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-Cwhich 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!

By **jjovann**
cheatography.com/jjovann/

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