

POSSIBLE MECHANISMS BY WHICH ELECTRICAL STIMULATION IMPROVES MEAT TENDERNESS

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INTRODUCTION

WHEN an electrical current is applied to a carcass shortly after death, the first noticeable change is the massive contraction that takes place in the carcass musculature. Although it is possible that the electrical current itself may have some effect, the major cause of the associated changes that take place in a carcass due to electrical stimulation is the depletion of muscle energy by the observed muscle contraction and subsequent attempts by the muscle to replenish this energy, or by the massive contractions themselves.

ACCELERATED POSTMORTEM GLYCOLYSIS AND RIGOR MORTIS

THE ATTEMPTS of the muscle to replenish the ATP used during electrical stimulation contractions, induce an acceleration of postmortem glycolysis and results in a rapid accumulation of lactic acid and a drop in pH (Carse, 1973; Bendall, 1976). In some studies a pH below 6.0 has been noted as early as one hour post-stimulation (Bendall, 1976; Chrystall et al., 1980). The alteration in pH itself may have an effect on tenderness by causing alterations in the various muscle proteins, but, more likely, the lowered pH produces an environment conducive for other changes in the muscle proteins. A low pH and rigor mortis formation occurs before heat can be removed from the carcass, resulting in rigor mortis at a higher temperature (George et al., 1980). The high temperature-low pH combination would also have an accelerating effect on acid proteases (Dutson et al., 1977). Development of rigor mortis at elevated temperatures has been associated with marked increases in tenderness (Locker and Daines, 1976; Dutson et al., 1977) and could be a major factor in tenderness changes of electrically stimulated muscle.

The events of rapid pH drop and rapid rigor formation of electrically stimulated muscle may not, in themselves, produce the tenderness changes, but they create the proper conditions for the tenderness changes to occur.

COLD SHORTENING

REPORTS in the literature have indicated that electrical stimulation causes an increase in tenderness by preventing the toughening associated with cold shortening or thaw rigor (Bendall et al., 1976; Chrystall and Hagyard, 1975, 1976; Davey et al., 1976). Measurement of the amount of shortening in electrically stimulated muscle by determination of sarcomere length has revealed that differences in shortening between electrically stimulated and non-stimulated sides of beef do not exist in most cases (Smith et al., 1977, 1979a; Savell et al., 1977, 1979; McKeith et al., 1979; Seideman et al., 1979; Will et al., 1980; George et al., 1980). However, differences in sarcomere lengths have been noted occasionally between electrically stimulated and non-stimulated muscle (Smith et al., 1977, 1979). George et al. (1980) found no difference in sarcomere length of stimulated and non-stimulated samples when using the laser technique of sarcomere measurement but did find differences when sarcomeres were measured on isolated fibers using the light microscope. Normally these two techniques produce identical sarcomere measurements (Cross et al., 1980). Sarcomere length values from various studies are presented in Table 1; results indicate that only two out of seventeen studies showed a significant effect ($P < 0.05$) of electrical stimulation on sarcomere length.

It appears from the number of studies that have been conducted, that electrical stimulation does not normally produce different sarcomere lengths than would have been present without the stimulation treatment. Thus, under normal chilling conditions, reduction of cold shortening is probably not a factor in the tenderness improvement caused by electrical stimulation. However, in some instances such as extreme chilling or in localized positions within a muscle, changes in sarcomere length may be prevented by electrical stimulation.

PROTEOLYSIS

PROTEOLYSIS has long been considered a possible mechanism for many of the tenderness changes that occur in postmortem muscle but the actual proof of this phenomenon has been elusive. Even so, studies have shown that conditions which favor proteolysis, such as acid pH and high temperature, also cause changes to take place that are associated with tender muscle and actually produce increased tenderness in some cases (Dutson et al., 1977; Locker and Daines, 1976).

Electrical stimulation has been shown by many researchers to produce a low pH condition in the muscle while the temperature remains fairly high (Dutson et al., 1977; Bendall, 1976; Chrystall et al., 1980; George et al., 1980). This condition, in addition to producing tenderness-associated changes, has also been shown to cause a release of lysosomal enzymes from the membrane-bound condition into a milieu favorable for their action (Moeller et al., 1976, 1977).

Investigations were conducted in our laboratory on ovine muscle to determine if electrical stimulation also caused a release of enzymes from the lysosome. The data presented in Table 2 (Dutson et al., 1980) indicates that a 24% to 30% increase in free activity of lysosomal enzymes is caused by electrical stimulation. At this point it is not certain if it is the electrical impulse itself or the lowered pH-high temperature that causes a rupturing of the lysosomal membrane. The release of these enzymes at an acid pH may be responsible for some of the tenderness change caused by electrical stimulation.

Research by Savell et al. (1978b), Nilsson et al. (1979) and George et al. (1980) has shown that electrical

stimulation causes an acceleration in the aging process of beef. If the aging process results from or is enhanced by proteolytic enzymes, then an increase in the aging process by electrical stimulation is further evidence that proteolysis may be activated by electrical stimulation.

STRUCTURAL ALTERATIONS

RESEARCH reported by Savell et al. (1978c) was the first to demonstrate that structural alterations occurred in muscles of animals that had been electrically stimulated. These alterations were demonstrated using both the light microscope and the electron microscope and appeared as contracture bands or contracture nodes.

Electron micrographs of electrically stimulated and non-stimulated muscle are presented in Figures 1 and 2, respectively. These micrographs show the characteristic differences between stimulated and non-stimulated muscle. Disappearance of the banding pattern and disruption of the Z line is clearly evident in the contracture band area of the stimulated muscle. Disruption of the sarcoplasmic reticulum and slight separations of the myofibrils are also evident in some areas. Many of the alterations in the stimulated muscles appear very much like those that are caused in muscles having rapid postmortem glycolytic rates (Dutson et al., 1974; Abbott et al., 1977), except for the contracture bands.

More recently, Will et al. (1980) and George et al. (1980) have made similar observations of structural alterations in electrically stimulated muscle. These authors both observed the structures we have termed contracture bands, but George et al. (1980) attributed these structures to precipitation of other protein material on the myofibrils. It is clearly evident from Figures 1 and 2 that the structures observed as contracture bands are continuous with the unaffected myofibrils and are derived from them. It is also evident that myofilaments and remnants of Z lines (arrows) are present in the contracture band and are continuous with their more normal counterparts in the unaffected portion of this micrograph. Thus, it is felt that the major portion of the structural abnormality in the contracture band area is due to extreme shortening of the sarcomeres and not due to precipitation of sarcoplasmic protein as described by George et al. (1980).

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Table 1. Sarcomere Length Values for Various Electrical Stimulation Experiments.

ES	NS	Level of probability	Study
1.83	1.84	NS	Savell et al. (1977)
1.73	1.75	NS	Savell et al. (1978b)
1.86	1.86	NS	Savell et al. (1979)
1.67	1.65	NS	Bowling et al. (1978)
1.67	1.68	NS	Bowling et al. (1978)
1.72	1.71	NS	Bowling et al. (1978)
1.82	1.86	NS	Smith et al. (1979a)
1.73	1.77	NS	Smith et al. (1979a)
1.74	1.74	NS	Seideman et al. (1979)
1.96	1.84	P<0.05	Smith et al. (1979b)
1.58	1.58	NS	McKeith et al. (1979)
1.65	1.70	NS	Will et al. (1979)
3.44	3.22	NS	Will et al. (1979)
1.65	1.76	NS	Will et al. (1979)
2.03	1.90	NS	Will et al. (1979)
1.60	1.58	NS	George et al. (1980)
1.85	1.87	NS	George et al. (1980)
1.60	1.46	NS	George et al. (1980)
2.09	1.80	P<0.01	George et al. (1980)

Table 2. Free Activity Changes in Lysosomal Enzymes Caused by Electrical Stimulation^a.

Enzyme	Free Activity Increase
β -glucuronidase	24%
Cathepsin-C	30%

^aAdapted from by Dutson et al. (1980).



Figure 1. Electron micrograph of electrically stimulated muscle immobilized and embedded at 6 hours postmortem. Arrows denote a continuation of Z line remnants. CB=contracture band area.

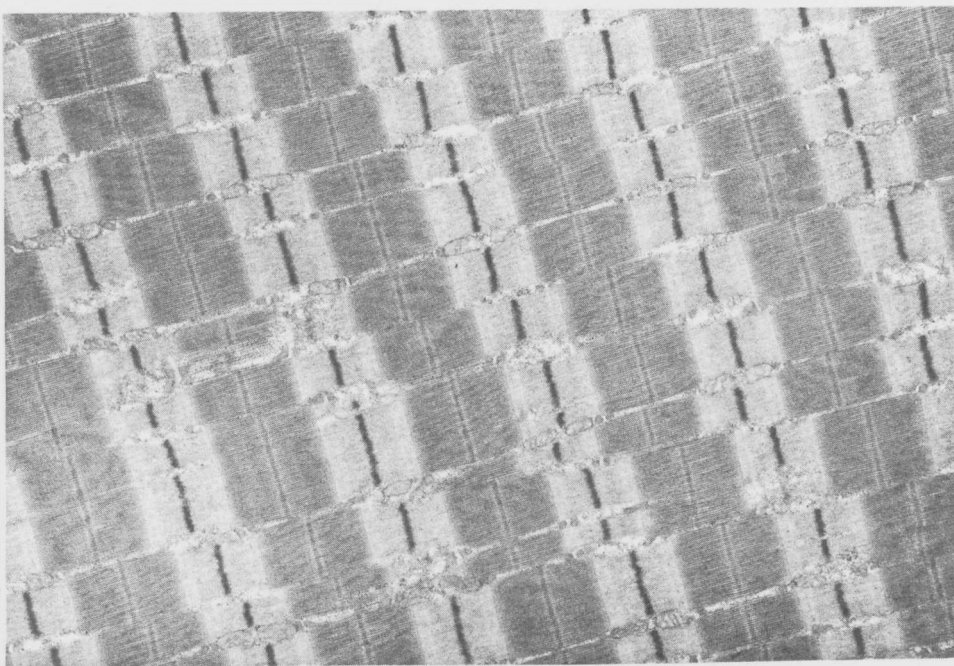


Figure 2. Electron micrograph of control muscle from the unstimulated side of the same carcass as shown in Figure 1.