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## D-58 Thematic Poster - Skeletal Muscle Cell Signaling

JUNE 2, 2011 3:15 PM - 5:15 PM

ROOM: 304

**695 Chair: Stuart M. Phillips, FACSM. McMaster University, Hamilton, ON, Canada.**  
(No relationships reported)

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### 696 Board #1 3:15 PM - 5:15 PM

#### Impaired Inhibition of eIF4E-BP1 in Skeletal Muscle Impacts Stretch-Shortening Contraction Maladaptation with Age

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Specific molecular signaling mechanisms governing protein synthesis/ degradation in skeletal muscle, which results in known adaptation or maladaptation following mechanical loading with aging are largely unknown.

**PURPOSE:** The purpose of this study was to determine the morphological localization, distribution, and quantity of eIF4E and its regulator eIF4E-BP1 (regulators of the initiation of protein translation and the inhibition of initiation of protein translation, respectively) and their collective influence/s on young and old skeletal muscle of rats following chronic high-intensity mechanical loading via stretch-shortening contractions (SSCs).

**METHODS:** Left dorsiflexor muscles of young (12 weeks, N=6) and old (30 months, N=6) Fischer Brown Norway Hybrid rats, were loaded 3 times/week for 4.5-weeks using a protocol of 80 maximal SSCs per exposure *in vivo*. Transverse sections of the tibialis anterior muscle midbelly were cut and prepared for eIF4E and eIF4E-BP1 immunofluorescence and quantified via microscopy/imaging using standard stereology and densitometry.

**RESULTS:** The % volume density of fibers per muscle section and the % affected area of eIF4E-BP1 both decreased significantly (~16% and ~83%;  $p < 0.05$  and  $p < 0.01$ ), respectively in young rats following SSC loading. Interestingly, following SSC loading the % affected area of eIF4E labeling remained elevated by ~150% ( $p < 0.05$ ) in old compared with young rats following SSC loading. Furthermore, the % affected area of eIF4E-BP1 remained elevated by ~400% ( $p < 0.01$ ) following SSC loading in old compared with young rats following SSC loading, while the % volume density also remained elevated by ~32% ( $p < 0.01$ ) in old versus young rats.

**CONCLUSIONS:** Our data suggest that SSC loading adaptation/ maladaptation is significantly impacted by the distribution and quantity of eIF4E-BP1 and its regulation on the initiation of protein translation via sequestration of eIF4E. Collectively, these findings indicate that eIF4E-BP1 may exert a chronic inhibitory effect on the availability of eIF4E to contribute to protein translation/synthesis in skeletal muscle of aged populations following repetitive mechanical loading.

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### 697 Board #2 3:15 PM - 5:15 PM

#### Endurance Exercise Does Not Impair mTOR Signalling After Resistance Exercise

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(No relationships reported)

Resistance exercise is known to stimulate muscle hypertrophy and this effect is mainly mediated by the mammalian target of rapamycin (mTOR) pathway. In contrast, endurance exercise results in a divergent phenotypic response which to a large extent is mediated by adenosine monophosphate-activated protein kinase (AMPK). Research indicates that molecular interference may exist, possibly through an inhibitory effect on mTOR signalling by AMPK, when these two modes of exercise are combined.

**PURPOSE:** To investigate the impact of subsequent endurance exercise on resistance exercise induced mTOR signalling.

**METHODS:** In a randomized and cross-over fashion, ten male subjects performed either heavy resistance exercise (R) or heavy resistance exercise followed by endurance exercise (RE) on two separate occasions. The R protocol consisted of thirteen sets of leg press exercise with 3 minutes of recovery allowed between each set. In the RE session, resistance exercise was followed by 15 minutes recovery after which 30 min of cycling was initiated at an intensity equal to 70 % of the subjects' maximal oxygen consumption. Muscle biopsies were collected before, 1 and 3 hours after resistance exercise in both trials. Samples were analyzed for several signalling proteins in the mTOR pathway using western blot technique.

**RESULTS:** Phosphorylation of mTOR increased approx. twofold at 1 h post resistance exercise and remained elevated at the 3 h time point ( $p < 0.01$ ) with no difference between the two trials. Phosphorylation of p70S6k, a downstream target of mTOR, was increased about 6-and18-fold at 1 h and 3 h post resistance exercise ( $p < 0.01$ ). There was no difference in p70S6k phosphorylation at any time point between the two trials. Phosphorylation of the eukaryotic elongation factor eEF2 was decreased 3- to 4-fold at both time points post resistance exercise ( $p < 0.01$ ) with no difference between trials. Phosphorylation of AMPK was unchanged at the 1 h time point but decreased approximately 30 % from pre-exercise values in both trials at 3 h post resistance exercise ( $p < 0.01$ ).

**CONCLUSIONS:** The signalling response following heavy resistance exercise is not blunted by subsequent endurance exercise. Supported by the Swedish National Centre for Research in Sports.

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### 698 Board #3 3:15 PM - 5:15 PM

#### Delayed Satellite Cell Activation in Relation to Myostatin Expression Following Acute Resistance Exercise in Older and Younger Adults

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(No relationships reported)

Muscle growth is regulated by a unique population of muscle stem cells (satellite cells: SC). Depletion or dysfunction of SC leads to a loss in muscle mass and capacity for muscle growth. It remains unknown if this cell population is adversely affected by the aging process.

**PURPOSE:** To quantify the SC pool size and SC cell-cycle response to acute resistance exercise in healthy young (Y) and Old (O) humans in relation to muscle myostatin.

**METHODS:** Subjects (young (Y) 21.3±3.1y; N=9; Older adults (O) 69.6±3.9y; N=9) performed 4 sets of 10 rep of unilateral leg extensions and leg press at 75% of 1RM. Blood and muscle biopsies (vastus lateralis) were obtained pre-exercise (PRE) and 3, 24 and 48h post exercise. SC number and cell-cycle kinetics