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Elevated Methylation Status in Old Rat Skeletal Muscle Supports Gene Expression Disparities in Response to Injury

Marshall Naimo Jr.[1,2], **James Ensey**[1] **and Brent Baker**[1]

[+] Author Affiliations

Abstract

Prior research in our lab has established an *in vivo* rodent dynamometer model to explore the effects of skeletal muscle injury following stretch-shortening contractions (SSCs). Previously, we have shown that the genomic response is altered in old versus young rats; however, the underlying molecular basis for this response is unclear. Thus, our purpose was to ascertain if there were pathway-specific changes in gene expression for inflammation and apoptosis in old versus young skeletal muscle following SSC-induced injury; and, if so, whether changes in methylation could help explain these differences. Dorsiflexor muscles of young (3 mo.) and old (30 mo.) male Fischer 344xBN rats were injured using 150 continuous SSCs at 500°/s. Seventy-two hours following loading, the left tibialis anterior muscle was harvested. Gene expression and methylation status were quantified via RT^2 Profiler and Methylation Arrays (Qiagen®, Valencia, CA). Following injury, expression of proinflammatory specific genes in old compared to young muscle was nearly 6.5 fold lower (1.62 vs. 10.16). Interestingly, methylation levels were higher in old versus young (4.3 vs 2.4%). Similarly, proapoptotic specific genes were 10 fold lower (0.05 vs. 0.5) in old compared to young muscle, with aging also resulting in higher levels of methylation (1.9 vs. 0.6%). These data reveal that old skeletal muscle has an attenuated inflammatory and apoptotic gene expression response to injurious loading, which may be attributed to a higher methylation status of pathway-specific genes. This process may help explain why there is a disparate response to injury with increasing age.