# Immune Response and Mitochondrial Metabolism Are Commonly Deregulated in DMD and Aging Skeletal Muscle



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Résumé en anglais

Duchenne Muscular Dystrophy (DMD) is a complex process involving multiple pathways downstream of the primary genetic insult leading to fatal muscle degeneration. Aging muscle is a multifactorial neuromuscular process characterized by impaired muscle regeneration leading to progressive atrophy. We hypothesized that these chronic atrophying situations may share specific myogenic adaptative responses at transcriptional level according to tissue remodeling. Muscle biopsies from four young DMD and four AGED subjects were referred to a group of seven muscle biopsies from young subjects without any neuromuscular disorder and explored through a dedicated expression microarray. We identified 528 differentially expressed genes (out of 2,745 analyzed), of which 328 could be validated by an exhaustive meta-analysis of public microarray datasets referring to DMD and Aging in skeletal muscle. Among the 328 validated co-expressed genes, 50% had the same expression profile in both groups and corresponded to immune/fibrosis responses and mitochondrial metabolism. Generalizing these observed meta-signatures with large compendia of public datasets reinforced our results as they could be also identified in other pathological processes and in diverse physiological conditions. Focusing on the common gene signatures in these two atrophying conditions, we observed enrichment in motifs for candidate transcription factors that may coordinate either the immune/fibrosis responses (ETS1, IRF1, NF1) or the mitochondrial metabolism (ESRRA). Deregulation in their expression could be responsible, at least in part, for the same transcriptome changes initiating the chronic muscle atrophy. This study suggests that distinct pathophysiological processes may share common gene responses and pathways related to specific transcription factors.

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