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Contribution of Protein Degradation and Sarcomere Length to Aged Pork Loin Warner-Bratzler Shear Force

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Objectives

Postmortem aging of fresh pork loins improves tenderness through protein degradation. Sarcomere length (SL) of postmortem muscle can vary between animals, and this could impact access and efficacy of proteinases to degrade proteins within, but not outside of the myofibril. The relationship between SL and protein degradation is not well documented in pork. Therefore, the objective of this experiment was to compare protein degradation of troponin-T with desmin and SL in aged pork loins over 21d.

Materials and Methods

Paired sides of fresh pork loins (n = 20) were collected 1 d postmortem. Criteria for inclusion in the study were a pH between 5.85 and 6.10 and a visual color score (NPPC) between 3 and 4. Eight loin chops (2.54 cm) containing only the longissimus muscle were fabricated. Two chops from each pair of loins were aged for 1, 8, 14 or 21 d and immediately evaluated. After aging, chops were cooked to 68°C and Warner-Bratzler shear force (WBSF) was measured. Whole muscle proteins were solubilized from samples at each aging period (10mM sodium phosphate, pH 7.0 and 2% wt/vol sodium dodecyl sulfate). Abundance of degraded troponin-T (30 kDa) and intact desmin (55 kDa) in the whole muscle sample was determined by immunoblotting. Abundance of troponin-T degradation product and intact desmin was normalized by a reference sample on each gel. A helium-neon laser diffraction method was used to determine SL (total of 36 SL per sample were recorded). The distance between primary diffraction bands was used to calculate SL. Correlation coefficients were determined using PROC CORR of SAS 9.4 and significance determined by $P < 0.05$.

Results

Overall and across all days aging, SL was not strongly correlated to intact desmin (r = –0.198; P = 0.07) or troponin-T degradation (r = 0.236; P = 0.04). Troponin-T degradation was not detected at d1 in any samples but overall and across all days was highly correlated with WBSF (r = –0.671; $P < 0.01$)). Intact desmin was correlated with WBSF (r = 0.661; $P < 0.01$). Across all samples, SL was correlated with WBSF (r = –0.445; $P < 0.01$). Intact desmin and troponin-T degradation were correlated (r = –0.818; $P < 0.01$).

Correlations within day of aging revealed that protein degradation was not significantly correlated with WBSF at d 1. In contrast, troponin-T degradation was correlated ($P < 0.01$) with WBSF at 8, 14, 21 d postmortem (r = –0.733, –0.641, and –0.772, respectively). Similarly, intact desmin was correlated ($P < 0.01$) with WBSF at 8, 14, and 21 d postmortem (r = 0.447, 0.553, and 0.824, respectively). SL was correlated ($P < 0.01$) with WBSF at each d postmortem (r = –0.445, –0.562, –0.714, and –0.512, respectively).

Conclusion

The correlation results suggest that SL is consistently correlated with WBSF across aging periods and is more strongly correlated with WBSF early postmortem than protein degradation. After aging, troponin-T degradation and intact desmin demonstrate greater correlations with WBSF than SL. Finally, SL correlation to troponin-T and desmin were generally similar and not strong, suggesting that SL does not affect the efficacy of proteinases to degrade proteins within the myofibril differently than extra-myofibrillar proteins.