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Identification of therapeutic targets for muscle atrophy via analyzing transcriptomic profiles with systems biology

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Skeletal muscle atrophy is defined as an unwanted loss of muscle mass. Muscle atrophy is one of the major side effects of high dose or long-term usage of glucocorticoids (e.g., dexamethasone); therefore, such a drug is often used to induce a model system for muscle atrophy research. This study was designed to explore the key factors and molecular mechanisms of muscle atrophy based on the analysis of transcriptomic profiles using systems biology approaches. Here, we generated and analyzed the RNA-seq data of gastrocnemius (GA) muscle and tibialis anterior (TA) muscle specimens from a group of C57BL/6 male mice treated with dexamethasone (DEX). As a result, we successfully identified 637 differentially expressed genes (DEGs) between GA muscles with atrophy (n=4) and healthy controls (n=5) and 619 DEGs between TA muscles with atrophy (n=6) and healthy controls (n=6). By running pathway enrichment analysis, we found interesting gene sets including circadian rhythm, protein digestion and absorption, AMPK signaling, and antigen processing and presentation pathways. Furthermore, our active subnetwork analysis also showed that the key molecules belonging to the pathways tightly interact with each other at the level of protein-protein interaction, assuring that these pathway genes provide a critical clue in understanding on the disease mechanism and subsequent therapeutic target identification. In conclusion, we believe that our study results will be an important step towards finding a treatment solution for muscle atrophy.