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Understanding the molecular mechanisms of muscle atrophy: A single-nucleus transcriptome analysis of mice with dexamethasone-induced skeletal muscle atrophy

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Skeletal muscle atrophy is the loss of muscle mass and function caused by a variety of factors, including aging, disuse, malnutrition, and disease. Dexamethasone (DEX)-induced muscle atrophy is a valuable model for understanding muscle atrophy because it is a rapid and reliable way to induce muscle wasting in animals. In this study, we generated single-nucleus RNA-seq (snRNA-seq) data of skeletal muscle from DEX-induced mice to decipher cell type-specific transcriptional changes associated with this disease. Our data analysis revealed that DEX treatment led to a decreased number of muscle cells and an increased amount of fibroblasts. Fibroblasts are known to play a crucial role in muscle atrophy through fibrosis which caused by chronic inflammation and oxidative stress. We identified altered gene expression related to protein degradation, inflammation, and extracellular matrix remodeling which are associated to muscle loss and fibrosis. Our study provides new insights into the molecular mechanisms of muscle atrophy and could inform the development of new strategies for preventing and treating this condition.