Post-Mortem Chemical Changes in Muscle - Meat Aging

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Introduction

Since the earliest days of muscle science, physiologists have been attracted to the phenomenon of rigor mortis. Because considerable quantities of lactic acid accumulate in post-mortem muscle, the characteristic stiffness of rigor was originally attributed to the denaturation and reduced solubility of proteins at the lowered pH. However, some 50 years ago (Lundsgaard, 1930) this comfortable view was discarded after the unequivocal demonstration that the stiffening of rigor also occurs in muscles from stressed animals — muscles in which there is little or no accumulation of lactic acid. This observation led to a thorough reappraisal of the phenomenon of rigor mortis, and, arising from the developing knowledge of the contractile process, it was soon evident that the primary biochemical cause of rigor development was the loss of ATP from post-mortem muscle (Bate-Smith and Bendall, 1947; 1949). The extensive research that has led us to a comprehensive understanding of the process of rigor development has been the subject of excellent recent reviews (Bendall, 1973; Marsh, 1981).

The phenomenon of rigor has assumed a special significance to the physiologist concerned with elucidating the mechanism of muscular contraction. It is now recognized that rigor, brought about by ATP disappearance, is a consequence of the cross-bridging of the heads of the myosin molecules of the thick filaments with the g-actin monomeres of the thin filaments. This cross-bridging is also a momentary but key step in the cyclical interaction of these sets of proteins during physiological contraction (Squire, 1981). Our knowledge of post-mortem biochemical changes in muscle will be advanced with further elucidation of the biochemistry of the cold- and heat-shortening phenomena (Locker and Hagyard, 1963) and the role of calmodulin in compartmentalizing the various control functions of Ca^{2+} (Klee and Vanamon, 1982).

Biologists are well aware that living tissue is in a state of continual replacement and that the complex and simple molecules that make up an organism undergo very active changes. In muscle, for instance, turnover time varies from months for collagen (Woessner, 1968) to days for the myofibrillar proteins (Millward, 1980). In the past, scientists pursuing the molecular basis of contraction have left rather unregarded the catabolic aspects of such protein turnovers. However, these catabolic processes are now receiving their due attention, and their elaboration owes much to the contributions of the meat scientist.

It is not my intention to review again the chemical changes of rigor development, but rather to offer a perspective of the structural and catabolic changes that occur in muscle after rigor mortis has been achieved. Particular emphasis is given to those changes associated with meat aging, since the study of these has contributed to our knowledge, not only of muscle structure but also of meat texture.

Factors Affecting Meat Tenderness

The results reported here depend heavily on studies using ox M. sternomandibularis. These studies were designed to identify the relationship between muscle structure and meat tenderness and have, therefore, required the use of a single, uniform muscle type. It is with no parochial intent that lesser mention is made of excellent work using different muscles and species.

Tenderness, or its converse toughness, is now recognized to be affected by a number of post-mortem events, of which aging is but one. Tenderness is a property of cooked meat. To relate tenderness to the fine structure of raw meat is an undertaking of doubtful use, so profound are the structural changes wrought by cooking. However, rather simple observations can be made on changes in tenderness with different post-mortem treatments; observations that offer insights into both muscle structure and general meat texture.

Tenderness is measured as the shearing force (expressed as a shear-force value) required to cleave a standard cross-sectional area of cooked meat across the muscle cells or fibers. The Warner-Bratzler machine cleaves the meat with a thin metal plate, whereas the MIRINZ tenderometer (Macfarlane and Marer, 1966) cleaves with a blunt wedge. Despite these design differences, the mechanical events occurring on cleaving are essentially the same. The wedge compresses the meat and the fibers are stretched, especially at the leading edge, where, with the further penetration of the wedge, they eventually reach their yield point and break. In other words, toughness, as measured by the tenderometer, is related to the tensile strength of the individual muscle fibers. However, the distortion patterns produced in meat during cleaving (Fig. 1) show that slip or strain is widely dispersed away from the tip of the wedge. Shearing develops initially between fibers and is presumed also to occur between myofibrils. The strain will, therefore, be

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Figure 1

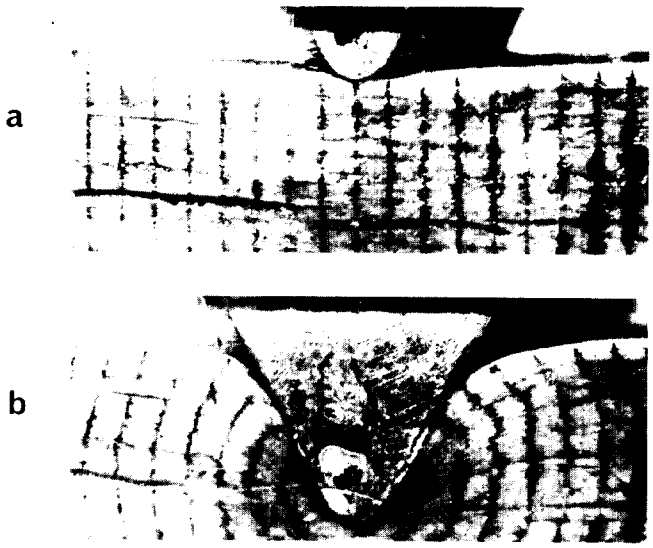

Figure 1. Distortion patterns developed on cleaving cooked ox. M. sternomandibularis. (a) Prior to penetration. (b) About 70% penetration.

absorbed in part in transverse linkages between both muscle fibers and adjacent myofibrils. The more widely the wedge load is dispersed amongst and within muscle fibers, the greater the load will have to be to bring the fibers to their yield point. In other words, tenderness or toughness depends not only on the tensile strength of the individual longitudinal elements of muscle but also on the adhesive strength of the linkages between them (Davey and Gilbert, 1977).

Figure 2

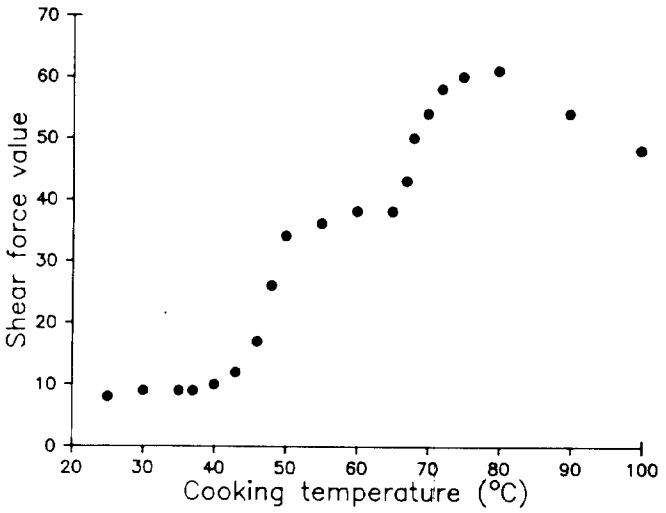

Figure 2. Toughening of ox M. sternomandibularis with increasing cooking temperature. The muscle samples were cooked in a water bath at the prescribed temperature for 40 minutes (after Davey and Gilbert, 1974).

During the cooking of meat, two separate phases of toughening occur (Fig. 2) as temperature increases (Bramblett et al., 1959; Davey and Gilbert, 1974). The first, between 40° and 50°C, coincides with the loss of myosin solubility and is presumed to indicate protein denaturation in the contractile apparatus of the muscle cell. Sarcoplasmic proteins are also largely denatured in this temperature range. The second phase, between 60° and 75°C, is assocated with a 25-30% shrinkage of the muscle fibers, largely through denaturation of connective tissue proteins, which results in shrinkage of the connective tissue. If cooking is prolonged, or the cooking temperature is raised further, the toughening induced in this second phase is overcome through the melting of collagen. Judged by the ease with which muscle fibers separate from each other, the mechanical strength of connective tissue is entirely lost once collagen has been converted to gelatin.

The setting of a muscle in rigor mortis when it is in a shortened state can induce a considerable degree of toughening (Locker, 1960). The force required to shear M. sternomandibularis cooked to 80°C rises three to four fold if the muscle is shortened by 40% of its excised equilibrium length (Marsh and Leet, 1966). With a further reduction in length towards 60% shortening, toughness undergoes an equally sharp decline (Marsh and Leet, 1966). Thus a prominent peak of toughness exists in the cooked muscle, at 40% shortening. It is the rising slope of this curve that will concern us here. The declining slope at higher shortenings is undoubtedly due to widespread breakdown in ultrastructure (Marsh et al., 1974), although why muscle structure should be disrupted at the very low levels of power development during cold shortening (Davey and Gilbert, 1975) is a curiosity worthy of address.

If rigor muscle is aged for a number of days before cooking, it usually becomes appreciably more tender. Shear-force values of unshortened M. sternomandibularis for example, held at 15°C, fall by 60% over a period of 2.5 days, with a slight additional decline on prolonging storage to 8 days. The effect of maximal aging on the shortening-toughening relationship is of considerable importance to our understandings of the aging phenomenon (Davey et al., 1967). Ox M. sternomandibularis that has shortened by no more than 20% tenderizes to a maximum degree, but at higher shortenings the tenderizing falls off, and approaches zero at 40% shortening. In other muscles that have been studied (Herring et al., 1967), the fall off in tenderizing with shortening, although pronounced, is not as marked as in M. sternomandibularis.

The fact that prolonged cooking destroys connective tissue collagen offers a way of identifying structural components that toughen meat due to shortening and tenderize it due to aging (Davey et al., 1976). The relationship between shear-force values and cooking time for ox M. sternomandibularis that has shortened to different degrees is shown in Fig. 3. Virtually all the considerable decline in shear-force values occurs in the first 12 hours of cooking. The muscle that has shortened to about 40% of its equilibrium length remains

Figure 3

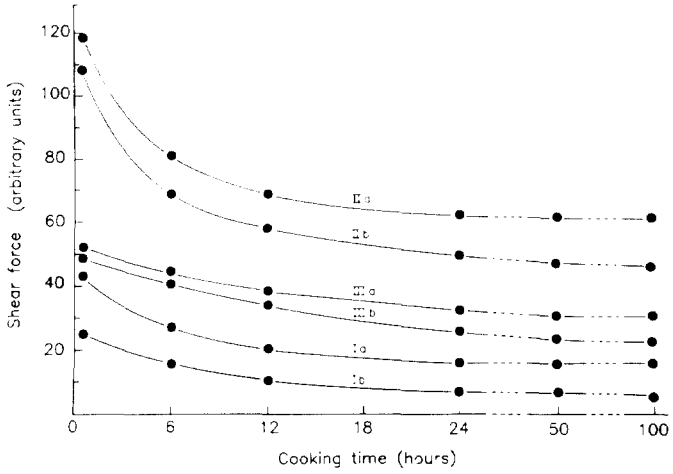

Figure 3. The effect of prolonged cooking on the toughness of unaged and aged ox M. sternomandibularis. Curves I, unshortened muscles; Curves II, muscles shortened by about 40%; Curves III, muscles shortened by about 60%; Curves (a) unaged muscle; Curves (b) aged muscle. (After Davey et al., 1976.)

tougher than either unshortened muscle or muscle shortened by 60%. In other words, despite prolonged cooking, the peaked relationship between shortening and toughening persists, although it is flattened by an approximate halving of shear-force values at all degrees of shortening (Davey et al., 1976). The reasonable conclusion is that the relationship between muscle shortening and cooked-meat toughness is an inherent property of the muscle cell and not of the connective tissue. Furthermore, the tenderizing from aging evident in briefly-cooked muscles is still apparent after prolonged cooking, even though prolonged cooking would have melted the collagen from the meat. This indicates that the majority of the tenderizing of aging also occurs in consequence of changes in the muscle cell and not in the connective tissue.

These simple observations on the shearing of cooked meat give useful ideas on how the toughness of meat changes through post-mortem treatments. In essence they suggest that:

- Toughness is induced — by cell components being interlocked into a tighter structural continuum through muscle shortening; by heat coagulation and denaturation of cellular proteins; and by heat shrinkage of connective tissue.
- Tenderness is induced — by meat aging, which breaks down various structural components within the muscle cell, and by prolonged cooking, which melts out the collagen of the connective tissue.

The Cytoskeletal Framework of the Muscle Cell

Recent evidence has brought to light the existence of a well-defined cytoskeletal framework in the muscle cell (Lazarides, 1980; Robson et al., 1981), separate from the well-characterized sarcoplasmic reticulum and the paracrystalline contractile apparatus. This filamentous cytoskeleton is largely made up of so-called desmin and connectin, although an increasing variety of other proteins seemingly associated with the cytoskeleton are being discovered and characterized (Greaser et al., 1981).

Desmin, sometimes referred to as skeletin or 10 nm filament protein, is a constituent of the salt-insoluble fraction of muscle. Either in isolation (Cooke, 1976; Small and Sobieszek, 1977) or in association with actin (Hubbard and Lazarides, 1979) desmin forms filaments in vitro. These filaments in vivo are thought to have a structural role in skeletal muscle. Immunological evidence (Lazarides and Hubbard, 1976; Granger and Lazarides, 1978) shows that desmin is present in the periphery of each Z-disc, forming a honeycomb network of collars within the Z-plane that presumably maintains the alignment of adjacent sarcomeres and unifies the contractile actions of all the separate myofibrils.

A second protein with a likely structural role in striated muscle is connectin, which has been studied in detail by Maruyama and co-workers (Maruyama et al., 1977; 1979). Immunoglocial evidence suggests that connectin is present throughout the sarcomere of skeletal muscle (Maruyama et al., 1977). Connectin behaves anomalously on SDS (sodium dodecylsulphate) polyacrylamide gels, banding as a doublet at high molecular weights (around 700,000 dalton) (King and Kurth, 1979) while also showing a marked tendency to smear. With this uncertainty as to its characterization, immunological results must be treated with caution.

Several authors have identified an array of filaments within the sarcomere in addition to the thick and thin filaments of the contractile apparatus (for example, see Huxley and Hanson, 1954; Locker and Leet, 1976; Dos Remedios and Gilmore, 1978). Locker and Leet (1976) have described fine filaments (termed G-filaments) seen spanning the gap region opened up between thick and thin filaments in fibers of overstretched beef muscle. These G-filaments are believed to be centered on the Z-discs and to extend out on each side and terminate within the thick filaments of A bands of adjacent sarcomeres. There is recent strong independent evidence for their existence. Strands (5 nm thick) showing a fine regular transverse striation (periodicity 4.2 nm) have now been shown emerging from or attaching to the ends of negatively stained thick filaments isolated from muscle (Trinick, 1981).

Aging and the Cytoskeleton

Desmin

Pronounced structural changes doubtless occur in meat during aging. It is possible, however, that such changes are due to a weakening rather than to a breaking of structural attachments, and therefore distinct and convincing histological changes may not be very obvious. For this reason meat is

best subjected to a period of controlled homogenization to break weakened bonds before histological examination. The muscle-fiber pieces from unaged meat subjected to such controlled homogenization retain their well-ordered state of laterally aligned myofibrils (Davey and Gilbert, 1969; Fukazawa and Briskey, 1969). As aging proceeds, the muscle cell apparently suffers a weakening of its lateral attachments and becomes more susceptible to breakdown during homogenization, shown in the first instance by the loss of precise ordering of the myofibrils and shown after prolonged aging by a complete breakdown to single myofibrils. Further fragmentation into sarcomeres does not happen so easily. Such weakening of lateral attachments, probably at the Z-discs, is the most distinctive physical change of aging, and it alone can be invoked to explain the low stretching modulus of aged meat (Davey and Dickson, 1970).

Despite marked loss of fine detail on prolonged cooking (12 h, 80°C), muscle retains its basic sarcomere structure and, in fact, new structural features are exposed. Prominent amongst these are quite dense bridges linking adjacent myofibrils at the Z-discs (Davey et al., 1976). These bridges are especially evident in unaged meat, which is more open structured. Their intensity and rather regular width tells against their being little more than protein condensed at these positions. They can hardly be confused with the transverse tubular system, which completely disappears from the cooked meat. We believe that these clearly defined lateral links are composed of desmin, probably decorated with denatured sarcoplasmic protein, and that they weaken during aging (Davey and Winger, 1979).

Desmin constitutes about 2% of the myofibrillar bulk and remains in the residue after extraction of washed myofibrils with KI (0.6 M), which solubilizes about 85% of the myofibrillar proteins (Young et al., 1980). Desmin (55,000 dalton) is, however, solubilized by 6M guanidine HCl at pH 8.0. In this respect, two-stage extraction of washed myofibrils with KI and guanidine is a useful way of separating the two sets of proteins — the first that makes up the contractile apparatus (KI extraction), and the second that makes up the cytoskeleton (guanidine extraction). Changes in the protein composition of each fraction during aging can then be followed. Based on SDS gel electrophoresis of guanidine extracts (Fig. 4), desmin progressively disappears during the aging of muscle and is presumed to be destroyed by proteolysis. Although a protein of molecular weight identical to that of desmin appears in KI extracts, it is thought not to be solubilized desmin.

Figure 4

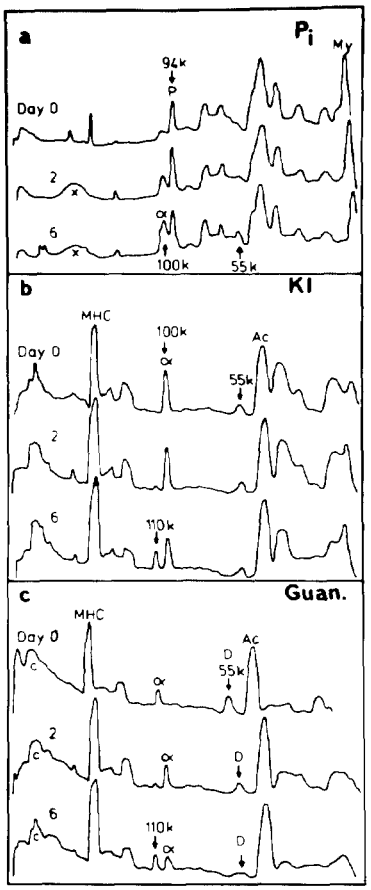

Figure 4. Absorbance traces from Coomassie blue-stained 10% polyacylamide gels, 1% cross-linked. Molecular weights and days of aging are indicated for the three fractions: a, phosphate (Pi); b, KI; c, guanidine (Guan). The two peaks labelled x are artifacts.

Myosin heavy chain (MHc), α-actinin (α), phosphorylase (P), actin (Ac), myoglobin (My), desmin (D), connectin (C). (After Young et al., 1980.)

Connectin

If a muscle is set in rigor mortis while restrained in a fully extended state (twice its equilibrium length), then another distinct structural change associated with muscle aging can be identified (Fig. 5) (Davey and Graafhuis, 1976). Heating fully stretched strips of rigor muscle causes gaps to form at the junction of the A- and I-bands as the A-bands shorten from 1.5 to 1.1 μm. Bridging filaments are then revealed crossing the gaps. Subjecting aged muscle strips to the same treatment causes the A-bands to shorten as before while the I-bands collapse loosely onto the Z-discs; but in this case the much wider gaps that form lack bridging filaments. These bridging filaments, which disappear on aging, are in all likelihood the G-filaments described by Locker and Leet (1976) and are probably constituted of connectin described by Maruyama et al. (1977; 1979).

Like desmin, connectin is refractory and rather insoluble. After myosin and actin, it is the most common of the 20 or so proteins identified in the muscle sarcomere, accounting for about 6% of the myofibrillar bulk (Greaser et al., 1981). On 10% polyacrylamide gels, connectin migrates in the high-molecular-weight region above myosin (Young and Davey, 1981). It appears initially in the guanidine extract but is lost from there with meat aging, possibly to appear in part in the KI extract. Its transfer from the guanidine extract might indicate a solubilization rather than a complete loss through proteolysis. Whatever the case, its disappearance from the extract supports Maruyama and co-workers' observation of low yields of connectin from aged chicken muscle (Maruyama et al., 1977).

Rapid proteolytic degradation of connectin has been shown to occur during the heating of meat at 55°C (King et al., 1981), implying to these authors that connectin contributes little to meat toughness. In contrast, it should be recalled that the aging of meat proceeds at its maximal rate at 55° to 60°C (Davey and Gilbert, 1976), suggesting at least a possible relationship between aging and connectin disappear-

Figure 5

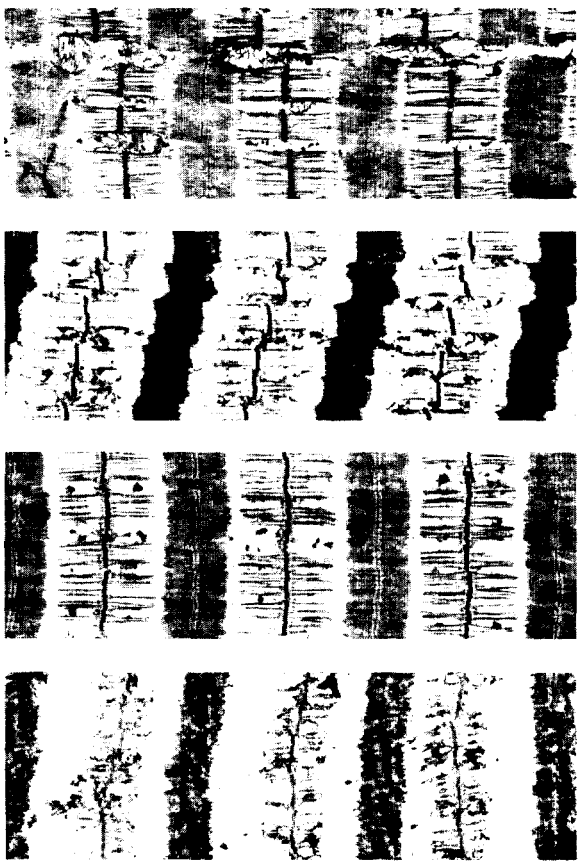

Figure 5. Micrographs of ox M. sternomandibularis stretched and set in rigor at twice equilibrium length. Magnification x10 000. (a) Uncooked; (b) Cooked; (c) Uncooked, aged; (d) Cooked, aged. (After Davey and Graafhuis, 1976.)

ance. Locker (1982) has considerably advanced our knowledge of the G-filaments and particularly their role in determining the toughness of meat. Based on load-extension studies of muscle strips cooked to different temperatures, he regards the G-filaments to be the main load bearers determining the tensile strength of the muscle cell. The G-filaments are vulnerable to a variety of proteases including the endogenous, calcium-activated factors (Dayton et al., 1981a), and their loss through proteolysis is considered to account for the tenderizing of aging.

Aging and the Contractile Apparatus

All who work in the fields of muscle and of meat science would have such a detailed understanding of the contractile apparatus within the sarcomere (Squire, 1981) that there would be no benefit to be gained in its further description.

The Z-Disc

Much past research into aging centered on the Z-disc, since its progressive disappearance was the first clearly identifiable structural change reported to occur (Davey and Gilbert, 1967; Fukazawa and Yasui, 1967). However, until a convincing description of its composition and structure was available, progress has been slow in relating Z-disc disappearance to aging. It appears that α-actinin (100,000 dalton) is the most likely component of the Z-disc to undergo change. The high α-helical content (74%) and axial ratio (∿10) of α-actinin indicate its fibrous character, and this molecule would fit well into the squared-lattice dimensions of the Z-disc (Robson et al., 1981). In unaged muscle, α-actinin appears largely in the KI extract, although there is some carryover into the guanidine extract. As Fig. 4 shows, α-actinin progressively disappears from the KI extract with aging and appears in small quantity in the phosphate-buffer fraction used to wash myofibrils free of sarcoplasm. In accordance with the observations of Chin-Sheng and Parrish (1978), the bonding of α-actinin to the Z-disc structure apparently is weakened during aging, and buffers of low ionic strength are then sufficient to release it into solution. Assuming that α-actinin has a key structural role in anchoring opposing thin filaments from adjacent sarcomeres, its loss from the Z-disc must lead to a considerable fall in myofibrillar tensile strength. Alternatively, if, as Locker believes, the G-filaments serve as the key tensile element in the sarcomere, loss of α-actinin from the Z-disc would be of less significance. Whichever is the case, myofibrillar breakages produced by stretching aged muscle strips occur predominantly at thin-filament insertions with the Z-discs (Davey and Dickson, 1970; Dayton et al., 1981a).

Thin Filaments

There is little doubt that calcium-activated factors attack many of the proteins of the myofibril in vitro. The neutral proteases hydrolyze tropomyosin, troponins I and T, the M and C proteins and probably myosin (Dayton et al., 1981a). In the in situ circumstance, the attack is greatly confined and only the hydrolysis of troponin T to a soluble subfragment (30,000 dalton) has been convincingly demonstrated (Penny, 1980). Because of the easy identification of this subfragment, it has been proposed as a useful index to the progress of aging. In early studies, before the characterization of the troponins, aging was followed by the progressive release of actin (as actomyosin) from washed myofibrils (Davey and Gilbert, 1968). It was shown then that tropomyosin was retained in the insoluble 'ghost'-myofibrillar fraction throughout aging, it appears from this that although troponin T serves as the tropomyosin-binding sub-unit, it does not have a strong role in preserving the structural integrity of the double coil of tropomyosin in the thin filaments.

Thick Filaments

Throughout the history of research into the aging phenomenon, there has been periodic preoccupation with demonstrating that the main site of proteolytic attack is the cross bridges between the thick and thin filaments (Fujimaki et al., 1965; Schwartz and Bird, 1977). With the so-called resolution of rigor, meat loses its stiffness and becomes flaccid, somewhat resembling pre-rigor muscle: it also becomes readily extendible with loads that are little more than are needed to stretch pre-rigor muscle (Davey and Dickson, 1970). However, the similarities between pre-rigor and aged, post-rigor muscle are superficial, for whereas the former is

reversibly extendible, the latter is irreversibly so and on load removal it remains in its fully extended state. In aged muscle, sarcomeres do not lengthen with stretch but break at the insertion of the thin filaments in the Z-discs (Davey and Dickson, 1970). There is contrasting evidence, however, in that during the aging of meat not cut from the carcass, sarcomeres lengthen (Taylor et al., 1972). One can only suppose that the very slow resolution of strains happens not as myofibrillar breakages, but as a lengthening of sarcomeres through withdrawal of thin filaments from between the thick filaments.

Solubilized myosin is readily hydrolyzed by papain, trypsin and chymotrypsin, the most vulnerable site of attack being the region of attachment of the H-meromyosin head to the L-meromyosin tail (Lowey et al., 1967). Despite such vulnerability, there is little convincing evidence that the cross bridges in the aging sarcomere are weakened or broken through proteolytic attack. Most studies are confounded by the apparent paradox that the endogenous enzymes extracted from muscle have a wide capacity to hydrolyse the structural proteins, but only if these themselves have been brought into solution. Presumably there are morphological constraints on the enzymes and their substrates that must be met to bring them into catabolic association. There is intriguing evidence of this. We have irrigated isolated thick filaments with a partially purified preparation of calcium-activated neutral protease. Quite distinctly and remarkably, the thick filaments become visually decorated with the enzyme (Fig. 6). The presence of Ca^{2+} (but not Mg^{2+}) is required, and after a period of incubation the H-meromyosin heads of the filaments seemingly are removed. The decoration is roughly in the form of a squared lattice with a side dimension of about 7.5 nm, indexing at about half the 14.3 nm myosin-head periodicity along the filament. While this may turn out to be a fortuitous aggregation, it does prompt speculation on catabolic control mechanisms in cells. In this case it seems possible that individual molecules, in the process of genesis, are protected from the catabolic enzyme, which must assemble on receptive macrostructures within the cell to carry out its hydrolytic role. A process of Ca^{2+} accretion in the lattice assembly may also occur, resolving the dilemma of how the proteases can carry out their functions in muscle where even in active contraction the concentration of free Ca^{2+} is orders of magnitude below that needed to achieve their maximal activation (Dayton et al., 1981a). There is one significant difficulty: Immunological evidence points to the Z-disc as being the exclusive domain of the calcium-activated neutral protease (Dayton et al., 1981b). However, this observation is not supported by biochemical evidence, which suggests that it is dispersed throughout the sarcoplasm (Penny and Ferguson-Pryce, 1979).

Figure 6

Figure 6. Micrographs of negatively stained thick filaments isolated from chicken M. pectoralis and decorated with a partially purified preparation of calcium-activated neutral factor.

Future Research Trends

Before an entirely convincing concept of causes of meat toughness is obtained, a much clearer view of the connectin-based cytoskeleton is required. As an instance, thick and thin filaments have, respectively, dihedral symmetry about the M-lines and Z-discs, so that the contractile proteins in each half sarcomere can present themselves to one another in the same stearic sense. Nature undoubtedly imposes the same symmetry demands upon the various longitudinal and lateral components of the cytoskeleton. How these demands are satisfied will be the subject of some daunting unravellings in the years ahead. Possibly some of the growing inventory of structural proteins now being identified may be found to serve as cementing agents for the key proteins of the cytoskeleton.

Many aspects of the nature and role of the endogenous proteases in muscle remain unresolved and are likely to remain so until a comprehensive picture of the cytoskeleton is obtained. It is not clear whether the primary attack of such enzymes is on the structural proteins themselves or on the cementing materials that anchor connectin and desmin into the cytoskeleton and that anchor α-actinin into the Z-disc lattice.

It now seems that a process of aging can be triggered in pre-rigor muscles held near body temperature, to overcome the toughening of cold shortening (Marsh et al., 1980). This observation has considerable practical potential and points to the need for a much deeper insight into the very early post-mortem biochemistry of muscle. Recent research demonstrating the generation of myosin subfragments in muscle above 25°C would appear to be related to this phenomenon

Figure 7

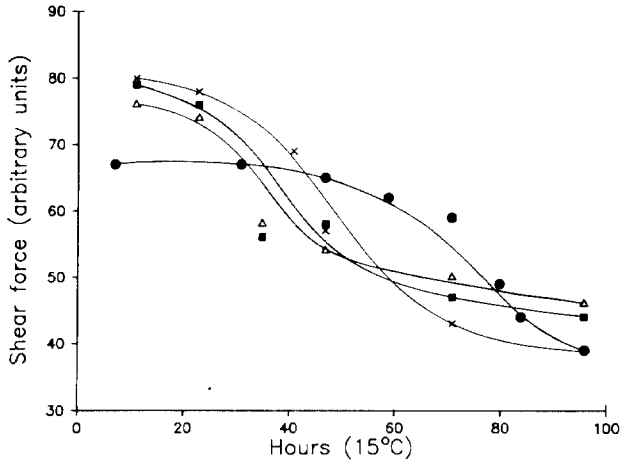

Figure 7. Aging time courses as measured by decreased shear-force values of cooked ox M. sternomandibularis.

(Chin-Sheng and Parrish, 1978; Bechtel and Parrish, 1983). It seems that aging can also be remarkably delayed. We have observed cases in which significant tenderness development in M. sternomandibularis held at 15°C does not begin until almost five days postmortem (Fig. 7), but tenderness is completely developed within the sixth. Such unusual cases offer opportunities for achieving unequivocal correlations between the changes in structure and the increases in tenderness in aging meat.

Aging studies are complicated by the fact that distinct structural changes are usually observed only after associated tenderizing has run its full course. It should be realized that the degradation of susceptible structural components will only lead to an increase in tenderness if the tensile strength of these components is not overridden by the strength of more refractory structural elements. For instance, in shortened muscle the tensile strength of the tight, interlocking coagulum of structural and sarcoplasmic proteins masks that of other structural elements under aging attack. Again, in unshortened muscle, connective tissue can be considered to assume this masking role if the changes of aging have moved so far that further change produces no additional tenderization.

Meat scientists are encouraged to address themselves to the important research now being undertaken into the mechanism of degradative processes in muscle and particularly those related to the turnover of the myofibrillar proteins. We must assume that the turnover rate of individual proteins in life will bear some relationship to the catabolic decay of the proteins in death and as such offer useful insights into the aging phenomenon.

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