[BIOINFO 2024]

Dexamethasone-Induced Muscle Atrophy: A Comparative Single-cell Transcriptomic Analysis

Bum Suk Kim1, Ahyoung Choi1, Yoomi Baek1, No Soo Kim2, Aeyung Kim3, Haeseung Lee4 and

Hyunjin Shin1*

[1]*MOGAM Institute for Biomedical Research, Seoul 06730, Republic of Korea*
[2]*Korean Medicine (KM) Convergence Research Division, Korea Institute of Oriental Medicine, Daejeon 34054, Republic of Korea*
[3]*Korean Medicine (KM) Application Center, Korea Institute of Oriental Medicine, Daegu 41062, Republic of Korea*
[4]*Department of Pharmacy, Pusan National University, Busan 46241, Republic of Korea*
**Corresponding author hyunjin.shin@mogam.re.kr*

Skeletal muscle atrophy is a condition characterized by the loss of muscle mass and function and significantly impacts both quality of life and overall health. It is associated with various factors including aging, disuse, malnutrition, disease, and drug administration. Dexamethasone (DEX)-induced muscle atrophy provides a valuable model for studying the mechanisms underlying muscle wasting due to its rapid and reliable induction. To elucidate the molecular mechanism involved in muscle atrophy, we generated and analyzed single-nucleus RNA-seq (snRNA-seq) data of skeletal muscle from the tibialis anterior (TA) muscle in DEX-treated mice. Our analysis revealed important pathways, including the circadian clock, protein synthesis, and glycolysis. Additionally, we observed a shift in muscle fiber type from type 2A/X to type 2B, a transformation known to contribute to a decline in muscle function. To understand the similarities and differences between DEX-induced muscle atrophy and other muscle wasting models, we compared our findings with existing transcriptomic data from aging, denervation, and Duchenne muscular dystrophy (DMD) models. In conclusion, 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.