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Mechanisms of muscle atrophy: from UPS implication in rodent models to human biomarkers

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The ubiquitin proteasome system (UPS) is a major player of skeletal muscle wasting, a common characteristic of many diseases (cancer, sepsis, heart failure, kidney diseases, etc.) that negatively impacts treatment and life prognosis. In the early 2000, the notion of “atrogenes” (atrophy-related genes) was proposed for genes that were systematically up regulated during catabolic situations in rodents, which included 2 founding members, the E3 ubiquitin ligases MAFbx/Atrogin-1 and MuRF1/TRIM63 [1]. Interestingly, MuRF1 is so far the only E3 ligase known for targeting several sarcomeric proteins (α -actin, MYHC, Troponin I, telethonin) and we recently identified the E2 ubiquitin conjugating enzymes that bring the catalytic activity for MuRF1-dependent protein degradation [2, 3].

Besides rodent models, we and others demonstrated that MAFbx and MuRF1 are also gold standard atrogenes in human pathologies and we recently found that numerous proteins may be part of a muscle atrophy program in chronic kidney disease and lung cancer patients [4]. In addition, we have identified blood markers that reflect skeletal muscle loss in patients suffering from several pathologies.

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