Post-mortem muscle metabolism is important in contributing to the following meat quality defects:

- pale, soft, exudative (PSE) meat;
- dark-cutting beef (DCB);
- dark, firm, dry (DFD) pork;
- tough meat;
- abnormal meat colour;
- excessive drip.

PSE, DCB and DFD meat are stress-related conditions. PSE meat occurs in pigs and turkeys. In pigs it is common in particular breeds which are known as stress-sensitive (SS) or halothane-positive (nn) genotypes. This chapter focuses on some of the key features in post-mortem muscle metabolism which lead to these meat quality problems, and so helps to explain how pre-slaughter stress influences meat quality.

When an animal is slaughtered, its muscle continues to metabolize energy, contract and produce heat. Some of the energy is used to power the convulsions, muscle twitching (fasciculations) and rigor contractions which occur in the carcass, and some is used in non-contractile biochemical changes. During muscle contraction, adenosine triphosphate (ATP) is utilized and it forms adenosine diphosphate (ADP) and free phosphate (P). ATP breakdown provides the energy needed for the contractions. ATP is also utilized in pumping mechanisms that regulate the concentration of ions in the cell.

ATP is resynthesized in two main ways. The first is from an energy store which is in the form of creatine phosphate (CP). Creatine phosphate passes on a high energy phosphate group to ADP, forming ATP, in a reaction that is catalysed by the enzyme, creatine phosphokinase (CPK):
CP + ADP ⇌ ATP + C

CPK is present in large amounts in muscle. In the live animal its leakage into the bloodstream can be a useful indicator of damage to muscle membranes or excessive activity of the muscle.

The other way in which ATP is resynthesized is through the mitochondrial respiratory chain. This involves an electron transport system which is catalysed by NAD-linked dehydrogenases, flavoprotein dehydrogenases and cytochromes (Fig. 6.1). Every time the electron transport chain is activated, three molecules of ATP are produced from three molecules of ADP and Pi, and one atom of oxygen is incorporated into water. It is important to note that this way of regenerating ATP requires oxygen.

The electron transport chain is linked to other metabolic processes as follows. In the sarcoplasm of the cell, the end product of the glycolytic pathway is pyruvate. After pyruvate enters a mitochondrion it joins the tricarboxylic acid (TCA) cycle as it is converted to acetyl CoA (see Fig. 5.6). This reaction is linked to the respiratory chain through NAD. For each molecule of pyruvate that is converted to acetyl CoA, one atom of oxygen is incorporated into water and three molecules of ATP are resynthesized from ADP. The conversion of isocitrate to oxalosuccinate fuels the respiratory chain in the same way, but in the case of the oxidation of succinate to fumarate the reaction is linked to a flavoprotein instead, and only two molecules of ATP are formed from ADP.

![Fig. 6.1. Electron transport chain and the regeneration of ATP in mitochondria.](image-url)
The relevant features of the above are that the reactions which are catalysed by the NAD-linked dehydrogenases and the flavoprotein dehydrogenases form a necessary link between the electron transport chain, which regenerates ATP, and the oxidation of metabolic fuels derived from carbohydrates, fats and proteins. It is the electron transport chain and the need to regenerate ATP which drives the whole system during post-mortem muscle metabolism.

The glycolytic pathway helps to fuel the TCA cycle and hence the electron transport chain. Exchange between glycolysis and the TCA cycle only occurs when pyruvate leaves the sarcoplasm, enters the mitochondria and is converted to acetyl CoA. This is a one-way process, which acts as a valve, maintaining the direction of flow of energy towards the TCA cycle. After slaughter, glycolysis is no longer fuelled by glucose derived from the bloodstream and instead it relies predominantly on the glycogen that is stored in muscle. FFA utilization post-mortem is greatly reduced in comparison with that occurring in the live animal, as there is limited ability to translocate FFA from the intramuscular lipid stores to the mitochondria post-mortem.

During death there are three processes which cause the normal metabolic processes in living muscle to slow down and eventually stop:

- depletion of oxygen;
- depletion of substrate;
- inhibition of enzymes.

When an animal is stuck, the blood supply to its muscles stops. The muscles no longer receive oxygen and the respiratory chain instead depends on the reserves of oxygen that were in the tissues at the time the animal was slaughtered. When these reserves are used up, the electron transport chain ceases to function. ATP resynthesis will continue for a short period from the store of creatine phosphate.

When the electron transport chain fails through lack of oxygen, the reactions in the TCA cycle slow down and eventually stop through inadequate supplies of NAD and FAD in their oxidized forms. This would lead to a build-up of pyruvate if it were not for the presence of lactic dehydrogenase in the sarcoplasm which converts the pyruvate to lactate. In so doing, it reoxidizes NADH to NAD, and the replenishment of NAD allows a further burst of glycolysis through the continuation of the reaction catalysed by glyceraldehyde 3-phosphate dehydrogenase. Thus, whilst anaerobic conditions cause the TCA cycle to stop, glycolysis continues. Glycolysis eventually comes to a halt through one of two effects. Either there is depletion of substrates (glycogen, glucose and hexosephosphates) or the build-up of acidity in the form of lactic acid inhibits the enzymes in the glycolytic pathway.

Putting these processes together, we can see a pattern in the depletion of ATP, CP and glycogen, and the accumulation of lactic acid in muscle during the early post-mortem period (Fig. 6.2).

ATP normally has two functions in muscle. It provides energy for muscle
contraction, which occurs when actin and myosin filaments interlock by sliding over each other. The contraction process is normally initiated by Ca\(^{2+}\), which is released into the sarcoplasm when the muscle is stimulated by a nerve.

ATP also provides energy that operates two ionic pumps within the muscle cell. These pumps reduce the concentration of Ca\(^{2+}\) in the sarcoplasm; one of the pumps is present on the sarcoplasmic reticulum and the other in the mitochondria. As the ATP level falls post-mortem, the ionic pumps start to fail. There is insufficient energy to operate the pumps and the Ca\(^{2+}\) level in the sarcoplasm gradually rises. When the Ca\(^{2+}\) level exceeds 10\(^{-6}\) M, the Ca\(^{2+}\) activates the muscle to contract. At this stage there is insufficient ATP to reverse the contraction by pumping the Ca\(^{2+}\) out of the sarcoplasm, and so the muscle remains in a contracted state. This permanent contracted state is known as rigor. The severity of rigor is important in influencing the toughness of meat. Once rigor has set in, the toughness it creates can only be released by breaking up the myofibres. Disruption of the myofibres can occur enzymatically during normal ageing of meat, or mechanically when a steak is beaten before cooking.

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**Fig. 6.2.** ATP, CP, glycogen and lactic acid changes during post-mortem muscle metabolism (after Tarrant et al., 1972).

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When an animal is stressed before slaughter, the reserves of glycogen and ATP at the time of slaughter are likely to be low. This means that ATP depletion is likely to occur shortly after the animal is killed, the sarcoplasmic Ca\(^{2+}\) levels will quickly rise and so rigor will set in earlier. A characteristic feature of carcasses from animals that have been severely stressed immediately before slaughter is that they enter rigor sooner. A simple way of distinguishing this in different carcasses is to select an appropriate point in the slaughterline, and to lift a forelimb of each carcass as it passes that point.

If muscle glycogen levels became depleted during a preslaughter stress, the muscle may have to switch to using free fatty acids as an energy source instead. The free fatty acids are utilized by breaking them down to acetyl CoA, which feeds into the TCA cycle, providing the muscle with much needed energy. The presence of acetyl CoA inhibits the conversion of pyruvate to acetyl CoA and glycolysis is greatly reduced. A common feature of aerobic but glycogen-depleted muscle is that all the glycolytic intermediates are suppressed except for pyruvate, which is either normal or has built up to higher than normal levels.

In some situations the metabolites in muscle provide a better indication of stress than measuring metabolites and hormones in blood. This is because the change in concentration in some metabolites in muscle provides an integration of stress over a longer period. Table 6.1 summarizes some commonly used measures of muscle exhaustion.

An important feature of glycogen-depleted muscle from stressed animals is that it has insufficient glycolytic substrates to allow the muscle to acidify properly when the animal is slaughtered. Failure in acidification can be measured from the pH of the meat 24 hours after slaughter. This is known as the ultimate pH, or pH\(_{ult}\). A high pH\(_{ult}\) (e.g. greater than 6.0) indicates that the muscle was glycogen-depleted at slaughter, that the animal was metabolically stressed before slaughter and that the meat is likely to be dark-cutting (i.e. DCB or DFD pork). Dark-cutting meat is objectionably dark in colour and it is prone to microbial spoilage.

The pH of the muscle at the time the animal is bleeding out is called the initial pH of the muscle. A low initial pH indicates either that there has been intense stimulation of the animal or muscle just before stunning, or that

<table>
<thead>
<tr>
<th>Table 6.1. Measures of exercise stress or exhaustion in muscle and meat.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Muscle (immediately after slaughter)</strong></td>
</tr>
<tr>
<td>↓ Glycogen</td>
</tr>
<tr>
<td>↓ ATP</td>
</tr>
<tr>
<td>↑ Lactate</td>
</tr>
</tbody>
</table>

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stunning itself caused excessive activation of the muscle as convulsions. The initial pH is also sometimes called the pH\textsubscript{5 min}. The importance of stress during the minutes before slaughter on initial pH was demonstrated experimentally by Bendall (1966), when pigs had nervous transmission to their muscles blocked with curare just before they were slaughtered. This resulted in a high initial pH (Fig. 6.3).

A third measure of pH which is important in meat science is the pH\textsubscript{45 min}. If the rate of post-mortem metabolism is accelerated, the pH\textsubscript{45 min} will be lower than normal. At the same time the lactate concentrations in the muscle will be higher. In pork, if the pH at 45 minutes is less than 6.0 the meat is likely to be PSE, and pH\textsubscript{45 min} measurements are often used as a way of classifying pig carcasses for PSE during commercial slaughtering operations. PSE meat can also be detected from the rapid onset of rigor, from the amount of drip released from the cut surface and from the reflectance (brightness) of light from the cut surface. In poultry, post-mortem glycolysis is relatively rapid and so the pH\textsubscript{15 min} is measured instead of the pH\textsubscript{45 min}.

PSE meat is particularly common in SS breeds of pig. These animals have an unusually reactive muscle metabolism. They develop PSE meat

![Fig. 6.3. Effect of pre-treatment with curare on post-mortem muscle pH decline in pigs.](image-url)
even when handled and transported in a gentle manner. An example of their super-sensitivity is the response that they show to anoxia. Anoxia causes rapid ATP depletion in their muscle, whereas it has less effect in stress-resistant (SR) pigs (Lister et al., 1970). Even when SS pigs are totally relaxed before slaughter and have no muscle contractions (for example, following treatment with curare), they still have accelerated muscle ATP utilization when bled out, in comparison with SR pigs (Sair et al., 1970). Their muscle has a higher resting rate of metabolism and it is unusually sensitive to even the mildest stimuli. The effect of preslaughter handling stresses on their muscle metabolism can, however, be blocked with carazolol, the β-adrenergic receptor blocking drug, which suggests that a large part of their excessive responsiveness during normal preslaughter procedures is mediated through the sympathetic nervous system (Warriss and Lister, 1982).

When an animal is slaughtered, muscle metabolism is stimulated through the release of Ca\(^{2+}\) into the sarcoplasm. In SS genotypes, the sarcoplasmic reticulum and mitochondria fail to extract (sequester) Ca\(^{2+}\) at a fast enough rate to control the accelerated metabolism. In extreme cases, SS pig carcasses can develop rigor before the slaughterfloor staff have had a chance to do the evisceration. The accelerated glycolysis results in rapid acidification of the muscle, and if it occurs whilst the muscle is still warm it becomes very pale. Chapter 9 describes this mechanism in more detail. The combination of low pH\(_{45}\) with a high muscle temperature is critical in the formation of PSE meat.

When SR pigs are given curare just before slaughter, the slope of the pH fall curve is not affected, but the development of acidification in muscle is altered because the whole curve has been displaced upwards (Fig. 6.3). At any point in time after slaughter, the pH was higher in the curarized pigs. The pH fall was delayed because the muscle had a higher initial pH. If such a delay allows a carcass to cool before a low pH develops, it will help to reduce the formation of PSE meat. To summarize, if preslaughter stress causes a pig to be acidotic at slaughter and have a low initial pH, it is likely to experience the high temperature–low pH conditions which cause PSE meat (see Fig. 9.6). If, on the other hand, the pig is rested before it is slaughtered, it will have a higher initial pH and the carcass has a chance to cool before the meat pH falls to the level where the risk of PSE meat is increased.

In general, reducing nervous activity in SR pigs is not likely to have a large effect on the rate of ATP utilization in their muscles (McLoughlin, 1974). However, increasing nervous activity and muscle contractions immediately after slaughter has an immense effect. The extent to which the muscles contract and twitch and the physical spasms that occur during the convulsions at slaughterhouses help to accelerate anaerobic glycolysis. In extreme situations they can cause a low pH at a high muscle temperature, and this leads to PSE meat. For this reason, excessive contractions following
stunning or during electrical stimulation of the carcass can cause PSE meat in pigs and turkeys.

Ischaemia (failure of blood supply) during bleeding out can help to accelerate ATP depletion in muscles. This is due to reduced resynthesis of ATP because of an inadequate supply of oxygen to the tissue, rather than an increased utilization of ATP by muscle. Before slaughter, muscle contractions are more important than hypoxia in causing low initial pH values in meat (McLoughlin et al., 1973).

Fish that are severely exercised during the catching procedure go into rigor more quickly than fish that have less exercise. Exercised fish also have a shorter rigor duration before they start softening during the resolution of rigor. Unlike beef, lamb and pork, exercise-stressed fish do not usually produce high pH\text{ult} meat. Instead, they tend to have a low pH\text{ult}. This is because, in live fish, lactic acid accumulates within the muscle, and often persists through to the post-mortem period.

Meat tenderness is influenced by three factors:

- The chemical nature and amount of connective tissue in the meat. For example, forequarter cuts have more intermuscular connective tissue, which makes them tougher.
- The extent of rigor. A strong rigor can cause tougher meat.
- Meat ageing. When left to age, meat softens enzymatically.

Immediately after slaughter, meat is tender and has a low shear force when it is compressed. The toughness that is present in this pre-rigor meat is influenced by its connective tissue and marbling content. However, once the meat goes into rigor, these two components together only account for 20% of the variation in meat tenderness (within an aged muscle). Variation between muscles is more likely to be explained by differences in their connective tissue content, especially if there is inadequate trimming.

Theoretically, chronic stress during rearing could affect meat texture by altering the ratio of myofibres to connective tissue. There are two ways in which this could occur. In long-term exercise-induced muscle hypertrophy there is an increase in the ratio of myofibrillar to connective tissue proteins and the meat is more tender (Aalhus et al., 1991). Alternatively, if there is no hypertrophy and instead the chronic stress is associated with catabolism of muscle protein, the meat may end up tougher (Bramblett et al., 1963). This may also explain why animals that are severely fasted before slaughter, as a way of reducing excessive carcass fatness, are reputed to produce tougher meat.

As rigor develops, the muscles shorten and they become tougher. The conditions under which the carcass is held are important in influencing the extent of rigor and subsequent toughness. There are five ways in which the extent of rigor can be affected:

- **Rigor shortening** - removal of muscle from the carcass before rigor has set in, allowing excessive shortening of the myofibres during rigor.
contraction. This can occur during hot deboning and in early portioning of poultry breast fillets.

- **Heat shortening** - combination of high temperature and acid accumulation in muscle leading to an excessive contraction as rigor sets in.
- **Cold shortening** - excessive cold-induced muscle contraction during rigor, which occurs when a carcass is rapidly chilled.
- **Mechanical stimulation** - mechanically induced rapid post-mortem glycolysis leading to heat shortening (e.g. plucker-induced toughness in poultry meat).
- **Thaw shortening** - delayed, excessive contraction during thawing which occurs in meat that was frozen pre-rigor.

In pigs and poultry, acute stress can lead to tougher meat through heat shortening. If the animal is exercised and gets hot before it is slaughtered, the combination of a high temperature and a low pH leads to an early, stronger rigor. In practice this can occur in pigs which develop PSE meat, and in poultry it develops in birds that flap their wings excessively before slaughter.

The relationship between $\text{pH}_{\text{ult}}$ and meat toughness is curvilinear (see Fig. 8.5). At low or high $\text{pH}_{\text{ult}}$ values the meat is more tender; it tends to be toughest at an intermediate $\text{pH}_{\text{ult}}$ (Purchas, 1990). The effect of pre-slaughter stress, and muscle glycogen depletion, depends on the extent to which it affects the $\text{pH}_{\text{ult}}$. If, as often happens in stressed cattle, the $\text{pH}_{\text{ult}}$ is only slightly raised, the meat may end up marginally tougher. Part of this effect is due to greater sarcomere shortening during rigor in meat which develops an intermediate $\text{pH}_{\text{ult}}$ (Purchas and Aungsupakorn, 1993; Olsson et al., 1995). If, on the other hand, the animals are stressed to exhaustion and the $\text{pH}_{\text{ult}}$ is very high, the meat will be more tender (Chrystall et al., 1982).

As meat ages it loses some of the strength that developed during rigor. This relaxation is known as resolution of rigor. It is not a true relaxation, as the myofilaments do not slide apart. Instead, the meat is partly digested by calpain enzymes and the myofibres break up, particularly through solubilizing of the Z lines in the sarcomeres.

The calpain enzyme system has three components: a low calcium-requiring enzyme ($\mu$-calpain), a high calcium-requiring enzyme (m-calpain) and a calpain inhibitor (calpastatin). Meat tenderness can be explained by this system in a number of situations. Ruminants have higher calpastatin activities than non-ruminant species, and this probably explains why the rate of tenderization is slow in beef and lamb. Addition of calcium to meat can help to activate the calpains and make the meat more tender. The activity of both calpains is greater at high $\text{pH}_{\text{ult}}$ values (Cena et al., 1992) and this partly explains why meat from severely stressed animals is often more tender. Genetic differences in meat texture are partly due to differences in calpastatin activity in the meat (Koohmaraie et al., 1995). Giving $\beta$-agonist
growth promoters to the live animal results in tougher meat through increased calpastatin activity. Similarly, intravenous infusion of adrenaline for 7 days before slaughter resulted in increased calpastatin activity (Sensky et al., 1996).

In both redmeat and whitemeat species, if the animals are stressed and muscle is glycogen-depleted before slaughter, and if ATP is rapidly depleted post-mortem, the pH of the meat will be elevated and Ca^{2+} levels will rapidly increase because of failure in ATP-powered calcium re-uptake mechanisms. The combination of high pH and high Ca^{2+} concentration usually enhances the calpain activity. Meat from severely exhausted animals is likely to be more tender because of this.

Ways that are used for tenderizing meat post-slaughter include:

- **Tenderstretch** - holding muscles in a stretched position before they go into rigor (as in hip suspension of the carcass).
- **Ageing** - endogenous enzymatic breakdown of myofibrillar proteins post-rigor (also known as conditioning).
- **Hot conditioning** - holding the carcass for up to 24 hours at a high temperature to encourage calpain-induced tenderization.
- **Electrical stimulation** - electrically induced accelerated post-mortem ATP utilization, which prevents cold shortening.
- **Mechanical tenderization** - mechanical tearing and disruption of myofibres post rigor (e.g. beating a steak before cooking).
- **Chemical tenderization** - injection of papain before slaughter to break down the myofibres enzymatically after slaughter.
- **High-temperature cooking** - solubilizing the collagen.

Meat colour is measured from its L-, a- and b-values. L is its lightness, which is directly proportional to its luminous reflectance. The a-value is its redness value; meat with a high a-value would be red. The b-value is a measure of its yellowness. PSE pork and meat from heat-stressed poultry have high L-values and low a-values. Dark-cutting beef has low L-, a- and b-values (Purchas and Grant, 1995). If a piece of normal beef and DCB are sliced very thinly across the fibres of the meat and the two slices are then placed on a white background, the slice of DCB appears to be more translucent than the normal beef. This is measured objectively in its lower reflectance (L-value). The reasons for the dark colour in high pHult meat are described in the section below on myoglobin. On cooking, dark-cutting semitendinosus can remain slightly darker but because of its high pH the myoglobin does not denature readily and the meat remains redder in colour than beef with a normal pH (Hawrysh et al., 1985).

Dark-cutting beef should not be confused with three other conditions which cause dark discoloration in beef. These are darkening of the meat surface due to desiccation, exercise-induced changes in haem pigments in the muscle and brown discoloration which develops on exposure to the atmosphere. Desiccation causes darkening because the muscle pigments
become more concentrated as water is lost. It can be controlled by using appropriate packaging. Allowing young bulls to exercise whilst they are growing, instead of being confined to cubicles with tethers, causes their meat to have a higher pigment concentration and to be darker (Ramsgaard Jensen and Oksama, 1996). Browning in uncooked meat is due to the formation of metmyoglobin. The rate of oxidation of myoglobin to metmyoglobin is pH dependent. For example, the rate of autoxidation in air doubles with a decrease in pH from 6.6 to 5.4 (George and Stratmann, 1954). This means that high pH_{ult} meat is less prone to metmyoglobin formation.

In some veal markets there is a preference for pale meat, and the methods used for producing the pale colour are contrary to good welfare. Calves that are grown for producing white veal are fed milk substitute that has a low iron content. The haematocrit in their blood is closely related to the haem iron content in the muscle ($r = 0.8$ to $0.9$), which suggests that the intensity of colour of muscle is related to the degree of anaemia in the calf (Charpentier, 1966). In extreme situations, anaemia is a weakening and debilitating disorder.

The post-mortem chemical reactions involving myoglobin have a profound effect on meat colour. Normally, myoglobin is oxygenated to oxymyoglobin shortly after meat is cut, and the fresh meat changes from purplish-red to a bright red. This process is known as blooming, and the bright red colour is synonymous with freshness. High pH_{ult} meat does not bloom when it is cut; instead, it remains a dark colour. The reasons for this are as follows. The proteins in high pH_{ult} meat are above their isoelectric point and this favours stronger water retention by the meat. The greater water retention makes the meat more turpid and firm, and it has a less open structure. This reduces the penetration of oxygen when the meat is cut. Poorer oxygen penetration reduces the oxygenation of myoglobin (dark purplish-red in colour) to oxymyoglobin (bright red colour), and so high pH_{ult} meat remains darker in colour. This effect may not be evident after cooking. During cooking, myoglobin and oxymyoglobin are denatured to brown pigments. However, at high pH, myoglobin is more heat stable and less prone to being denatured. So, the dark purplish-red pigment in high pH_{ult} meat is also more likely to persist during cooking. In summary, high pH_{ult} meat is dark in colour when it is uncooked and it retains more redness when it is cooked, in comparison with normal pH_{ult} meat.

Preslaughter stress can also enhance the rate at which myoglobin turns green in vacuum-packaged meat. Meat from severely stressed animals is more prone to putrefying, and the mechanisms behind this are explained in Chapter 7. When it goes off, H_{2}S is liberated from the sulphur-containing amino acids and combines with myoglobin to form sulphmyoglobin, which is green in colour. Sulphmyoglobin formation only occurs when the meat pH is above 6.0 (Nicol et al., 1970).

The water-holding capacity of meat is a measure of the strength with which it retains water. It can be assessed by pressing a slice of meat on to
a filter paper and measuring the spread of moisture out of the meat in terms of the area that is covered. The mechanisms that influence water retention are described in Chapter 12, and the relationship between pH<sub>ult</sub> and water-holding capacity is shown in Fig. 12.1. This relationship is fundamental in the link between preslaughter stress and weight loss from meat as water. Meat which develops a high pH<sub>ult</sub> retains charges on its proteins which hold water electrostatically, whereas at a normal pH<sub>ult</sub> the proteins are near their isoelectric point where they lose their charge and do not bind water so strongly. Water-holding capacity is reduced in PSE meat, which can release considerable amounts of fluid as drip. The drip acts as a good medium for bacterial growth; it is unsightly; and in wholesale cuts it contributes to weight loss and reduced yield.

Meat juiciness can be affected by fat content and the amount of moisture left in the meat after it has been cooked. If a carcass is excessively lean (e.g. a pig carcass with a P2 backfat thickness of 8 mm), the meat is less juicy. The reason for this is best explained by analogy with hamburger manufacture. If fat is added in varying amounts to a batch of ground hamburger meat, juiciness improves as the fat level increases from 9% to 20% (Table 6.2). The fat imparts a self-basting effect during cooking and molten fat gives succulence, provided it is not burnt off during cooking. Tenderness can improve with the increase in fat content simply through a dilution effect. The fat is more tender than ground lean and so the more fat there is, the more tender the product. Over this range of fat content (9-20%), flavour is not likely to be affected. The juiciness of meat is spoilt if the meat is cooked to dryness, and when meat is being overcooked its water-holding capacity can influence the stage at which juiciness is lost. High pH<sub>ult</sub> meat is less prone to loss of moisture during cooking than normal pH<sub>ult</sub> meat, and so with prolonged roasting or grilling it will retain its moisture for longer and this could, theoretically, delay the point at which it loses juiciness. Whilst high pH<sub>ult</sub> meat can retain moisture and juiciness, PSE pork is less likely to be juicy than normal pork (Sayre et al., 1964).

Raw meat has a salty, metallic flavour. The meat-like flavour develops as the meat is being cooked and it is largely due to the formation of Maillard

### Table 6.2. Effect of fat level in hamburgers on their eating quality.

<table>
<thead>
<tr>
<th>Fat in hamburgers</th>
<th>9%</th>
<th>20%</th>
<th>28%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Juiciness</td>
<td>7.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10.5&lt;sup&gt;b&lt;/sup&gt;</td>
<td>11.3&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Tenderness</td>
<td>9.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>11.8&lt;sup&gt;b&lt;/sup&gt;</td>
<td>13.1&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Flavour</td>
<td>9.9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>9.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>9.0&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Subjective score ratings were used.

<sup>a</sup>-<sup>c</sup> Columns without a common superscript letter were significantly different at $P = 0.05$. 

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reaction products and caramelized products. Inosinic acid (IMP) helps to enhance flavours within meat. Different cooking methods result in different meat-like flavours. Meat cooked in a microwave oven or meat that is stewed has less meat-like flavour compared with roasted, fried or grilled meats. This is because the cooking temperature is lower during microwave cooking and stewing, and there is less surface browning. High cooking temperatures are usually associated with stronger flavours. Part of this effect could be due to the formation of more Maillard reaction products and part could be due to the greater release of IMP (Cambero et al., 1992).

Maillard reaction products are formed on heating amino acids, peptides, sugars, sugar phosphates, nucleotides and nucleosides. Functional groups on meat proteins, lipids and carbohydrates also take part in Maillard reactions. Amino acids and reducing sugars combine to form N-sugar amines which are modified by elimination of water and the amine group to deoxyreductones, deoxyosones and aldehydes. These contribute meat-like flavours, along with products belonging to the furan, furanthiol, furanone and ethanethiol groups of compounds, and they all help to provide character impact (Bailey, 1992). Sulphur-containing Maillard reaction products are thought to be particularly important in providing meat-like aromas. Cooking conditions that favour Maillard reaction product formation also appear to favour the formation of mutagenic compounds on the surface of the cooked meat (Holtz et al., 1985).

Warmed-over flavours (WOFs) can displace meat-like flavours when meat has been cooked and then stored for 48 hours or more in a refrigerator. Paint-like, cardboard and oxidized flavours take its place. These flavours are formed in a similar manner to the oxidative rancidity that develops in frozen meat, except that they occur much more rapidly. Some Maillard reaction products can inhibit WOF development by acting as antioxidants. Pro-oxidants that favour WOF formation include ultraviolet light, iron and activated oxygen species such as superoxide and hydroxyl radicals.

There are three counteracting ways in which animal welfare and meat flavour could theoretically be linked. Firstly, if preslaughter stress exhausted the animal and reduced the level of glycolytic intermediates in the muscle, there would be fewer hexose and triose aldehyde groups available for the formation of Maillard reaction products. This would result in the meat being more bland. Secondly, if the stress caused extensive conversion of ATP to AMP, there could be enhanced meat flavour. This would be due to the gradual conversion of AMP to IMP post-mortem through the action of AMP aminohydrolase which is present in fresh meat. IMP has a flavour-enhancing effect, but during prolonged storage this is lost as the IMP is degraded to inosine. The third way is by enhancing oxidative rancidity and the formation of WOFs. When animals are forced to take strenuous exercise, the subsequent susceptibility of the muscle to develop lipid peroxidation is increased (Alessio et al., 1988). The mechanism is as follows. During exercise, oxygen uptake by muscle increases by up to 200 times its resting rate.
The oxygen helps to regenerate energy by oxidative phosphorylation in the mitochondria. However, about 2–4% of the oxygen escapes from the respiratory chain as superoxide (O₂⁻). Superoxide is converted to toxic hydroxyl radicals (OH⁺) by the Haber-Weiss reaction, as follows:

\[ \text{H}_2\text{O}_2 + \text{O}_2^- \rightarrow \text{O}_2 + \text{OH}^- + \text{OH}^+ \]

Hydrogen peroxide is needed for this reaction and it is produced from superoxide by dismutation reactions in the mitochondria and microsomes. Hydroxyl radicals are important because they cause peroxidation of unsaturated phospholipids in cell membranes with the formation of lipid peroxides, which in turn give rise to hydroperoxides, carbonyl compounds and hydrocarbons, all of which contribute to rancid flavours. This process can be inhibited by either chemical reduction of the oxidized membrane lipids (e.g. with vitamin E), OH⁺ scavengers (e.g. thiourea), complete removal of superoxide by dismutation to H₂O₂ and O₂ (e.g. with superoxide dismutase), or removal of H₂O₂ (e.g. by catalase, or glutathione peroxidase). Thus, pre-feeding rats with vitamin E or injecting them with superoxide dismutase helps to reduce muscle lipid peroxidation during severe exercise stress (Goldfarb et al., 1994; Radak et al., 1995). Protecting muscle from hydroxyl radical injury also helps to reduce the muscle soreness that can develop after strenuous exercise (Ørtenblad et al., 1997).

Research into the effect of preslaughter exercise on meat flavour has not produced any consistent findings. However, in general, high pHᵢᵤᵢᵦ beef has less beef-like flavour and high pHᵢᵦᵦ lamb has less distinct aroma. If bruised tissue which is rich in haem iron is included in mince, there is a risk that the added iron will act as a pro-oxidant and favour oxidative rancidity and WOF formation.

Broilers reared at low stocking densities have been found to produce thigh meat with a stronger odour (Farmer et al., 1997), and broilers raised with a reduced intestinal flora have been found to have a weaker breast meat flavour. It is possible that these effects are due to differences in the litter, which may impart flavours to the meat through the skin.

There is a growing consumer trend away from fresh meat and towards processed meat products. An important technical feature which influences the appearance and eating quality of processed meats is the way in which the meat pieces bind and hold together. Clearly, nobody wants a hamburger that falls apart when it is cooked. It should also have an appropriately meaty mouth-feel. A large component in the mouth-feel of meat is due to the alignment of fibrous proteins. This makes the mechanical and chewing properties highly directional, or anisotropic. In particular, the strength of fibres when pulled along their length is much greater than pulling at right angles to their length. When unprocessed meat is chewed, the initial stages are the breaking down of the epimysium and perimysium (see Fig. 5.2). In later stages of chewing, an appreciation of the anisotropic nature of the
fibres develops. In manufactured meats the aim is to retain these anisotropic properties but to lose the connective tissue structures. In practice this is achieved by trimming the larger pieces of connective tissue and cutting the meat into sufficiently small pieces to disrupt the intramuscular connective tissue. The cut pieces are then stuck together again.

The main adhesive used in sticking the pieces together to make a re-formed meat is myosin. During the manufacturing procedures soluble myosin is extracted and coats the surface of the pieces of meat. When the re-formed meat is cooked, the myosin forms a gel that binds to adjacent meat particles, holding them together. The binding can be ruptured in one of three ways:

- Gel can be pulled apart - tensile fracture.
- Gel can break by sliding apart - shear fracture.
- Gel or meat can break by tension or shear - cohesive fracture.

The strength of the gel can be important in influencing the shape or deformability of the product as well as its holding-together properties during cooking. High pH meat is likely to produce strong myosin gels, whereas PSE meat produces weak myosin gels.