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Determinants of meat quality: tenderness

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Meat quality is a term used to describe a range of attributes of meat. Consumer research suggests that tenderness is a very important element of eating quality and that variations in tenderness affect the decision to repurchase. The present paper highlights recent information on the factors that affect tenderness. While the precise aetiology is not fully understood, a number of factors have been shown to affect tenderness. Of these factors, postmortem factors, particularly temperature, sarcomere length and proteolysis, which affect the conversion of muscle to meat, appear most important. However, it is now becoming clear that variation in other factors such as the muscle fibre type composition and the buffering capacity of the muscle together with the breed and nutritional status of the animals may also contribute to the observed variation in meat tenderness.

Meat quality: Tenderness: Postmortem factors: Muscle fibre characteristics

Meat quality is a generic term used to describe properties and perceptions of meat. It includes attributes such as carcass composition and conformation, the eating quality of the meat, health issues associated with meat such as *Escherichia coli* 0157 contamination and bovine spongiform encaphalopathy, and production-related issues including animal welfare and environmental impact. These factors combine to give an overall assessment of meat quality by the ultimate arbiter, the consumer. The critical point of appraisal of meat quality occurs when the consumer eats the product, and it is this outcome, together with views of colour, healthiness and price, that determines the decision to repurchase (Boleman *et al*. 1997). Hence, consumer evaluation of eating quality is the major determinant of meat quality, with tenderness, juiciness and flavour of meat being the most important elements. However, the main source of consumer complaint and the primary cause of failure to repurchase is the variability in eating quality, especially tenderness (Tarrant, 1998; Bindon & Jones, 2001).

Despite the efforts to control and optimise the peri-slaughter environment (Meat and Livestock Commission, 1991; Tatum *et al*. 1999; Moloney *et al*. 2001), which has a particular impact on tenderness (Ferguson *et al*. 2001), there is still unacceptable variation in eating quality, suggesting that determinants of meat eating quality are multifactorial and complex. This situation is not surprising since muscle is intrinsically a highly organised and complex structure, so that the properties of meat are likely to be determined at different levels ranging from the molecular to the mechanical.

In the light of this complexity and consumers' concerns about tenderness, the present paper will focus on the major determinants of tenderness and overall acceptability, and will consider some of the intrinsic characteristics of muscle that influence these attributes.

The conversion of muscle to meat

It is generally agreed that postmortem events are the main determinants of tenderness. Consequently, while other factors such as nutrition and selective breeding may be used as *in vivo* strategies to optimise meat quality, all efforts may be in vain if, during the conversion of muscle to meat post mortem, other factors are suboptimal.

Early postmortem changes

As muscle is converted to meat a number of metabolic and structural changes occur. In the immediate postmortem period, as the muscle attempts to maintain homeostasis, muscle glycogen is metabolised via anaerobic glycolysis, thus phosphorylating ADP to supply ATP. Anaerobic glycolysis generates lactate that accumulates, lowering the intracellular pH, so that by 24 h post mortem the pH has fallen to an ultimate pH (pHu) of about 5·4–5·7. Muscle is highly sensitive to both ATP and Ca^{2+}, which are both involved in the contraction–relaxation process. Consequently, as ATP levels are reduced and Ca^{2+} levels rise post

Abbreviations: FG, fast-twitch glycolytic; FOG, fast-twitch oxidative glycolytic; GP, glycolytic potential; MMP, matrix metalloproteinases; RN, Rendement Napole mutation; SO, slow-twitch oxidative; pHu, ultimate pH.

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mortem, irreversible cross-bridges form between myosin heads and actin, and rigor mortis occurs in the tissue. Formation of rigor bonds is associated with an increase in toughness. Under normal postmortem conditions these events occur over a period of hours. If, however, carcass temperature falls below 10–15° in the early postmortem period when lactate accumulation has not led to a major reduction in pH (pH approximately 6·0–6·4) and there is sufficient residual ATP, the muscle will shorten and lead to toughening. It is clear then that shortening, and hence sarcomere length, together with temperature are central to the early determination of tenderness (Bruce & Ball, 1990; Geesink *et al*. 2000), and it is suggested that a rigor temperature of 15° has greatest beneficial effects on tenderness (Devine *et al*. 1999). The importance of both sarcomere length and temperature is such that recommended limits for temperature with time, and best practice for carcass suspension to ensure stretching are included in the recommendations set out by the UK Meat and Livestock Commission (1991, 1993, 1995).

pH and drip loss

The rate of pH fall and pHu have major consequences for eating quality, but the precise relationship between tenderness and pH is complex and not fully understood (Van Laack *et al*. 2001). Meat with a high pHu (often >6·5), described as dark cutting or dark, firm and dry, occurs when animals have lower than normal muscle glycogen levels at slaughter and as a result lactate production is low (40 μM-lactate/g muscle at pHu 6·2 compared with 100 μM-lactate/g muscle in normal meat). The tenderness of meat with a high pHu is a matter for considerable debate. Some studies show that it may be more tender than normal (Dransfield, 1981) because the reduction in glycolytic substrate availability causes more rapid ATP depletion and early rigor (the latter reducing susceptibility to cold shortening; Wanatabe *et al*. 1996) and allows prolonged activity of proteases. On the other hand, other studies report that dark, firm and dry beef has higher shear force values (+3·11 kg *v*. 0·63 kg) and is less palatable than normal beef (Wulf *et al*. 2002). In contrast, meat with a low pHu is likely to be of poorer eating quality; the enzymes involved in postmortem tenderization are inhibited by the acidification, and low pHu is also associated with increased drip loss resulting in meat with lower overall acceptability.

There appears to be a strong correlation between the pHu of the muscle and its ability to produce lactate, or glycolytic potential (GP; 2 × (glucose + glycogen + glucose-6-phosphate) + lactate; Van Laack & Kauffman, 1999). Moreover, it has been shown that higher phosphorylase *a* activity is related to lower pHu and higher AMP deaminase is associated with higher pHu, and that a combination of GP and phosphorylase *a* activity explained about 45 % of the variation in pHu (Van Laack *et al*. 2001). These data, while explaining a proportion of the variation, suggest that there is still considerable unexplained variation in pHu that is likely to arise from variation in the intrinsic characteristics and metabolic nature of the muscle fibres.

Muscle fibre types

Interest in the possible association between eating quality and fibre type characteristics has arisen from observations that both variables vary between muscles (Karlsson *et al*. 1993, 1999; Koch *et al*. 1995; Zamora *et al*. 1996; Maltin *et al*. 1997, 1998*a*, 2001*b*; Shackelford *et al*. 1997*a*; Klont *et al*. 1998; Sinclair *et al*. 2001). However, identifying the optimum fibre type composition for best eating quality remains a tough challenge.

The different types of muscle fibre which comprise muscle form and differentiate *in utero* (Maltin *et al*. 2001*a*), so that in the adult fibres can be classified on the basis of their contractile and metabolic activities into at least three groups or types depending on the methods used (Maltin *et al*. 1997; Bee *et al*. 1999; Lefaucheur *et al*. 2002). The most basic classification involves slow-twitch oxidative (SO; type I), fast-twitch oxidative glycolytic (FOG; type IIa) and fast-twitch glycolytic (FG; type IIb) fibre types. However, this classification is limited, and it is important to realise that muscle fibres have dynamic plastic responses, and changes can occur in fibres that affect their phenotypic homeostasis but which may not result in changes to their histochemical or immunochemical reactivity. Discussions in relation to fibre type can then only be taken in the context of the method used for classification. Based on histochemical and immunochemical staining methods it has been shown that fibre type proportions and sizes vary both within and between muscles, but in general glycolytic fibres attain a greater size than oxidative fibres because of the higher requirement for O_2 diffusion of the latter.

Both the contractile and metabolic nature of the fibres may relate to eating quality. Fast-twitch fibres tend to have higher levels of stored glycogen, a more extensive and efficient sarcoplasmic reticulum, with higher amounts of Ca^{2+}-activated myosin ATPase than slow-twitch fibres which have higher numbers of mitochondria, higher concentration of myoglobin and thicker Z lines. Consequently, fast- and slow-twitch fibres have different abilities to generate ATP anaerobically and to sequester and release Ca^{2+}, which may have implications for the onset of cold shortening and rigor. It is not surprising then that it has been suggested that muscles with a high frequency of anaerobic fibres, and hence high GP, have a higher probability of being of poor eating quality due to more rapid accumulation of lactate, decline in pH and a lower pHu (Rosenvold *et al*. 2001; Van Laack *et al*. 2001). However, this speculation is not fully substantiated and relies on substrate availability being the limiting factor, whilst the activity of enzymes such as phosphorylase and AMP deaminase are not considered. Nevertheless, if GP is driven by the metabolic nature of the fibre types then it might be speculated that muscles with predominantly glycolytic fibre types (FG) would be of poorer eating quality than those with a mixed or oxidative character (FOG, SO).

Peri-mortal stress will increase glycogen breakdown through the effects of adrenaline, such that the proportion of glycogen-depleted FG fibres relates to meat quality. Muscles with high numbers of glycogen-depleted fibres have a tendency to be dark, firm and dry with a high initial

pH, or pale soft and exudative with a low initial pH, whereas meat with high numbers of glycogen-containing fibres gives good ultimate quality (Essén-Gustavsson, 1992). It is important, therefore, to reduce peri-mortal stress in order to limit variability in tenderness.

Hence, there is clear evidence of the importance of GP in muscle, but despite the metabolic differences between fibre types with respect to GP, the precise relationships between fibre types and tenderness or overall acceptability are not clear. Several studies of beef, lamb and pork have shown a positive relationship between proportions of SO fibres and either tenderness or juiciness (for example, see Valin *et al*. 1982; Ockerman *et al*. 1984; Maltin *et al*. 1998*a,b*, 2001*b*), supporting the contention that GP may be important. Other studies of beef report associations between proportions of fast-twitch fibres and tenderness (Koch *et al*. 1995) and between increased oxidative status and toughness (Zamora *et al*. 1996). Whereas in pig results of studies on *m. longissimus dorsi* suggest that the proportion of FOG fibres have major effects on toughness (Henckel *et al*. 1997; Maltin *et al*. 1997), and selection for leanness and high numbers of large muscle fibres may select for FG fibres, resulting in poorer vascularisation and aerobic supply, greater postmortem accumu- lation of lactate and reduced pork quality (Karlsson *et al*. 1999).

It would be tempting to speculate as to a possible general relationship between muscle metabolism or fibre type composition and overall acceptability or tenderness, particularly in the light of the difference between breeds and genotype. However, it is clear that no universal relationship between fibre characteristics and tenderness or overall acceptability exists, particularly when between-muscle variations are considered (Fig. 1), and evidence from a number of studies (Morrison *et al*. 1998; Vestergaard *et al*. 2000; Wheeler *et al*. 2000; Maltin *et al*. 2001*b*) shows very clearly that the variation in overall acceptability between muscles cannot be attributed simply to fibre type differences. The relationship between fibre type and tenderness is clearly complex, and it is likely that other variables interact with fibre type characteristics to determine eating quality.

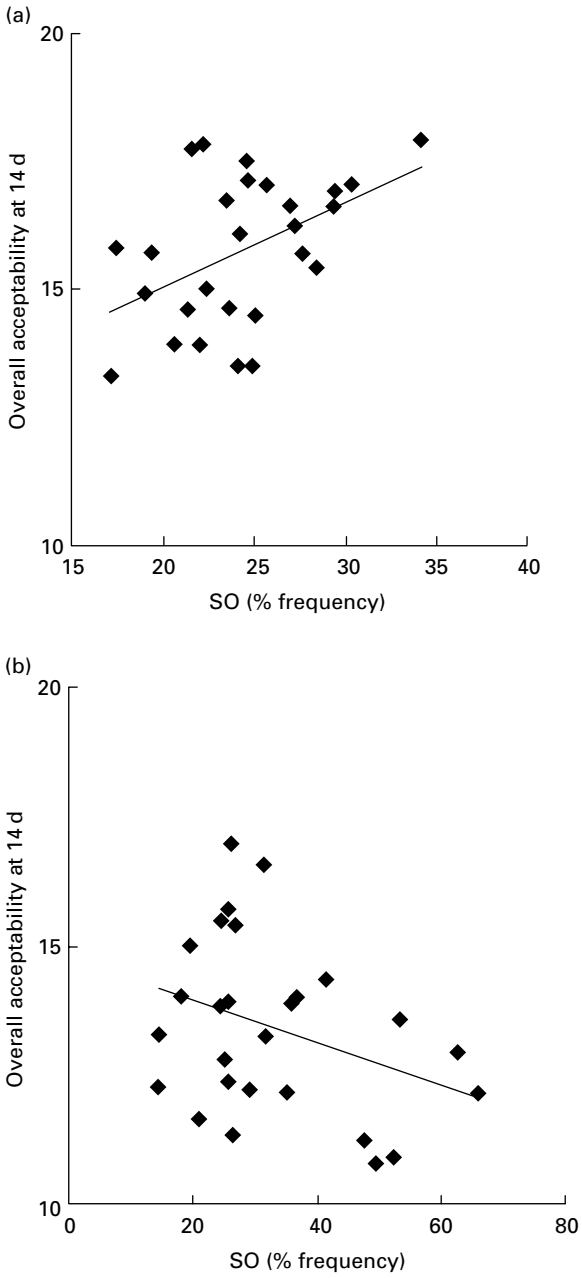

Fig. 1. Regression analysis showing the relationship between slow-twitch oxidative (SO) fibre frequency and overall acceptability at 14 d after slaughter in (a) *m. longissimus lumborum* (R^2 0·2135) and (b) *m. vastus lateralis* (R^2 0·1142) from beef cattle. Both muscles measured were obtained from the same group of animals to reduce genetic variation. (Some of the data are taken from Maltin *et al*. 2001*b*.)

Buffering capacity

One source of variation in tenderness could arise from variation in the buffering capacity within the different fibre types. The major buffering components in skeletal muscle are phosphates, proteins and histidine-related compounds, which are all found at different concentrations in pigs, cattle and chickens (Abe, 2000). The histidine-related compounds carnosine and anserine are found at relatively high levels in muscle, with levels in man and animals being higher in fast-twitch muscles than in slow-twitch muscles (Abe, 2000). Indeed, in horses and camel levels of carnosine are significantly higher ($P < 0{\cdot}05$) in FG fibres than in SO fibres, leading to the conclusion that carnosine could act to provide the greater buffering capacity required in anaerobic fibres (Abe, 2000).

Very little is known about the role of histidine-related compounds as buffers in meat-producing species. In a recent study in which pure-bred Charolais and Aberdeen Angus steers were fed to achieve either moderate or high rates of growth (Sinclair *et al*. 2001), samples of *m. longissimus lumborum* were analysed for their contents of histidine-related compounds. The data show that levels of carnosine in *m. longissimus lumborum* were significantly lower ($P < 0{\cdot}05$) in Aberdeen Angus steers compared with Charolais steers; anserine levels did not differ significantly between breeds (C Maltin, unpublished results). This finding is interesting because *m. longissimus lumborum* from Aberdeen Angus steers had significantly higher ($P < 0{\cdot}05$) proportions of SO fibres (and hence lower

proportions of fast-twitch fibres; Maltin *et al*. 2001*b*) and the meat was significantly more tender ($P<0{\cdot}05$) than that from Charolais after ageing (Sinclair *et al*. 2001). These data support the proposed relationship between anaerobic fibres and high levels of carnosine but do not support a relationship between carnosine levels and tenderness. Indeed, given that there is evidence that carnosine in muscle is associated with proteins, its role as a mobile proton buffer therefore being impeded, it is more likely that carnosine performs one of its other roles, i.e. as a hydrophilic antioxidant, a metal chelating agent or a regulator of enzyme activity (Boldyrev & Severin, 1990).

Postmortem proteolysis

The conversion of muscle to meat entrains changes in tenderness due to changes in the properties (mechanical) of muscle fibres and connective tissue. Initially, toughness increases into rigor, then as proteolysis progresses and rigor is resolved, tenderness increases during ageing (Taylor *et al*. 1995). There are several endogenous proteolytic systems in muscle, including the cathepsin–lysosomal system, the ATP-dependent ubiquitin–proteasome system, the calpain–calpastatin system and the matrix metalloproteinases (MMP). In relation to postmortem proteolysis there is little evidence that the ubiquitin system is involved in the ATP-depeleted tissue, nor do the lysosomal enzymes appear to be active, even when there is a rapid acidification of the tissue (Marsh *et al*. 1988).

Studies over the last 20 years have suggested that tenderization is primarily a result of calpain-mediated degradation of myofibrillar and cytoskeletal proteins (Koohmaraie, 1992, 1996; Taylor *et al*. 1995; Boehm *et al*. 1998; Wheeler *et al*. 2000). The calpain system comprises two ubiquitously-expressed isoenzymes μ- and m-calpain (activated at micro- and millimolar levels of Ca^{2+} respectively; Table 1), the muscle-specific homologue p94 and the calpain-specific inhibitor calpastatin (Sorimachi *et al*. 1997), all of which appear to be involved in a range of cellular functions *in vivo*. There is rapid autolysis of μ- and m-calpain on incubation *in vitro* in the presence of Ca^{2+}, which reduces the Ca^{2+} requirements for half-maximal activation, as shown in Table 1.

Despite the extensive evidence implicating calpains as central to tenderization, there is little consensus as to which of the isoenzymes is involved or which proteins are targeted by the calpains. Nevertheless, it is broadly agreed that μ-calpain, rather than m-calpain, plays the primary role in tenderization (Boehm *et al*. 1998; Ilian *et al*. 2001; Veiseth *et al*. 2001; Kanawa *et al*. 2002). However, recent data have added further complexities to the proposed roles for the calpains. The data suggest that most proteolysis occurs between 3 and 14 d post mortem, when the activity of μ-calpain is very low, and that μ-calpain may be bound to the myofibril and inactivated during postmortem storage (Boehm *et al*. 1998; Delgado *et al*. 2001), indicating that m-calpain (or other proteases) are active at this time.

It is clear that both calpains are active at physiological levels of Ca^{2+}, which are much lower than those used to assay calpain activation *in vitro*. Moreover, levels *in vivo* vary between muscle fibre types; in slow fibres levels are 100–300 nM, in fast fibres resting levels are 50 nM but transiently reach approximately 1 μM during motor nerve activity. This observation suggests that either *in vitro* estimates of requirements for half-maximal activation are misleading, or *in vivo* Ca^{2+} levels activate the proteases because cofactors or activators, or mechanisms such as autolysis, alter their Ca^{2+} sensitivity for activation (Suzuki & Sorimachi, 1998). If the proposed activators or mechanisms for activation are present post mortem then the apparent activation of the calpains at physiological Ca^{2+} levels may question the long-held assumption that postmortem Ca^{2+} concentration is too low to activate m-calpain. Moreover, since free Ca^{2+} levels rise from 0·1–0·2 μM to >100 μM as the muscle enters rigor (Jeacocke, 1993) and continue to rise to as high as 970 μM by 10–14 d post mortem (Parrish *et al*. 1981), it is likely that these levels would be sufficient to fully activate m-calpain. In addition, as the enzyme loses little of its activity with postmortem storage, it is conceivable, therefore, that to date the relative

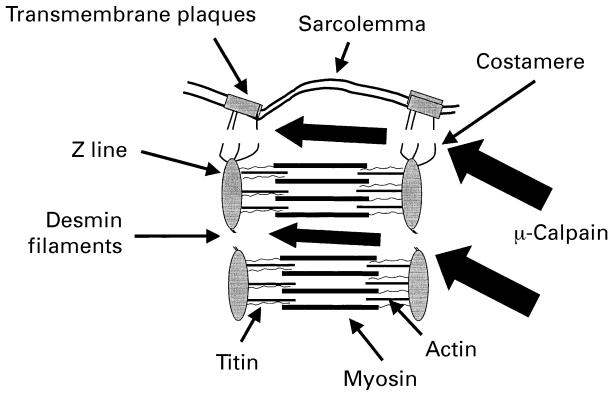

Fig. 2. A schematic representation showing the major structural components of the sarcomere and the proposed sites of cleavage by μ-calpain (←). (Adapted from Oddy *et al*. 2001.)

Table 1. General properties of the calcium-dependent proteases μ- and m-calpain (taken from Goll *et al*. 1992; Sorimachi *et al*. 1997; Suzuki & Sorimachi, 1998; Goll, 2000; Cottin *et al*. 2001)

	μ-Calpain	Autolysed μ-calpain	m-Calpain	Autolysed m-calpain
Molecular mass	110 118	99 292	108 304	98 530
Large subunit	81 890	78 914	80 006	78 152
Small subunit	28 316	20 396	28 316	20 396
pH optimum	7·5	7·6	7·6	7·4
Ca^{2+} concentration for half-maximal activity (μM)	1–50	0·5–2·0	200–1000	30–150
Ca^{2+} concentration for binding calpastatin (μM)	42	0·042	400	24
Theoretical isoelectric point	5·36	5·31	4·94	4·90

contribution of m-calpain activity to postmortem proteolysis has been underestimated.

Clearly, further understanding of the roles of the calpains and their activation post mortem is needed. However, on balance it would appear that μ-calpain is active in the early stages of proteolysis, acting on the costameres, intermediate filaments and the sarcolemmal membrane in the first 1–2 d post mortem (Fig. 2), and as Ca^{2+} levels rise m-calpain becomes activated, leading to further degradation of cytoskeletal proteins over the subsequent days. The role of calpastatin in this scenario remains to be elucidated, but the postmortem fall in calpastatin activity due to proteolytic cleavage would only serve to augment the proposed action of m-calpain (Doumit & Koohmaraie, 1999).

A role for p94 in postmortem proteolysis has also been proposed due to its affinity for titin at the N_2 line, a proteolytically susceptible site linked to tenderization. Studies in porcine muscles of divergent composition showed a positive correlation between p94 expression levels and FG fibres (Jones *et al*. 1999), but subsequent work only in *m. longissimus* provided no evidence that variability in p94 abundance explained variation in tenderness (Parr *et al*. 1999). In contrast, a study of calpain gene expression in various muscles from cattle and sheep suggests that between-muscle variation in tenderness in both species may be partly explained by variations in the expression of p94 (Ilian *et al*. 2001).

Cytoskeletal proteins, including titin and desmin found in the costamere, transmembrane plaques and in association with the Z line, have been shown to be substrates for the action of both μ- and m-calpain (Koohmaraie *et al*. 1991; Taylor *et al*. 1995). Indeed, evidence suggests that desmin cleavage in the costameres by calpains is an early postmortem event that is important in the development of tenderness, increased water-holding capacity and hence juiciness (Kristensen & Purslow, 2001). It is suggested that water appearing as drip has been transported to the extracellular space via the conduit formed by the costameres, and that in pork proteolysis of these cytoskeletal proteins reduces this movement of water to the extracellular space, thereby improving the water-holding capacity in storage (Kristensen & Purslow, 2001) and hence the quality of the meat.

Analysis of desmin degradation shows variation in rates both between species (Koohmaraie *et al*. 1991) and within species between different muscles (Wheeler *et al*. 2000). The variation is quite striking, with a mean degradation of about 40 % in *m. longissimus* compared with 0·2 % in *m. triceps brachii* and *m. semitendinosus* in the pig (Wheeler *et al*. 2000). It is very tempting to assume that variation in desmin degradation could account for the variation in eating quality; however, the data show that this is not the case, with *m. semitendinosus* and *m. triceps brachii* having significantly higher ($P < 0{\cdot}05$) mean tenderness scores than *m. longissimus* (Wheeler *et al*. 2000). Clearly, other factors are important, and the authors (Wheeler *et al*. 2000) highlight that, in addition to proteolysis and sarcomere length, collagen content (and the interactions between these factors) may be an important determinant of tenderness in individual pork muscles.

Connective tissue

As an animal matures to slaughter weight, fibre hypertrophy is accompanied by maturation of the endomysium, perimysial thickening and the formation of non-reducible cross-links between the collagen molecules (Robins *et al*. 1973). Studying these changes in pigs Fang *et al*. (1999) have concluded that the thickening of the perimysium during growth is closely related to the increase in toughness of pork during growth. Surprisingly few recent studies have examined collagen degradation in relation to tenderness, but it is possible that the MMP are important. MMP are a family of secreted enzymes that are regulated through transcription, zymogen activation and tissue-specific endogenous inhibitors and are present and active in muscle both *in vivo* and post mortem (Balcerzak *et al*. 2001; Sylvestre *et al*. 2002). MMP activity appears to vary with extremes of growth rate, such that in rapidly-growing lambs (approximately 275 g/d) compared with slow-growing lambs (approximately 25 g/d) there was a higher proportion of soluble collagen and free hydroxyproline (an indicator of collagen metabolism; Sylvestre *et al*. 2002). Moreover, there was a significant increase ($P < 0{\cdot}05$) in free hydroxyproline between slaughter and 21 d post mortem in the rapidly-growing lambs, suggesting that MMP activity persisted post mortem (Sylvestre *et al*. 2002). These findings are important because generally collagen degradation post mortem is quite limited (1–5 % total), and if collagen content and degradation are important determinants of eating quality, then the opportunities to increase collagen degradation through manipulation of the MMP could be considerable.

Selective breeding and genotype

Modern breeding strategies, particularly in pigs and meat-type chickens, have been targeted to achieve maximum lean muscle mass in a cost-efficient manner. Postnatally, muscle mass depends on muscle protein accretion, determined by the balance between the fractional rates of protein synthesis and protein degradation. In adult animals the protein accretion rate slows, but in muscle predominantly comprising FG fibres net accretion rates remain positive, while in those comprising large numbers of SO fibres rates tend to become negative (Garlick *et al*. 1989). The limitation on the growth of oxidative fibres is presumably related to the requirements for O_2 delivery to the centre of the fibres. Thus, it is not surprising that selection pressures for high lean growth and increased muscle mass select for animals with high numbers of fast fibres rather than slow fibres (Maltin *et al*. 1997) and FG fibres in particular (Brocks *et al*. 2000; Wegner *et al*. 2000), and in many cases this type of selective breeding has compromised both muscle colour and eating quality, particularly tenderness (Karlsson *et al*. 1999; Oksbjerg *et al*. 2000; Lonergan *et al*. 2001).

Examination of the changes in porcine muscle following generations of domestication or selective breeding gives some interesting insights. For example, comparison of wild boar (Sus *scrofa scrofa*) with domesticated German Landrace (Sus *scrofa domesticus*) showed, as expected, that the domesticated pigs had higher growth rates and larger

Table 2. Areas (% total) of fibre type in longissimus muscles from wild, semi-wild and domesticated pigs (taken from Solomon & West 1985; Rahelic & Puac, 1981; Fiedler *et al*. 1998)

	Area (% total)		
Type of pig	SO	FOG	FG
Wild boar: Hunted	100	0	0
Farmed	44	16	40
Trapped	35·8	14·4	49·8
Semi-wild	16·9	9·4	76·4
Large white	7·2	8·7	84·1

SO, slow-twitch oxidative; FOG, fast-twitch oxidative glycolytic; FG, fast-twitch glycolytic.

muscle fibres, with higher numbers of FG fibres in both *m. longissimus* and *m. psoas major* (Bader, 1983).

Similarly a range of studies (Table 2; Rahelic & Puac 1981; Essén-Gustavsson & Lindholm, 1984; Solomon & West, 1985; Fiedler *et al*. 1998) show that selection pressure for fast lean growth leads to a decrease in the proportion of slow-twitch fibres and an increase in the overall mean fibre diameter (Rahelic & Puac, 1981) consistent with a shift towards a fast-twitch fibre profile and the observed reduction in capillarization (Essén-Gustavsson, 1992; Karlsson *et al*. 1999), further supporting the contention that selection for lean growth results in muscles with larger fibres and higher proportions of glycolytic fibres. These observations are also consistent with the data from a comparison of Duroc and Large White pigs. Duroc pigs are less 'improved' for carcass quality than the British White pig, and in general yield fatter carcasses with a lower lean meat yield than Large White pigs. A study comparing pigs with 0·50 Duroc inclusion level with pigs with no Duroc inclusion showed that despite lower proportions of muscle and higher levels of fat in the carcass, the animals with 0·50 Duroc inclusion level produced meat of higher eating quality (Blanchard *et al*. 1999*b*). Similar results were found for pure-bred animals (Čandek-Potokar *et al*. 1998). Interestingly, Duroc pigs have been shown to have higher proportions of SO fibres in *m. longissimus* (Maltin *et al*. 1998*b*), consistent with the proposed higher oxidative capacity indicated by the higher haem content. Moreover, these SO fibre characteristics show Mendelian inheritance (Maltin *et al*. 1998*b*), further supporting the contention that selective breeding for a lean carcass may select against SO fibres and favour FG fibres, with possible penalties for meat quality. In cattle few studies have compared pure-bred animals, but in a recent study where Aberdeen Angus, Charolais and Holstein steers were compared, tenderness and overall acceptability assessments by a trained sensory panel were highest for Aberdeen Angus for all muscles tested (Sinclair *et al*. 2001). In addition, it was found that of the three breeds the proportion of SO fibres was highest in Aberdeen Angus; the difference being significant ($P < 0{\cdot}05$) compared with values for Holstein steers (Maltin *et al*. 2001*b*).

These data raise the questions of whether these breed differences in cattle selected for different purposes (milk *v*. meat) are real or meaningful, and whether (and how) selection strategies, albeit not for lean mass in this case, may have led to altered proportions of muscle fibre types.

A number of gene mutations affect meat tenderness. Double muscling in cattle such as the Belgian Blue, arises from a mutation in the myostatin gene (McPherron & Lee, 1997). Myostatin, a member of the transforming growth factor β superfamily of secreted growth and differentiation factors, is inactivated by the mutation. This effect leads to an increase in both hyperplasia and hypertrophy, and in particular the number and size of the FG fibres appear to be increased, suggesting that the mutation particularly affects secondary myogenesis (Maltin *et al*. 2001*a*). Meat from cattle expressing this mutation appears to have improved tenderness but lowered scores for juiciness (Wheeler *et al*. 2001). The effects on juiciness may be related to the recent finding that myostatin-deficient mice have a significant ($P < 0{\cdot}05$) reduction in fat accumulation (McPherron & Lee, 2002). Commercial interest has been considerable, due to the increase in muscle mass with few penalties for meat quality, but breeding programmes have to consider the welfare and cost implications of the high requirements for parturition by caesarian section (Murray *et al*. 2002).

A spontaneous mutation leading to muscle hypertrophy in the hindquarters of sheep has been identified and termed 'callipyge' (from the Greek, meaning beautiful buttocks). In the callipyge phenotype the hypertrophy is not accompanied by hyperplasia, but in common with the myostatin mutation there is a particular hypertrophy of the FG fibres. The hypertrophy in callipyge is due to reduced protein degradation (Lorenzen *et al*. 2000), with increased calpastatin activity leading to an overall reduction in calpain proteolytic activity (Koohmaraie *et al*. 1995; Shackelford *et al*. 1997*b*) and an increase in meat toughness (Freking *et al*. 1999; Duckett *et al*. 2000).

Two further mutations have been identified in pigs. The well-known porcine stress syndrome, in which stressed pigs show massive glycogenolysis leading to increased lactic acid and raised body temperature and muscle rigidity, has been shown to be due to a single autosomal recessive gene known as the halothane (*hal*) gene because of the animals lethal sensitivity to halothane anaesthesia. A point mutation in the sarcoplasmic reticulum Ca^{2+}-release channel protein, the ryanodine receptor, allows release of Ca^{2+} from the sarcoplasmic reticulum at approximately twice normal rates. Post mortem the mutation contributes to the incidence of pale, soft and exudative meat resulting from the early and unregulated release of Ca^{2+}, and a reduced capacity for its re-sequestration. This outcome can lead to myosin denaturation, loss of water-holding capacity and tough meat (Monin *et al*. 1999). In addition, the toughening of meat found in heterozygous animals is caused by a reduction in abundance and activity of m-calpain with no change in calpastatin (Sensky *et al*. 1999). However, it is not known whether these reductions are a direct result of the mutation or arise from the changes in metabolism due to the mutation.

The Rendement Napole (RN) or Hampshire mutation in pigs was first identified because of the apparent benefits for the production of cured cooked hams. The gene maps to chromosome 15 and has been identified as a mutation in the protein kinase AMP-activated γ_3 subunit (Ciobanu *et al*. 2001). The gene exists as two alleles: one recessive (rn^+); one dominant (RN^-). The RN^- is characterized by an

increased growth rate and higher than normal level of muscle glycogen at slaughter, which leads to a higher rate of lactic acid production and a more acid pH, high drip loss and pale colour (Lundstrom *et al*. 1996). However, despite greatly increased intracellular glycogen in the muscle of these animals, evidence suggests that the presence of the RN^- allele leads to a general increase in the relative area of the oxidative fibres at the expense of the glycolytic fibres (Lebret *et al*. 1999). The commercial value of the meat from these animals for processing is well established, but because of the pale colour and dry texture the value for unprocessed meat is less clear.

Growth rate and nutrition

Nutritional strategies to improve tenderness generally attempt to increase the activation of the calpain system *in vivo* before slaughter. Particular attention has been focused on manipulating growth rate and protein turnover to achieve tenderness benefits. It is well known that protein turnover varies with genotype (Oddy, 1999) and that both nutrition and hormones are central to the control of muscle metabolism (Lobley, 1998). The concept that growth rate and protein kinetics may be important determinants of tenderness arose from observations that faster growth, with associated high rates of protein turnover, resulted in a lower proportion of matured proteins in muscle and a lower proportion of stable non-reducible cross-links (Aberle *et al*. 1981). These results and data showing that double-muscled cattle exhibiting compensatory growth had lower shear force measurements (Hornick *et al*. 1998) were taken to indicate that there was a positive correlation between lean growth rate and tenderness.

Recent studies in cattle using growth rates at or above those used commercially showed that neither preweaning (Allingham *et al*. 2001) nor preslaughter growth rates (Moloney *et al*. 2001; Sinclair *et al*. 2001) affect tenderness. Moreover, even when compensatory changes were evident in terms of elevated rates of both protein synthesis and protein degradation (Lobley *et al*. 2000), no improvements in eating quality were observed and the major effects appeared to relate to genotype (Lobley *et al*. 2000; Sinclair *et al*. 2001). In contrast, however, where more extreme strategies are used to manipulate protein turnover, clear associations between growth rate, turnover rates and the activity of proteolytic systems can be seen. For example, in lambs feed restriction not only increased protein degradation but also decreased the extractable activities of calpastatin, whilst insulin-like growth factor 1 treatment significantly ($P < 0{\cdot}05$) decreased the activity of μ-calpain but did not affect that of calpastatin (McDonagh *et al*. 1999). In contrast, administration of the β-adrenoceptor agonists, which induce a muscle-specific protein anabolism through an initial increase in synthesis followed by a decrease in degradation, has been shown to up regulate calpastatin and to reduce μ-calpain activity, leading to increased toughness (Bardsley *et al*. 1992; McDonagh *et al*. 1999).

Although Oddy *et al*. (2001) suggest that nutritional history or early patterns of growth may impact on tenderness in cattle, the types of growth pattern described include nutritional restrictions of varying severity and duration. Commonly, cattle in temperate regions are not subjected to such regimens of compromise, and the main body of evidence concurs that normal variations in growth patterns do not account for the observed variation in eating quality. Consequently, the comment made by Moloney *et al*. (2001) that cattle management strategies during finishing, including feeding pattern and ration composition, generally have little impact on tenderness seems appropriate.

Given that non-experimental nutritional manipulations have few major effects on tenderness in ruminants, other nutritional approaches have targeted the activation of the calpain system more directly. Supranutritional administration of cholecalciferol with or without Ca^{2+} has been used in cattle and sheep to increase muscle Ca^{2+} levels and hence lead to greater activation of the calpain system (Scanga *et al*. 2001; Wiegand *et al*. 2001). Although this approach does lead to increases in serum Ca^{2+} levels, no improvement in tenderness was observed (Scanga *et al*. 2001; Wiegand *et al*. 2001). These data clearly indicate that the homeostatic regulation of muscle intracellular Ca^{2+} levels *in vivo* is complex and robust, and that manipulation strategies must take this factor into account.

However, the effects of growth rate and nutrition appear to differ somewhat between ruminants and non-ruminants. Work in pigs tends to support more strongly the relationship between high growth rates and improved tenderness, because feeding *ad libitum* produced more tender meat than restricted feeding; feeding *ad libitum* throughout the finishing period or immediately before slaughter improved tenderness (Blanchard, 1994; Ellis *et al*. 1996; Blanchard *et al*. 1999*a*). However, a recent study suggests that a compensatory growth achieved by restricted feeding during the growing period and *ad libitim* feeding in the finishing period altered DNA and RNA concentrations but did not affect technological measures of meat quality (Oksbjerg *et al*. 2002). In addition to affecting tenderness through growth rate, other approaches have included creatine monohydrate supplementation (Maddock *et al*. 2002; O'Quinn *et al*. 2000) or manipulation of intramuscular glycogen stores (Rosenvold *et al*. 2001; Leheska *et al*. 2002). In relation to creatine monohydrate supplementation, the concept here is that increasing available energy for ATP production without involving glycolysis will improve meat quality by slowing the fall in pH. However, while creatine monohydrate supplementation had a transient effect on pH, no differences in pHu or drip loss were evident.

Similarly, the concept that high intramuscular glycogen levels at slaughter will result in more lactic acid build-up, lower pHu and higher drip loss has formed the basis of dietary manipulation strategies to reduce drip loss. For example, in pigs feeding a diet low in carbohydrate with or without high protein before slaughter led, in certain cases, to a reduction in muscle glycogen (Rosenvold *et al*. 2001) but had no effect on pHu (Rosenvold *et al*. 2001; Leheska *et al*. 2002).

While some of these observations further substantiate the potential value of manipulating growth rate to improve product quality, it is important to consider that for ruminants the majority of studies in which responses in tenderness have been achieved have used extreme manipulations, intact male animals or pharmaceutical agents, and many of them

have not used sensory panel evaluation of the meat. Caution should be applied, therefore, when considering the commercial value of such strategies, particularly in view of the growing consumer concerns for animal welfare and 'natural' products.

Conclusions

The evidence shows that postmortem factors such as sarcomere length, temperature, pH and proteolysis have a major impact on tenderness. Thus, it is clear that unless perimortal handling procedures are optimised, any manipulations of animal-related factors may be without benefit to the ultimate meat eating quality. However, although the precise relationships remain to be defined, it is also clear that the intrinsic properties of muscle, i.e. the fibre type composition and the collagen content, together with factors related to breed, genotype, growth rate and nutrition may also play a part in determining tenderness. These findings illustrate the importance of gaining further understanding of how the intrinsic characteristics and metabolism of muscle are affected by, and respond to, perimortal handling procedures.

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