

MEAT YIELD AND QUALITY

Effects of calcium chloride injection and pre-rigor storage temperature on tenderness indicators in bull Longissimus lumborum and Semitendinosus muscles

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Introduction

Classic work on muscle shortening phenomena demonstrates that meat stored at 12-15°C until the muscle has entered rigor produces the least shortening and, as a consequence, most tender meat. Coupled with known effects of calcium chloride (CaCl₂) injection, the objective of the current study was to establish whether pre-rigor injection of a CaCl₂ would overcome the toughening effects of storing meat for the first 24 h postmortem (pm) at temperatures either below 10°C (cold shortening) or above 15°C (heat shortening).

Methods

From both sides of 10 bull (5 Friesian x Hereford and 5 Murray Grey, 18-24 months) carcasses were removed both the Longissimus lumborum (LL; striploin) and Semitendinosus (ST; eye of round) at 30 min pm. All muscles were transported to Massey University (Palmerston North, New Zealand) by 50 min pm. Muscle pH was measured at 60 min pm, and again at 24 h and 7 d pm. A treatment group consisting of half of the total number of muscles of each type was injected (Fomaco 40 needle injector) to 105% of initial weight with a 2% CaCl₂ solution maintained at 15°C. Both untreated and injected muscles were then sliced into 6 equal lengths, vacuumed packaged, and allocated to 24 h storage treatments at 0, 4, 10, 15, 20, or 25°C after which time all samples were stored at 8°C for 6 days. At the end of the complete storage period, instrumental tenderness of 1 cm³ strips was measured with a MIRINZ Tenderometer after samples were cooked in a waterbath

to an internal temperature of 72°C. Myofibrillar fragmentation index (MFI) was measured at the same time on subsamples located adjacent to those prepared for tenderness evaluation.

Results and Discussion

Control and CaCl₂ treated steaks from both the LL and ST showed the characteristic relationship between temperature and tenderness first reported in the classic works of this nature. With the exception at 20°C for injected ST, minimum toughness was evident at 15°C. Maximum toughness was observed at 0° and 25°C for the LL and ST, respectively (Figs. 1a, b), and that this relationship was still evident after 6 d at 8°C, seems to confirm evidence that cold shortened meat does not tenderise significantly with ageing. Other researchers who injected LL and Semimembranosus with CaCl₂ have concluded that the greatest improvement in tenderness occurred between day 2 and 7 with little improvement thereafter. So, the fact that there was no significant difference between the control and injected samples after 7 d at 8°C was not unexpected.

The MFI of the injected muscles increased in a linear fashion with increasing temperature until reaching an asymptote at 10°C, after which little further fragmentation was observed (Figs. 2a, b). MFI of control samples, however, tended to increase in a linear fashion over the temperature range of 0-20°C in the LL and 0-25°C in the ST. The peak MFI was expected to occur at 15°C as previous work has shown that both m- and m-calpain are substantially depleted at temperatures in excess of this level as a consequence of early and rapid proteolytic activity, whilst enzymatic activity was slowed at temperatures below 15°C. The CaCl₂ injected LL samples had undergone a greater degree of fragmentation than the corresponding controls for all but the 0°C and 20°C stored samples and the difference in fragmentation was significantly different ($P < 0.05$) for the 10°C and 15°C stored muscles. In the case of the ST muscle samples the difference in MFI between the CaCl₂ injected samples and the respective controls was not as clear cut. Furthermore, the LL muscles were significantly more fragmented at all storage temperatures than the ST, a result that was not unexpected given that numerous studies have shown the LL to be more tender than the ST.

Irrespective of injection treatment, pH in both the LL and ST at 7 d was dependent on initial storage temperature with a minimum at 4°C rising to a maximum

at 15°C before declining again with increasing temperature (Figs 3a,b). In general, the CaCl₂ injected steaks had a lower 7 d pH than the comparable controls, although the differences were not significant. Increased myofibrillar fragmentation with consequential exposure of the amide groups on the myofibrillar fragments could explain the rise in pH for samples stored between 0-15°C, but such an explanation fails to explain the decrease in pH at temperatures above 15°C where myofibrillar fragmentation at the higher temperatures was comparable to that at 15°C for all treatments and for both the LD and ST muscles. Other authors have shown that as a consequence of early proteolysis of m-calpain and m-calpain in muscles stored at temperatures higher than 15°C, subsequent ageing is compromised compared to muscles stored at 15°C, and this could explain why meat pH failed to rise with temperatures above 15°C. The pH curves for both the LL and ST tend to be a mirror image of the tenderness and MFI curves indicating some relationship between final meat pH and toughness with maximal toughness and a reduction in MFI occurring at pH 5.9-6.1 with improvements as pH became more alkaline. Work by other authors corresponds with these results and indicates that meat tenderisation is affected by ultimate pH.

Conclusions

The most striking result from this study is that storage conditions during the pre-rigor period have a large impact on the tenderness, degree of fragmentation, and ultimate pH. The study confirms earlier work that showed cold shortened meat did not tenderise and that meat stored initially at 20° and 35°C was not as tender as meat that had been stored at 15°C. While some authors have attributed the toughening on either side of 15°C to be the result of enzymatic breakdown, the present study indicates that over the temperature range of 0-20°C, pre-rigor storage conditions simply controlled the rate of myofibrillar fragmentation and, by implication, enzymatic breakdown, to such an extent that subsequent storage at 8°C had little impact on further tenderisation. Injection of CaCl₂ did improve muscle tenderness, but did not materially affect the influence of pre-rigor temperature conditions as the main determinant of meat toughness. It would appear that the biochemical events over the first 24 h postmortem have a major bearing on subsequent meat toughness. From the commercial perspective, the benefits of CaCl₂ injection must be taken advantage

of within a 7 d window. Extrapolation of trends in the current results suggests that beyond this 7 d period, there appears to be no commercial advantage to the process and, given the extra handling and processing required, the treatment could be more costly than the conventional accelerated conditioning that has been used by the New Zealand meat industry for the past 22 years.

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Figure Captions

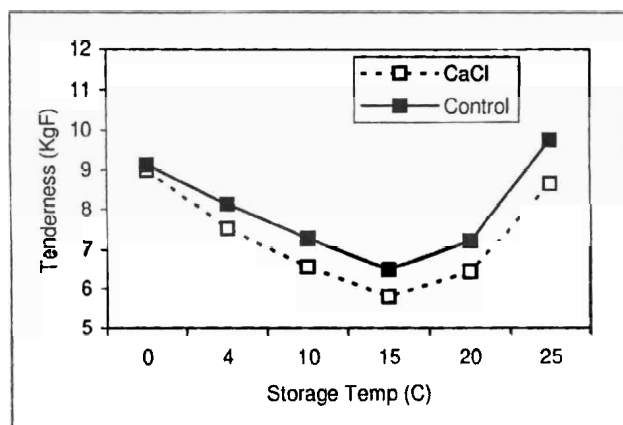


Fig 1a. Effect of pre-rigor storage temperature and calcium chloride injection on MIRINZ Tenderometer values of bull *Longissimus lumborum* measured 7 d postmortem.

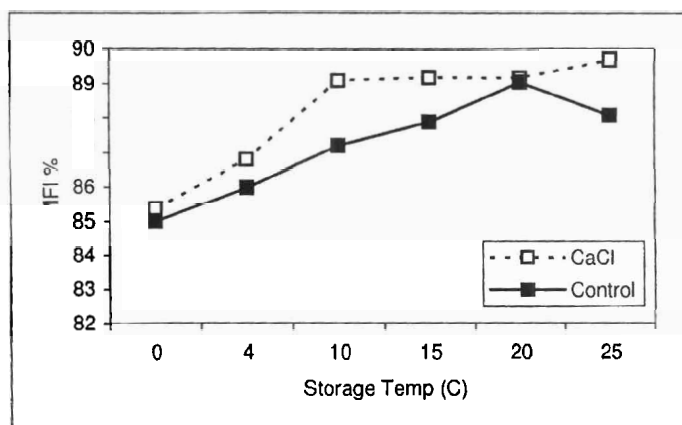


Fig. 2a. Effect of pre-rigor storage temperature and calcium chloride injection on myofibrillar fragmentation index values of bull *Longissimus lumborum* measured 7 d postmortem.

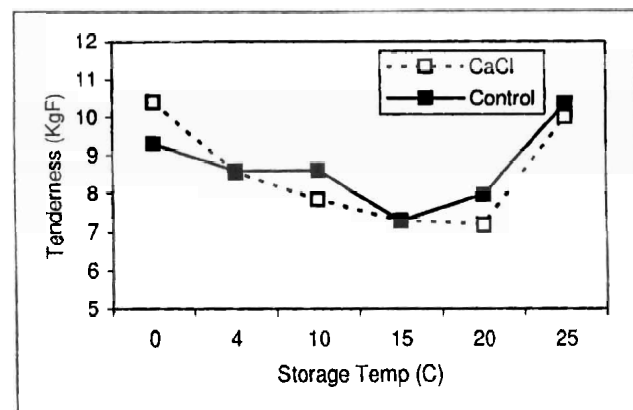


Fig. 1b. Effect of pre-rigor storage temperature and calcium chloride injection on MIRINZ Tenderometer values of bull *Semitendiosus* measured 7 d postmortem.

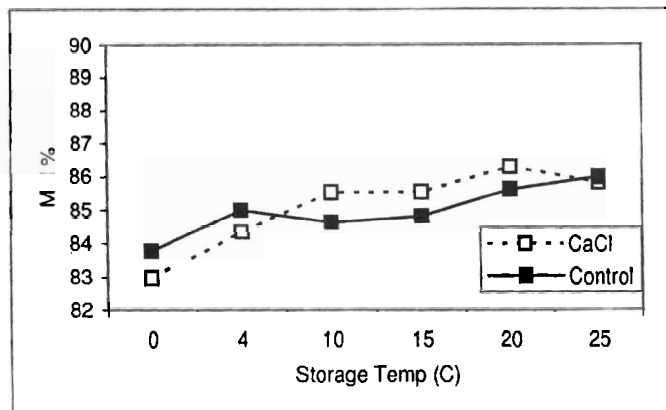


Fig. 2b. Effect of pre-rigor storage temperature and calcium chloride injection on myofibrillar fragmentation index values of bull *Semitendiosus* measured 7 d postmortem.

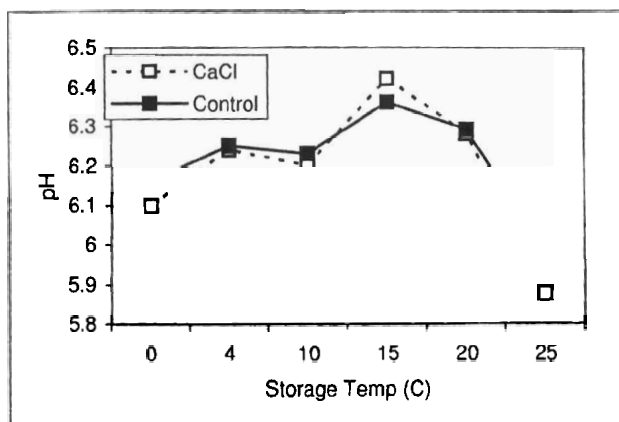


Fig. 3a. Effect of pre-rigor storage temperature and calcium chloride injection on pH_{7d} of bull *Longissimus lumborum*.

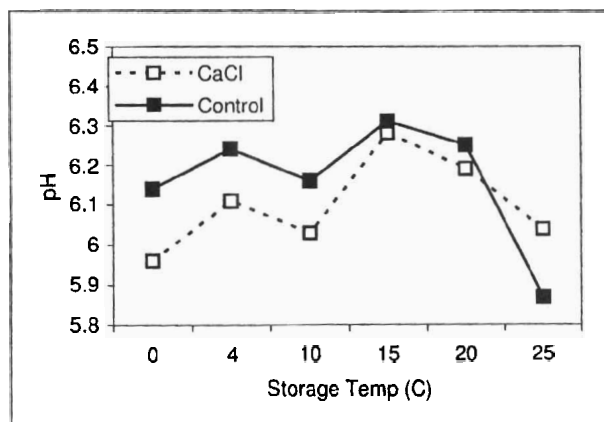


Fig. 3b. Effect of pre-rigor storage temperature and calcium chloride injection on pH_{7d} of bull *Semiten-dionsus*.
