Medicine & Science in Sports & Exercise
Issue: Volume 36(5) Supplement, May 2004, p S2

Publication Type: [Annual Meeting Abstracts: A-15 – Free Communication/Slide: Cellular/Molecular Aspects of Muscle Injury]
ISSN: 0195-9131
Accession: 00005768-200405001-00008

[Annual Meeting Abstracts: A-15 – Free Communication/Slide: Cellular/Molecular Aspects of Muscle Injury]

Effects of Range of Motion on Skeletal Muscle Morphology due to Stretch-Shortening Cycle-Induced Injury

Baker, Brent A.; Mercer, Robert; Krajnak, Kristine; Geronilla, Kenneth B.; Miller, Gerald R.; Cutlip, Robert G.

Author Information
NIOSH, Morgantown, WV.
Email: bwb3@cdc.gov
(Sponsor: Stephen A. Alway, Ph.d., FACSM)
0055

Quantification of skeletal muscle damage in response to injurious stretch-shortening cycles (SSC's) would be beneficial in elucidating the effect of different biomechanical exposures on the amount of muscle damage. **Purpose:** To investigate the effect of stretch-shortening cycle range of motion (ROM) on skeletal muscle damage in rats.

Methods: Testing was performed on the dorsiflexor muscles of Sprague-Dawley rats *in vivo*. Animals (n = 36) were randomly assigned to a long ROM injury group (L-Inj), short ROM injury group (S-Inj), or isometric group (Iso). The injury protocol consisted of 7sets of 10 SSC's at 500°/s. The S-Inj group received SSC's between 70°-120° ankle angle, whereas the L-Inj group received SSC's between 90°-140° ankle angle. The Iso group received equivalent muscle stimulation at 90° ankle angle. Dorsiflexor muscles were stimulated for 2.8 s each set and administered every minute. Rats were sacrificed at 6 and 48 hours post exposure. Following sacrifice, tissue was excised, weighed, sectioned, quick-frozen, and stored at -80°C. Transverse sections (12 µm) were cut, mounted on pre-coated microscope slides, and stained using a routine procedure with Harris Hematoxylin & Eosin. Tissue sections were evaluated on a Leica DMLB microscope. Stereology was used to quantify the degree of myofiber damage in muscle from each group; and, also was used to measure the volume fraction, surface densities, and average thickness of normal myofibers, degenerative myofibers, and the interstitial space. **Results:** No degenerative fibers were present in the contra-lateral control tissue, Iso group at 6h or 48h post-injury, nor the S-inj and L-inj at 6h post injury. In contrast, there was an increased volume of degenerative myofibers in the S-inj and L-inj at 48h post injury. The volume of cellular interstitium (cell infiltrates) and non-cellular interstitium (edema) increased ($p = 0.0082$ and $p = 0.0053$; respectively) from muscle exposed to the S-inj and L-inj at 48h post injury; and, thus the average thickness of the extracellular matrix increased with these groups. **Conclusions:** Increased degenerative myofibers, cellular infiltrates, and edema occur temporally in response to both S-inj and L-inj. However, a more dramatic increase occurs with the L-inj at 48h as assessed by the increase in volume of cellular and non-cellular interstitium.