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Identification of DNA-Damage-Inducible Transcript 4 in Young and Old Skeletal Muscle Following Stretch-Shortening Contractions: 608: June 2 10:00 AM - 10:15 AM

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The molecular mechanisms governing skeletal muscle adaptation/ 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 DNA-damage-inducible transcript 4 (DDIT4 - a marker of protein degradation and apoptosis that resides within the IGF/PI3K/Akt pathway) and its influence 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=5) and old (30 months, N = 5) 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 DDIT4 immunofluorescence and quantified using standard stereology and densitometry.

RESULTS: SSC loading increased the volume density and % affected area of DDIT4⁺ labeling, irrespective of age. The volume density of fibers per muscle section, which expressed DDIT4 increased by 20% after SSC loading in young rats compared with old rats ($p < 0.05$). However, the average percent area of each fiber that was labeled with DDIT4 was 31% greater in fibers of old rats compared with young rats ($p < 0.05$).

CONCLUSIONS: SSC loading increased the distribution of DDIT4⁺ labeling in young rats compared with old rats, which may reflect normal protein turnover following repeated bouts of mechanical stress. However, old rats increased the percent area of tissue labeled with DDIT4, and this may indicate that aged tissue may be susceptible to increased protein degradation and apoptosis. Collectively, these findings suggest that DDIT4 affects protein degradation as previously reported in skeletal muscle, and may represent a novel apoptotic factor residing within the Akt pathway in skeletal muscle.

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