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Elucidating the Role of Apoptosis in Meat Tenderization Using the Callipyge Lamb Model

T. Cramer[1]*, M. Penick[1], J. Waddell[2], and Y. H. B. Kim[1]

[1]Department of Animal Science, Purdue University, West Lafayette, IN, USA;
[2]Department of Animal Science, Tarleton State University, Stephenville, TX, USA

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Objectives

Apoptosis is the process of mediated cell death occurring immediately after exsanguination of animals. It is initiated by release of cytochrome C to the cytoplasm, ultimately activating caspase 3. Since caspase 3 is an enzyme that cleaves calpastatin, a known inhibitor of calpain (primary proteolytic enzyme), a potential involvement of apoptosis in meat tenderness development has been proposed. Previously, we presented that a higher activity of heat shock protein (HSP) 27, which has an anti-apoptotic function, and greater toughness were found in callipyge lamb loins compared with normal lamb loins. Therefore, in this study, we aimed to further investigate the role of apoptosis in the postmortem meat tenderization process using the callipyge lamb model.

Materials and Methods

As previously reported, a total of 16 lambs four different genotypes [callipyge (CN) and normal (CC, NC, NN)] was slaughtered. Loins (*M. longissmus*) were collected at 15 min, 3, 6, and 9 d postmortem for protein extraction and tenderness measurement. Warner-Bratzler shear force (WBSF) and cook loss were measured. Western blots were performed to determine the extent of degradation of the myofibrillar proteins desmin and troponin T over aging, as well as calpain 1 autolysis, calpastatin, HSP27, caspase 3 and cytochrome C. The experimental design was a randomized complete block design, and data were analyzed using mixed procedure of SAS (SAS Inst. Inc., Cary, NC) to compare the traits across genotypes and aging times. Means were separated (F-test, $P < 0.05$) by least significant differences and a Pearson correlation was conducted for all traits.

Results

A lower extent of degraded HSP27 was found in CN compared to other genotypes across aging ($P < 0.001$), indicating HSP27 was more active in CN muscle compared to normal counterparts. HSP27 degradation was negatively correlated with intact desmin ($r = –0.44$, $P = 0.0003$), troponin T ($r = –0.50$, $P < 0.0001$), and calpain 1 autolysis ($r = –0.69$, $P < 0.0001$). This observation suggests that HSP27 would likely involve the myofibrillar protein degradation process and possibly proteolytic activity of calpain 1. This was also supported by the negative correlation of HSP27 degradation with WBSF ($r = –0.33$, $P = 0.02$) and cook loss ($r = –0.36$, $P = 0.01$). CN showed the most procaspase 3 at the 32 kDa band across aging ($P < 0.05$), indicating that less caspase 3 was activated in CN during aging. Further, procaspase 3 was positively correlated with intact troponin T ($r = 0.26$, $P = 0.04$) and WBSF ($r = 0.47$, $P = 0.0008$). Cytochrome C was present in lesser amounts in CN compared to the normal genotype counterpart throughout aging, suggesting that cytochrome C was less released into cytosol in CN.

Conclusion

The results from the current study suggest the involvement of apoptosis in myofibrillar protein degradation and subsequent meat tenderness development. Positive correlations of anti-apoptotic activities (shown by elevated HSP27 and procaspase 3, and subsequently

less cytochrome C) with increased toughness in callipyge lamb loins indicate that apoptosis would be likely involved in postmortem proteolytic systems and subsequent meat tenderization. This study provides novel insight into the well-established toughness of callipyge lamb loins by brining apoptosis perspective. Further studies to elucidate the extent of this mechanism as compared to myofibrillar chaperoning should be warranted.