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Special Issue Website Information Requirement

Journal: *Cells*
Section: Cellular Pathology
Special Issue: Skeletal Muscle Atrophy: Mechanisms in a Cellular Level
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Special Issue Entry Information

Skeletal muscles constitute the largest of body organs and make up about half of mammals' body weight. Several conditions, spanning from neuromuscular disorders, aging, cancer, and toxins, cause a loss of muscle mass and function. This acquired condition, referred to as muscle atrophy, is an emerging health concern and burden for human health. Notwithstanding the cellular and molecular clues of muscle atrophy are still a relatively young fields of research, great efforts have been made over the last two decades to decipher the pathophysiological bases underlying muscle loss. A wide range of cellular (e.g. myocites and satellite cells), and subcellular compartments (e.g. neuromuscular junction), organelles (mitochondria, ER, SR), degradation pathways (UPS and autophagy), molecular signaling networks (e.g. AKT, mTOR, etc), and genes (e.g. atrogenes) have been identified as critical players in the regulation of muscle mass and atrophy, thus contributing fundamental insights onto the plasticity and vulnerability of the muscle tissue in physiological and pathological conditions.

This Special Issue aims to provide a general overview on the cellular and molecular mechanisms responsible for muscle atrophy and to set novel questions stimulating the identification of novel strategies that tackle conditions or disorders associated with muscle loss.

Keywords:

- Muscle atrophy
- Muscle proteostasis
- Muscle disuse
- Atrogenes
- Sarcopenia
- Neuromuscular disorder
- Myopathies
- Muscle degeneration

- Neuromuscular paralysis
- Cancer cachexia