



HAL
open science

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

Daniel Taillandier, Dulce Peris-Moreno, Cécile Polge

► To cite this version:

Daniel Taillandier, Dulce Peris-Moreno, Cécile Polge. Mechanisms of muscle atrophy: from UPS implication in rodent models to human biomarkers. 2022 Padua Days on Muscle & Mobility Medicine, University of Padova, Mar 2022, Padova, Italy. hal-03858980

HAL Id: hal-03858980

<https://hal.inrae.fr/hal-03858980v1>

Submitted on 18 Nov 2022

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

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

Daniel Taillandier, Dulce Peris Moreno, Cécile Polge

Université Clermont Auvergne, INRAE, UNH, Unité de Nutrition Humaine, F-63000 Clermont-Ferrand, France

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.

References

[1] Peris-Moreno, D.; Taillandier, D.; Polge, C. MuRF1/TRIM63, Master Regulator of Muscle Mass. *Int. J. Mol. Sci.* 2020, 21, 6663. [doi:10.3390/ijms21186663](https://doi.org/10.3390/ijms21186663)

[2] Polge, C.; Cabantous, S.; Deval, C.; Claustre, A.; Hauvette, A.; Bouchenot, C.; Aniort, J.; Béchet, D.; Combaret, L.; Attaix, D.; Taillandier, D. A Muscle-Specific MuRF1-E2 Network Requires Stabilization of MuRF1-E2 Complexes by Telethonin, a Newly Identified Substrate. *J. Cachexia Sarcopenia Muscle* 2018, 9, 129–145. [DOI: 10.1002/jcsm.12249](https://doi.org/10.1002/jcsm.12249)

[3] Peris-Moreno, D.; Malige, M.; Claustre, A.; Armani, A.; Coudy-Gandilhon, C.; Deval, C.; Béchet, D.; Fafournoux, P.; Sandri, M.; Combaret, L.; Taillandier*, D.; Polge, C*. UBE2L3, a Partner of MuRF1/TRIM63, Is Involved in the Degradation of Myofibrillar Actin and Myosin. *Cells* 2021, 10, 1974. <https://doi.org/10.3390/cells10081974>

[4] Aniort, J.; Stella A.; Philipponnet, C.; Poyet, A.; Polge, C.; Claustre, A.; Combaret, L.; Béchet, D.; Attaix, D.; Boisgard, S.; Filaire M.; Rosset E.; Bulet-Schiltz O.; Heng, A-E; Taillandier, D. Muscle wasting in patients with end-stage renal disease or early-stage lung cancer: common mechanisms at work. *J. Cachexia Sarcopenia Muscle* 2019, 9, 129–145. [DOI: 10.1002/jcsm.12376](https://doi.org/10.1002/jcsm.12376)