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Review

Biochemistry of postmortem muscle — Lessons on mechanisms of meat tenderization

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ABSTRACT

It is certain that meat tenderness is a highly valued consumer trait and thus definition of the multiple processes that influence meat tenderness will provide clues toward improving meat quality and value. The natural process by which meat becomes tender is complex. Tenderness development is dependent on the architecture and the integrity of the skeletal muscle cell and on events that modify those proteins and their interaction. Specifically protein degradation and protein oxidation have been identified as processes that modify proteins as well as the tenderness of meat. The intracellular environment is a major factor that controls these events. Ultimately, the interplay between these events determines the rate and extent of tenderization. Given the intricacy of the structure of the muscle cell, coupled with the complexity of the regulation of protein modification and the ever-changing intracellular environment it is not surprising that this area of research is a very dynamic field. Just as the overall integrity and function of muscle cells does not depend on a single protein, but rather on the coordinated interaction of several proteins, the structural weakening of muscle cells during postmortem aging also must not depend on the degradation of a single myofibrillar or other cytoskeletal protein. The proteins mentioned in this review are located in different regions of the muscle cell, and most have been implicated in some manner as being important in maintaining the structure and function of the muscle cell. Oxidation of myosin heavy chain, a predominant protein in the myofibril, is known to promote aggregation and toughening of meat. Degradation of proteins such as desmin, filamin, dystrophin, and talin (all located at the periphery of the Z-line) may disrupt the lateral register and integrity of the myofibril themselves as well as the attachments of the peripheral layer of myofibril to the sarcolemma. Degradation of the proteins within the myofibril that are associated with the thick and thin filaments may allow lateral movement or breaks to occur within the sarcomeres of postmortem aged samples. Titin, nebulin, and troponin-T, by their ability to directly interact with, or modulate the interaction between, major proteins of the thick and thin filaments and (or) the Z-line, play key roles in muscle cell integrity. Disruption of these proteins, especially titin and nebulin, could initiate further physicochemical and structural changes that result in myofibril fragmentation and loss of muscle cell integrity, and ultimately in tenderization of the muscle. In order to make real progress in this area, the scientific community must have a global appreciation of how both the structural proteins and the key proteases are influenced by the vast changes that occur during the conversion of muscle to meat.

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1. Introduction

From one point of view, the question of meat tenderness is quite simple; it is either tender or it is not. However, the explanation of observed variation in meat tenderness often exposes a process with many different pathways that contribute to the development of meat tenderness. From a physical standpoint, the development of tenderness is dependent on the architecture, the integrity of the skeletal muscle cell, the activity of endogenous proteases within the cell and the extracellular matrix (McCormick, 2009). To make matters more complex, the intracellular environment, the availability and the timing of the availability of metabolites are additional controlling factors that must be considered. In fact, it is in actuality, the interplay between these systems that is the critical factor in determining the rate of tenderization. Given the intricacy of the structure of the muscle cell, coupled with the complexity of the regulation of protease activity, and the ever-changing intracellular environment it is not surprising that this area of research is a very dynamic field, and has been for numerous years. While the extracellular matrix is also critical to tenderness, discussion of it is beyond the scope of this review. Readers are encouraged to read these references to gain a greater appreciation of this area of research (Nishimura, 2010; Purslow, 2005).

It is interesting to that in 1948, Bate-Smith (Bate-Smith, 1948) noted "because of the bewildering rate of growth of this fundamental knowledge (of physiological and biochemical properties and behavior of muscle) and the constantly changing conception of muscle which has resulted, there has not been so striking an advance in knowledge of the particular processes involved in the prolonged storage of meat, nor any striking application of the principles of modern biochemistry to the technology of handling of animals and meat" (Bate-Smith, 1948). Even though the scientific community has continued to make great progress in understanding the physiology and biochemistry of muscle, we are still discovering a bewildering array of factors that can influence meat quality, and still are struggling with the ideal predictors of beef tenderness. Lowe (Lowe, 1948) noted that in poultry, microscopic changes were noted that seemed to parallel changes in tenderness. This work has landmark and has been shown numerous times to be the same in multiple species including beef and point out the importance of understanding muscle structure and its relationship to tenderness.

2. Muscle structure and metabolism

Muscle cells are among the most highly organized cells in the animal body. This is because they perform a diverse array of mechanical functions. They are required for movement of limbs for locomotion and other gross movements, but also, they must perform finer tasks such as maintaining balance and coordination. Muscle movement and metabolism are also associated with other diverse functions such as aiding in maintaining body heat and movement of blood and lymph. Few cells are required to generate as much force and undergo as dramatic shifts in rate of metabolism as muscle cells. Thus the organization, structure and metabolism of the muscle are keys to its function and to the maintenance of its integrity both during contraction and during the early postmortem period.

Muscle cells are striated, meaning that when viewed under a microscope, distinct banding patterns or striations are observed. This appearance is due to specialized organelles, myofibrils, found in muscle cells. Myofibrils are the contractile "machinery" of the cell, and, like the cells they reside in are very highly organized. When examining a myofibril, one of the first observations that can be made is that the cylindrical organelle is made up of repeating units. These repeating units are known as sarcomeres. Contained in each sarcomere are all of the structural elements needed to perform the physical act of contraction at the molecular level. Current proteomic analysis estimates that over 65 proteins make up the structure of the sarcomere (Fraterman, Zeiger, Khurana, Wilm, & Rubinstein, 2007) however, the actual number is likely far greater than this. Given that this the sarcomere is the most basic unit of the cell and that the number quoted in this analysis did not take into account the multiple isoforms of the proteins, this number is quite high. Many of the proteins interact with each other in a highly coordinated fashion, and some of the interactions are just now being discovered. Proteolysis or other modification of relatively minor or less studied proteins may impact the structure of the sarcomere/myofibril in a subtle manner that may not be realized until a later time postmortem. This paper will describe some of the major changes in the myofibril during postmortem storage and will explore some of the possible changes that may be related to tenderness.

The structure of the sarcomere is responsible for the striated appearance of the muscle cell. The striations arise from alternating, protein dense A-bands and less dense I-bands within the myofibril. Bisecting the I-bands are dark lines known as Z-lines. The structure between two Z-lines is a sarcomere. The less dense I-band is made up primarily of thin filaments while the A-band is made up of thick filaments and some overlapping thin filaments (Goll, Robson, & Stromer, 1984). The backbone of the thin filaments is made up primarily of the protein actin while the largest component of the thick filament is the protein myosin. Myosin consists of a tail or rod region that forms the backbone of the thick filament and a globular head region that extends from the thick filament and interacts with actin in the thin filament. In order for contraction to occur, the thick and thin filaments interact via the head region of myosin. The complex formed by the interaction of myosin and actin is often referred to as actomyosin. In electron micrograph images of contracted muscle or of post-rigor muscle the actomyosin bond looks very much like cross-bridges between the thick and thin filaments, indeed, it is often referred to as such. In postmortem muscle these bonds become irreversible and are also known as rigor bonds as they are the genesis of the stiffness that develops in postmortem muscle. The globular head of myosin also has enzymatic activity; it can hydrolyze ATP and liberate energy. In living muscle during contraction, the ATPase activity of myosin provides energy for myosin bound to actin to swivel and ultimately pull the thin filaments toward the center of the sarcomere. This shortens the myofibril, the muscle cell and eventually the muscle to produce contraction. The myosin and actin can disassociate when a new molecule of ATP is bound to the myosin head (Goll et al., 1984). In post-rigor muscle, the supply of ATP is depleted, resulting in the actomyosin bonds becoming essentially permanent. In meat, the amount of shortening of the myofibrils will