NPB 101 Skeletal Muscle Cheat Sheet Cheat Sheet

by kbui04 via cheatography.com/213313/cs/46479/

Sarcomere structure

Structure

-Z-lines (titin) hold thin filaments
-Thick filaments slightly overlap thin filaments
-Middle of thick filaments is A-band, middle of that is M-line
-Zone of thick filament not overlapping thin is H-zone
-Thick filaments have myosin head with ATPase/actin binding site and myosin tail
-Thin filaments have actin/troponin/tropomyosin
-Troponin binds C -> I -> T

Skeletal Muscle

What is Skeletal Muscle?

-In opposition to cardiac and smooth
-Striated and voluntary (cardiac is striated/involuntary while smooth is unstriated/involuntary)
-50% of body strength
-40% body weight in men
-32% body weight in women
-Allows us to purposefully move external objects around
-Squeezes internal hollow organs
-Empties certain organs to external environment

Cross-Bridge Cycle

Cycle

-ATP binds to myosin head ATPase, breaking myosin/actin filament cross bridge
-ATPase splits ATP
-Cross-bridge forms in presence of Ca^{2+} from troponin binding and forming tropomyosin, will not form if no Ca_{2+}
-Pi released to form stronger cross bridge between actin/myosin
-ATP binding again drives power stroke

Calcium transport

Transport

-T-tubules are tubes in the SR, "invaginations", transports Ca^2+
-SR is modified ER with network of T-tubules where Ca^2+ is transported/stored
-Lateral sacs are parts of SR that touch T-tubules
-Foot proteins (ryanodine receptors) span the gap between lateral sacs and T-tubules, modify permeability of t-tubules
-Dihydropyridine receptors are receptors in T-tubule membrane that change foot protein permeability to Ca^2+

Calcium Transport

Muscle tension

Tension

Tension opposes load
-Single action potential is twitch
-Tension develops from frequency, length of fiber, extent of fatigue, thickness of fiber
-Twitch summation increases tension from elevation of cytosolic calcium from repetitive stimulation, duration of action potential shorter than twitch
-Tetanus smooth sustained contraction of maximal strength, 3-4x stronger than twitch
-Optimal muscle length for maximal tension

Muscle metabolism

Metabolism

-Creatine, oxidative phosphorylation, glycolysis
-Type 1 is slow contraction and uses oxidative phosphorylation
-Type 2a is fast contraction, still ox.
-Type 2b is very fast and uses glycolysis, high in glycogen
-Fatigue is CNS no longer activating motor neurons
-Deplete glycogen reserves and inorganic phosphate makes fatigue
-Excess post-exercise oxygen consumption elevated is O2 uptake after exercise

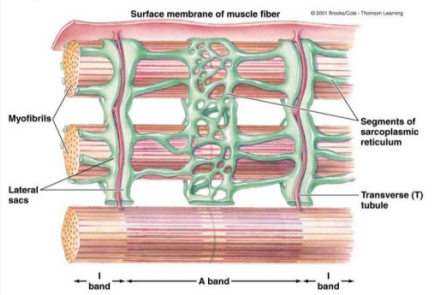
Characteristics

Characteristics of one fiber

-Multiple mitochondria
-Multinucleated
-T tubules
-Myofibrils and sarcomeres
-Sarcolemma (plasma membrane)
Structure from largest to smallest goes muscle fiber, myofibril, A/I bands, sarcomere, Z/M lines and H zone, thick (myosin) filaments, thin (actin) filaments
-Sarcoplasm (cytoplasm)
-Sarcoplasmic reticulum (smooth ER)

Structure

-Myofibrils are elongated, cylindrical contractile elements made of



Motor Unit

Motor Unit

-One motor unit multiple muscle fibers, but muscle fibers only have one motor neuron
-One motor neuron activated, triggers all innervated fibers
-Weak simultaneous contraction of whole muscle if one motor neuron triggered, need recruitment for stronger contraction
-Single motor unit may have 1.5-2k muscle fibers if strong
-Recruitment large increase in tension
-Strength =/= precision

sarcomeres (smallest contractile unit)
-Each sarcomere goes from Z-line to Z-line
-Made of partially overlapping thick and thin filaments
-Each thick filament has 6 adjacent thin filaments
-Each thin filament has 3 adjacent thick filaments
-T-tubules extend membrane throughout muscle cell
-Sarcoplasmic reticulum (SR) surrounds T-tubules and myofibrils

Structure of a muscle fiber

Structure

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Neuromuscular Junction

NMJ sequence (1-4)

1. Action potential enters into terminal button
2. Depolarization of button opens voltage-gated Ca^2+ channels
3. Ca^2+ ions causes vesicles of acetylcholine (Ach) to fuse with the plasma membrane
4. Ach vesicles transported across synaptic cleft which causes binding at motor end plate

Neuromuscular junction

NMJ sequence (5-7)

5. Ach receptor binding opens Na+ channels which depolarizes the end plate
6. Depolarizing current flows to adjacent membrane with voltage gated Na+ channels
7. Ach degraded by acetylcholinestrase (Ach-esterase), terminating Ach action

Cross-Bridge consequences

Consequences

Binding -> power stroke -> detachment -> binding

1. Sarcomere shortens
2. H-zone shortens
3. I-band shortens
4. A-bands stay the same
5. Actin/myosin fibers stay the same

Sarcomere