RIGOR ONSET BEFORE CHILLING

by
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Early research on the changes occurring in muscles during development of rigor mortis and particularly during rigor shortening (Bate-Smith, 1948; Bate-Smith and Bendall, 1949; Bendall, 1951; Marsh, 1954), have provided a background for an understanding of the factors involved in the onset of rigor mortis and the reasons for rigor shortening. These early workers determined that post-mortem muscle undergoes shortening in association with rigor mortis, and that shortening is highly temperature-dependent, with much more drastic shortening taking place at higher temperatures. Locker (1960), reported that the degree of muscle contraction was definitely a factor in determining the ultimate eating quality of meat, and suggested that meat toughness could be prevented by placing carcasses in a configuration that would stretch the muscles and prevent their shortening. Locker and Hagyard (1963) extended the earlier findings of Bate-Smith (1948), Bendall (1951) and Marsh (1954) by the discovery that shortening in beef muscle not only increases as temperature is raised toward 37°C, but also increases as temperatures are lowered toward 0°C, with a minimum amount of shortening taking place between 14 and 19°C.

Based on a knowledge of the phenomena of cold-shortening and the relationship of shortening to tenderness, a considerable amount of research was initiated to determine methods for alleviating or minimizing cold-shortening. This research has primarily been in two areas, that of preventing shortening due to restraining the muscles (which is the subject of a companion paper by Dr. Stouffer), and that of eliminating the stimulus to cold-shortening by holding carcasses at higher temperatures for a period of time prior to onset of rigor mortis. Stouffer (1977) has discussed the research concerned with alleviating shortening by placing physical restraint upon the muscle which has received considerable emphasis in our laboratory.

Numerous articles have been published on the relationship of post-mortem temperature decline and muscle tenderness (Locker and Hagyard, 1963; Marsh and Leet, 1966a, 1966b; Marsh *et al.*, 1968; McCrae *et al.*, 1971; Busch *et al.*, 1967; Parrish *et al.*, 1969; Smith *et al.*, 1971; Bouton *et al.*, 1973; Bouton *et al.*, 1974; Harris, 1975; Fields *et al.*, 1976; Smith *et al.*, 1976), with most of these articles relating tenderness differences to differences in cold-shortening. Although the relationship of shortening to tenderness has been shown in numerous experiments, a recent study by Locker and Daines (1976) has shown that by altering the temperature of sternomandibularis muscles in the final stages of rigor, markedly different tenderness can be produced in muscles of identical sarcomere lengths.

Recent research in our laboratory (Table 1) also indicates that differences in tenderness can be produced in muscles having identical sarcomere lengths. It is evident from these data that holding carcasses at elevated temperatures early post-mortem, increases sensory panel tenderness rating and decreases the shear force of longissimus muscles. In the first three groups of animals, this tenderness increase could be attributed to increases in sarcomere length of myofibrils from the high temperature treated sides. However, the animals which had both sides suspended via the obturator foramen, had identical sarcomere lengths in the control and treated sides, yet there was a significant difference in tenderness. This indicates that, although a portion of the increased tenderness resulting from delayed chilling and elevated temperatures during rigor onset is caused by increased sarcomere lengths, another factor affects tenderness which is not related to sarcomere length.

This "other tenderness factor" could be related to increased rates of autolytic proteolysis in the elevated temperature treated sides and it seems logical that the increased temperature and decreased pH (Cassens and Newbold, 1967) would have an effect on the rupture of lysosomes and release of lysosomal enzymes (Hirsch and Cohn, 1964; Wiseman, 1964). Experiments in our laboratory where animals were treated similar to those described in Table 1, have shown that the free activity of β-glucuronidase and cathepsin C (both lysosomal enzymes) was increased significantly by the delayed chilling treatment (Moeller *et al.*, 1976). Additional experiments were con-

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TABLE 1

MEAN SARCOMERE LENGTH, OVERALL TENDERNESS AND SHEAR FORCE VALUES FOR LONGISSIMUS MUSCLES FROM CONTROL (C) AND HIGH TEMPERATURE CONDITIONED (HT) SIDES.

Animals			Method of carcass	Sarcomere length (μm)		Overall tenderness[b]		Shear force (N)[c]	
N	Age	Description	suspension	C	HT[a]	C	HT	C	HT
17	2-4 wk.	Veal	Achilles	1.75***	1.92	5.1***	6.3	63.2***	45.8
6	9-12 mo.	Beef	Achilles	1.77***	1.96	5.0*	6.3	76.1	68.1
20	18-26 mo.	Beef	Achilles	1.78***	1.93	4.8***	5.6	84.1***	69.4
10	9-12 mo.	Beef	Obturator foramen	2.22	2.20	6.7*	7.1	48.9**	42.3

a HT sides were held at 22°C for 4 hr., 16°C for 8 hr. and 1°C for 36 hr., while the opposite side (control) was held at 1°C for the entire 48 hr. period.

b Determined by an 8-member sensory panel, (8 = extremely tender; 1 = extremely tough).

c Force expressed as Newtons

*$P < .05$

**$P < .01$

***$P < .001$

ducted where muscle samples were held at 37°C or 2°C for 12 hr. post-mortem (Moeller *et al.*, 1977), with the 37°C treated samples having a much more rapid decline in pH (Figure 1), a greater release of lysosomal enzymes from the lysosome and much higher fragmentation values than the 2°C controls. Thus, treatment of carcasses with high pre-rigor temperatures causes a release of lysosomal enzymes at an acid pH environment conducive to proteolysis, as well as reducing shortening of the myofibrils.

A logical explanation for increased tenderness with no difference in sarcomere shortening would be changes in the connective tissue component of tenderness. Changes in the connective tissue were investigated by determining percentages of alpha and beta subunits in samples held at 37°C or 1°C for 12 hr (Wu and Dutson, 1977). Very little difference was found in collagen subunits between the 1°C and 37°C treatments; however, the 37°C treatment caused a more marked difference in collagen subunits between 12 and 24 hr post-mortem. This indicates that changes take place in the collagen; however, these changes appear to be small in relation to the tenderness differences observed.

In order to determine if changes in myofibrillar proteins were associated with higher temperatures and lower pH's, myofibrils were purified from muscle samples incubated at 37°C or 4°C and purified myofibrils were applied to SDS gels for electrophoresis (Yates, 1977). SDS gels were scanned using a densitometer to quantitate the relative amount of the proteins present (Table 2). When samples were incubated at 37°C significantly less protein appeared in the myosin region and significantly more protein in the α-actinin region than when samples were incubated at 4°C. Less α-actinin may be a result of less

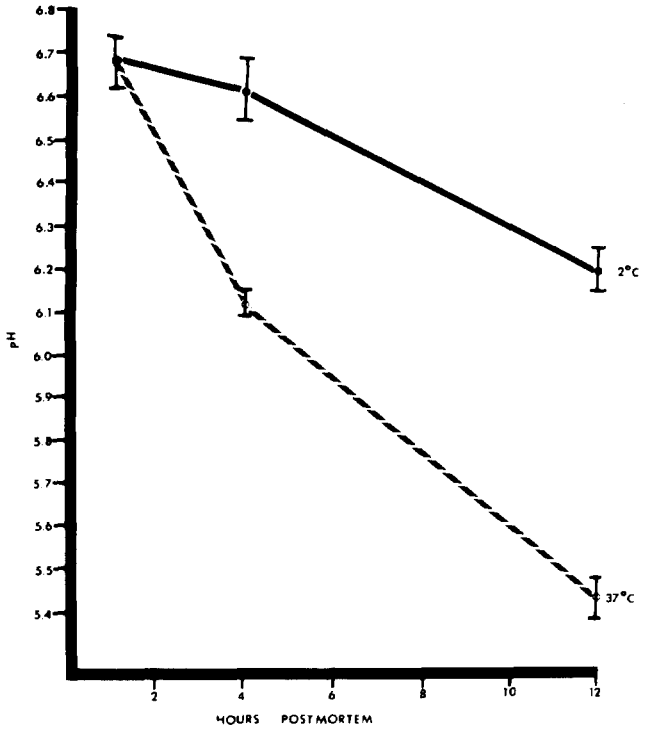

FIGURE 1

A plot of pH vs time post-mortem (hr) for 2°C and 37°C incubated muscle samples showing the increased rate of pH decline for the 37°C incubated muscle (average of six animals). The pH at 4 and 12 hr is significantly less ($P < 0.05$ and $P < 0.01$, respectively) for the 37°C incubated samples. Vertical bars indicate standard deviations. From Moeller et al. (1977).

calcium activated factor (CAF) activity in the 37°C treatment due to the rapid drop in pH observed in these samples (Figure 1). In comparing gels of myofibrils prepared from muscle incubated at 4°C or 37°C (Figure 2), the actual differences in the amount of protein in the various band areas can be observed, especially the decrease in the amount of myosin and an alteration in the M and C protein areas. A change in the amount of protein in the area of α-actinin, increased amounts of material between α-actinin and actin (particularly at about 50,000 daltons, just above actin), an alteration in the troponin-T and tropomyosin peak and an increase in the material in the 28,000 to 32,000 dalton region can also be observed (Figure 2).

In order to determine whether the lowered pH or the increased temperature caused the major effect on myofibrillar proteins, pre-rigor ground muscle samples were incubated at 37°C for 12 hr at pH 5.4 and pH 7.0. In general, the same type of changes occurred at pH 5.4 as occurred due to incubation of whole muscle at 37°C, whereas the changes in the samples incubated at pH 7 were small when compared to that of control samples (Figure 3). The latter finding indicates that the lower pH and associated lysosomal catheptic activity is probably responsible for changes observed in myofibrillar proteins due to 37°C incubation, and that the enzyme CAF which is active at a neutral pH and not at an acid pH (Dayton *et al.*, 1976), was probably not a factor in changes found in myofibrillar proteins (Yates, 1977).

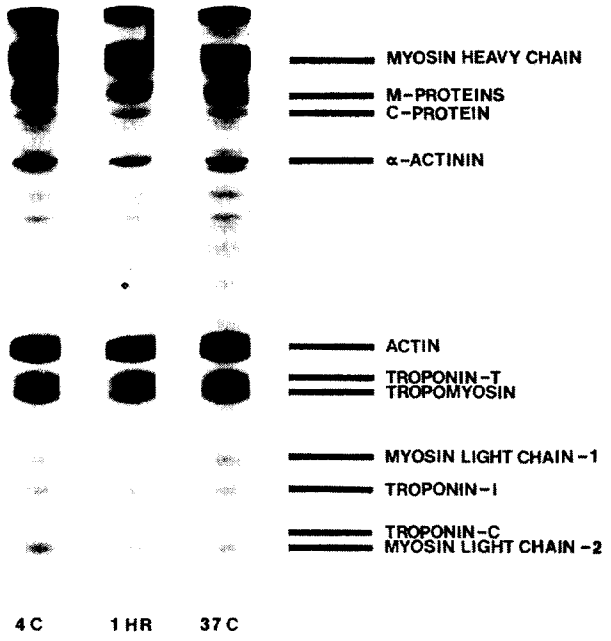

FIGURE 2

SDS polyacrylamide gels of myofibrils prepared from 1 hr post-mortem muscle and 12 hr post-mortem muscle incubated at 1°C and 37°C. (From Yates (1977).

TABLE 2

MEAN VALUES FOR THE PERCENTAGE PROTEIN COMPOSITION AS DETERMINED BY DENSITOMETRY OF SDS POLYACRYLAMIDE GELS OF THE MYOFIBRILLAR FRACTION PREPARED FROM 4° AND 37°C INCUBATED LONGISSIMUS MUSCLE SAMPLES.[c]

Region	Percentage of myofibrillar protein	
	4°C	37°C
Myosin	29.5[a]	24.8[b]
M-proteins	8.3[a]	9.0[a]
C-proteins	3.0[a]	3.2[a]
α-actinin	3.6[a]	6.6[b]
50,000-100,000 Dalton region	1.6[a]	3.0[b]
Actin	27.8[a]	26.6[a]
Troponin-T + Tropomyosin	17.3[a]	16.0[a]
28,000 - 32,000 Dalton region	1.1[a]	1.8[b]
MLC A-1	2.4[a]	3.2[b]
Troponin I	2.3[a]	2.4[a]
Troponin C	3.4[a]	3.5[a]

a, b Mean values in the same row bearing a common superscript letter are not ($P > .05$) significantly different.

c Values are means of duplicant gels from 6 different animals. Adapted from Yates (1977).

Electron micrographs taken of samples incubated at 37°C and at 1°C are presented in Figure 4. In the longitudinal section the Z line appears much more dense in the 37°C treatment than in the 1°C treatment, indicating that CAF was probably more active in the 1° samples which had a much slower pH decline which is consistent with the SDS gel data. It appears that the samples incubated at 37°C also have thinner, more thread-like appearing thick filaments when compared to either controls or 1°C incubated muscle. This is also consistent with the finding of alterations in the myosin molecule as observed on SDS gel electrophoresis (Figure 2). When observing cross-sections of muscle incubated at 37°C or 1°C, the 37°C incubated samples differ extremely in appearance from either control or 1°C incubated muscle. It is difficult to discern thick vs. thin filaments in the 37°C incubated samples; whereas, both thick and thin filaments can be observed in the cross sections of control and 1°C incubated muscle, again indicating that changes in the filament structure have taken

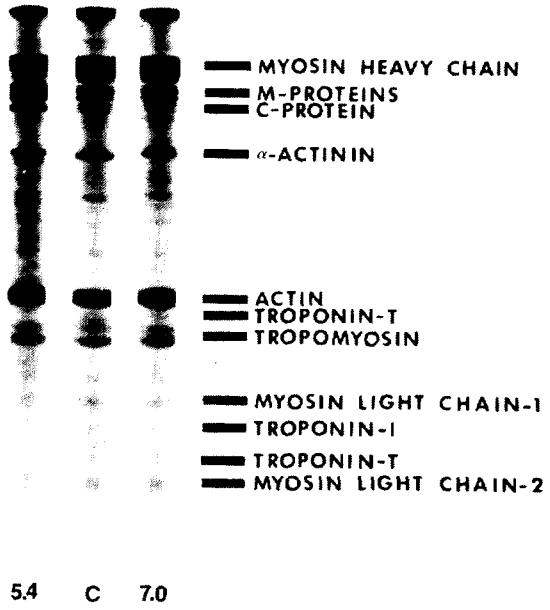

FIGURE 3

SDS polyacrylamide gels of myofibrils prepared from control muscle (C) and muscle incubated at pH 5.4 and pH 7.0 for 12 hr. From Yates (1977).

place as a result of incubation at 37°C. Electron micrographs of muscle incubated at pH 5.4 and pH 7.0 presented by Yates (1977) revealed that alterations occurring at pH 5.4 were similar to those observed in 37°C incubated muscle, while appearance of samples incubated at 7.0 were more nearly like those of control samples and those incubated at 1°C.

In order to determine the specific changes occurring in myosin, myofibrils were incubated with papain. Papain is known to hydrolyze myosin between the heavy meromyosin subunit 2 and heavy meromyosin subunit 1 (S1) portion of the molecule, leaving the S1 heads attached to actin in post-rigor muscle (Cooke, 1972). The S1 heads of myosin can then be released by pyrophosphate extraction, which results in a characteristic protein band at approximately 95,000 daltons on SDS gels as shown in Figure 5 (Cooke, 1972). Muscle tissue samples which had been previously held at 37°C or 1°C were also extracted with pyrophosphate and a similar protein band appeared at approximately 95,000 daltons on SDS gels of the extract from 37°C treated muscle but not on gels from 1°C treated muscle (Figure 6). This indicates that 37°C incubation of muscle probably hydrolyzes myosin by proteolysis at the papain-sensitive region of the myosin molecule. Muscle samples which had been previously incubated at pH 5.4 or pH 7.0 have also been extracted with pyrophosphate and a protein fragment similar to S1 has been observed in extracts of the pH 5.4 treatment, but no such fragment was found in extracts of the pH 7.0 or control samples (Yates, 1977). This indicates that the combination of low pH and high temperature activates proteolysis and enhances breakage of myosin at the papain-sensitive region, removing the S1 head from the remainder of the myosin molecule (Yates, 1977).

Another type of treatment for producing rapid rigor onset which has recently been investigated rather extensively in New Zealand, England and the U.S. is electrical-stimulation. The use of electrical-stimulation to increase tenderness of meat is not a new idea. It was used as early as 1749 (Lopez and Herbert, 1975) and a patent was granted to Harsham and Deatherage (1951) to use electrical-stimulation for tenderizing meat. After 1951 very little attention was paid to electrical-stimulation until New Zealand researchers used this method to alleviate the toughening of meat caused by cold-shortening (Carse, 1973; Chrystall and Hagyard, 1975, 1976; Davey *et al.*, 1976). More recently in the United States (Grusby *et al.*, 1976; Savell *et al.*, 1976, 1977a, b; Smith *et al.*, 1977a, b), research involving voltages and stimulating conditions different from those used in New Zealand has been conducted.

Table 3 summarizes some of the research conducted in our laboratory on electrical-stimulation of various species. An increase in tenderness was found in each case when electrical-stimulation was applied. Although other studies (Locker, 1976; Chrystall and Hagyard, 1976; Davey *et al.*, 1976; Chrystall, 1976) have attributed the increased tenderness of electrically-stimulated muscle to prevention of cold-shortening, our studies indicate that, in many cases, the sarcomere length of electrically-stimulated carcasses was not different from that of control carcasses (Table 4). Therefore, with our method of stimulation (Electro-Sting model 160-ESS set at 100 volts, 5 amps and 50 cycles per second, which generates approximately 440 volts between electrodes; 50 impulses of 0.5 to 1 second each are administered) there must be some mechanism, in addition to or other than prevention of cold-shortening, which produces the increased tenderness of electrically-stimulated carcasses. Due to the fact that electrical-stimulation produces a rapid pH drop (Carse, 1973; Jambers, 1977) and the fact that a lowered pH at high temperatures causes a release of lysosomal enzymes (Moeller *et al.*, 1977), these enzymes may cause changes in muscle proteins which are similar to those which have been shown to occur when muscle is incubated at 37°C (Yates, 1977). Recent evidence indicates that an increase in

FIGURE 4

Electron micrograph of 1 hr post-mortem muscle (C and D) and muscles incubated at 1°C (E and F) and 37°C (A and B). From Yates (1977).

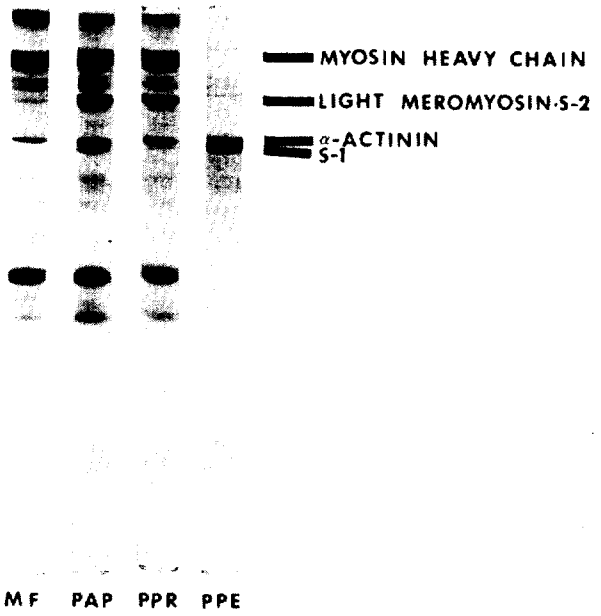

FIGURE 5

SDS polyacrylamide gels of myofibrils (MF), myofibrils treated with papain (PAP), residue from extraction of PAP with pyrophosphate (PPR) and the pyrophosphate extract of PAP (PPE). From Yates (1977).

the free activity of lysosomal enzymes is caused by the proper electrical-stimulation treatment (Dutson *et al.*, 1977). However, the actual effect of the lowered pH on myofibrillar proteins of electrically-stimulated muscle has yet to be determined.

We have also observed (Savell *et al.*, 1977b) that under certain electrical-stimulation conditions almost always contraction bands appear along the muscle fibers of electrically-stimulated carcasses (Figures 7, 8, 9 and 10). These contraction bands are associated with extreme shortening of sarcomeres in certain sections of the muscle fiber and concomitant stretching of sarcomeres in other sections of the fiber. Also, the severe shortening in the area of the contraction bands produces some tearing or rupture of muscle fibers, which may also be a factor related to the increased tenderness of the electrically-stimulated muscle (Figures 7, 8, 9 and 10).

It presently appears that electrical-stimulation produces tenderization by physical alterations of the muscle cells and by increasing the free activity of lysosomal enzymes, in addition to the tenderness increase caused by the alleviation of cold-shortening.

TABLE 3

EFFECTS OF ELECTRICAL-STIMULATION ON MEAN VALUES FOR SHEAR FORCE AND TENDERNESS

Carcasses	Shear force value (kg)		Tenderness rating[a]	
	Control ($\bar{x}$)	Electrically stimulated ($\bar{x}$)	Control ($\bar{x}$)	Electrically stimulated ($\bar{x}$)
Grain-fed goat	6.3	4.7	3.5	4.5
Grain-fed lamb	3.8	2.9	6.0	6.7
Forage-fed beef	8.5	6.4	5.0	6.2
Grain-fed beef	10.0	5.4	2.8	5.0
Forage-fed beef	7.8	4.6	3.1	4.8
Hot-skinned calf	5.5	4.3	5.0	5.8
Cold-skinned calf	5.8	4.8	4.9	5.8

a Sensory panel ratings based on an 8-point scale (8 = extremely tender; 1 = extremely tough), (Smith et al., 1977a).

TABLE 4

EFFECT OF ELECTRICAL STIMULATION ON SARCOMERE LENGTH

Carcasses	Sarcomere length (microns)	
	Control	Electrically stimulated
Grain-fed goat	1.76	1.85
Grain-fed lamb	1.80	1.83
Forage-fed beef	1.84	1.83
Forage-fed beef	1.84	1.96
Hot-skinned calf	1.86	1.83
Cold-skinned calf	1.77	1.73

(Smith et al., 1977a)

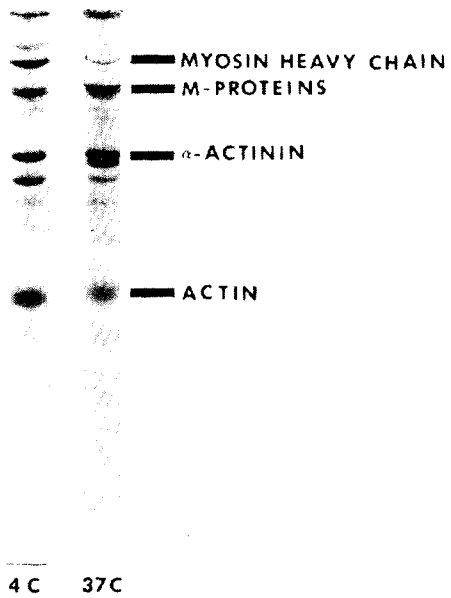

FIGURE 6

SDS polyacrylamide gels of pyrophosphate extracts of myofibrils prepared from muscle incubated at 4°C and 37°C. From Yates (1977).

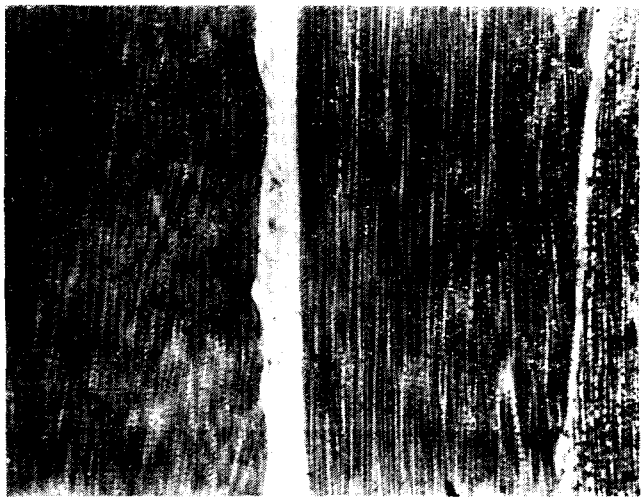

FIGURE 7

Light micrograph of muscle fibers from a control carcass. X 1,000. From Savell et al. (1977b).

FIGURE 8

Light micrograph of muscle fibers from an electrically stimulated carcass. CB = contraction band. X 1,000. From Savell et al. (1977b).

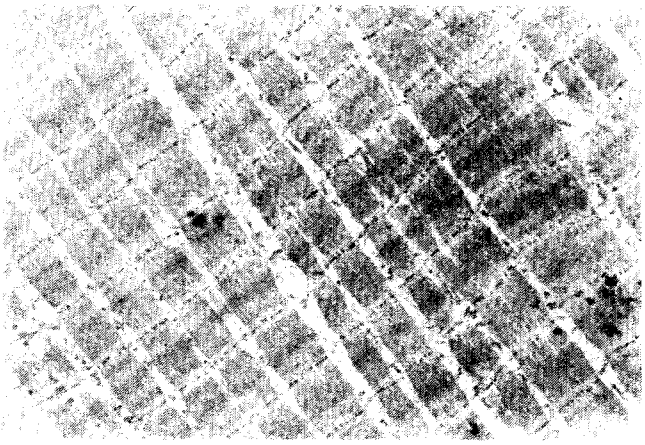

FIGURE 9

Electron micrograph of a muscle fiber from a control carcass. X 9,250. From Savell et al. (1977b).

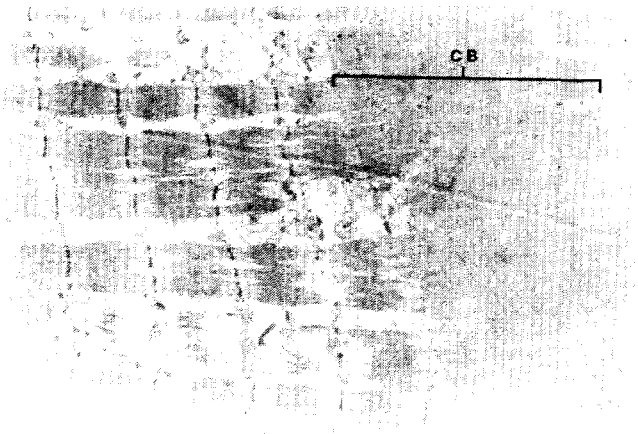

FIGURE 10

Electron micrograph of a muscle fiber from an electrically stimulated carcass. CB = contraction band. X 9,250. From Savell et al. (1977b).

SUMMARY

Rigor-onset before chilling can be produced by holding carcasses at elevated temperatures during the initial post-mortem period, allowing the muscle to go into rigor mortis at a higher temperature and preventing cold-shortening. However, when muscles are restrained to produce identical sarcomere lengths, elevated temperature conditioning produces increased tenderness without affecting sarcomere length. Although cold-shortening is prevented or minimized by high temperature pre-rigor conditioning, other factors which add to the increased tenderness must be operative. Increased activity of lysosomal cathepsins (due to the low pH and high temperature of the muscle) which hydrolyze myosin at the papain-sensitive region, releasing the S1 head from the myosin molecule is probably one operative factor. Other changes taking place in myofibrillar proteins include an alteration in the troponin-T band and the production of a 28-32,000 molecular weight subunit. These changes are a direct result of the lowered pH and higher muscle temperature and are probably caused by acidic catheptic activity and not by the neutral protease CAF.

Electrical-stimulation is another method by which the rate of rigor-onset can be increased, with rigor mortis occurring before chilling of the muscles reaches a point critical to cold-shortening. However, investigations in our laboratory have indicated that under certain conditions of electrical-stimulation, no difference in shortening of sarcomeres occurs between control and stimulated sides. Thus, electrical-stimulation must be producing increases in tenderness by other modes of action in addition to the minimization of cold-shortening. We have postulated that

the increase in free lysosomal enzymes resulting from lowered pH and concurrent high temperature of electrically-stimulated muscle increases the proteolysis of muscle proteins. Also, electrical-stimulation produces structural alterations in the muscle with contraction bands, stretched sarcomeres and torn muscle fibers being evident. These structural alterations may have a major effect in increasing the tenderness of electrically-stimulated carcasses.

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