

Events Involved in Initiating Muscle Contraction

- Nerve impulse reaches axon terminal voltage-gated calcium channels open ACh released to synaptic cleft
- ACh binds to its receptors on sarcolemma opens ligand-gated Na⁺ and K⁺ channels end plate potential Opens voltage-gated Na⁺ channels AP and AP propagation across the sarcolemma down into the t-tubules
- Voltage-sensitive proteins in T tubules (DHP receptors) change shape in response to AP and activate the Ryanodine receptors SR releases Ca²⁺ to cytosol

Muscle Proteins

- Ca²⁺ binds to troponin-C which changes the shape of the troponin-tropomyosin complex and uncovers the myosin binding sites on actin
- Myosin heads of the thick filament will then attach to the myosin binding sites of the thin filament as long as the proper molecules are present

Cross-Bridge Cycle

- Continues as long as sarcoplasmic Ca²⁺ concentrations remain high and adequate ATP present
- Cross-bridge formation—high-energy myosin head (has ADP and inorganic phosphate bound to it) attaches to thin filament
- Power Stroke—myosin head pivots and pulls thin filament toward M line
- Releases ADP and Pi in the process

Length-Tension Relationship

- The tension a muscle fiber is capable of producing is dependent upon the amount of overlap between thick and thin filaments
- Too short (understretched)
- Too much overlap between thick and thin filaments
- Tension generation decreases the more a muscle shortens
- Thin filaments begin to overlap

The Sliding-Filament Mechanism

- Rigor Mortus
- Muscles can become stiff shortly after death
- Can begin 3-4 hours after expiration
- Maximum stiffness at around 12 hours post expiration
- Can take 48-60 hours to subside
- Caused by lack of ATP post death
- Heart stops, blood flow stops, no oxygen/nutrient delivery and waste removal from tissue
- ATP production declines and then ends as cells die
- ATP is not available to bind to the myosin head
- This means the head does not dissociate from the active site of the actin
- ATP no longer available to power calcium pumps that pump calcium out of the sarcoplasm and into terminal cisternae
- This means that intracellular calcium concentration stays high enough to allow the active sites of the thin filament to stay unblocked
- Can be used to determine time of death

Role of Calcium (Ca²⁺) in Contraction

- At low intracellular Ca²⁺ concentration
- Tropomyosin blocks active sites (myosin binding sites) on actin of thin filaments
- Myosin heads of thick filaments cannot attach when binding sites of thin filament are blocked
- Muscle fiber in relaxed/resting state

Muscle Proteins: Thin Filament

- In this graphic the troponin-tropomyosin complex has shifted into the "gutters" of the actin molecule unblocking the myosin binding site
- The troponin-tropomyosin complex can slide back and forth depending on the concentration of Ca²⁺ in the sarcoplasm

Cross Bridge Cycle

- Cross bridge detachment—ATP attaches to myosin head and cross bridge detaches
- "Cocking" of myosin head—energy from hydrolysis of ATP into ADP and inorganic phosphate (Pi) cocks myosin head into high-energy state

Length-Tension Relationship

- Too long (overstretched)
- Too little overlap between thin and thick filaments•Not enough actin/myosin crossbridges are able to be formed
- Tension decreases the more a muscle is stretched

Role of Calcium (Ca²⁺) in Contraction

- At higher intracellular Ca²⁺ concentrations–Ca²⁺ binds to troponin C of the troponin-tropomyosin complex
- Troponin changes shape and moves tropomyosin away from myosin-binding sites
- Unblocks the myosin binding sites
- Myosin heads bind to actin and undergo power stroke causing sarcomere shortening and muscle contraction
- When nervous stimulation ceases (no more ACh release and AP stimulation), the majority of Ca²⁺ in the sarcoplasm is pumped back into SR by SERCA pumps (active transport)
- This causes intracellular calcium concentration to decrease and contraction to end
- Muscle fiber begins to relax
- Some calcium is pumped out of the cell by Ca pumps (active transport) in the sarcolemma

Sliding Filament Model of Contraction

- Myosin heads bind to actin (cross bridge formation)
- Sliding of the thin filaments with respect to the thick begins as power stroke of the myosin heads take place
- Cross bridges form and break several times, ratcheting thin filaments toward center of sarcomere–Causes shortening of muscle fiber
- Pulls Z discs toward M line
- I bands shorten; Z discs closer; H zones disappear; A bands of adjacent sarcomeres move closer together (A band length stays same)

Length-Tension Relationship

- Sarcomere shortening produces tension within a muscle

Length-Tension Relationship

- Optimal resting length
- The ideal thin and thick filament overlap that allows for the maximum number of actin/myosin cross bridge formations
- Maximum tension (force) generated during contraction
- Your muscles actually rest at this length!

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