NSAP 47th Annual Conference (JOS 2022) CONFERENCE PROCEEDINGS
THEME: SECURING ANIMAL AGRICULTURE AMIDST GLOBAL CHALLENGES

MEAT CONTRACTION AND RELAXATION: A REVIEW OF EFFECT ON MEAT TENDERNESS

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ABSTRACT

The demand for high-quality meat has increased. Flavor, juiciness, and tenderness are the three main attributes that influence the sensory enjoyment of meat. Tenderness has been shown to have the largest role in consumer purchasing decisions. The tenderness of a piece of meat is a result of several factors which can be considered sequentially. After an animal is slaughtered, blood circulation enough energy to contract the muscles, and also produces lactic acid. With no blood flow to carry the lactic acid away, the acid builds up in the muscle tissue. If the acid content is too high, the meat loses its water-binding ability and becomes pale and watery. If the acid is too low, the meat will be tough and dry. The lactic acid buildup also releases calcium, which causes muscle contraction. As glycogen supplies are depleted, ATP regeneration stops, and the actin and myosin remain locked in a permanent contraction called rigor mortis. Freezing the carcass too soon after death keeps the proteins all bunched stops, and muscles exhaust their oxygen supply. Muscle can no longer use oxygen to generate ATP (adenosine triphosphate) and turn to anaerobic glycolysis, a process that breaks down sugar without oxygen, to generate ATP from glycogen, sugar stored in muscle. The breakdown of glycogen produces together, resulting in very tough meat. Aging allows enzymes in the muscle cells to break down the overlapping proteins, which makes the meat tender. Without ATP, actin and myosin remain locked in a permanent contraction called rigor mortis.

Keywords: Meat, contraction, relaxation, tenderness, ATP (adenosine triphosphate).

INTRODUCTION

Meat is mostly the muscle tissue of an animal. Most animal muscle is roughly 75% water, 20% protein, and 5% fat, carbohydrates, and assorted proteins. Muscles are made of bundles of cells called fibers (The Accidental Scientist (2002). In a live animal, these protein filaments make muscles contract and relax. Both actions require enormous amounts of energy, which they get from the energy-carrying molecule ATP (adenosine triphosphate). The most efficient generation of ATP requires oxygen, which muscles get from circulating blood. ATP powers muscle contraction Each cell is crammed with filaments made of two proteins: actin and myosin. (The Accidental Scientist (2002). The source of energy that is used to power the movement of contraction in working muscles is adenosine triphosphate (ATP) – the body's biochemical way to store and transport energy (Anon (2019). The ATP is not stored to a great extent in cells. So once muscle contraction starts, the making of more ATP must start quickly. Since ATP is so important, muscle cells have several different ways to make it (Anon (2019). These systems work together in phases. The three biochemical systems for producing ATP are, in order: using creatine phosphate, using glycogen, aerobic respiration.

Creatine Phosphate (with oxygen)

All muscle cells have a little ATP within them that they can use immediately – but only enough to last for about 3 seconds! So all muscle cells contain a high-energy compound called creatine phosphate which is broken down to make more ATP quickly (Anon (2019). Creatine phosphate can supply the energy needs of a working muscle at a very high rate, but only for about 8–10 seconds (Anon (2019).

Glycogen (without oxygen)

Fortunately, muscles also have large stores of a carbohydrate, called glycogen, which can be used to make ATP from glucose. But this takes about 12 chemical reactions so it supplies energy more slowly than from creatine phosphate (Anon (2019). It's still pretty rapid, though, and will produce enough energy to last about 90 seconds. Oxygen is not needed – this is great, because it takes the heart and lungs some time to get increased oxygen supply to the muscles. A by-product of making ATP without using oxygen is lactic acid (Anon (2019).

Aerobic Respiration (with oxygen again)

Within two minutes of exercise, the animal body starts to supply working muscles with oxygen. When oxygen is present, aerobic respiration can take place to break down the glucose for ATP. This glucose can come from several places (Anon (2019). Remaining glucose supply in the muscle cells, glucose from food in the intestine, glycogen in the liver fat reserves in the muscles in extreme cases (like starvation), the body's protein. Aerobic respiration takes even more chemical reactions to produce ATP than either of the above two systems. It is the slowest of all three systems – but it can supply ATP for several hours or longer, as long as the supply of fuel lasts. (Anon (2019)

Contraction phase

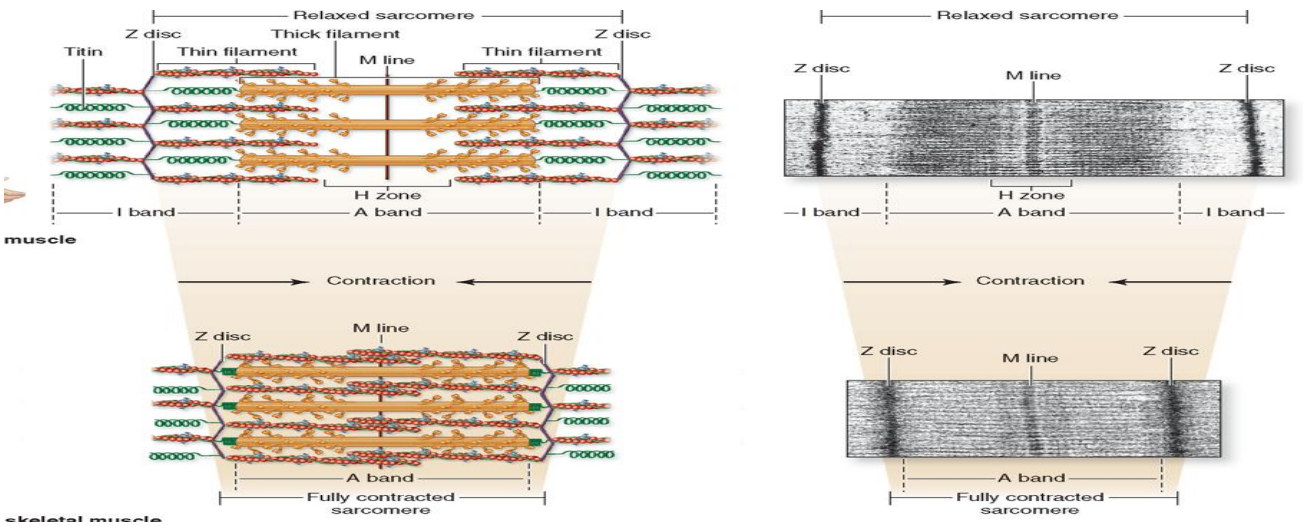
The sequence of events leading to contraction is initiated in the central nervous system, either as voluntary activity from the brain or as reflex activity from the spinal cord (Gerrard *et al.*, 2012). A motor neuron in the ventral horn of the spinal cord is activated, and an action potential passes outward in a ventral root of the spinal cord. The axon branches supply a number of muscle fibers called a motor unit, and the action potential is conveyed to a motor endplate on each muscle fiber (Gerrard *et al.*, 2012). At the motor endplate, the action potential causes the release of packets or quanta of acetylcholine into the synaptic clefts on the surface of the muscle fiber (Gerrard *et al.*, 2012). Acetylcholine causes the electrical resting potential under the motor end plate to change, and this then initiates an action potential that passes in both directions along the surface of the muscle fiber. At the opening of each transverse tubule onto the muscle fiber surface, the action potential spreads inside the muscle fiber (Gerrard *et al.*, 2012). At each point where a transverse tubule touches the part of the sarcoplasmic reticulum, it causes the sarcoplasmic reticulum to release Ca++ ions. The calcium ions result in the movement of troponin and tropomyosin on their thin filaments, and this enables the myosin molecule heads to "grab and swivel" their way along the thin filament. This is the driving force of muscle contraction. The Contraction is turned off by the Acetylcholine at the neuromuscular junction is broken down by acetylcholinesterase, and this terminates the stream of action potentials along the muscle fiber surface. The sarcoplasmic reticulum ceases to release calcium ions and immediately starts to sequester all the calcium ions that have been released. In the absence of calcium ions, a change in the configuration of troponin and tropomyosin then blocks the action of

the myosin molecule heads, and contraction ceases. In the living animal, an external stretching force, such as gravity or an antagonistic muscle, pulls the muscle back to its original length. For the next 8–10 seconds, your muscles use creatine phosphate stores to provide ATP.

- Motor nerve action potential arrives at the motor endplate
- Acetylcholine released, sarcolemma and membranes depolarized (Na+ flux into fiber)
- Action potential transmitted via T-tubules to SR
- Ca++ released from SR terminal cisternae into the sarcoplasm
- Ca++ bound by troponin
- Myosin ATPase activated and ATP hydrolyzed
- Tropomyosin shift from actin binding site
- Actin-myosin crossbridge formation

Relaxation Phase

In subsequent relaxation, contractile impulses from the CNS ceases which relaxing impulses arrive at the motor endplate causing the massive preferential release of the enzyme acetylcholinesterase at the myoneural junction (Lindsay *et al.*, 2021). This enzyme destroys acetylcholine thus setting in train a wave of repolarization. The sarcolemma becomes less permeable to Na^+. Intracellular Na^+, therefore, diffuses outwards into the extracellular fluid. K^+ migrate inwards across the cell membrane. The wave of repolarization spread along the length of the sarcolemma and through the T-tubules to the SR. The SR vesicles release a special protein called the relaxing factor. The relaxing factor enhances the removal of excess Ca^{2+} from the sarcoplasm and trapping these ions in the SR. As free Ca^{2+} concentration in the sarcoplasm falls below 10^{-7}M, troponin molecules release the Ca^{2+} bound to them during contraction (Lindsay *et al.*, 2021). Myofibrillar ATPase (myosin and actomyosin ATPases) are inhibited by the loss of Ca^{2+} from troponin and therefore cease to break down ATP. ATP concentration therefore increases and binds again to Mg^{2+} to form the Mg-ATP complex which prevents the cross-bridges from binding to the actin filaments. These filaments can now passively slide in and out over the myosin or thick filaments thus reestablishing the resting state within the muscle cells (Lindsay *et al.*, 2021).



Source: Lumen (2021)

- Cholinesterase released and acetylcholine breakdown
- Sarcolemma & T-tubules repolarized
- SR Ca++ pump activated & Ca++ returned to SR terminal cisternae
- Actin-myosin crossbridge formation terminated
- Return of tropomyosin to actin binding site
- Mg++ complex formed with ATP
- Passive sliding of filaments Sarcomeres return to resting state

CONCLUSION

To enhance good meat quality such as flavor, juiciness, and tenderness, aging of meat must be allowed. Aging allows enzymes in the muscle cells to break down the overlapping proteins, which makes the meat tender.

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