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Evidence for autophagy attenuation during post-mortem maturation of hypertrophied muscle in myostatin deficient mice

Rim Nassar1,2, Barbara Vernus1, Gilles Fourtet1, Bénédicte Goustadt1, François Casas1, Lionel Tintignac1, Isabelle Cassar-Malek1, Brigitte Picard1, Iban Seliez1, Arnaud Chatonnet1, Aline Hamade1, Fadia Najjar1, Anne Bonnieu1 and Béatrice Chabi1

1- INRA, UMR 866 Dynamique Musculaire et Métabolisme, Montpellier, France ; 2 - Laboratoire d’innovation thérapeutique, Université Libanaise, Beyrouth, Liban ; 3 - Département de Biomédecine, Université de Bâle, Bâle, Suisse ; 4 - INRA, UMR1213 Herbivores, Saint-Genès-Champangene, France; 5 - INRA, UMR1419 Nutrition, Métabolisme, Aquaculture, Saint-Pée sur Nivelle, France.

Introduction

The conversion of skeletal muscle into meat, i.e. maturation, is a complex process where muscle undergoes different biochemical and physiological changes (Quaî et al., 2006). In agronomic field, the study of these events is of particular interest, in order to improve the quality of the final product put on the market. If the characterization of the proteolytic mechanisms involved in skeletal muscle maturation is still ongoing, the participation of autophagy in this process is still under debate. The aim of the study was therefore to assess the involvement of autophagy during skeletal muscle maturation and to study the interaction between autophagy and myostatin, a negative regulator of skeletal muscle mass (Mc Pheron et al., 1997), within a 72h postmortem (PM) time frame in mice.

Animal model

Healthy 6 month-old Mstn+/+ (WT) and Mstn−/− mice (N=72 for each genotype) were used in this study.

Muscle sampling

Mice were sacrificed using cervical dislocation. After decapitation and bleeding, mice were stored at room temperature (22±2°C) during 1h and then at 4°C until the dissection time.

Longissimus dorsi and gastrocnemius muscles were harvested and used for pH and western-blotting analyses respectively.

Methods and model validation

1) pH evolution in the Longissimus muscle

2) Myofibrillar protein degradation

Results

1) Phosphorylation state of proteins involved in autophagic signaling pathway (AMPK, FOXO andULK)

2) Autophagy flux measurement

Colchicine administration resulted in LC3Bl accumulation (NaCl 0.9% vs. colchicine)

The decrease in ULK1 (Ser757) phosphorylation in Mstn KO mice suggests an early activation (1h PM) of autophagy

Basal and postmortem autophagic fluxes are greater in WT versus Mstn KO mice.

Colchicine administration resulted in LC3Bl accumulation (NaCl 0.9% vs. colchicine)

In conclusion, our data showed that autophagy is preserved during skeletal muscle PM maturation, but to a lower extent in Mstn-deficient mice, suggesting a relationship between myostatin and autophagy. This work will be completed with microscopic analyses of skeletal muscle tissue sections to visualize in situ autophagic events.