

牛肉宰后嫩化机制的研究

Mechanism of postmortem tenderization of beef

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中文摘要:

为了阐明牛肉的成熟机理, 本试验模拟尸僵后牛肉的内在环境和成熟温度, 将纯化后的钙激活酶 I (μ -Calpain)、内源性钙激活酶 I 专一抑制剂(Calpastatin)、肌原纤维和钙激活酶 I 外源性抑制剂抑亮酶肽(Leupeptin)用于6个不同的处理组合, 反应不同时间后, 分别做变性聚丙烯酰胺凝胶电泳(SDS-PAGE)和蛋白质印迹分析(Western-blotting)。结果表明, Ca^{2+} (100 μM)对肌原纤维没有降解作用, 而在含 Ca^{2+} (100 μM)的反应体系中, μ -Calpain对肌间线蛋白(Desmin)和肌钙蛋白T(Troponin-T)都有明显的降解作用, 且降解产物和宰后牛肉自然成熟条件下的降解产物类似; Calpastatin不能完全抑制 μ -Calpain的活性; 离体反应和自然成熟的牛肉中的肌动蛋白都没有发生变化。以上结论表明 Ca^{2+} 很可能是通过激活 μ -Calpain而间接发挥对肌原纤维的降解作用, 而溶酶体组织蛋白酶在牛肉成熟的早期对嫩度的改善贡献不大。

英文摘要:

The objective of this study was to elucidate the mechanism of beef tenderization. Purified μ -Calpain, calpastatin, myofibrils and leupeptin were used in six different reaction mixtures. Myofibrillar SDS-PAGE and western blotting analysis were conducted after being incubated for a period of time in mixed salt solution(MSS) formulated on the basis of the post rigor condition. The results showed that desmin and troponinT were degraded by μ -Calpain into smaller fragments which were similar to the polypeptides degraded by aged beef, while calcium(100 μM) alone had no effects on myofibrils. It was also found that Calpastatin was not capable of completely inhibiting μ -Calpain activity and no degradation occurred for actin. It is concluded that cytoskeletal proteins were degraded by μ -Calpain during aging process, which might be the major reason for improving beef tenderness. Nevertheless, there was little contribution of Lysosomal cathepsins to beef tenderness at early period of aging.

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