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Short Communication

Muscle Post-Mortem Proteolysis and Meat Tenderization: Lessons from Myostatin-Deficient Skeletal Muscle

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ABSTRACT

Identifying the mechanisms that regulate post-mortem proteolysis in skeletal muscle is of economic and environmental importance because it is a crucial process during meat conversion and tenderization. Among the catabolic systems discussed, the calpain proteolytic system has received much attention. In addition, the muscle of cattle with double musculature, bearing natural deletions or mutations in the Myostatin gene, is more tender providing an interesting agro-ecologic way to answer both to reduction of livestock and enhanced quality. In this context, the study of the contribution of the calpain proteolytic system during post-mortem proteolysis, using the model of myostatin knock-out mice, brings new answers and poses current reflections, as discussed in this article.

Keywords: Agriculture; Muscle post-mortem homeostasis; Physiology; Animal production

ABOUT THE STUDY

In the muscle of meat-producing animals, a set of enzymatic and physico-chemical processes leads to the Post-Mortem (PM) transformation of muscle into meat. These processes are of great importance for the sensory qualities of meat, especially tenderness [1]. Among others muscle properties (lipid and collagen content, contractile and metabolic type, and structural organization); tenderness also depends on the content and activity of endogenous proteases in post-mortem muscle [2]. Work carried out over the last twenty years has established that the calpain proteolytic system is responsible for post-mortem proteolysis, and the subsequent meat tenderness. Indeed, *in vitro* degradation pattern of myofibrillar proteins in incubations with calpains are the same as that seen in post-mortem muscle [3,4]. Moreover, *in vivo*, the perturbation of calpain function (by overexpressing calpastatin, the endogenous inhibitor of the calpain enzymes or by inactivating μ-calpain) led to a reduction in post-mortem proteolysis of muscle proteins [5,6]. While the mechanisms responsible for muscle post-mortem proteolysis are not fully understood, recent data suggest that others catabolic systems, such as the Ubiquitin Proteasome System (UPS) and macro autophagy could also contribute to this proteolytic process [1,7,8]. The incubation of bovine muscle fibres with UPS enzyme resulted in proteolysis of myofibril proteins including nebulin, myosin, actin and tropomyosin and the activity of UPS has been shown to be maintained post-mortem [9]. Protein expression and activities of several autophagic markers BECN1, LC3, cathepsin D and cathepsin B are increased in two Spanish beef breeds during muscle to meat conversion [10]. Collectively the above studies on muscle post-mortem proteolysis make the point on the importance of the calpain system in this process and the possible contribution of 20S proteasome and autophagy. However, the regulation of this proteolytic potential of post-mortem muscle remains to be investigated.

It is widely known that Myostatin (MSTN) a powerful negative regulator of muscle mass plays a key role in the muscle hypertrophy/atrophy balance and currently appears as a major modulator of catabolic pathways (Ubiquitin-Proteasome, autophagy) [11]. MSTN negatively regulates the activity of the AKT pathway (Protein kinase B), which promotes protein synthesis, and increases the activity of the ubiquitin-proteasome system to induce atrophy. Evidences have also suggested that MSTN could trigger protein degradation *via* the stimulation of the autophagy-lysosomal system. Natural or experimental mutations in the gene encoding this factor are the cause of a hypermuscular phenotype, especially in double-muscled cattle

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[12,13]. In cattle, these mutations in the *Mstn* gene often result in improved tenderness [14]. However, the existence of a link between MSTN and muscle proteolysis during early post-mortem ageing was not, so far, determined by scientific studies. The existence of such a link has just been tested in mice lacking myostatin.

The study of Nassar et al published in Meat Science in 2021 performed a detailed proteolysis characterization of the myostatin-deficient muscle during early post-mortem storage and aimed to identify key proteolytic systems involved in post-mortem conditions [15]. This study showed that myostatin deficiency in skeletal muscle led to increased protein breakdown in post-mortem muscles and a preserved antioxidant status. It is worth mentioning that within the proteolytic systems analyzed in this study, autophagy and proteasome are active in post-mortem muscle, but not responsible for the difference of post-mortem proteolysis between the two genotypes. In line with previous evidence on the importance of the calpain system in this process, the myostatin deficient muscle displays an improvement of calpain activity. This study therefore indicates that calpain activity can explain the effect of MSTN absence on muscle post-mortem proteolysis. Previous data have demonstrated that proteolysis can be correlated with the variation in tenderness in some of the muscles but not of all the muscles [16], and that tenderness is improved in all muscles with MSTN inactivation [17]. The study of Nassar et al reinforces the idea of a positive relationship between proteolysis and tenderness in this double-muscled model of MSTN inactivation.

The results of this study raise many questions. What are the mechanisms regulating calpain activity in the absence of myostatin? One can also ask whether the muscle redox state controls the post-mortem degradation of myofibrillar proteins. A characteristic of the process of converting muscle into meat is the gradual increase in ROS production and decrease in antioxidant capacity, leading to oxidative stress and cell damage [18,19]. Oxidation plays a direct role the calpain activity and oxidative modifications of myofibrillar proteins might change their susceptibility to proteolysis [20,21]. The observations described here suggest that the redox state when reduced could directly participate in the activation of muscle post-mortem proteolysis.

Finally, we can ask ourselves the extent to which the data described here in *Mstn* KO mice can be translated in double-muscled cattle breeds harbouring *Mstn* mutations. In livestock animals calpain activity in fast glycolytic muscles, is associated with increased protein proteolysis in post-mortem muscle [22, 23]. However, some results reported reduced calpain/calpastatin levels in double-muscled cattle breeds [14]. Thereby, the obtained results need further considerations and appropriate comparative studies to be applied to bovine cases. However, the maturation of muscle into meat is a very complex phenomenon, which also depends on the muscle typology and intramuscular fat content. The influence of myostatin deficiency on all of these aspects remains to be specified.

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