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POST-MORTEM CHANGES IN MUSCLE WITH REGARD TO PROCESSING OF HOT-BONED BEEF

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INTRODUCTION

Hot boning of carcasses, which was quite common in earlier times before the era of refrigeration, offers a number of advantages such as facilitation of centralised processing, reduction of cooling space, energy input and chilling time, reduced shrinkage, improved sanitation and shelf live etc. [13, 55, 101]. Furthermore "hot" meat is particularly suitable for the production of emulsion-type sausages [33, 36]. Hot processing of pork was shown to be advantageous with regard to tenderness, and to colour development, colour stability and colour penetration of cured products [13, 55].

Immediately after hot-boning; the muscle is usually still in the *pre-rigor* state; from the biochemical point of view, this state is highly unstable because muscle metabolism is continuing under anaerobic conditions, causing contracture of muscle fibres and the development of *rigor mortis*. The rate of these *post-mortem* changes, which is affected by conditions of storage and processing, does influence important features of meat quality such as tenderness and water-holding capacity. Knowledge in this field is necessary, in order to use optimum conditions for the processing (storage, packaging, freezing, curing, sausage production etc.) of hot-boned meat.

In this paper, *post-mortem* changes in muscle with regard to hot-boned beef (and also lamb) but not to pork are discussed because corresponding knowledge on pork is very limited although a considerable research on the technology of hot-boned pork has been carried out [13, 45, 55, 89, 90, 91]. Much more research must be done to understand exactly the relationship between the *post-mortem* changes in hot-boned porcine muscle and the quality of pork and pork products.

BIOCHEMICAL AND STRUCTURAL CHANGES IN BOVINE MUSCLE *POST-MORTEM* UNTIL COMPLETION OF *RIGOR MORTIS*

CONTRACTURE AND *RIGOR MORTIS*

Immediately after a well-rested animal is slaughtered its muscle contain ATP *), creatine phosphate, and have a pH of 6.9 to 7.2. In living muscle the ATP is continuously turned over to maintain resting metabolism but when the oxygen-carrying blood supply is cut off, the muscle becomes anaerobic and can no longer maintain the level of ATP by oxidative phosphorylation. At first, the level of ATP in muscle is maintained by conversion of ADP to ATP at the expense of creatine phosphate but when the latter is exhausted the ATP level falls. The loss of ATP also triggers the anaerobic conversion of glycogen to lactate with the result that after 24 h the pH falls to about 5.5 (3.81). As a consequence of this process in normal muscle, pH and the glycogen level decrease and the lactate level increases continuously p.m. The ATP level remains rather constant for a certain period until it drops [3, 42, 51, 72] (Fig. 2).

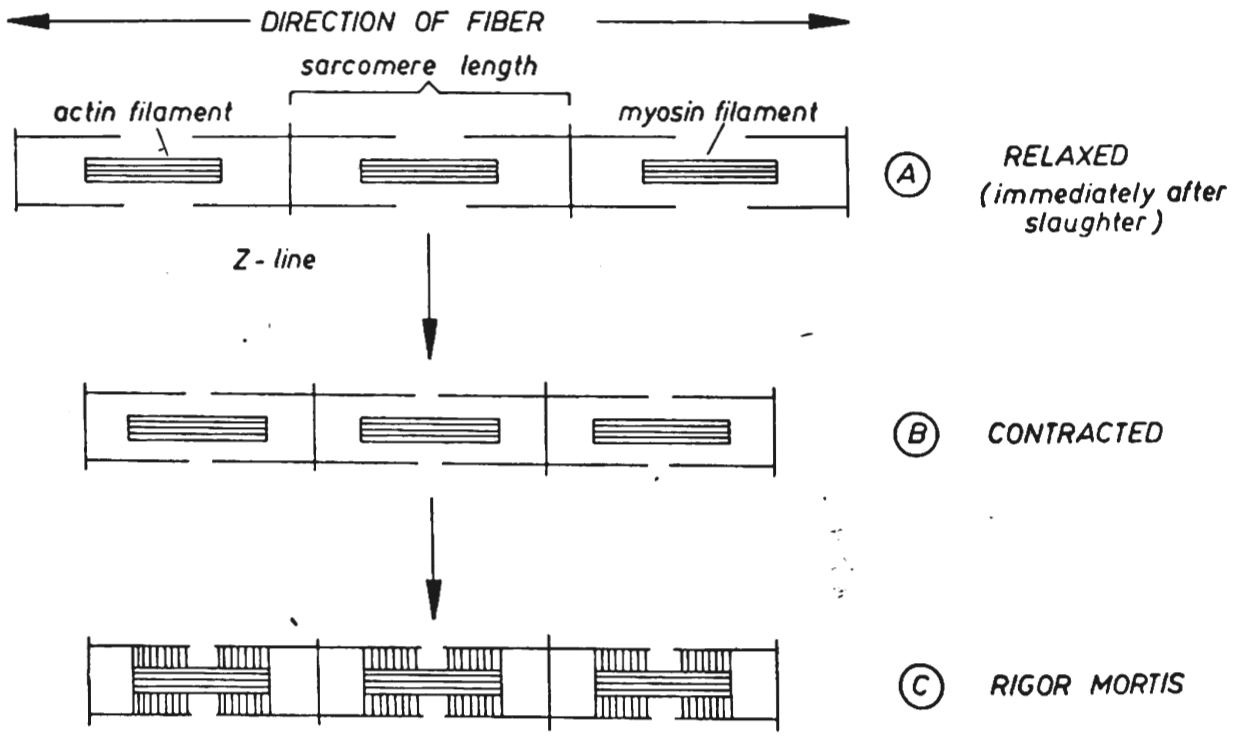

Fig. 1. Scheme of *post-mortem* contracture and *rigor mortis* (from Ref. 42)

After death of the animal, calcium ions are released from muscle mitochondria. As long as sufficient ATP is present, the calcium ions are actively transported into the vesicles of the sarcoplasmic reticulum (SR) by an ATP-driven calcium pumping system localised in the membranes of SR. With a falling level of the ATP the calcium pump becomes inactive and consequently the concentration of free Ca^{2+} around the myofibrils

*) Abbrevations used: ADP = adenosine diphosphate, ATP = adenosine triphosphate, IP = isoelectric point, SR = sarcoplasmic reticulum, p.m. = *post-mortem*, WHC = water-holding capacity.

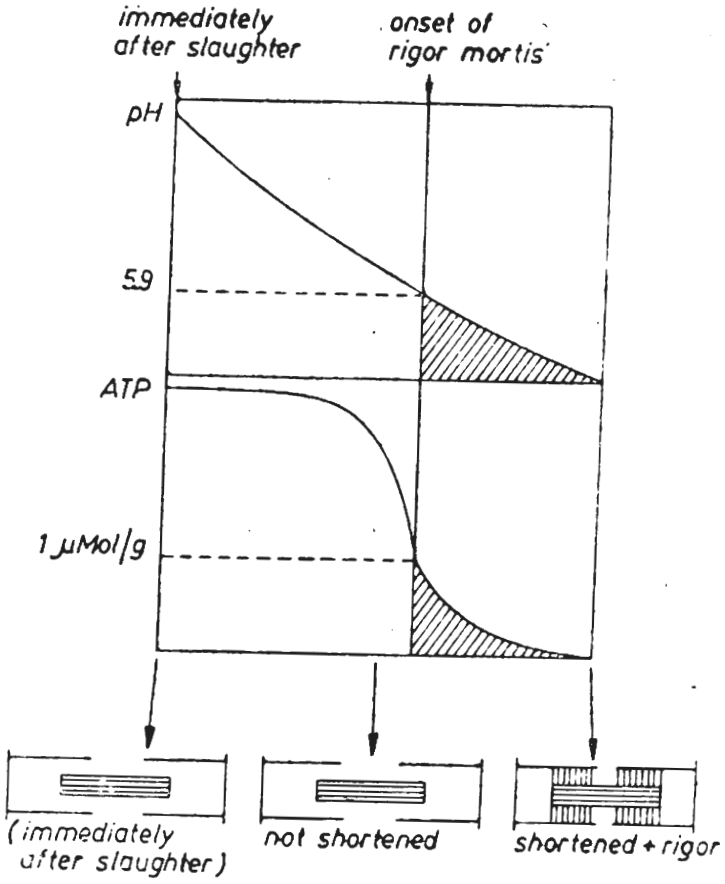

Fig. 2. Scheme of biochemical and structural changes in bovine muscle p.m. at temperature 20°C. The abscissa, indicating the time p.m., has no units because the rate of *post-mortem* changes depends on the temperature (from Ref. 42)

increases. An increase of free Ca^{2+} from about 10^{-8} M to about 10^{-6} initiates contracture by a mechanism probably similar to the stimulation by the nerve which induces contraction in the living muscle (for references see 35, 78).

The most important changes in muscle structure p.m. are contracture and *rigor mortis*. These are two different processes. The thin actin filaments and the thick myosin filaments are arranged along the fibre direction. The thin filaments are connected with the Z-discs which are arranged perpendicular to the fibre direction. The distance between two Z-lines is called the sarcomere length (Fig. 1).

In the resting living muscle as well as in the muscle immediately after death, the interaction between myosin and actin is prevented by the effect of the troponin-tropomyosin protein system which is located in the thin filament. This system is effective as long as the Ca^{2+} concentration in the sarcoplasmic fluid is low (10^{-6} M). The release of Ca^{2+} from the SR caused by neurostimulation of the living muscle or by inactivation of the calcium pump in the muscle p.m., inactivates the troponin-tropomyosin system by binding of Ca^{2+} to one of the three protein moieties of the troponin (troponin C) and, consequently, myosin and actin can react with each other. As a result contraction occurs. During contraction of the muscle fiber the actin filaments glide along the myosin filaments whereby a series of fast interactions between the filaments occurs and the sar-

comere length decreases (Fig. 1). The presence of ATP is necessary for contraction because the energy needed for the sliding process is derived from enzymic dephosphorylation of ATP to ADP. Contrary to the conditions in living muscle, after death the contraction is usually irreversible (contracture). As long as sufficient ATP is present, the myofilaments remain mobile and, therefore, the muscle is extensible (for references see 43). The extent of muscular contracture p.m. can be determined by measuring the decrease of length of the unloaded muscle or by the observation of changes in sarcomere length either under the microscope or with a laser technique.

When the ATP level falls below about 0.1 μmole/g wet tissue, the myosin filaments of the myofibril form bonds with the overlapping actin filaments and the muscle looses its extensibility and goes into *rigor* [3] (Fig. 1). The onset of *rigor mortis*, i.e. the decrease in extensibility, however, starts at a higher ATP level, namely at about 1 μmole/g. At this ATP concentration the pH of muscle has usually reached a value around 5.9 provided that at the time of death the muscle glycogen was at a normal level (about 700 mg/g wet weight) [42, 51] (Fig. 2).

Of course, contracture is not possible after development of *rigor mortis*. It must be realised, however, that *rigor mortis* will not occur in all fibres of a muscle at the same time. Thus, the extensibility of muscle starts at an average ATP level in the tissue of about 1.0 μmole/g (pH 5.9) and decreases continuously until the ATP level has fallen below 0.1 μmole/g (pH 5.5) [51].

INFLUENCE OF CONDITIONING TEMPERATURE ON *POST-MORTEM* CHANGES IN MUSCLE

The rate of *post-mortem* metabolism is strongly influenced by the temperature of conditioning. Lowering the tissue temperature from 37°C (immediately after death) to 6-8°C results in a continuous decrease in the rate of ATP turnover as is to be expected but a further decrease of the tissue temperature, to the freezing point (about −1°C), causes an acceleration of *post-mortem* metabolism in the *pre-rigor* muscle [4, 48, 52, 54, 72, 92] (Figs. 3 and 4).

The phenomenon of accelerated muscle metabolism at lower temperatures is caused by "cold shortening". A fall in temperature in the *pre-rigor* muscle from about +10°C to 0°C causes increased shortening of bovine and ovine muscle [65, 66, 73] (Fig. 6). Cold-shortening is also found to occur in porcine muscle [6, 23] but its onset starts at lower temperatures [23]. Cold-shortening is more marked in so-called red muscle than in the white ones.

The cold shortening phenomenon can be explained by changes at low

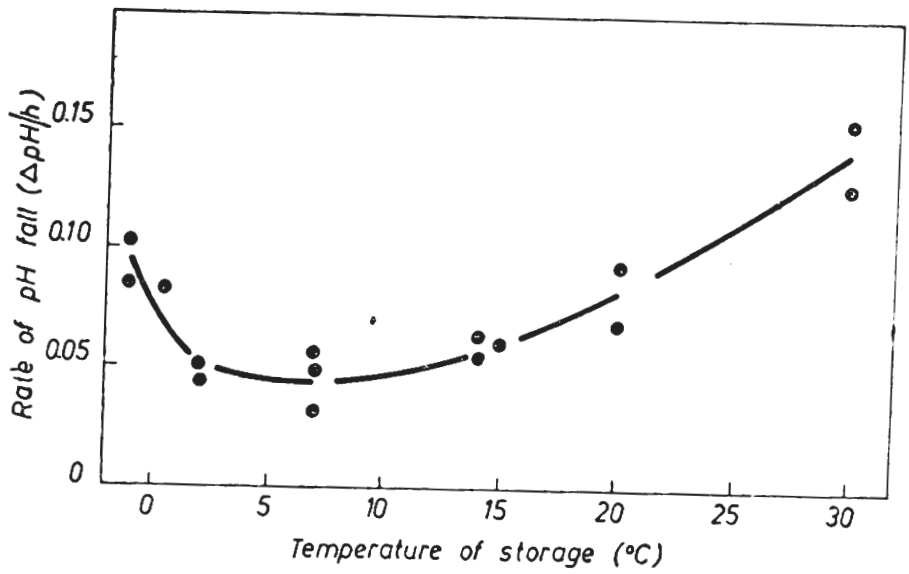

Fig. 3. Rates of pH fall p.m. between pH 6.8 and 6.1 in bovine neck muscles at various temperatures (from Ref. 54)

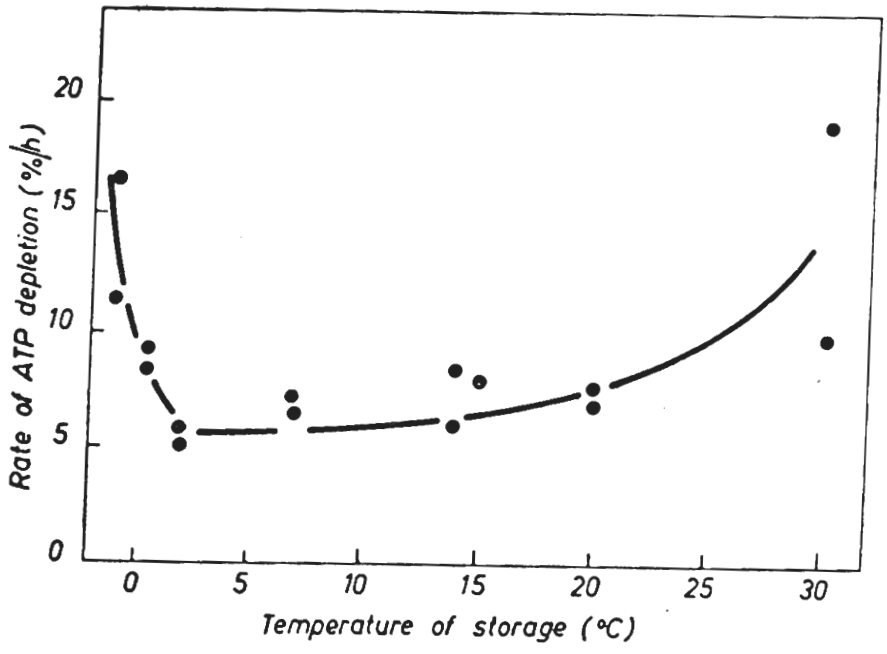

Fig. 4. The rates of ATP depletion in bovine neck muscles held at various temperatures. The ATP concentration in the "delay phase" (first period p.m., cf. Fig. 2) was taken as 100 percent (from Ref. 54)

temperatures in the lipoprotein system of membranes. These inactivate the ATP-driven calcium pump of the SR and/or increase the permeability of membranes of SR or mitochondria to Ca^{2+}. The enhanced concentration of Ca^{2+} in the myofibrillar space, together with still appreciable levels of ATP, initiates muscular contracture before the onset of *rigor mortis* [10, 14, 35, 48, 78, 79]. The energy necessary for the muscle shortening, is supplied by an increased ATP turnover which is accompanied by accelerated glycolysis.

As mentioned above, in bovine muscle *rigor mortis* usually occurs by the time the pH has reached a value around 5.9. Thus the course of pH fall at different conditioning temperatures is of particular interest. As Fig. 5 shows, the rate of pH fall at 0.5°C during the first hours p.m.

is higher than at 7°C or 14°C. This is certainly due to an accelerated turnover of ATP as mentioned above [52] (Figs. 3 and 4). Increased shortening of sarcomeres caused by raising the temperature above 16°C [8, 52] (Fig. 6, upper curve) is preceded by an accelerated decrease of pH during the first hours p.m. [52] (Fig. 5). Contrary to the cold-shortening effect, however, this acceleration of *post-mortem* metabolism, which is also characterised by an increased turnover of ATP [8, 54] (Figs. 3 and 4), is not combined with *pre-rigor* shortening (within the first 3 h p.m.) (cf. Fig. 6, upper curve), probably because insufficient Ca^{2+} is

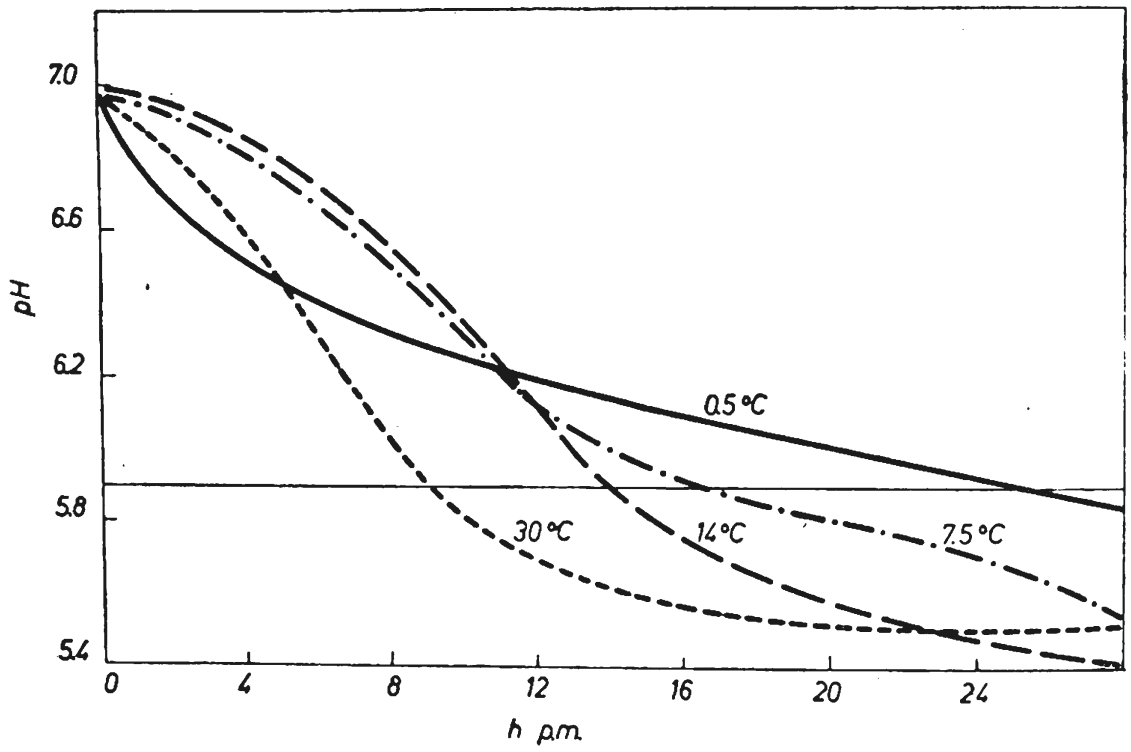

Fig. 5. Relation between the pH fall and the time p.m. in bovine neck muscles at various temperatures (from Ref. 52)

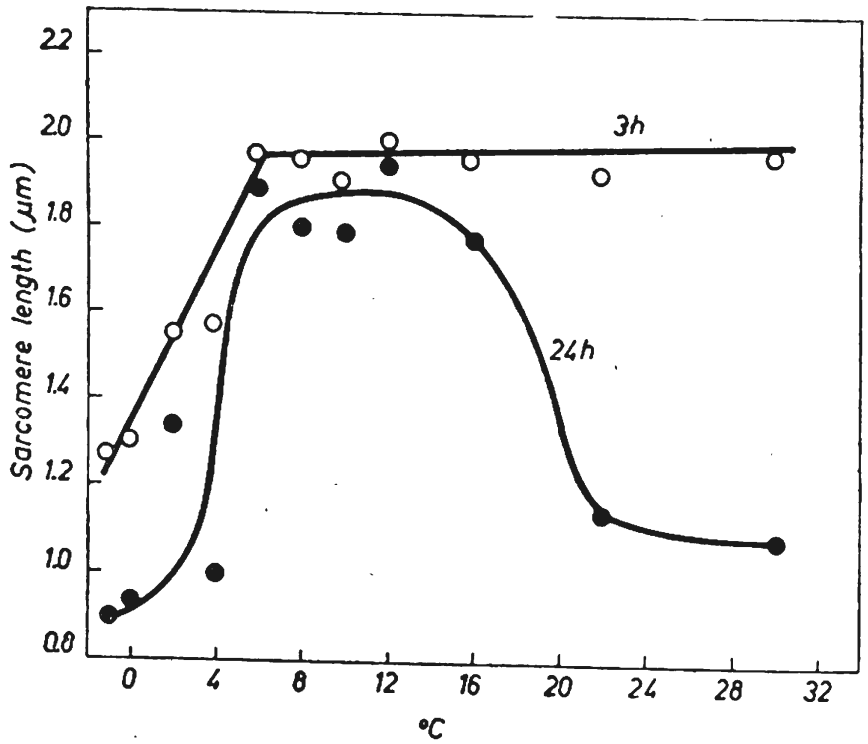

Fig. 6. Sarcomere length of bovine neck muscles at 3 and 24 hours p.m. at different incubation temperatures (from Ref. 52)

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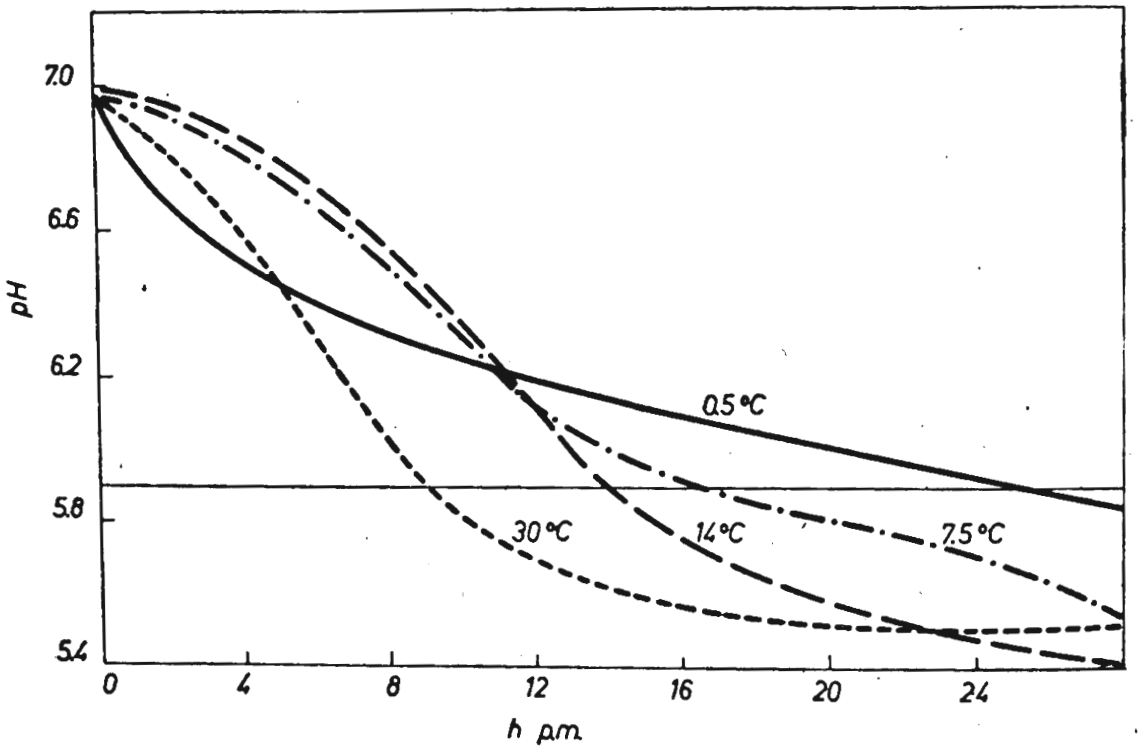

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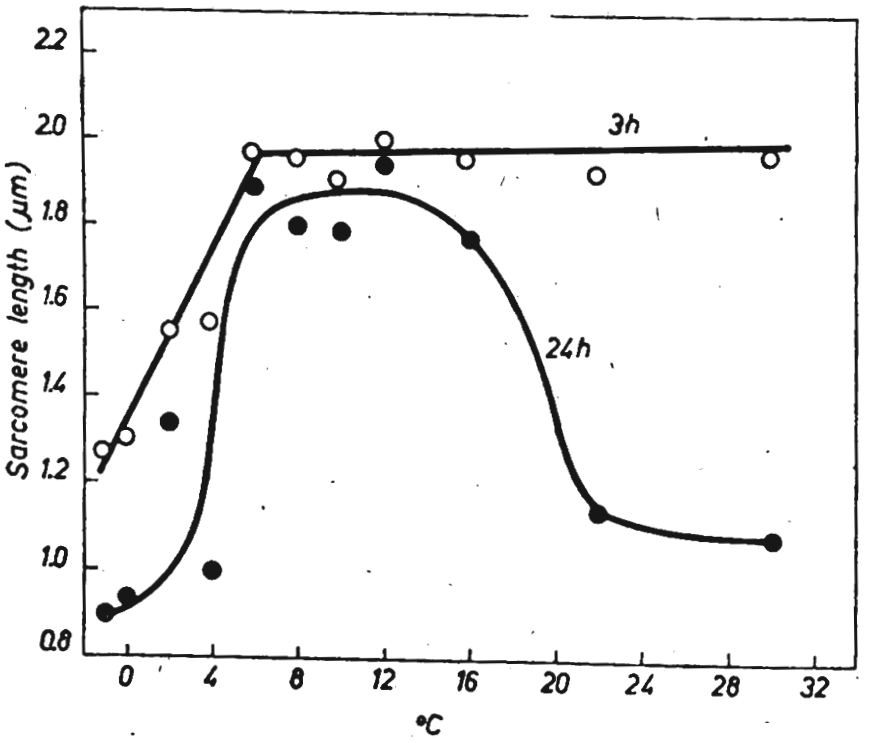

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released from calcium-accumulating organells at these temperatures [48]. Just before depletion of ATP, so-called *rigor*-shortening (contracture immediately before *rigor mortis*) occurs [52, 61] (see also scheme Fig. 2). This *rigor* contracture may well be initiated by the increased Ca^{2+} concentration — itself a reflection of the inability of the system to pump calcium back into the SR since ATP is now scarce.

The accelerated *post-mortem* metabolism at low temperature lasts for only a few hours p.m. Thereafter, further decrease of pH at 0°C is much slower than at 7°C or higher temperatures [52, 102] (Fig. 5). The onest of *rigor mortis*, as measured by the loss of extensibility, occurs in bovine muscle with normal initial glycogen content around pH 5.9 independently of incubation temperature [50, 52], but the time necessary for reaching this pH decreases continuously with rising temperature (Fig. 5). It has been suggested that, after a few hours p.m. at all temperatures between 0°C and 30°C, similar amounts of Ca^{2+} ions are released and that the rate of *post-mortem* metabolism is then determined by the normal influence of temperature on biochemical reactions [52].

INFLUENCE OF CUTTING AND COMMINUTING ON THE *POST-MORTEM* CHANGES IN BEEF

The rate of *post-mortem* metabolism during chilling of carcasses varies considerably between muscles and within muscles [94].

Relatively slow metabolic rates occur in the superficial, rapidly cooling parts of the carcass while in deeper musculature, where cooling is delayed, relatively rapid rates are observed. Hot-boning has a major effect on the rate of metabolism in the muscle p.m. [93]. A relatively uniform rate of glycolysis and ATP-breakdown can be observed throughout the hot-boned muscle compared to the carcass muscles in which the rate of metabolism increases with depth. This, of course, is due to a more uniform temperature distribution in the hot-boned cuts. At cooler-room temperatures below +10°C cold-shortening conditions are reached more easily in the hot-boned beef than in the intact side. For sausage manufactured from hot-boned beef, the influence of comminuting *pre-rigor* beef on the rate of *post-mortem* changes is of practical interest. In the ground muscle, *post-mortem* metabolism is faster than in the intact muscle. It can be shown that grinding *pre-rigor* bovine muscle increases the rate of ATP-turnover manifested by a faster decrease of the levels of ATP and glycogen and an accelerated drop of pH [32]. *Rigor mortis* does occur not only in the intact muscle but also in the fibre fragments of beef ground in the *pre-rigor* state [28, 32, 40]. The same is true for the cold-shortening effect, by lowering the tissue temperature from 10°C to 0°C a continuous acceleration of *post-mortem* metabolism can be ob-

served not only in the intact muscle (Figs. 3 and 4) but also in the comminuted tissue [48]. Therefore, by fast cooling the ground *pre-rigor* beef, the critical level of 1 μmole ATP/g, at which the development of *rigor mortis* starts, may be reached within a few hours p.m. [48]. It has been suggested that the acceleration of *post-mortem* metabolism due to comminution is caused by damage to the sarcoplasmatic reticulum resulting in a release of Ca^{2+} [32]. This view, however, is still questionable [35].

INFLUENCE OF FREEZING AND THAWING ON THE *POST-MORTEM* CHANGES IN BEEF

Under practical conditions cuts of hot-boned meat are sometimes frozen before onset of *rigor mortis*. For this reason the influence of freezing and thawing on *post-mortem* metabolism in muscle tissue is of interest. If muscle is frozen before onset of *rigor mortis*, a severe contracture of muscles fibres occurs during thawing. This "thaw *rigor*" is accompanied by an accelerated breakdown of ATP and glycogen [14, 20, 44, 47, 74] (Fig. 7). Thaw contracture occurs in both red and white muscles. Thaw *rigor* occurs even if only 5-20 percent of the original ATP is present. Only at an ATP level as low as 0.1 μmole/g is thaw *rigor* prevented [14]. Of course, the level of ATP in the frozen tissue depends on the rates of chilling and freezing which is influenced by the size of cuts. With decreasing rate of freezing the ATP turnover and the rate of ATP depletion increase and in comminuted muscle ATP breakdown and

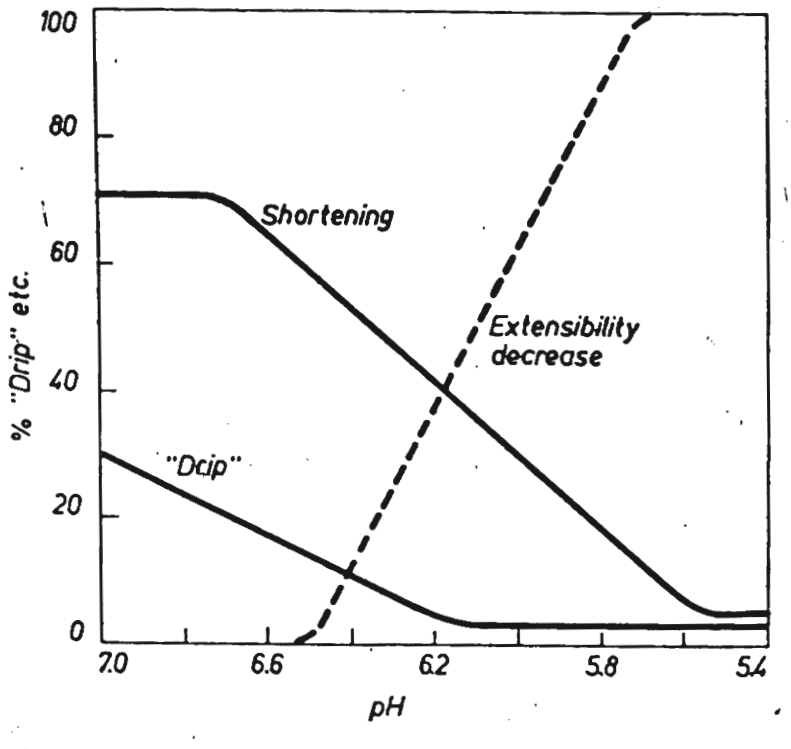

Fig. 7. Thaw-shortening and drip (expressed as percent of initial length and weight respectively) in relation to the pH at which freezing occurs (m. *longissimus dorsi* of lamb) (from Ref. 74)

glycolysis during freezing occur faster than in the intact tissue [21]. Freezing does stop the *post-mortem* metabolism, but only at about −18°C and lower temperatures. Above −18°C increasing temperature of frozen-storage causes an increased rate of ATP turnover and glycolysis which is higher in ground muscle than in the intact tissue [22]. If the ATP concentration in the frozen tissue falls below 1 μmole/g no contracture or *rigor* can occur because they are prevented by the rigid matrix of ice.

It is suggested that thaw *rigor* is caused by a release of Ca^{2+} from SR and/or mitochondria by membrane damage during freezing and thawing [16, 20, 35, 58, 59, 78]. The capacity of the SR for accumulating Ca^{2+} is not lowered by freezing and thawing [100]. But the irreversibility of thaw *rigor* is likely due to the release of such high amounts of calcium ions from freeze-damaged organells that not enough Ca^{2+} can be repumped into the SR in order to prevent fast contracture and — after reaching an ATP level of about 1 μmole/g — *rigor mortis* [20].

During slow thawing of *pre-rigor* frozen beef (e.g. from −20°C to −1°C within 10-12 h) a slow breakdown of ATP and glycogen occurs. If the critical ATP level of about 1 μmole/g is reached while the tissue is still frozen, no contracture of muscle fibres can occur because such contracture is prevented by the rigid matrix of ice. During the process of thawing the lack of ATP causes the normal development of *rigor mortis* without contracture [47]. For similar reasons it is possible to prevent thaw *rigor* by storage of the *pre-rigor* frozen meat for several days at −3°C [5] or for at least 20 days at −12°C [16].

INFLUENCE OF EARLY *POST-MORTEM* CHANGES IN MUSCLE ON THE QUALITY OF MEAT AND MEAT PRODUCTS

TENDERNESS

Shortening of muscle and the development of *rigor mortis* affect the tenderness of meat [71]. *Pre-rigor* meat can be used for the preparation of pre-cooked ready-to-eat, meat-based foods, therefore the question arises in which way the cooking of hot-boned meat soon after slaughter influences the tenderness of cooked meat. Most of the published data indicate that beef cooked in the *pre-rigor* state is more tender than cooked *rigor*-meat [70, 80, 84, 99]. But there are also findings which show the opposite effect of *pre-rigor* cooking [11, 85, 86]. It must be realized that during cooking of *pre-rigor* muscle the rising temperature induces more or less pronounced rigor shortening of muscle (cf. Fig. 6) which could influence the tenderness in a way similar to cold-shortening and results in increased thoughness [85]. It is also conceivable that during fast

heating no *rigor* or *rigor* shortening occurs because the myofibrillar proteins coagulate before the ATP concentration has reached the critical level. In this case the meat can be expected to be more tender than meat cooked after development of *rigor mortis*. But it has also been suggested that a relatively high rate of heating could result in supercontracture of muscle structure and, therefore, an increase in tenderness. So, it can be shown that after rapid heating of bovine muscles soon after slaugter the meat is more tender than after slower heating [9, 18, 85]. Prevention of shortening during cooking led to a greater toughness of beef than in samples cooked free [62]. These results all indicate that hot-boned cuts have to be cooked as fast as possible in order to obtain acceptable tenderness.

If the cross-linking between thin and thick filaments causes the toughening of meat as a result of *rigor mortis*, it is to be expected that with increasing amounts of cross-linkages, i.e. with increasing shortening of muscle, the tenderness decreases. Of all the *post-mortem* changes taking place before or during *rigor mortis*, it is indeed the extent of shortening which is of over-riding importance to meat tenderness [17, 60, 71, 76, 96]. The length/toughness relationship, however, is not a simple one. Experiments on excised muscles have revealed the magnitude of toughening caused by cold-shortening in beef [73] and lamb [67]. The rate of toughening with length decrease is small at first, but rises with increasing shortening until, at about 40 percent change, the meat may be four times as tough as its unshortened control (Fig. 8). With still further shortening, toughness

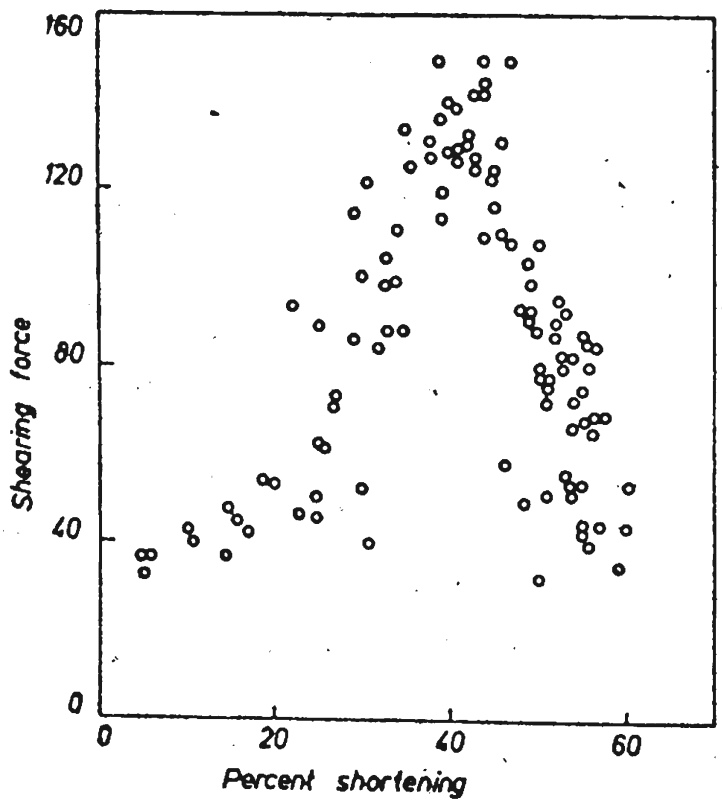

Fig. 8. Relative tenderness in relation to the shortening induced by thawing samples of bovine neck muscle (previously frozen very rapidly at different stages of room temperature *rigor* onset) at several rates. Thaw shortening as percent initial excised length (from Ref. 73)

decreases (Fig. 8) because of a major rupturing of the structure produces alternating zones of supercontraction and fracture [75]. The toughening effect of cold-shortening is not eliminated by *post-rigor* ageing of meat [97].

The shortening which accompanies the rapid thawing of a muscle frozen before *rigor* completion, can produce very appreciable toughening [11, 76]. Thaw-*rigor* becomes even more critical to meat tenderness if meat frozen *pre-rigor* is subjected to cooking from the frozen state [76].

The detrimental effect of cold shortening or thaw rigor on the tenderness of meat can be prevented or reduced by several procedures: (a) by delayed chilling until *rigor* onset has occurred [71], e.g. by storage of the cuts of hot-boned meat at 8-12°C for about 24 hours before the temperature of the chilling room is lowered to 0-4°C [19, 24, 69, 87, 88]. As a general guide in commercial practice, it is considered that cold-shortening is safely avoided if no part of the musculature is allowed to fall below 10°C at least 10 h following death [5, 13], (b) by freezing so rapidly that cold-shortening has no opportunity to take place, and then maintaining a slightly sub-freezing temperature, allowing ATP breakdown to proceed in presence of a restraining ice matrix (see above) [1, 2, 22, 47, 71, 74, 76], (c) by accelerating glycolysis and *rigor* onset so that only a relatively brief pre-chilling delay will be necessary. The simplest way is to elevate the temperature, but bacterial proliferation and the possible heat-shortening make this a hazardous course [71]. A brief application of pressure of about 1000 atmospheres to *pre-rigor* muscle greatly hastens glycolysis and *rigor* onset, and the meat is very significantly tenderised despite immediate post-pressure chilling [57, 68]. The pressure produces shortening of the same order as that achieved during cold-shortening but without the accompanying increase in toughness [68]. More suitable for practical application and already used on an industrial scale is *rigor* acceleration by *post-mortem* electrical stimulation of muscles either in situ or in the excised state [cf. e.g. 7, 46, 98]. The effect of electrical stimulation on muscle metabolism p.m. and quality of meat, however, shall not be discussed in this paper.

Finally it should be mentioned that raising the temperature in cold-shortened beef to 37°C in the final stages of *rigor* completely eliminates the toughening seen in cold-shortened meat without affecting the shortening. The effects are not due to ageing but may arise from modification of actin-myosin bonding [64].

WATER-HOLDING CAPACITY

The influence of the early *post-mortem* changes in muscle excised in the *pre-rigor* state on water-holding capacity (WHC) are of practical

interest with regard to the formation of exudate ("drip") in packaged hot-boned meat cuts, to drip loss during cold-shortening and after freezing and thawing, to the release of juice during cooking (cooking loss) and to the production of sausages from "hot" beef.

UNSALTED MEAT

It has been known for a long time that WHC of beef decrease within the first 24 h p.m. [27, 28, 31]. However, detailed studies on the dependence of WHC of beef on *post-mortem* metabolism and the development of contraction and *rigor mortis* have not been published until recently [51, 52]. It has been shown that the development of *rigor mortis* (pH 5.9), in the excised muscle has no significant effect on the WHC of muscle pieces and of unsalted muscle homogenates [51]. The cooking loss (Fig. 9) as well as the amount of fluid expressible from the unheated tissue [54] decrease slightly and continuously with the *post-mortem* fall of pH. The question arises as to why the development of *rigor* has no significant effect on the WHC of unsalted meat. During the *post-mortem* drop of pH from 7 to 5.9, when the onset of *rigor mortis* occurs, the myofibrillar protein is approaching its I.P., this means an increase of oppositely charged groups and, therefore, an increase of intermolecular ionogenic

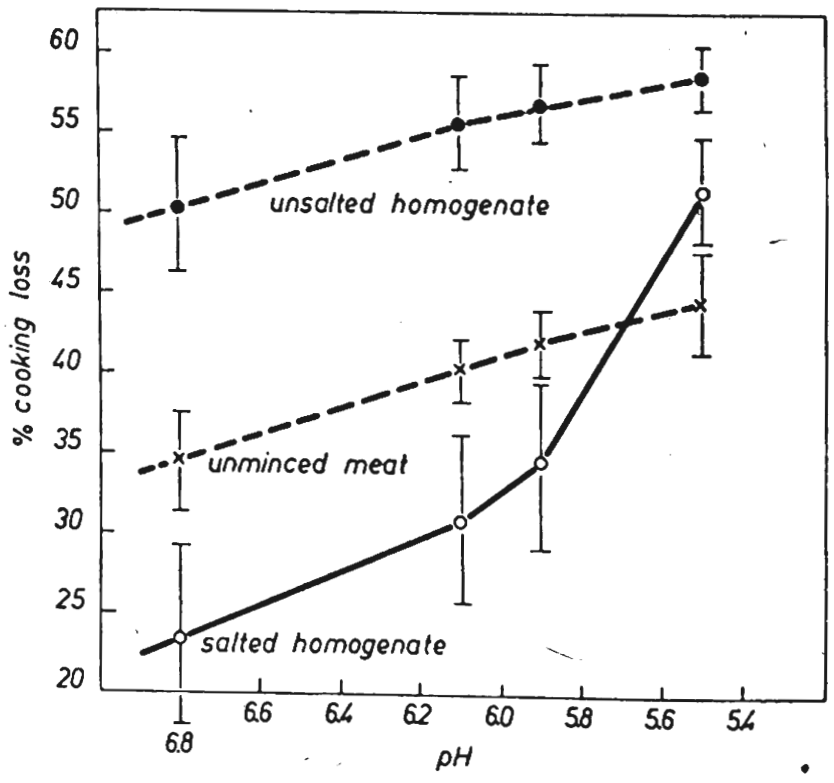

Fig. 9. Cooking loss of intact bovine neck muscles, unsalted and salted homogenates in dependence on the *post-mortem* pH fall in the intact muscle at temperature between 0° and 30°C. The homogenates were prepared after the corresponding pH value in the intact muscle was reached. The bars indicate the standard deviation (including all incubation temperatures) (from Ref. 52)

cross-linkages. Less water can be immobilized in the network of myofibrillar proteins, tightened in this way, than in the looser network existing at higher pH. The result is a decrease of WHC in agreement with the general concept of the influence of protein charges on WHC and swelling of muscle [27, 28, 31]. This pH-dependent type of intermolecular cross-linking is so strong that an additional cross-linking between myofilaments caused by *rigor* development cannot exert an additional significant effect on WHC in the absence of salt [51].

The results of earlier work on the influence of cold-shortening on the WHC of meat have been contradictory [15, 63, 64, 83] but recent studies have clarified this situation [50, 52]. The WHC in terms of cooking loss and juice expressible from the raw tissue, measured 24 hours p.m. in the intact muscle as well as in unsalted muscle homogenates, was not significantly influenced by the temperature of conditioning and, therefore, by the rate of *post-mortem* metabolism. Neither cold-shortening (*pre-rigor* contracture) nor shortening at higher temperatures (*rigor* contracture) exert an effect on WHC of beef 24 hours p.m. [52]. Of course, the WHC decreases faster p.m. if the rate of pH fall increases, but the relationship between *post-mortem* pH and WHC is not influenced by the rate of pH decline [52].

Pre-rigor contracture (cold shortening) has no significant effect, and *rigor* shortening at elevated temperatures only a small influence, on the drip loss within the first 24 hours p.m. However, after a longer period of storage (0-4°C) higher amounts of drip are released from shortened muscle than from normal muscles [50] (Fig. 10). A comparison of Fig. 10 with Fig. 6 shows that the amount of drip loss after 7 days storage increases with the extent of shortening. Apparently, muscle contracture does not result in a significant change of muscle volume until 24-48 h p.m. but then shrinkage occurs which is higher in the more contracted

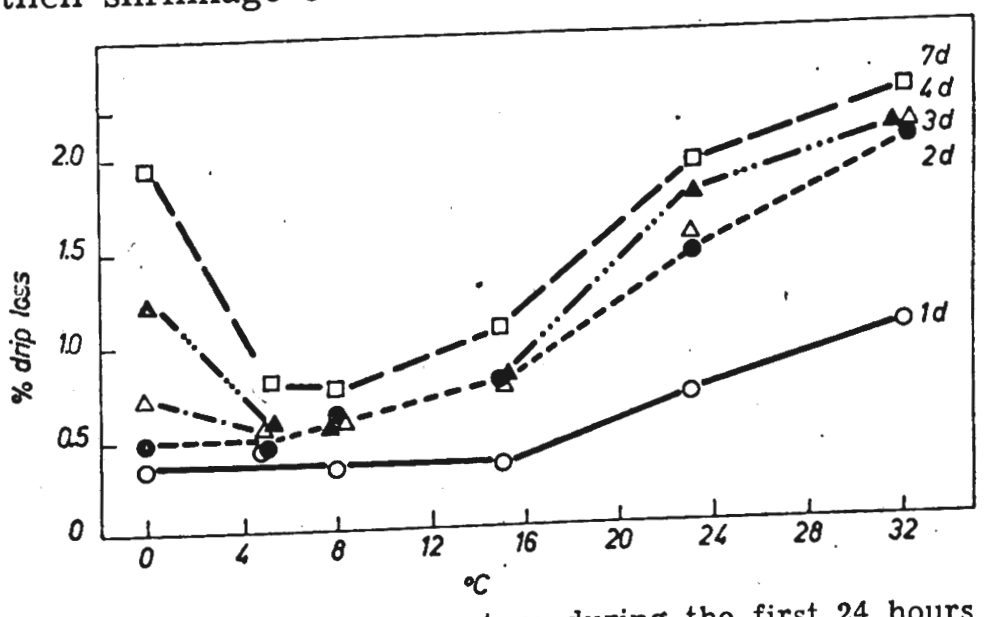

Fig. 10. Influence of incubation temperature during the first 24 hours p.m. on the drip loss from boyine neck muscles during further storage at +4°C for 7 days (from Ref. 50)

muscle. As it is shown below, the increased release of drip is not due to a lower WHC of the myofibrillar proteins in the shortened muscle because the storage of cold shortened muscle for 7 days has no detrimental influence on the binding properties of sausages prepared from such meat [50].

It is interesting that the cooking loss (including drip loss before cooking) of cold-shortened beef measured after 7 days storage at 0°C was about the same as at 48 h p.m. (Honikel et al., unpublished). Apparently the influence of pressure, caused by heat coagulation, on the myofibrillar system is not altered during ageing and must be much higher than the influence of shrinking of the raw tissue during storage. In hot-boned beef, packaged under vacuum, cold-shortening caused an increase in the amount of exudate 24 h p.m. The volume of exudate increased further during storage for 7 days (0°C) (Honikel et al., unpublished).

If pieces of *pre-rigor* meat are cooked a more or less extensive contracture, depending on cooking conditions, can take place as it was explained above. The *pre-rigor* muscle shortens considerably more during cooking than muscle in which *rigor* is established [9, 86] but its cooking loss is less [9, 11, 80, 85, 86]. Similar results have been obtained with ground beef [12]. Presumably both the higher pH and the higher content of ATP of the early *post-mortem* tissue contributes to fluid retention, the two influences being adequate to counteract the pressure which is undoubtedly generated in the tissue by drastic shortening [9].

Thaw contracture causes a remarkable decrease of WHC and a strong drip formation [20, 27, 28, 74] (Fig. 7). The drop of WHC during thaw contracture is determined by shortening of sarcomeres rather than by the development of *rigor mortis* [20]. As mentioned above, cold-shortening does influence the drip formation during longer storage of muscle but not the WHC of the muscle proteins, thaw *rigor*, however, results in a loss of WHC of the myofibrillar proteins because it lowers considerably the WHC of salted muscle homogenates [20, 47] and of sausages frankfurter type [47]. Thus the damage of the myofibrillar protein system seems to be much more severe after thaw *rigor* than after cold-shortening.

The drip formation after fast thawing of *pre-rigor* frozen beef is higher than that obtained after very slow thawing (0°C). The amount of drip increases during storage of the thawed meat and is not higher after slow thawing than the drip obtained after storage of cold-shortened beef [47]. As was mentioned above, slow thawing prevents or reduces thaw-contracture.

All procedures preventing cold-shortening and thaw *rigor*, which were discussed above with regard to tenderness, improve also the WHC of meat. So, cooking losses from hot-boned beef are the same as those

from cold-boned beef — or even lower — if the hot-boned cuts are subjected to delayed chilling procedures [13, 19, 24, 56, 95].

SALTED, COMMINUTED MEAT

Meat in the *pre-rigor* state has a higher WHC and better "fat emulsifying" properties than in the *rigor* or *post-rigor* state. It thus produces sausages of the frankfurter of bologna type with reduced release of moisture and less rendering out of fat when cooked [27, 28, 29, 31, 33]. These superior processing properties are lost during the development of *rigor mortis* but this loss can be retarded for several days by salting the ground *pre-rigor* muscle, or for several months by fast freezing tissue, either salted or unsalted, or even for years by lyophilisation of meat, ground and salted in the *pre-rigor* state [27, 28, 29, 31, 33, 37, 38, 39]. For optimum processing of hot-boned meat it is important to understand the influence of early *post-mortem* changes in muscles on the WHC of comminuted salted meat [36].

Contrary to unsalted meat, development of *rigor mortis* in the intact muscle exerts a substantial effect on the WHC (e.g. cooking loss) of salted muscle homogenates. At the onset of *rigor mortis*, i.e. after reaching pH 5.9, a considerable increase in cooking loss (loss of WHC) [51, 52] (Fig. 9) or of the fluid, which is expressible from the unheated homogenate [54], occurs. Sausages manufactured from *pre-rigor* beef show a significantly lower release of moisture and fat during cooking than those prepared from *post-rigor* beef [34].

Addition of salt to the tissue homogenate causes an increase of WHC (decrease of cooking loss) which is much more pronounced in homogenates with *pre-rigor* muscle than in those with *rigor* or *post-rigor* muscle [48, 51] (Fig. 9). This fact can be explained by the electrostatic theory of swelling [27, 28, 31].

It can be demonstrated that the rather similar decrease of WHC of salted and unsalted muscle homogenates (prepared from intact muscle at different times p.m.), during the *pre-rigor* phase (above pH 5.9) (Fig. 9) is caused by the fall of pH only, and that at least two thirds of the substantial total loss of WHC of the salted muscle homogenates between pH 6.8 and 5.5 p.m. must be due to the development of *rigor mortis* [25, 51]. What is the reason for this effect of *rigor* on salted beef? Addition of salt at pH values higher than the I.P. of the myofibrillar protein causes a strong increase of WHC and swelling of muscle which is related to a shift of the I.P. to lower values [27, 28, 31]. The binding of salt ions increases the electrostatic repulsion between adjacent protein molecules. The resulting loosening of the protein network causes an increase of the immobilisation of water after heat coagulation. The formation of interfilamental cross-linkages during *rigor* will hinder this

swelling effect of NaCl. Therefore, the effect of NaCl in increasing WHC of muscle homogenates or sausage emulsion is diminished with progressive development of *rigor mortis* [51, 52] (Fig. 9).

Similar to the unsalted beef, the WHC (cooking loss) of salted muscle homogenates is not significantly influenced by cold shortening or by *rigor* contracture at elevated temperature (30°C) [52]. But the development of *rigor mortis* in the intact muscle results in a remarkable decrease of WHC of salted muscles homogenates regardless of the temperature at which muscle has gone into the state of *rigor* [52] (Fig. 9).

It can be concluded that longitudinal alterations in muscle fibres as they occur during cold-shortening or *rigor* contracture have much less influence on the WHC of comminuted salted beef than transversal changes as they are caused by interactions between the charged groups of adjacent protein molecules (e.g. effect of pH or ion binding) and particularly by the formation of cross-linkages between the myofilaments during development of *rigor mortis* [52]. It is interesting that the extent of shortening of sarcomeres, i.e. the degree of overlapping of the thin and thick filaments (Fig. 1), does not influence the WHC of salted muscle homogenates [52] or of emulsion-type sausages [50]. Apparently the formation of relatively few cross-links between actin and myosin filaments suffices to decrease the WHC of comminuted salted muscle after onset of *rigor mortis*, the number of cross-links seems not to be of importance.

The detrimental effect of thaw *rigor* on the WHC of salted muscle homogenates and sausages was explained above. This effect can be at least partially prevented by processing (chopping) of the *pre-rigor* frozen beef without prior thawing [28, 47] or by thawing very slowly [47].

The high WHC of "hot" beef can be maintained for several days by coarsely grinding the lean *pre-rigor* muscle, salting with 2 to 4 percent NaCl (or nitrite curing salt) and storing under refrigeration. From such material sausages of the frankfurter or bologna type of excellent quality can be prepared. It is interesting that salt addition to *pre-rigor* tissue causes an irreversible increase of WHC although it accelerates the breakdown of ATP [32]. This can be explained by the exchange of Ca^{2+} from the SR (or mitochondria) against Na^{+} of the NaCl, the calcium ions released accelerate the enzymatic ATP dephosphorylation [32, 49]. This increase in the concentration of free Ca^{2+} is also the reason that storage of *pre-rigor* comminuted and salted beef at temperatures decreasing from 15°C to 0°C does not result in an increase of ATP turnover (no cold-shortening effect) [48]. NaCl also exerts a stimulating effect on *post-mortem* ATP hydrolysis in ground pork [77]. It should be mentioned that the acceleration of ATP breakdown in presence of salt can be observed during freezing of ground beef [21] and during storage of ground beef at freezing temperatures above −18°C [22].

In order to obtain the beneficial pre-salting effect, the salt has to penetrate the tissue before the ATP concentration has fallen to a level at which the onset of *rigor mortis* takes place. Therefore, it is important to salt the "hot" beef either before or immediately after grinding and before cooling the ground material.

The irreversibility of the effect of NaCl on the WHC of *pre-rigor* beef is caused by prevention of *rigor mortis* in the fibre fragments. This inhibition of *rigor* is probably due to a strong repulsion between adjacent protein molecules caused by the combined effect of ATP, high tissue pH and high ionic strength, the result are irreversible changes in the conformation of the myofibrillar proteins [20, 28, 30, 32, 37, 40, 41]. The idea that an irreversible solubilisation of myofibrillar proteins causes the presalting effect is probably not correct [26, 37].

The principle of preventing the *post-mortem* decrease of WHC by salting the comminuted beef in the *pre-rigor* state can be applied also to freezing and to freeze-dehydration. Breakdown of ATP p.m. during freezing as well as during thawing of *pre-rigor* salted and frozen beef and during rehydration *pre-rigor* salted and then freeze-dehydrated ground beef is not accompanied by shortening and *rigor* development, consequently no or only little decrease of WHC occurs [20, 21, 22, 28, 33, 38, 47, 82]. From such presalted frozen or freeze-dried material sausages of the frankfurter or bologna type with very satisfying quality can be manufactured.

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POST-MORTEM ZMIANY W MIĘŚNIACH WOŁOWYCH ODKOSTNIONYCH NA CIEPŁO

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Streszczenie

Wyjaśniono krótko współzależność pomiędzy metabolizmem w mięśniach zachodzącym po uboju (rozpad ATP i glikogenu), skurczem mięśni i stężeniem pośmiertnym występującym w różnych warunkach środowiska. Wyjaśniono również rolę jonów wapnia. Przedyskutowano wpływ zmian pośmiertnych w mięśniach na jędrność i wodochłonność (WHC) wołowiny. Przedstężeniowy skurcz mięśni występujący w niskich temperaturach powoduje wzrost twardości mięsa i w rezultacie wyższe straty wycieku podczas składowania wołowiny w stanie zamrożonym, ale skurcz chłodniczy nie ma ujemnego wpływu na wodochłonność białek mięśniowych i jakość frankfurterów przygotowanych z tego surowca. Stopień skurczu stężeniowego (stężenie w temperaturze powyżej 20°C) wpływa na jędrność i wyciek, ale nie wpływa na wodochłonność białek mięśniowych lub jakość kiełbas. Wpływ obróbki cieplnej wołowiny w okresie przed stężeniem na jędrność i wodochłonność (ubytki termiczne) zależy od szybkości ogrzewania, która wpływa na stopień kontrakcji. Wpływ zmian pośmiertnych w mięśniach na wodochłonność wołowiny niesolonej jest zdeterminowany raczej spadkiem pH niż rozwojem stężenia pośmiertnego. Wystąpienie *rigor mortis* daje jednak znaczne obniżenie WHC w solonej wołowinie i utratę jakości kiełbas kutrowanych. Stężenie rozmrożeniowe, które pojawia się podczas rozmrażania mięsa zamrożonego w stanie przed stężeniem pośmiertnym, powoduje zazwyczaj twardnienie wołowiny i obniżenie WHC mięsa niesolonego (powstawanie dużego wycieku) lub solonego (wędliny niskiej jakości).

Silny skurcz mięśni zarówno podczas rozmrażania lub gotowania może prowadzić jednak do poprawy jędrności. Związane to jest z rozerwaniem struktur mięśniowych. Rozważono metody zapobiegające skurczom chłodniczym i stężeniom rozmrażalniczym (z wyjątkiem stymulacji elektrycznej). Ponadto przedyskutowano korzystny wpływ solenia rozdrobnionej przed stężeniem wołowiny na jakość kiełbas typu frankfurter i bologne i przetworzenie tego materiału w stanie świeżym, mrożonym lub liofilizowanym.