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ULTRASTRUCTURE OF MECHANICALLY TENDERIZED PORK MEAT

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Please refer to Folio 58B. [Ed. note: Folio identification of S7P37.WP is in error.]

INTRODUCTION

It has been previously observed (Tyszkiewicz and Olkiewicz, 1992) that mechanical tenderization causes an increase in the water and brine holding capacity of meat which results in a better yield on cooking, as well as enhanced firmness and cohesiveness of the product. The technological effects of mechanical tenderization of meat were very similar to those caused by tumbling (Tyszkiewicz and Olkiewicz, 1992; Siegel *et al.*, 1978) but much more pronounced. The tumbling process, by applying energy to large pieces of meat, causes the development of a creamy, tacky exudate on their surface. The exudate, containing mainly salt-soluble proteins, contributes most to the binding strength of product. Muscle destruction caused by mechanical tenderization potentiates the availability of salt-soluble proteins and formation of a high protein exudate (Motycka and Bechtel, 1983).

The aim of the work was to examine the kind and degree of ultrastructure damage that occurs in mechanically tenderized meat in the context of protein availability.

MATERIALS AND METHODS

Excised well trimmed pork *biceps femoris* muscles cut off from the ham 48 hours post-mortem were mechanically tenderized by being treated with meat a activator, knife tenderizer and meat grinder with a kidney plate. In the meat activator (Lutterberg, Germany), the tubular attachment mounted on the outlet of meat grinder, the muscles were transported by a conveying worm and squeezed out through a narrow slide that caused a sudden explosive expansion of the meat. In the knife tenderizer (model IT-1, Belam, the Netherlands) the muscles passed between two knife rolls having 59 knives each, making a series incisions in the meat. In the meat grinder that was equipped with a kidney plate the muscles were transported by a conveying worm and coarsely ground. As a control, the untreated 48 hours postmortem muscle was used in each run. To study the ultrastructure, two or three muscles were examined following every type of treatment. Four samples of each muscle were randomly taken from the deeper muscle areas, which had not been directly touched by the apparatus. Samples of about 30mm in length (i.e., 3mm in diameter) were excised along the long axis of the muscle fibres. These were then attached to plastic rods, fixed in glutaraldehyde-paraformaldehyde, embedded in Epon, and prepared for electron microscopy. All procedures were previously described in detail (Jakubiec-Puka *et al.*, 1981; Jakubiec-Puka, 1985).

RESULTS AND DISCUSSION

In the ultrastructure of the 48 post-mortem control muscles numerous changes were found: kinked fibres, disrupted sarcolemma, destruction of mitochondria, fibre fragments devoid of contractile material and organelles, and swelling

of fibres. The contractile structure of several fibres was in supercontraction and contraction bands were frequent. Several irregularities and impairments of myofibrils were present, such as breaks in the continuity of the Z-line, complete disappearance of the Z-line or fractures of myofibrils at the Z-line level (Figure 1). Most of these changes are known to occur be in the postmortem muscle. In all experimental muscles they were also present; some of them were much more frequent, especially the broken myofibrils (Figure 2). Some other changes seemed to be peculiar to or characteristic of the particular treatment method. Namely, the meat activator produced considerable expansion of the fibre fragments, up to the disruption of the I-A band connection (i.e., loss of contact between the myosin and the actin filaments) (Figure 3). The knife tenderizer produced some focal damage and expansions of the contractile structure (Figure 4). The meat grinder disrupted the muscle fibre content irregularly, though, often with preserved continuity of the myofibrils (Figure 5). As it could be stated, the severity of meat tissue damage varied due to different techniques of mechanical tenderization. In all cases, however, the expansion of the contractile system, fractures across the myofibrils, and disruption of the contractile apparatus might allow free movement of intra- and extra-cellular materials, such as to cause an increase in the availability of proteins to extraction. Simultaneously, the pressure-induced disaggregation and conformational changes of proteins might occur, together with the breaking of divalent cations protein bonds (Macfarlane 1985). Offer and Knight (1988) proposed an explanation of water uptake in salted meat as being caused by the expansion of the lattice due to chloride ions being bound to proteins. The expansion was limited to some extent because the filaments were held together by other structural elements, especially the Z-line. Thus, the main forces acting on the filaments in intact postmortem muscle were the electrostatic repulsive forces and the restraining forces that result from transverse structural elements. In mechanically tenderized meat, where several linkages in the contractile system were broken, the increasing repulsive forces between filaments were no longer restrained allowing a severe rise in the amount of water uptake.

CONCLUSIONS

The ultrastructure of mechanically tenderized pork *biceps femoris* muscles exhibits severe damage to the contractile apparatus: fractures across the fibre; breaking of myofibrils at the Z-line level; disconnection of the A- and I-bands; and myofibrillar expansion and irregular disruption. Thus the ultrastructural damage caused by mechanical tenderization can be responsible, by destroying several linkages in myofibrils, for lattice expansion and an increase in the uptake of brine in meat.

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