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Muscle Proteins

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Introduction

Muscle proteins are an important part of the diet in many cultures, as they make up the major proportion (other than water) of lean meat. These proteins provide most of the texture, color and nutritive value of meat and most meat products. In life, muscle proteins provide motive power to the animal. Following slaughter, muscle tissue forms meat.

Meat and meat products are today regarded in the developed world as a luxury, and in many Western countries meat consumption is on the decline due to both the price and the environmental cost. But strong demand for meat and meat products is expected to continue because of strong meat-eating traditions in many cultures, a general recognition of meat as one of the best sources of high quality bioaccessible protein and iron, and a general preference for the textures and flavors of meat-containing dishes. Further to this, meat plays an important part in many developing countries, particularly meat from pigs and chickens that can convert waste materials, and from ruminants that can convert plant materials that are inedible to humans, into high quality food.

Structure of Muscle

Skeletal muscles consist of multinucleated muscle fibers made up of bundles of elongated myofibrils in a parallel configuration (**Fig. 1**) (Gault, 1992; Strasburg et al., 2008). Depending on animal species, muscle size and anatomical location, muscle fibers differ in length (a few millimeters to 30 cm), diameter (10 μm to 100 μm), and orientation (parallel or at a specific angle to the length). Most of the skeletal muscles are composed of muscle fiber type I, IIA, IIX and IIB (Pette and Staron, 2000; Schiaffino and Reggiani, 2011; Spangenburg and Booth, 2003). These muscle fiber types are characterized based on their contraction speed (slow-twitch or fast-twitch) and preferred metabolic pathway for glycogen degradation (glycolytic or oxidative). Different muscle types vary in ATPase activity, which reflects their contractile characteristics as well as enzyme activities involved in their metabolic pathways. Type I fibers are slow-twitch, red, with oxidative metabolism; type IIA fibers are fast-twitch, red, with intermediate (oxidative glycolytic) metabolism; whereas type IIB and IIX fibers are fast-twitch, white, with glycolytic metabolism (Lefevre et al., 1999; Peter et al., 1972).

A muscle fiber is ensheathed by a fine connective tissue layer, the endomysium, which contains blood capillaries and nerves. Each muscle fiber is composed of a few dozen myofibrils and is enclosed by the muscle plasma membrane, called the sarcolemma, which is made up of a phospholipid bilayer with embedded proteins, glycoproteins and glycolipids. The myofibrils are bathed in

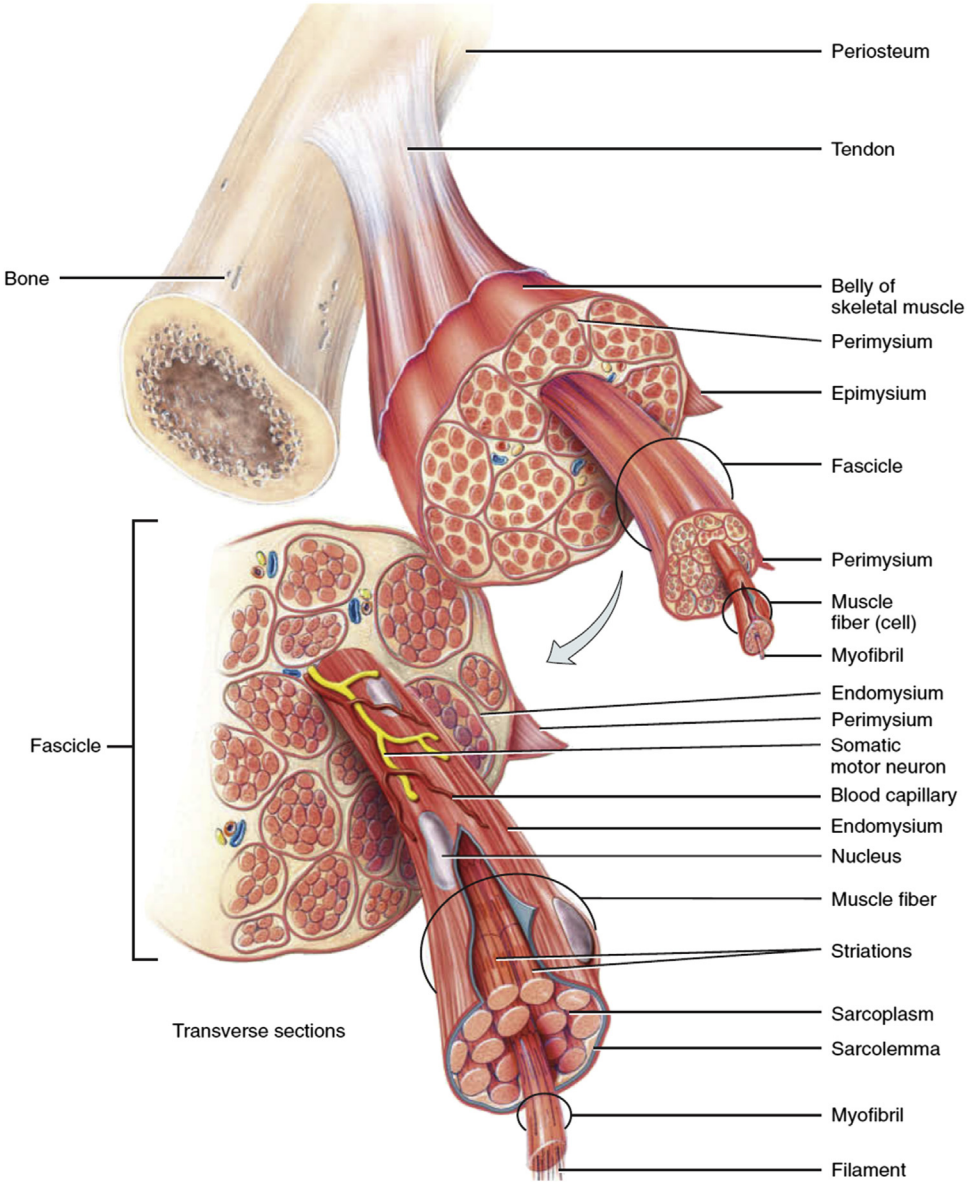

Figure 1 Structure of skeletal muscle. Adapted from Tortora, G.J., Derrickson, B.H., 2013. Principles of Anatomy and Physiology, fourteenth ed. Wiley Global Education, New York.

sarcoplasm containing several nuclei, sarcoplasmic reticulum, Golgi apparatus, mitochondria, lysosomes, glycogen granules, enzymes and other soluble constituents, that are vital for muscle function. The sarcoplasmic reticulum acts as a calcium ion reservoir for muscle contraction. Bundles of muscle fibers are organized into fascicles that are encased in another layer of connective tissue containing larger blood vessels and nerves called the perimysium. Several fascicles are assembled into a whole muscle by an outer layer of connective tissue known as the epimysium, which extends into tendons to join the muscles and bones together.

Unlike those of terrestrial animals, the muscles of fish are much shorter and are segmented into myotomes by a fine connective tissue layer called the myocommata, or myosepta, as depicted in **Fig. 2** (Johnston, 2001; Venugopal and Shahidi, 1996). Each myotome consists of muscle fibers organized in parallel to the length axis of the fish body. Fish muscle fibers are enclosed by the sarcolemma. However, the connective tissue of the extracellular matrix in fish fuses with myocommata at the myotome-myocommata junction, in contrast to terrestrial animal muscles, that are tapered into a tendon.

Regardless of the animal species, myofibrils in muscle fibers consist of longitudinal myofilaments comprising thick and thin filaments (**Fig. 3**) (Strasburg et al., 2008; Tortora and Derrickson, 2013). Thick filaments are made up of myosin molecules and several cytoskeletal proteins such as titin, while thin filaments are composed of actin, tropomyosin and troponin. Alternating light isotropic (I-band) and dark anisotropic (A-band) bands are seen on myofibrils under a polarized light microscope. At ultrastructural scale, each I-band is separated into two by a dark and narrow band called the Z-disk. The region between two Z-disks is known as a sarcomere, which is the repeating longitudinal contractile unit of the myofibril. A sarcomere comprises an I-band consisting purely of thin filaments, an A-band containing alternatively overlapping thin and thick filaments, an H-zone that is in the center region of the A-band, where the thin filaments are absent, and an M-line that is in the middle of the H-zone. When a muscle contracts, the Z-disks shift closer together due to shortening of the I-bands, and the length of the sarcomere is decreased (Gault, 1992).

Skeletal muscle proteins can be classified into myofibrillar (50%–60%), sarcoplasmic (30%) and stromal (10%–20%) proteins, based on their solubility at varying salt concentrations (Strasburg et al., 2008).

Myofibrillar Proteins

The myofibrillar proteins consist of contractile, structural and regulatory proteins (Hopkins, 2014). The contractile proteins are myosin and actin, which form the thin and thick filaments that control the skeletal muscle contraction and relaxation. The regulatory proteins include troponin and tropomyosin. The structural proteins comprise mainly titin, nebulin, α-actinin, β-actinin, tropomodulin, desmin, filamin, C-protein, H-protein and myomesin.

Contractile Proteins

Myosin (molecular weight of 520 kDa) is a filamentous protein that forms the thick filaments of muscle cells and is the principal protein of the A-band (Gault, 1992; Strasburg et al., 2008). A myosin molecule has a quaternary structure consisting of six subunits, which include two myosin heavy chains (MHC), two essential myosin light chains (MLC1) and two regulatory myosin light chains (MLC2), with molecular weights (MW) of approximately 220 kDa, 23 kDa and 20 kDa correspondingly (Clark et al., 2002; Swartz et al., 2009). Each myosin molecule can be hydrolyzed by enzymatic digestion into two portions, which are light meromyosin (LMM) and heavy meromyosin (HMM) (**Fig. 4**) (Choi and Kim, 2009; Pearson and Young, 1989a; Strasburg et al., 2008). LMM is important for the construction of thick filaments from myosin molecules while HMM is responsible for the regulation of ATPase activity and actin binding ability. HMM can be further broken down into fragment S1 and S2 where the former contains the globular myosin heads with the binding site of MgATP and actin, while the latter comprises the α-helix region which binds

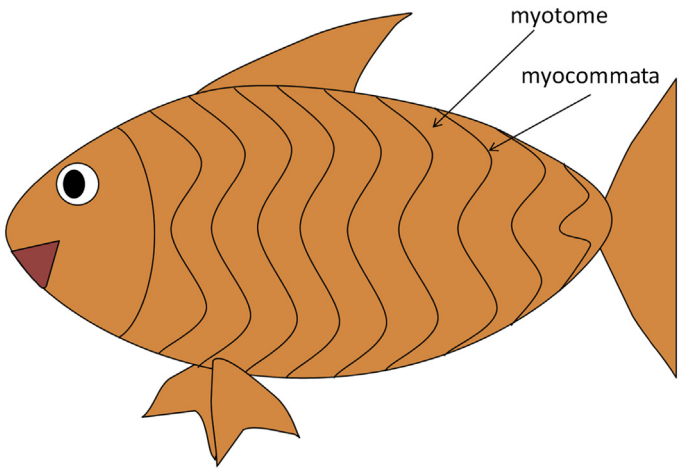

Figure 2 Schematic drawing of fish muscle displaying the orientation of myotomes and myocommata.

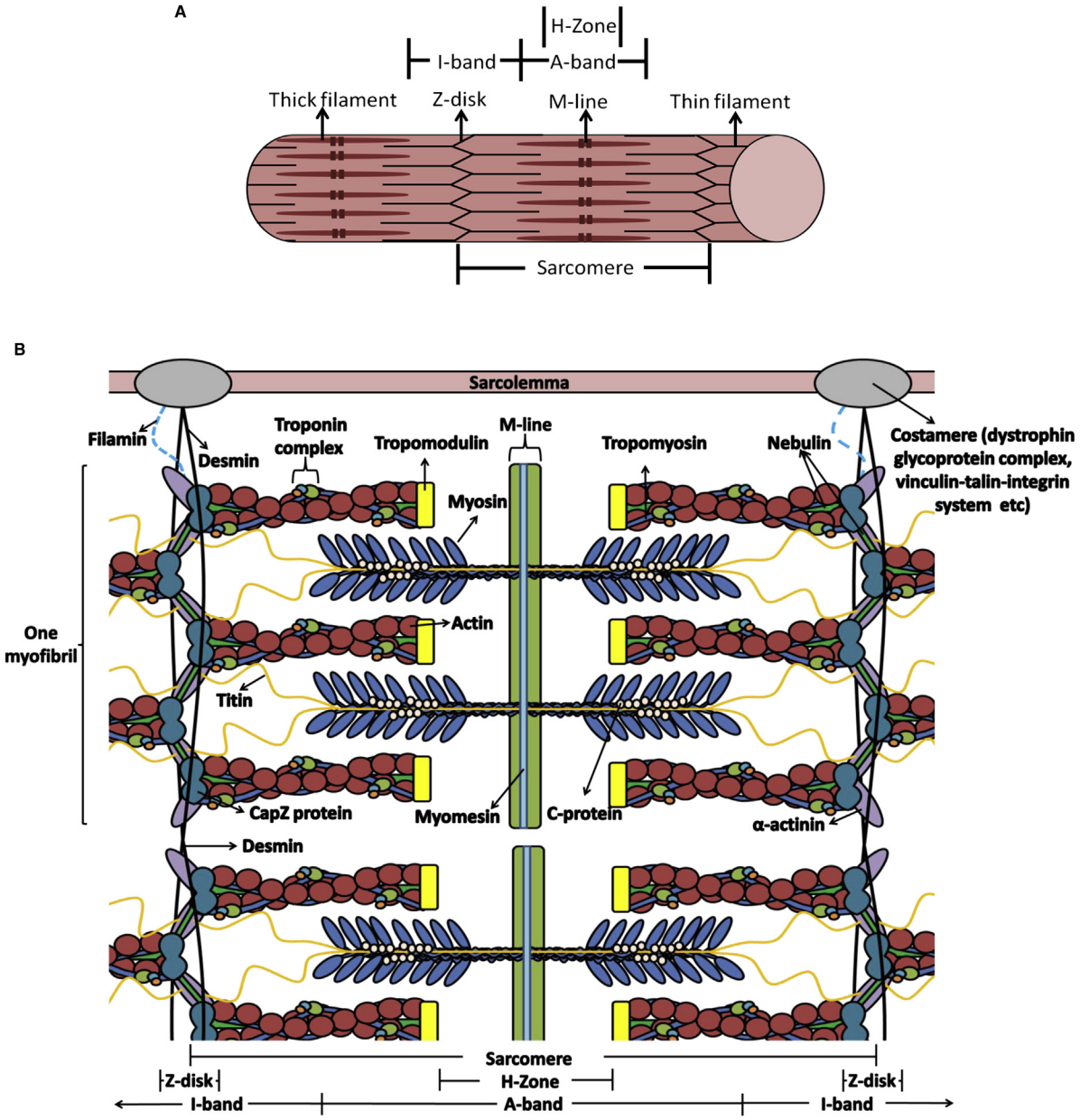

Figure 3 (A) Basic functional unit of a myofibril and (B) the arrangement of filaments within a sarcomere. Figure based on Au, Y., 2004. The muscle ultrastructure: a structural perspective of the sarcomere. Cell. Mol. Life Sci. CMLS 61, 3016–3033; Tortora, G.J., Derrickson, B.H., 2013. Principles of Anatomy and Physiology, fourteenth ed. Wiley Global Education, New York; Henderson, C.A., Gomez, C.G., Novak, S.M., Mi-Mi, L., Gregorio, C.C., 2017. Overview of the muscle cytoskeleton. Compr. Physiol. 7, 891–944.

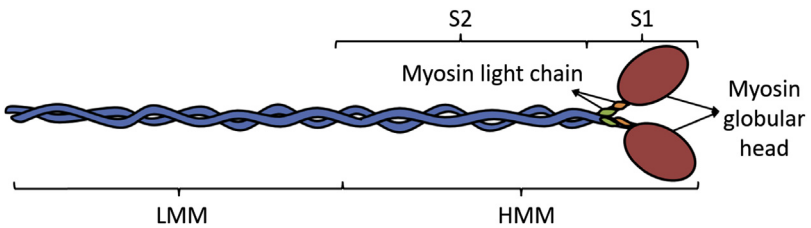

Figure 4 Schematic diagram representing a myosin molecule. Modified from Adamovic, I., Mijailovich, S.M., Karplus, M., 2008. The elastic properties of the structurally characterized myosin II S2 subdomain: a molecular dynamics and normal mode analysis. Biophys. J. 94, 3779–3789.

both MLC1 and MLC2. In adult mammalian skeletal muscle, there are primarily four MHC isoforms, namely MHC I, IIa, IIb, and IIx, which exist in pure slow-twitch fiber type I, fast-twitch fiber type IIa, IIb and IIx, correspondingly (Kohn et al., 2007; Pette and Staron, 2000). Hybrid fiber types containing different myosin isoforms have also been reported.

Actin (molecular weight of 42 kDa) is the building block of thin filaments and is present in two forms namely globular actin (G-actin) and filamentous actin (F-actin) (Gault, 1992; Strasburg et al., 2008). F-actin is formed by the polymerization of G-actin into double-stranded, coiled filaments. F-actin is bound to tropomyosin and troponin as shown in **Fig. 5**.

During muscle contraction, actin binds myosin to form actomyosin cross-bridges, which activate the myosin ATPase, leading to the pulling of thin filaments by myosin toward the M-line, resulting in shortening of the sarcomere (Lawrie, 2006).

Regulatory Proteins

Tropomyosin and troponin are two main proteins that regulate muscle contraction and relaxation (Choi and Kim, 2009; Zot and Potter, 1987). They prevent the activation of actomyosin ATPase in the absence of calcium ions by interacting with actin filaments to block the myosin binding site. Tropomyosin is a long, coiled protein (MW 65 kDa) that comprises two α-helix polypeptide subunits, called α- and β-tropomyosin. Tropomyosin molecules bind head-to-tail along the F-actin filament. Each tropomyosin molecule is attached to a troponin complex (MW 80 kDa) which is made up of troponin C (MW 18 kDa), troponin I (MW 21 kDa) and troponin T (MW 31 kDa) (**Fig. 5**). Troponin C acts as the calcium binding site; troponin T connects troponin complex to tropomyosin while troponin I inhibits actomyosin ATPase activity when it is bound to actin (Lehman and Craig, 2008). At high calcium ion concentration, calcium ions bind to Troponin C, which initiates a conformation change in the tropomyosin-troponin complex, dislocating troponin I, allowing the action of actomyosin ATPase for muscle contraction.

Structural Proteins

The structural proteins control the filamentous structure and integrity of myofibrils (**Fig. 3**) (Obinata et al., 1981). Titin, also known as connectin, with a molecular weight of 4200 kDa, serves as the backbone of thick filaments in the A-band. It also acts as a molecular spring in the I-band, which provides elasticity to the sarcomere during muscle contraction (**Fig. 3**) (Labeit and Kolmerer, 1995).

Nebulin (MW 800 kDa) is a structural protein that regulates the length of the thin filaments (McElhinny et al., 2003; Strasburg et al., 2008).

α-Actinin (MW 95 kDa) is the principal constituent of the Z-disk, supporting and attaching actin to the Z-disk (Obinata et al., 1981).

β-Actinin, also known as CapZ protein, is a heterodimer consisting of α- and β-subunits (MW 37 and 34 kDa respectively). It binds α-actinin in the Z-disk and prevents network formation between the actin filaments (Swartz et al., 2009).

Tropomodulin (MW 40 kDa) binds tropomyosin and actin as well as controlling the length of thin filaments by maintaining the number of G-actin monomers (Clark et al., 2002).

Both desmin (MW 55 kDa) and filamin (MW 300 kDa), play a major role in linking the myofibrils to the sarcolemma as well as stabilizing muscle structure (Capetanaki et al., 1997; Strasburg et al., 2008; Vander Ven et al., 2000).

C-protein (MW 140 kDa) and H-protein (MW 58 kDa) are myosin-binding proteins that are found in the A-band of thick filaments (Koretz et al., 1993; Xiong, 1997). These proteins are believed to contribute to the alignment and stabilization of the thick filaments.

Myomesin (MW 185 kDa) is the major protein in the M-line. It is responsible for the binding of titin and myosin and for maintaining the structure of the thick filaments.

Stromal Proteins

Stromal proteins constitute the connective tissue, which provides mechanical support and protection to the muscle in the form of tendons, epimysium, perimysium and endomysium (**Fig. 1**) (Gault, 1992). Connective tissue is composed mainly of collagen

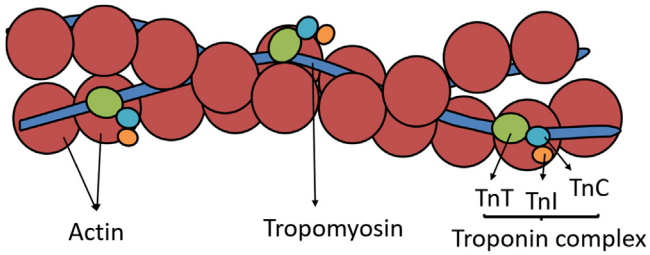

Figure 5 . Schematic diagram of a thin filament comprising actin, tropomyosin and troponin complex. TnT (troponin T), TnI (troponin I) and TnC (troponin C). Modified from Strasburg, G., Xiong, Y.L., Chiang, W., 2008. Physiology and chemistry of edible muscle tissues. In: Damodaran, S., Parkin, K.L., Fennema, O.R. (Eds), Fennema's Food Chemistry, fourth ed. CRC Press, Boca Raton FL, pp. 923–973.

(90%) along with other fibrous proteins including elastin, laminin and fibronectin, and proteoglycans (Chagnot et al., 2012; Voermans et al., 2008). Connective tissue contains different types of cells, including fibroblasts, macrophages, lymphoid cells, mast cells and eosinophils. Collagen and elastin are bound to an amorphous ground substance formed by proteoglycans and glycoproteins for the reinforcement of the connective tissue network.

Collagen

Collagen is the predominant stromal protein in skeletal muscles and is synthesized by fibroblasts (Bailey and Light, 1989; Duance et al., 1977; Gault, 1992). It is classified into four major types based on its aggregation characteristics: striated and fibrous (Types I, II, III, V and XI); non-fibrous and network forming (Type IV); microfibrillar or filamentous (Type VI), and fibril-associated collagen (Type VII) (Bailey, 1991).

Collagen consists of three polypeptide alpha chains with -Gly-X-Y- repeating units, where X is commonly proline and Y can be any amino acid (except tryptophan), but is often hydroxyproline, that coil to create a triple helix structure, forming tropocollagen (Astruc, 2014a; Bailey and Light, 1989; Gault, 1992; Strasburg et al., 2008). Tropocollagen molecules are polymerized into collagen fibers via covalent intermolecular cross-links that offer substantial tensile strength to collagen fibers. As the collagen fibers age, the divalent reducible cross-linkages interact to form mature trivalent, non-reducible, more heat-stable cross-links, which further enhances their stability and mechanical strength.

Collagen has been linked to the toughness of muscle-based food, and its content and extent of cross-linking differ among different animal species and breeds, age, muscle function, history of exercise and treatment with growth promoter (Strasburg et al., 2008). Small aquatic animals like fish generally have a lesser amount and less cross-linking of collagen compared to larger terrestrial animals that require higher weight-bearing strength. Some authors have reported an increase in meat toughness as the animal aged due to an increase in collagen cross-linking and a decrease in collagen solubility (McCormick, 1999; Purslow, 2005; Taylor, 2004). It was also found that the tender meat cut, bovine Longissimus dorsi, contains only half to two-thirds of the total collagen content and hydroxylysylpyridinoline cross-links of that found in the tougher cut, Biceps femoris. Various studies have shown improved meat quality by partial solubilization of collagen in tough meat cuts such as Semitendinosus (Christensen et al., 2011a,b; Combes et al., 2004; Sullivan and Calkins, 2010).

Elastin

Elastin is a minor constituent of connective tissue that offers elasticity to the blood vessels and ligaments in the muscles (Debelle and Alix, 1999). Elastin is an insoluble, hydrophobic, heat-stable and cross-linked protein fiber that behaves in a highly elastic manner in the presence of water. Elastin content varies among different muscle types. In Semitendinosus muscle, epimysium and perimysium are rich in coarse elastin fibers which are believed to be one of the contributors in meat toughness (Rowe, 1986). In contrast, Longissimus dorsi has limited coarse elastin fibers present in the epimysium and even less in the perimysium.

Sarcoplasmic Proteins

The sarcoplasmic proteins occur in the sarcoplasm surrounding the myofibrils (Pearson and Young, 1989b). They are involved in various metabolic functions such as protein metabolism, fatty acid oxidation, electron transportation, glycolysis, glycogenesis and glycogenolysis (Pearson and Gillett, 2012; Smith, 2000; Strasburg et al., 2008). The sarcoplasmic proteins include the heme pigments (myoglobin), the glycolytic enzymes (glyceraldehyde phosphate dehydrogenase), the mitochondrial oxidative enzymes (such as succinate dehydrogenase, cytochrome), the lysosomal enzymes (notably cathepsin), the nucleoproteins and others. Among them are the proteolytic enzymes that are involved in the post-mortem muscle tenderization process, and myoglobin that is responsible for meat color. The amount of different sarcoplasmic proteins is largely dependent on the muscle fiber type, which in turn depends on the animal species, breed, age, genetics, and muscle anatomical position and function (Strasburg et al., 2008). Sarcoplasmic proteases are vital in protein catabolism and post-mortem muscle softening. Both calpains and cathepsins are responsible for post-mortem proteolysis, with calpains and more specially calpain 1 or μ-calpain considered to be playing a major role (Ouali et al., 2006; Veiseth and Koohmaraie, 2005).

Calpains

The calpains are a family of calcium-activated, cysteine proteases that have maximum activity at neutral pH (Sentandreu et al., 2002). Calpains degrade myofibrillar proteins during protein turnover for muscle growth (Goll et al., 2008; Huang and Forsberg, 1998). There are two types of calpains responsible for post-mortem proteolysis, which are ubiquitous: μ-calpain and *m*-calpain (**Table 1**) (Raynaud et al., 2005; Strasburg et al., 2008). The calcium ion concentrations for the activation of μ-calpain and *m*-calpain are in the range of micromolar and millimolar respectively (Camou et al., 2007). Calpains are found and act along the Z-disk. Calpastatin is the natural inhibitor of μ-calpain and *m*-calpain.

Table 1 Characteristics of cathepsins and calpains associated with meat tenderness during ageing[a,b]

	Calpains	*Cathepsins*
Nature	Cysteine proteases; calcium-dependent	Acid proteases
Occurrence	Cytosol	Lysosomes (in sarcoplasm)
Class/Isoform	*m*-Calpains, μ-calpains and calpain 3.	Main cathepsins involved in muscle ageing are cathepsins B, L, H and D
pH for optimum activity	Neutral	4–6.5 (varies with enzyme class)
Target proteins	Proteins in the Z-disk and cytoskeleton (mainly titin and desmin). Some breakdown of nebulin, troponin and tropomyosin.	Troponin T, collagen cross-links and mucopolysaccharides of connective tissue. Actin and myosin at pH < 5

[a]Warriss (2010).
[b]Kemp et al. (2010).

Cathepsins

Cathepsins are sarcoplasmic proteins that are released from the lysosomes in post-mortem muscle, which are active at an acidic pH (**Table 1**) (Geesink and Veiseth, 2008). Among the family of cathepsins, cysteine cathepsin B, H and L and aspartic cathepsin D are the most abundant in muscles. Cathepsins break down MHC, troponin T, troponin I, tropomyosin and collagen (Bechet et al., 2005) and collagen (Goll et al., 1983). The proteolytic activity of cysteine cathepsins and cathepsin D can be inhibited by cystatins (Geesink and Veiseth, 2008) and pepstatin (Bohley and Seglen, 1992) respectively.

Myoglobin

Myoglobin is a heme protein that acts as an oxygen carrier in muscle cells and is responsible for the color of both raw and cooked meat. The amount of myoglobin varies between different muscle types. Oxidative muscle fibers type I & IIA have higher myoglobin content than glycolytic muscle fibers type IIB and IIX (Hoekstra, 1969). Poultry has a paler appearance than beef due to a higher content of fast glycolytic fibers and consequently a lower level of myoglobin.

Four forms of myoglobin exist in the muscle, depending on the state of the heme group: deoxymyoglobin (purplish red), oxymyoglobin (cherry red), metmyoglobin (brown) and carboxymyoglobin (cherry red) (**Fig. 6**) (Suman and Joseph, 2013). The color of the muscle depends on the ratio of these forms of myoglobin (Baldwin, 2012). Cooking of meat results in denaturation of myoglobin and causes the oxidation of heme which induces a change in meat color.

Other Sarcoplasmic Proteins

Most of the enzymes involved in the metabolic processes in muscles are sarcoplasmic proteins (Pearson and Young, 1989b; Strasburg et al., 2008). For instance, the enzymes creatine kinase and adenylate kinase take part in the synthesis and dissociation of high energy phosphate compounds such as ATP, ADP and AMP. Caspases, metalloproteases, thrombin and plasmin have been reported to be involved in post-mortem proteolysis (Sentandreu et al., 2002). Other sarcoplasmic proteins such as hemoglobin play an essential role in the exchange of oxygen and carbon dioxide (Pearson and Young, 1989b; Tahergorabi et al., 2011).

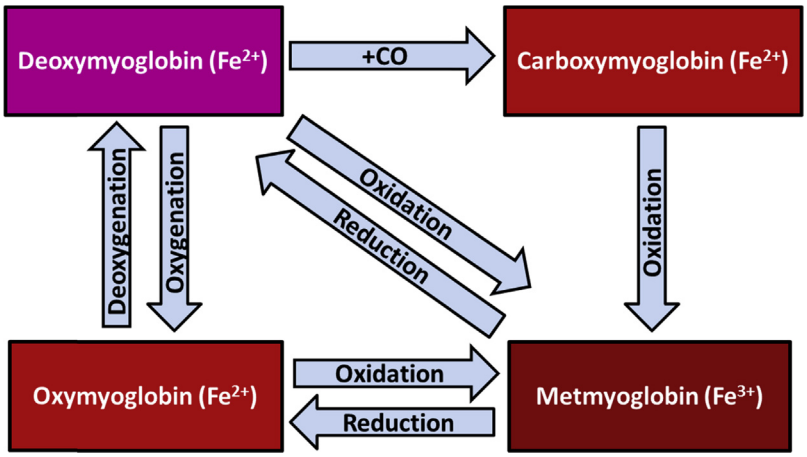

Figure 6 Interconversion of myoglobin redox forms in fresh meats. Modified from Suman, S.P., Joseph, P., 2013. Myoglobin chemistry and meat color. Annu. Rev. Food Sci. Technol. 4, 79–99.

From Muscle to Meat

Postmortem Biochemical Changes

Meat comes from the transformation of skeletal muscle after slaughter. After bleeding, cells survive for a short time by regenerating ATP (essential for maintaining ionic and potential membrane gradients) through anaerobic metabolic pathways. The synthesis of ATP is mostly based on the degradation of phosphocreatine and anaerobic glycolysis. When phosphocreatine is depleted, the poor performance of glycolysis does not maintain ATP at a constant value. Its cellular concentration gradually decreases. As the ATP level decreases and glycogen is degraded, protons and lactate molecules accumulate in muscle cells resulting in decreased muscle pH (Bendall, 1973; Greaser, 1986; Monin, 1988). When the ATP level falls by half the resting muscle content, the actin and myosin molecules bind to form an actomyosin complex. The relative sliding of the filaments becomes impossible and the whole of the myofibrillar apparatus is transformed into a rigid system called rigor. Subsequently, the muscle stiffness gradually fades, but not the inextensibility. This 'resolution' of rigor results from the lysis of the proteins constituting the myofibrillar structure during the so-called maturation period.

When the glycogen is depleted and/or when the enzyme co-factor AMP disappears, the pH stabilizes at a value called the ultimate pH (usually close to 5.7). The acidification of the muscle reduces the net charge of myofibrillar proteins and is close to their isoelectric point (around 5). The slightest repulsion of the proteins comprising the myofilaments leads to a tightening of the latter and a lateral contraction of the myofibrils (Pearce et al., 2011), which expels part of the intramyofibrillar water into the sarcoplasm that then migrates into the extracellular space. In addition to the decrease in pH, the osmotic pressure of the muscle evolves after slaughter. Its value doubles during the onset of rigor mortis (from 270–300 mosmoles to 500–600 mosmoles) (Ouali, 1990a, 1990b, 1992). This increase is directly related to the decrease in pH (Ouali, 1992). The resulting increase in ionic strength, close to 0.30, is sufficient to damage myofibrillar proteins and to promote the action of muscle proteases.

Fast Postmortem Metabolism Effects on Muscle Proteins and Meat Quality

Pronounced stress just prior to slaughter, coupled with the effects of stunning (especially electrical stunning), accelerates the muscle metabolism that continues after the animal dies and leads to a rapid drop in pH while the muscle temperature is still high (Charpentier and Goutefongea, 1963). The low pH/high temperature combination leads to the denaturation of the sarcoplasmic proteins (Bendall and Wismerpedersen, 1962; Charpentier, 1969; Charpentier and Goutefongea, 1963; Fischer et al., 1979; Lawrie et al., 1963; Scopes, 1964; Scopes and Lawrie, 1963) and the myofibrillar proteins, with myosin being the most sensitive (Bendall, 1973; Offer and Knight, 1988; Penny, 1967; Stabursvik et al., 1984). The denaturation of muscle proteins leads to reduction in their water holding capacity. The myofilament lattice retracts, expelling water to the extracellular space (Bendall and Swatland, 1988; Fialik, 1983; Guignot et al., 1993), increasing the ability of meat to reflect light (Macdougall, 1970, 1982). These meats are characterized by strong exudations, a lighter color and a soft texture, hence their name PSE (Pale Soft Exudative) (Adzitey and Nurul, 2011). This phenomenon was first observed in meat from pigs (Bendall and Wismerpedersen, 1962; Briskey, 1964; Hamm, 1960) and then in meat from poultry (Barbut, 2009; Owens et al., 2000; Swatland, 2008) and more recently in meat from sheep (Kim et al., 2014a,b; Warner et al., 2014) and cattle (Aalhus et al., 1998; Guignot et al., 1993; Warner et al., 2014).

Protein denaturation is also observed when the PSE trait is induced by maintaining a muscle at 37 °C for a few hours post slaughter (Penny, 1967, 1977).

Postmortem Proteolysis

Proteolytic Systems

Conditions after slaughter and during carcass chilling favor degradation of intracellular muscle proteins by proteolytic systems. Calpains seem to represent the major system involved in tenderizing meat (Goll et al., 2003, Koohmaraie, 1994, Koohmaraie and Geesink, 2006, Raynaud et al., 2005, Taylor et al., 1995a). It has been concluded that post-mortem muscle tenderization is due mainly to the action of μ-calpain and to a lesser extent the action of *m*-calpain.

Cathepsins and proteasome seem less involved (Sentandreu et al., 2002; Koohmaraie and Geesink, 2006; Koohmaraie, 1994; Taylor et al., 1995b). Other proteolytic systems such as caspases, metalloproteases, thrombin or plasmin could also be involved in the postmortem degradation of muscle tissue (Sentandreu et al., 2002; Ouali et al., 2013).

It has been reported that low pH, elevated temperature and electrical stimulation can all accelerate the release of cathepsins from lysosomes and eventually facilitate muscle tenderization.

Postmortem Muscle Protein Degradation

The consequences of postmortem proteolysis on the ultrastructure of muscle are well understood (Davey and Dickson, 1970; Davey and Gilbert, 1969; Koohmaraie, 1994; Koohmaraie and Geesink, 2006; Ouali, 1990b; Taylor et al., 1995a). Surprisingly, myosin and actin are little affected by post mortem proteolysis, and it is rather the degradation of cytoskeleton proteins that causes a - modification of myofibril ultrastructure and tenderness of the meat. Overall, post mortem proteolysis leads to a transverse rupture of the myofibrils at the connecting junction of the I-band to the Z-disks. This zone is essentially composed of actin, titin, and nebulin. Their postmortem degradation is likely to be responsible for myofibril breakdown (Koohmaraie and Geesink, 2006, Robson et al., 1997, Taylor et al., 1995a).

The cytoskeleton is also disorganized by the degradation of the desmin and the filamin, which bind the Z-disks of myofibrils to each other and also to the sarcolemma from peripheral myofibrils (Huff Lonergan et al., 2010, Taylor et al., 1995b, Koohmaraie and Geesink, 2006). The degradation of the constitutive proteins of the costamere (dystrophin-vinculin-talin-integrin) which constitutes the anchor point of desmin on the sarcolemma (Taylor et al., 1995a; Koohmaraie and Geesink, 2006) is also suspected to change the muscle fiber ultrastructure. The intermediate filaments which bind the myofibrils at the level of the M-lines are also partially hydrolyzed.

Deterioration of these major cytoskeletal proteins causes misalignment of the myofibrils relative to each other, detachment of the sarcolemma and rupture of the myofibrils along the Z-disks. Nevertheless, it appears that the degradation of desmin and titin are the most important of the mechanisms underlying development of tenderness during meat maturation (Koohmaraie and Geesink, 2006). Troponin T, incorporated into the thin filaments, is also degraded during the meat maturation phase, and its disappearance is related to the appearance of a 30 kDa peptide and the tenderness of the meat (Huff Lonergan et al., 2010). This protein is involved in the mechanism of muscle contraction, but it is also suspected to stabilize interactions between myofilaments. Its degradation may favor the disorganization of myofibrils.

Structural changes in intramuscular connective tissue become significant only after 10 days of maturation (Nishimura et al., 1995). In general, maturation increases the solubility of collagen (Purslow, 2005), causing an improvement in the texture of raw meat. However, the effects of postmortem changes in connective tissue on the texture of cooked meat are controversial (Nishimura, 2010; Purslow, 2005).

Postmortem Protein Oxidation

An increase in myofibrillar protein oxidation is observed during ageing (Martinaud et al., 1997; Rowe et al., 2004a,b), that results in carbonyl derivative formation (Levine et al., 1994; Martinaud et al., 1997) and protein disulfide cross-links (Martinaud et al., 1997; Stadtman, 1990). In general, both of these changes reduce the functionality of proteins (Xiong and Decker, 1995). Calpains are particularly susceptible to inactivation by oxidation (Lametsch et al., 2008). Therefore, oxidizing conditions in postmortem muscle decrease the calpain activity and are likely to affect the tenderization of meat. Myoglobin also undergoes oxidation during the refrigeration of carcasses and meat, with significant effects on the color of the meat (Renerre, 2000).

Effects of Processing on Meat Proteins

Meat processing affects the physical and chemical properties of the product by the action of physical forces, heat or the addition of salts, additives or processing aids (Lewis, 1992). For instance, meat tenderization through electrical stimulation, ultrasonic waves, blade tenderization and pressure treatment have been reported to modify the muscle structure and protein profile (Hopkins, 2014). These processes decrease the overlapping of actin and myosin, cause physical damage to the sarcomere and connective tissues or improve proteolysis rates through the activation of calpains by calcium ions released after membrane disruption.

Electrical Stimulation of Carcasses

Electrical stimulation of carcasses has no detectable influence on the degradation of desmin and troponin T (Ho et al., 1997), but could slightly accelerate the degradation of the cytoskeletal proteins titin and nebulin in postmortem muscle (Ho et al., 1996).

Pulsed Electric Field

There has been recent interest in meat tenderization applications of PEF. PEF causes the electroporation of muscle cell membranes, which potentially facilitates the meat ageing process by releasing the calcium ions from sarcoplasmic reticulum, which triggers the calcium-activated proteases and/or speeds up pre-rigor glycolysis, and also by releasing the cathepsins from the lysosomes (Warner et al., 2017). Compared to untreated muscles, PEF-treated cold- and hot-boned beef Longissimus lumborum muscles had increased degradation of troponin T and desmin (Suwandy et al., 2015a, b).

Exogenous Enzyme Technology

Exogenous proteases have been applied in meat tenderization for many years (Payne, 2009; Sullivan and Calkins, 2010). Most plant-based proteases can hydrolyze myofibrillar proteins efficiently (Bekhit et al., 2014; Kim and Taub, 1991). For the stromal proteins, under heating conditions, both papain and ficin were found to be effective in hydrolyzing elastin and collagen whilst bromelain degraded only collagen (more actively than papain and ficin) (El-Gharbawi and Whitaker, 1963; Miyada and Tappel, 1956; Payne, 2009). These enzymes have wide substrate specificity and can break down many of the peptide bonds present in meat proteins (Bekhit et al., 2014; Huff-Lonergan, 2014). Excessive proteolytic action can cause over-tenderization, leading to mushy meat texture (Ashie et al., 2002; McKeith et al., 1994; Weir et al., 1958).

Shockwave Hydrodynamic Processing

Hydrodynamic processing generates a shockwave up to 1 GPa which travels through water in fractions of a millisecond (Bolumar et al., 2014; Hopkins, 2014). A shockwave process can be set up by subjecting the sealed meat in a water-filled container to electrically-generated shockwaves (Hopkins, 2014). Shockwave treatment caused increased intramyofibrillar and endomysium spaces between muscle fibers (Zuckerman and Solomon, 1998; Bolumar et al., 2014) myofibrillar fragmentation alongside the Z-disk (Zuckerman and Solomon, 1998), destruction of collagen fibrils of the endomysium (Zuckerman et al., 2013) and degradation of C-protein, which is responsible for the integrity of the thick filaments (Spanier and Fahrenholz, 2005a). The physical disruption of muscles may lead to the release of endogenous proteases such as calpains and cathepsins, or their activators such as calcium ions, thus enhancing the tenderization effect. This was seen in a study where troponin T of shockwave-treated samples was degraded more than in an untreated control after ageing (Bowker et al., 2008a,b). Conversely, no enhancement in the activity of endogenous proteolytic enzymes (cathepsins and peptidases) was detected after the shockwave process, suggesting that the tenderization effect is mainly due to physical disruption. The effect of shockwave processing on the protein profile is debatable. It has been reported that there was no distinct difference in the myofibrillar, sarcoplasmic and stromal protein profiles between control and shockwave-treated samples (Bolumar et al., 2014; Schilling et al., 2002). However, it has also been reported that shockwave-treated muscle had higher myofibrillar solubility (Bowker et al., 2008a,b; Spanier and Fahrenholz, 2005a, 2005b) and increased collagen solubility.

High Pressure Processing

HPP is one of the technologies applied in the meat industry to improve shelf life, safety, and quality characteristics (texture and color) of foods (Bajovic et al., 2012). HPP subjects the meat to high pressure, usually ranging from 100 MPa to 800 MPa, via a surrounding liquid and is sometimes accompanied by heat treatment at 60 °C (Hopkins, 2014; Troy et al., 2016). HPP causes the destruction of microorganism, leads to protein denaturation and inactivation of endogenous enzymes (Sikes and Warner, 2016; Torres and Velazquez, 2005).

HPP alters protein secondary, tertiary and quaternary structures in the muscles by disrupting their conformations and molecular interactions (Campus, 2010; Sikes and Warner, 2016; Strasburg et al., 2008). For example, at pressures above 100 MPa protein tertiary structure unfolds and protein aggregation occurs (Cheftel and Culioli, 1997). However, covalent bonds are mostly unaffected. High pressure has been reported to denature myofibrillar proteins (Anderson and Parrish, 1989), cause depolymerization of actin (Ikkai and Ooi, 1966), and release cathepsins into the cytoplasm by degrading muscle membranes (Homma et al., 1994; Jung et al., 2000). For example, at pressures between 100 MPa and 300 MPa, myosin and actin were denatured (Angsupanich and Ledward, 1998; Sikes et al., 2010). The effect of high pressure on muscle structure depends on the applied pressure, processing temperature and time, and the muscle type (pre- or post-rigor) (Cheftel and Culioli, 1997). For instance, increases in intermyofibrillar spaces and myofibril shrinkage were observed in Atlantic salmon meat with increasing applied pressure (from 400 to 900 MPa) and time (Gudbjornsdottir et al., 2010).

The tenderness of pre-rigor meat can be improved by HPP (Sikes and Warner, 2016). Accelerated glycolysis of pre-rigor meat may occur when high pressure is applied, resulting in shear force reduction of myofibrillar-based food (Elgasim and Kennick, 1982; Hopkins, 2014; Kennick et al., 1980). The effect of high pressure on the texture of post-rigor meat depends on the processing temperature (Sikes and Warner, 2016). In most studies, high pressure treatment at 30 °C or below did not have a tenderization effect in post-rigor meat. The tenderness of post-rigor sheep muscle (Macfarlane et al., 1981; Macfarlane and Morton, 1978) and chicken breast (Kruk et al., 2011; Zamri et al., 2006) remained unchanged after high pressure, low temperature processing. In contrast, high pressure processing combined with heat treatment was effective in improving meat tenderness (Bouton et al., 1977; Rusman et al., 2007; Sikes et al., 2010).

High pressure processing affects the color of meat (Cheftel and Culioli, 1997). At pressures higher than 400 MPa, the oxidation of heme iron and denaturation of myoglobin in raw meat occur and cause an increase in lightness and decrease in the redness of meat. This increase in lightness was also observed in bluefish Pomatomus saltatrix as the applied pressure increased from 100 to 300 MPa (Ashie and Simpson, 1996).

Effects of Cooking on Meat Proteins

Sous Vide Cooking

Sous vide is a culinary technique developed in fine dining restaurants, where food is sealed under vacuum and cooked at a controlled temperature, usually in a water bath (Baldwin, 2012; Schellekens, 1996). Cooking in this way results in uniform and efficient heat transfer from the water to the food, overcoming the drawback of uneven heating encountered in conventional cooking processes, while retaining water, soluble components and volatiles in the food and avoiding oxidation. During the cooking process, heat denatures proteins, leading to physical changes in the meat. The effect of cooking on muscle texture largely depends on the cooking time and temperature as well as the heating rate.

The myofibrillar proteins (mostly myosin and actin) shrink during heating, causing the contraction and shrinkage of the muscle fibers (Baldwin, 2012; Christensen et al., 2011a,b). When meat is subjected to heat from 40 °C to 60 °C, muscle fibers shrink

transversely which causes a widening of the gap between them (Palka and Daun, 1999; Tornberg, 2005). As the temperature further increases, muscle fibers shrink longitudinally, and the water held between thick and thin filaments is expelled. The sarcoplasmic proteins start to aggregate and gel when heated at 40 °C to 60 °C (Baldwin, 2012). At sous vide cooking temperatures, most of the endogenous proteases remain active and contribute to enhancing the tenderness of the meat (Laakkonen et al., 1970a,b). At 55 °C, cathepsins B and L were found to be active during up to 24 hours of cooking, whereas both μ- and *m*-calpains were inactivated within 10 minutes (Ertbjerg et al., 2012). Connective tissues, such as collagen, denature at around 60 °C (Baldwin, 2012). This leads to the formation of water-soluble random-coiled gelatin, decreasing the adhesion between muscle fibers. Collagen in beef perimysium was observed to melt when cooked at 50 to 60 °C, while elastin remained heat stable at 100 °C (Taylor, 2004). DSC analysis showed that prolonged heating at 53 °C (for 19.5 hours) resulted in diminishing of myosin and collagen peaks of meat from bull Semitendinosus muscle in the thermograph, suggesting potentially improved meat tenderness (Christensen et al., 2013).

The temperature of sous vide cooking should be set where it is high enough for collagen solubilization and microbiological inactivation, yet has minimum myofibrillar shrinkage to achieve optimum tenderization action (Ruiz et al., 2013). When a tough meat cut is cooked at a temperature between 55 °C and 60 °C for 24 hours, the tenderness is improved as the collagen is converted to gelatin and the myofibrillar proteins are hydrolyzed by the endogenous proteolytic enzymes (Bouton and Harris, 1981; Tornberg, 2005). A decrease in shear force was observed with an increase in cooking temperature from 50 °C to 65 °C, but the shear force increased when the temperature exceeded 65 °C and up to 80 °C (Baldwin, 2012). It has been suggested that the reduction in shear force is due to the gel formed from the sarcoplasmic protein, which filled the channels between fiber bundles, resulting in a decrease in elastic modulus that requires lesser tensile stress to fracture the meat (Tornberg, 2005).

Other Cooking

Cooking of meat other than sous vide does not have the same precise control of temperature and generally exposes at least part of the meat to much higher temperatures than encountered in sous vide cooking, although other parts may be hardly heated at all (for example in the center of a "blue" steak). Chefs and consumers often judge the "doneness" of meat by its color, and this is mostly due to the degree of heme oxidation of myoglobin. During the cooking process, deoxymyoglobin, metmyoglobin and oxymyoglobin undergo oxygenation, oxidation and reduction reactions, and the ratio between them determines the color of final product (Liu and Chen, 2001). The color of the cooked meat also depends on the rate in achieving the designated core temperature and the duration held at that temperature. Cooked meat tends to be redder when the rate of heating is faster and paler when it is held at specific temperatures longer (Baldwin, 2012).

Important temperature effects are summarized in **Table 2** (McGee, 2004).

Nutritional Value and Digestion

Muscle-based food is an excellent source of protein nutrition. The compositions of lean tissue in muscle-based foods are consolidated in **Table 3** (Foegeding et al., 1996; Strasburg et al., 2008; Williams, 2007). About 17% to 23% of lean muscle is made up of protein that contains all the dietary essential amino acids. Consuming meat together with plant-based foods, such as cereals and legumes, can compensate for the lower levels of lysine (cereals), and sulfur amino acids (legumes) in the diet (Bodwell and

Table 2 Effects of heat on meat proteins and qualities

Temperature (° C)	*Meat qualities*	*Proteolytic enzymes*	*Fibrillar proteins*	*Collagen*	*Protein-bound water*	*Myoglobin status*
40	Soft Smooth Translucent	Active	Beginning to unfold		Begins to escape	
50	Firming Becoming opaque	Very active	Myosin begins to denature			
55	More fibrous, Juicy when cut	Denature and lose activity	Myosin coagulated			
60	Shrinking Losing resilience Losing juice Pink		Other proteins denature	Collagen sheaths shrink, squeeze cells	Flows from cells under collagen pressure	Begins to denature ("medium" doneness)
70	Shrinking Stiff Little juice Gray-brown			Begins to denature and dissolve	Flow ceases	
80			Actin denatures Cell contents compacted			

Adapted from McGee, H., 2004. On Food and Cooking – The Science and Lore of the Kitchen, second ed. Scribner, New York.

Table 3 Composition of lean muscle tissues

	Proximate composition (%)			
Species	*Water*	*Protein*	*Lipid*	*Ash*
Beef[a,b,c]	70–73	20–23	3–8	1
Pork[b,c]	68–70	19–20	9–11	1.4
Chicken[b,c]	73.7	20–23	4.7	1
Lamb[a,b,c]	73	20–22	5–6	1.6
Cod (lean fish)[b,c]	81.2	17.6	0.3	1.2
Salmon (fatty fish)[b,c]	64	20–22	13–15	1.3

[a]Williams (2007).
[b]Strasburg et al. (2008).
[c]Foegeding et al. (1996).

Anderson, 1986). Protein from meat is highly digestible and bioavailable compared to plant protein (Černá, 2011), except after severe processing which can modify amino acid side chains (Soladoye et al., 2015). Muscle-based foods, especially red meats, are a rich source of highly bioavailable iron, due to their myoglobin content.

Protein digestibility, which is largely dependent on the source and processing history of food, is considered to be an important factor determining the amount of undigested dietary protein reaching the large intestine (Yao et al., 2016). It has been reported that increasing cooking temperature and time can decrease meat protein digestibility (Astruc, 2014b; Li et al., 2017; Kaur et al., 2014). High pressure-treated meat was found to be more digestible than the untreated control sample, both in vitro (Kaur et al., 2016) and in vivo (Elgasim and Kennick, 1980). Long-time salting and drying rendered the meat protein to be less digestible by pepsin in the stomach (Li et al., 2017).

Sustainability of Meat as a Source of Protein

Real Cost of Meat, Water and Carbon Footprints

There has been much comment on the environmental cost of producing meat. A commonly quoted "fact" is that it takes 10 kg of plant protein to produce 1 kg of meat protein. This is a worst-case figure and largely incorrect because:

- It assumes the animal is eating food that could otherwise have been used by humans
- It ignores other contributions made by the animal – often a lifetime of milk or egg production or of providing motive power, as well as the other products from the carcass – offal, hides and gelatin, for example
- Animals are often farmed on land that is too steep or inaccessible to use for cropping, thus any opportunity cost for land use is, in the case of these animals, illusory
- In developing countries (and increasingly in developed countries) animals are fed on waste food that is no longer fit for human consumption.

Production of meat requires a lot of water. Meat animals are often farmed in regions where there is an excess of water available through plentiful rainfall – where no opportunity cost for water exists. Water management will be an increasingly important consideration for all kinds of farming. Farming for animal products, including meat, is appropriate under certain conditions.

The carbon footprint of meat production arises mostly because of production of methane by the animals during their lifetime. Methanogenesis occurs mostly in the rumen from the metabolism of methanogenic bacteria in the rumen population. It is generally undesirable, not only because it produces atmospheric methane, but because it results in lost energy and carbon that could otherwise benefit the animal. Research is underway globally to address this issue and there is hope that in the future ruminants will be able to be largely methanogen-free.

Importance of the Ruminant

It is a simple fact that almost all food must originate from plant material, because photosynthesis is the ultimate source of almost all organic material. The route by which this material can become food for humans depends on the palatability and digestibility of the plant material. Ruminant animals, by virtue of their completely different digestive systems, can consume plant material such as grasses and the leaves of bushes and trees and convert it into forms that can be valuable nutrition for humans (milk and meat). Without the intervention of the animal, these food sources would be lost to us.

The Future of Meat

The future of meat and the prospects of meeting future demand for meat and other animal-based sources of protein have been addressed by several groups, and potential solutions include more production through intensification and increasing grasslands

(at an environmental cost that may be unacceptable), recycling food waste to feed animals and using alternative sources of animal fodder, such as insects. Laboratory-grown meat has also made an appearance, albeit only as a prototype, but may be expected to have an influence in the future, as will plant-origin meat substitutes, but meat will continue to occupy an important place in the planet's food ecosystem.

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