior to RE, and immediately pre- and post-RE. The subjects performed 4 sets of leg press and leg extension at 80% 1 RM to failure. Rest periods between sets and exercises were approx. 150 seconds. Muscle biopsies (via the Bergstrom technique) were obtained from the vastus lateralis at four time points: baseline and 30 min, 2 hrs, and 6 hrs post RE and sampled for MEK1 and ERK1/2 activation (via phosphoELISA kits). Participants were fasted for the duration of the investigational period. Other variables (serum markers and other skeletal muscle proteins) were analyzed as part of a larger investigation, but only MEK1 and ERK1/2 and their activation/phosphorylation state are presented here. Skeletal muscle variables (MEK1 and ERK1/2) were transformed to delta values and analyzed via a 3 (group) × 4 (time points) repeated measures MANOVA. Univariate ANOVAs (Bonferroni adjusted) were conducted as follow-up tests to the MANOVA. Post-hoc tests of the interaction

effects demonstrated in the ANOVA were investigated via an independent samples T-test.

### Results

There was a main effect for time for MEK1 at the 2 hr time point ( $p = .005$ ). No main effect for group or a group × time interaction was observed for MEK1. Relative to ERK1/2, there were no main effects for time. A main effect for group revealed that the BCAA supplementation significantly elevated ERK1/2 activation as compared to the leucine ( $p = .001$ ) and placebo groups ( $p = .001$ ). A group × time interaction revealed that BCAA supplementation significantly elevated ERK1/2 activation at the 2 hr post and 6 hr post time points in comparison with the Leucine ( $p = .045$ ) and placebo groups ( $p < .001$ ).

### Conclusion

BCAA supplementation increased the phosphorylation status of ERK1/2 in conjunction with RE at 2 and 6 hours post-exercise. Leucine supplementation did not have any effect on ERK1/2 activation.