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Effects of Postmortem Aging on Small Heat Shock Protein Degradation of 3 Bovine Muscles

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Objectives

Meat tenderness is a result of enzymatic degradation of muscle structural proteins. Small heat shock proteins (HSPs) are a family of molecular chaperones that could be involved in postmortem meat tenderization through its protective role in programmed cell death, namely anti-apoptotic activity. The rate and extent of proteolytic enzyme activity (particularly calpain1) change and its subsequent impacts on myofibrillar protein degradation during aging have been well established. However, the impact of postmortem aging on small HSP degradation of different beef muscles has not been fully understood. Therefore, the objective of this study was to determine the effect of postmortem aging on small HSP dynamics and its relevance to meat quality attributes of different beef muscles.

Materials and Methods

At 1 d postmortem, 3 muscles [*longissimus lumborum* (LL), *semimembranosus* (SM), and *psoas major* (PM)] from 8 beef carcasses were divided into 5 sections, vacuum-packaged, and assigned for 1, 2, 9, 16, and 23 d of aging. Warner-Bratzler shear force (WBSF) and water-holding capacity (WHC) including drip loss, cook loss, and purge loss were determined at each aging point. Western blots were performed to determine the extent of myofibrillar protein degradation (desmin and troponin-T), calpain1 autolysis, and small HSPs including HSP27, HSP20, and αβ-crystallin intact/degradation. The experimental design was a split-plot design with muscle effect as whole plot and aging time as sub-plot. The data were analyzed by using PROC MIXED procedure of SAS (SAS Inst. Inc., Cary, NC). Spearman ranking correlations between protein dynamics and meat quality attributes were analyzed by using PROC CORR of SAS.

Results

Postmortem aging improved WHC of beef muscles indicated by decreased cook loss and drip loss of beef samples ($P < 0.05$). Shear force values decreased with aging as expected ($P < 0.05$). However, the different aging response for tenderness development was observed in a muscle specific manner, where PM exhibited the most rapid WBSF decrease, followed by LL and SM ($P < 0.05$). A significant decrease in intact desmin and troponin T along with increased degradation products of these proteins were found with prolonged aging ($P < 0.05$). Desmin and troponin T degradation were positively correlated with WHC, tenderness and degradation of HSP20, HSP27 and αβ-crystalin ($P < 0.05$). HSP20 and αβ-crystalin exhibited similar dynamics, where significant decreases of intact proteins were observed during aging ($P < 0.05$). An increase in HSP27 degradation product of all beef muscles was found with aging in general ($P < 0.05$), but LL showed the most degradation products, while PM showed the least HSP27 degradation ($P < 0.05$).

Conclusion

The result of the current study suggests that small HSP degradation of beef muscles increase with aging, but the extent of degradation would be different in a muscle-specific manner. A different degradation pattern of HSP in LL compared to PM could be coincided with greater myofibrillar degradation of LL compared to that of PM during aging. The increase in small HSP degradation could indicate loss of protective anti-apoptotic activity from delaying myofibrillar protein degradation. Further investigation of upper stream (mitochondrial) apoptotic factors and caspase system dynamics over aging and their relationship with HSP and meat tenderness development would be warranted.

