Net calpain activity is positively correlated with myofibril fragmentation rate

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Tenderisation of meat arises primarily from postmortem proteolytic changes in structural myofibrillar proteins. In particular the calpain proteolytic system has been implicated in postmortem proteolysis of myofibrillar cytoskeletal proteins and myofibrillar fragmentation (1). The calpain proteolytic system consists of calpain I, calpain II and their specific inhibitor calpastatin. This study was designed to determine the link between variations in activities of the calpain system and subsequent rates of post mortem meat tenderisation. We hypothesizd that differences in net calpain activity produced through nutritional and other treatments in the live animal should be reflected by variations in postmortem rate of tenderisation.

Twenty four Dorset cross bred wether lambs with an average starting weight of 27.0 ± 3.3 kg were assigned randomly to one of three treatment groups, control, b-agonist or IGF-analog treatment. Within treatments lambs were fed at either 0.6 or 1.8 X maintenance level. After being held on experimental diets with continuous feeding for 1 week, animals were slaughtered and the Biceps femoris and Longissimus dorsi muscles removed for the determination of calpain system component activities and rate of tenderisation studies.

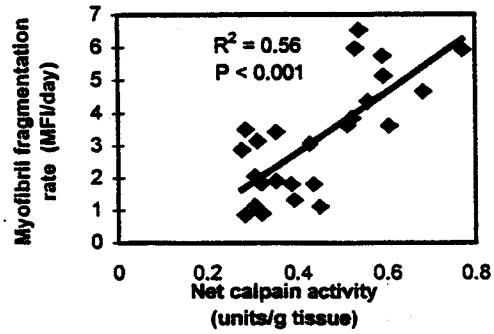

Figure. Net calpain activity (units/g tissue) expressed as the ratio of calpain I to calpastatin is positively correlated to myofibril fragmentation rate between 1 and 9 days postmortem.

Calpastatin levels were increased by high level of nutrition ($P<0.01$) and dietary inclusion of a b-agonist ($P<0.05$). Calpain I activity was significantly decreased by b-agonist treatment ($P<0.05$) and high nutrition IGF-analog treatment ($P<0.01$), but increased by low nutrition IGF-analog treatment ($P<0.01$). There was no effect of treatment on calpain II activity. Net calpain activity, defined as the ratio of calpain I to calpastatin, was significantly correlated with rate of myofibrillar fragmentation ($P<0.001$). Net calpain proteolytic activity, accounted for 56 percent of variation in myofibrillar fragmentation rate between 1 and 9 days postmortem.

These findings show that altering the activities of calpain I and calpastatin through nutritional treatments premortem has significant effects on the rate of postmortem fragmentation of muscle. Decreased net calpain activity reduces postmortem proteolysis.

1. Taylor RG, Geesink GH, Thompson VF, Koohmaraie M, Goll DE. Is Z-Disk degredation responsible for postmortem tenderisation. J Anim Sci 1995;73:1351-67.