

Scientists from the University of Alabama at Birmingham, AL discovered that despite the same stress placed on muscle, older men demonstrated an exaggerated immune and inflammation response. It was remarkable that, after the same mechanical stress of resistance exercise, 318 genes were differentially expressed among old vs. whereas only 87 genes among young, with the two age groups showing similar changes in only two genes. These age differences in response to resistance exercise occurred despite similar degrees of muscle damage as indexed by nearly identical percent increases in serum creatine kinase (a marker of muscle damage). ***Basically, this mean that the muscle fibers of older adult muscle were not more susceptible to muscle damage; yet they responded to the insult with a remarkably different gene expression profile that may help us begin to understand why regenerative function is impaired in old.***

This study supports the concept that the muscles of old may have experienced a degree of stress far exceeding that in young despite being exposed to the exact same stressor. Taken together, the numerous age differences noted strongly suggest the threshold of mechanical stress required to induce changes in the molecular signature of skeletal muscle is much lower in old. The muscles of old (vs. young) were much more sensitive to an equal and modest degree of damage—launching a robust transcriptome-level response that may begin to reveal key differences in the regenerative capacity of skeletal muscle with advancing age.

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DIFFERENTIAL GENOMIC RESPONSES IN OLD VS. YOUNG HUMANS DESPITE SIMILAR LEVELS OF MODEST MUSCLE DAMAGE AFTER RESISTANCE LOADING.

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Across numerous model systems, aging skeletal muscle demonstrates an impaired regenerative response when exposed to the same stimulus as young. To better understand the impact of aging in a human model, we compared changes to the skeletal muscle transcriptome induced by unaccustomed high-intensity resistance loading (RL) sufficient to cause moderate muscle damage in young (37 y) vs. older (73 y) adults. Serum CK was elevated 46% 24 h after RL in all subjects with no age differences, indicating similar degrees of myofiber membrane

Older Men Can't Recuperate from Exercise as well as Younger Men

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wounding by age. Despite this similarity, from genomic microarrays, 318 unique transcripts were differentially expressed after RL in old vs. only 87 in young. Follow-up pathways analysis and functional annotation revealed, among old, up-regulation of transcripts related to stress and cellular compromise, inflammation and immune responses, necrosis, and protein degradation; and changes in expression (up- and down-regulation) of transcripts related to skeletal and muscular development, cell growth and proliferation, protein synthesis, fibrosis and connective tissue function, myoblast-myotube fusion and cell-cell adhesion, and structural integrity. Overall the transcript-level changes indicative of undue inflammatory and stress responses in these older adults were not mirrored in the young. Follow-up immunoblotting revealed higher protein expression among old for NF-kappaB, HSP70, and IL-6 signaling (total and phosphorylated STAT3 at Tyr705). Taken together, these novel findings suggest that young and old are equally susceptible to RL-mediated damage; yet the muscles of old are much more sensitive to this modest degree of damage-launching a robust transcriptome-level response that may begin to reveal key differences in the regenerative capacity of skeletal muscle with advancing age. Key words: microarray, aging, muscle damage, resistance exercise.