



AP Biology

Why Do We Need All That ATP?





Animal Locomotion

What are the advantages of locomotion?

sessile







Lots of ways to get around...









Lots of ways to get around...









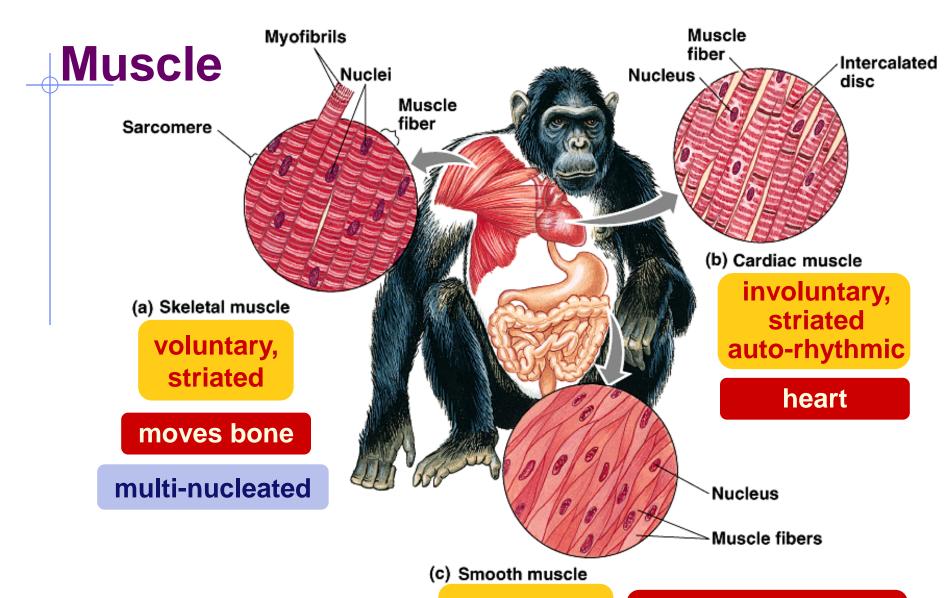
Lots of ways to get around...









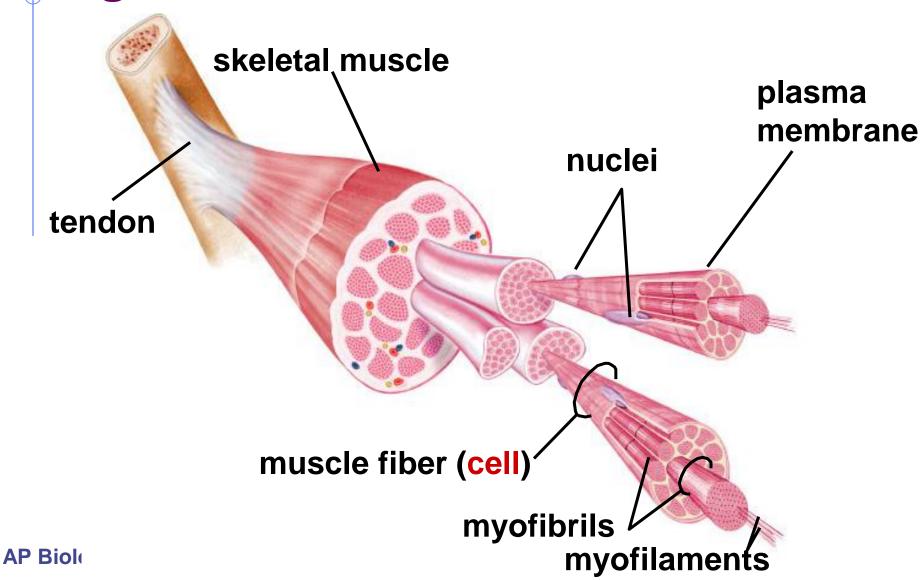


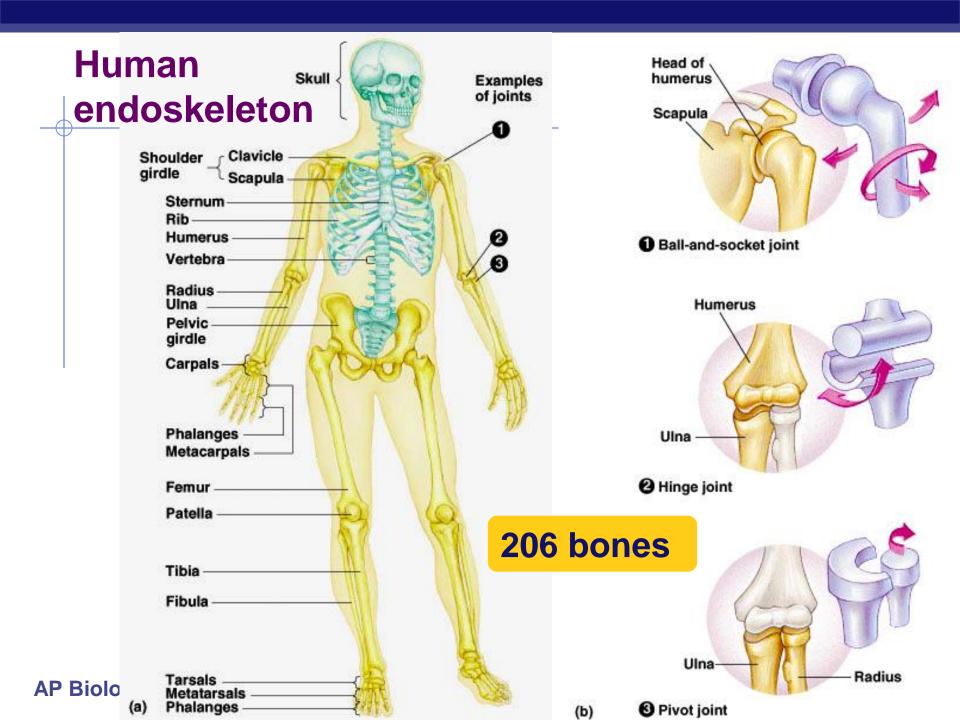
evolved first

involuntary, non-striated

digestive system arteries, veins

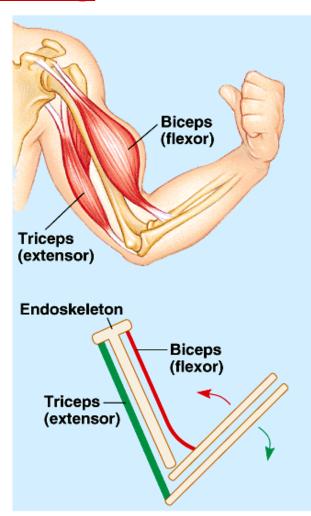
Organization of Skeletal muscle

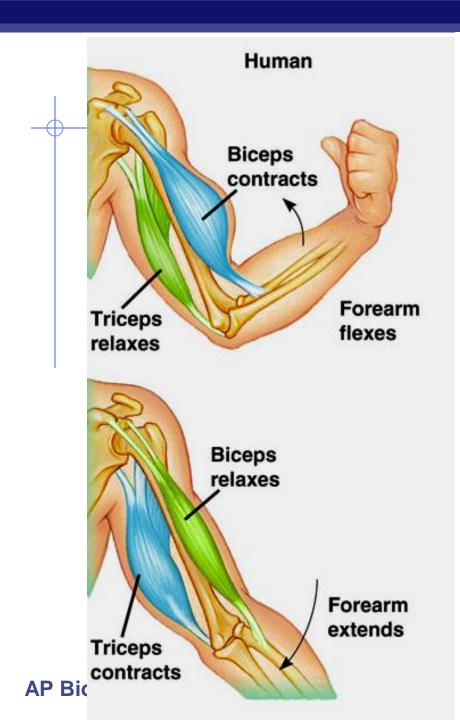


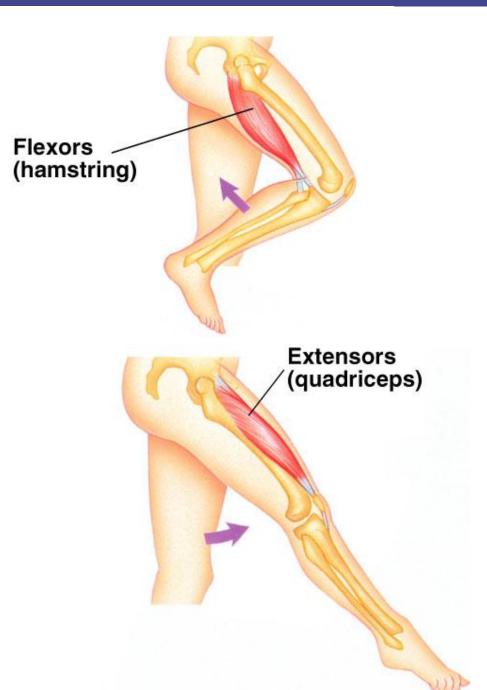


Muscles movement

- Muscles do work by contracting
 - skeletal muscles come in antagonistic pairs
 - flexor vs. extensor
 - contracting = shortening
 - move skeletal parts
 - tendons
 - connect bone to muscle
 - ligaments
 - connect bone to bone







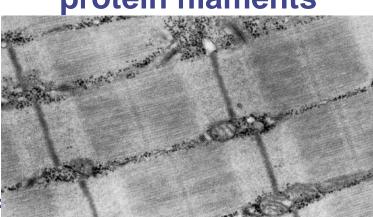
Structure of striated skeletal muscle

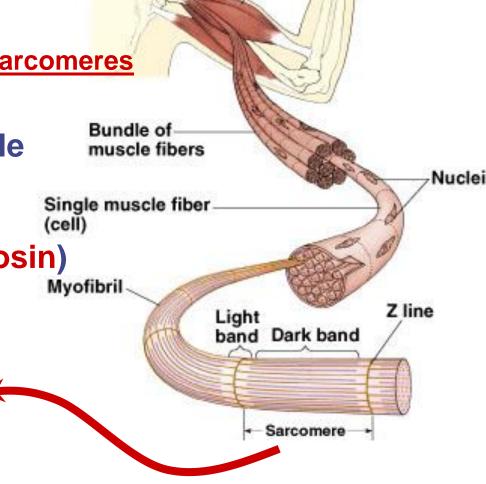


- muscle cell
 - divided into sections = <u>sarcomeres</u>

Sarcomere

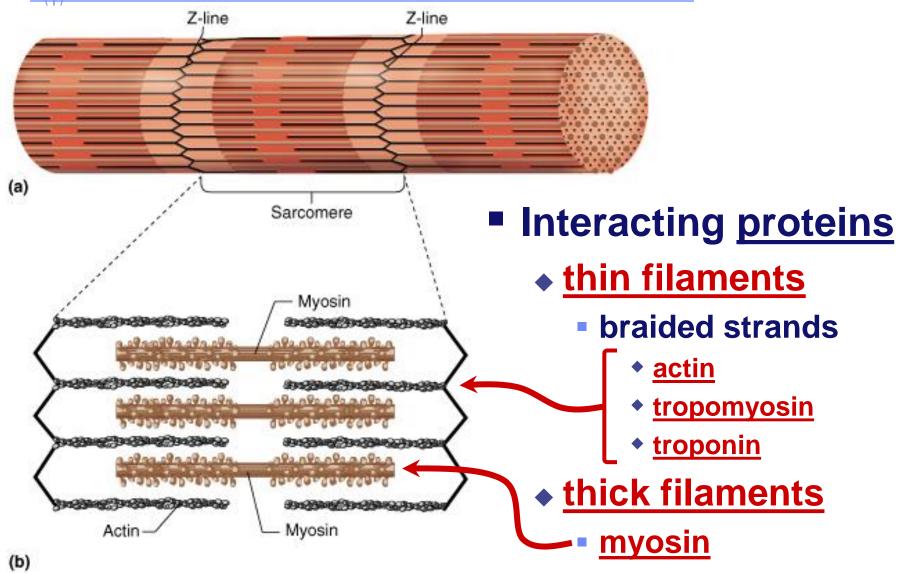
- functional unit of muscle contraction
- alternating bands of thin (actin) & thick (myosin) protein filaments





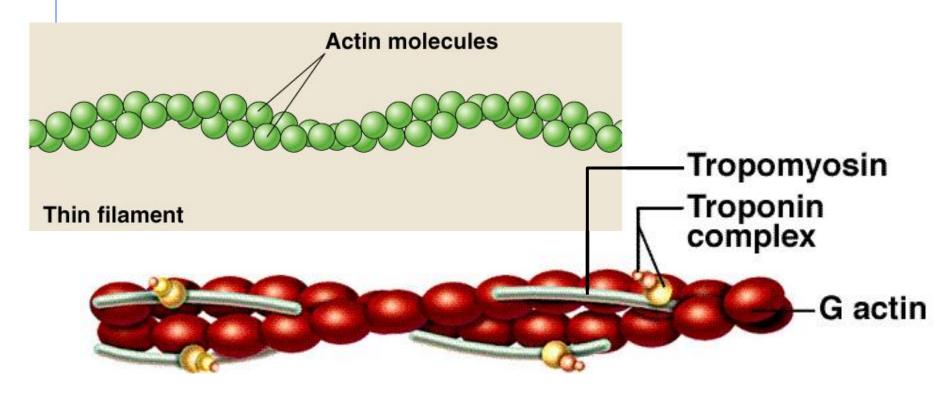
Muscle

Muscle filaments & Sarcomere



Thin filaments: actin

- Complex of proteins
 - braid of <u>actin</u> molecules & <u>tropomyosin</u> fibers
 - tropomyosin fibers secured with <u>troponin</u> molecules



Thick filaments: myosin

- Single protein
 - myosin molecule
 - long protein with globular head

Myosin head

Myosin molecule

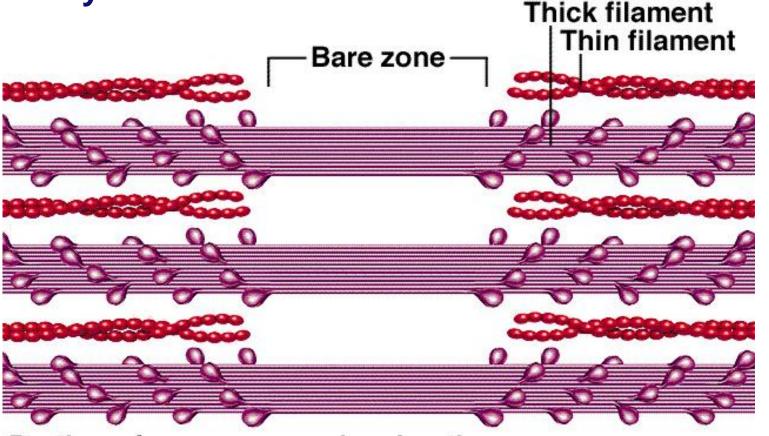
Myosin head

bundle of myosin proteins: globular heads aligned

Thick filament

Thick & thin filaments

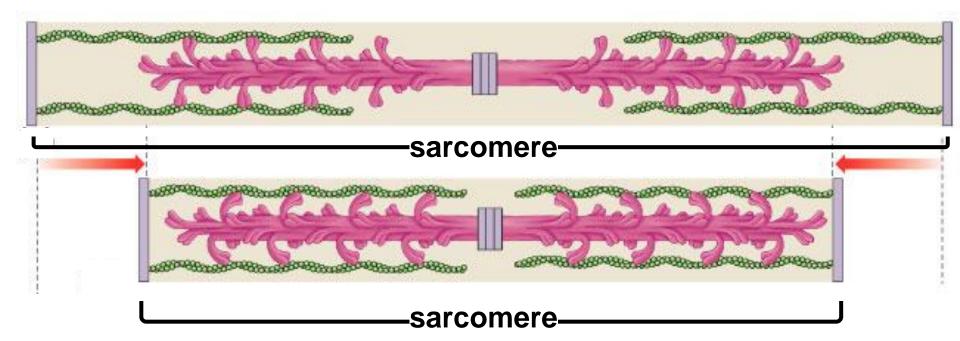
 Myosin tails aligned together & heads pointed away from center of sarcomere_

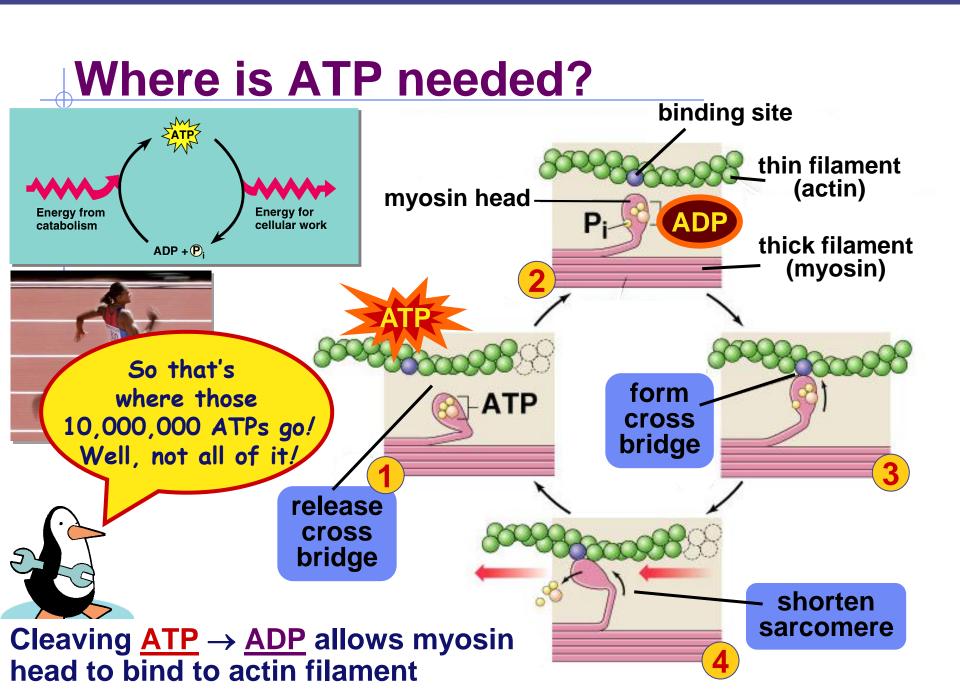


Portion of a sarcomere showing the overlap of thick and thin filaments

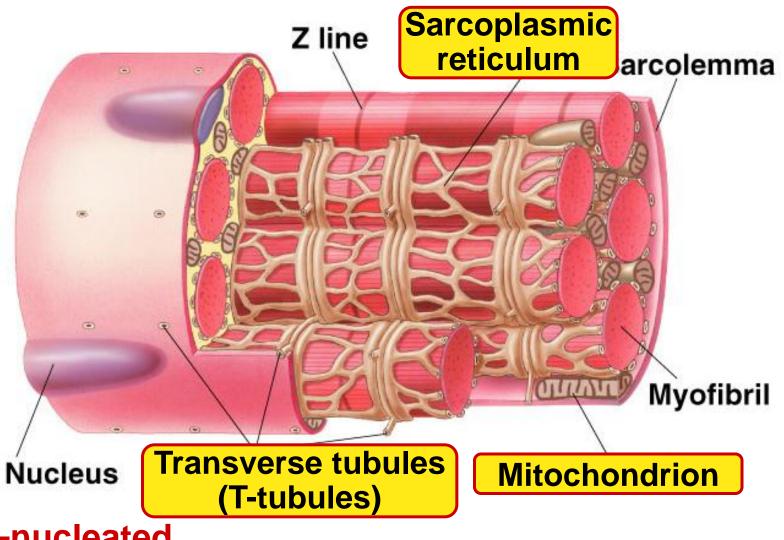
Interaction of thick & thin filaments

- Cross bridges
 - connections formed between <u>myosin heads</u>
 (thick filaments) & <u>actin</u> (thin filaments)
 - cause the muscle to shorten (contract)





Closer look at muscle cell

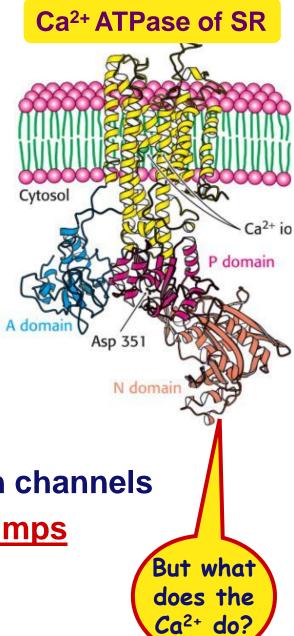


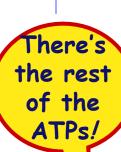
multi-nucleated

Muscle cell organelles

- Sarcoplasm
 - muscle cell cytoplasm
 - contains many mitochondria
- Sarcoplasmic reticulum (SR)
 - organelle similar to <u>ER</u>
 - network of tubes
 - stores Ca²⁺
 - Ca²⁺ released from SR through channels
 - Ca²⁺ restored to SR by Ca²⁺ pumps
 - pump Ca²⁺ from cytosol
 - pumps use ATP

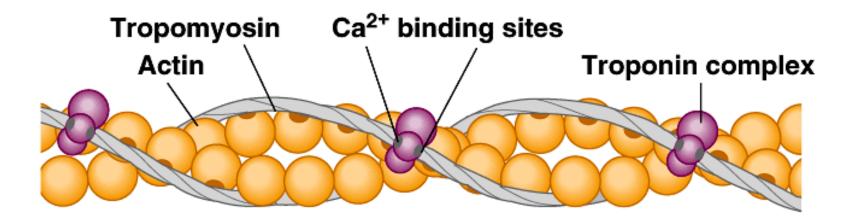






Muscle at rest

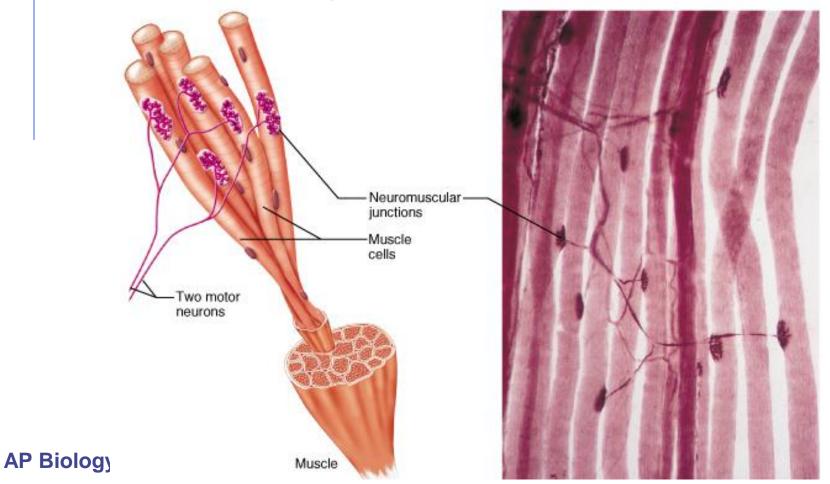
- Interacting proteins
 - at rest, <u>troponin</u> molecules hold <u>tropomyosin</u> fibers so that they cover the <u>myosin-binding</u> <u>sites</u> on <u>actin</u>
 - troponin has Ca²⁺ binding sites



(a) Myosin binding sites blocked; muscle cannot contract

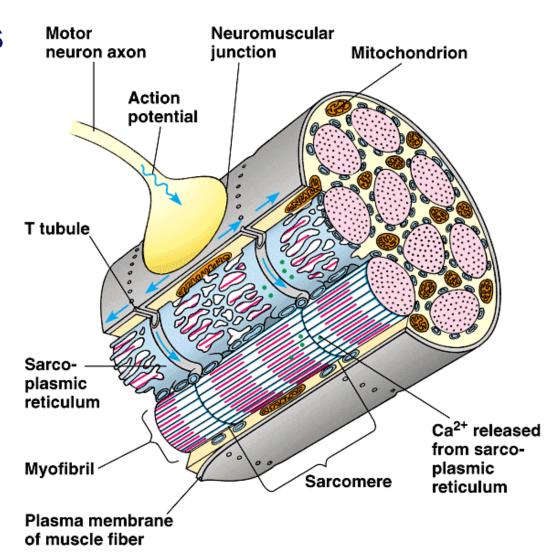
The Trigger: motor neurons

- Motor neuron triggers muscle contraction
 - release acetylcholine (Ach) neurotransmitter



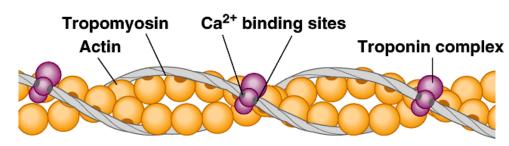
Nerve trigger of muscle action

- Nerve signal travels down T-tubule
 - stimulates
 sarcoplasmic
 reticulum (SR) of
 muscle cell to
 release stored
 Ca²⁺
 - flooding muscle fibers with Ca²⁺

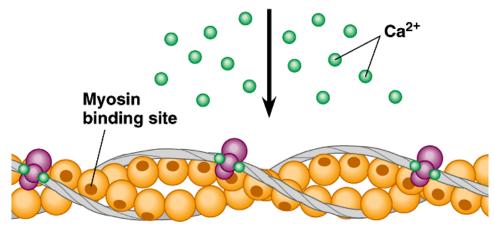


Ca²⁺ triggers muscle action

- At rest, <u>tropomyosin</u>
 <u>blocks myosin-binding</u>
 <u>sites</u> on actin
 - secured by troponin
- Ca²⁺ binds to troponin
 - shape change causes movement of troponin
 - releasing tropomyosin
 - exposes myosinbinding sites on actin



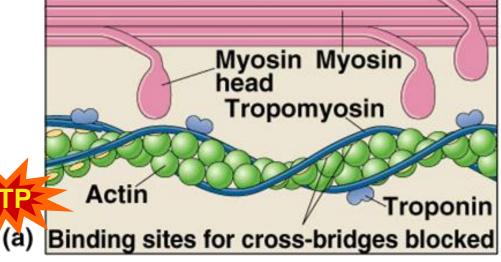
(a) Myosin binding sites blocked; muscle cannot contract

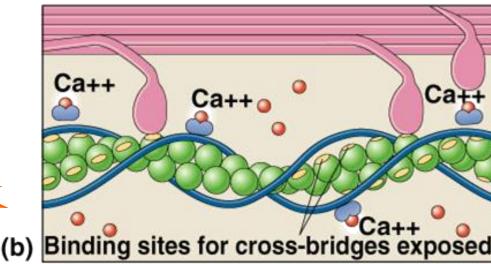


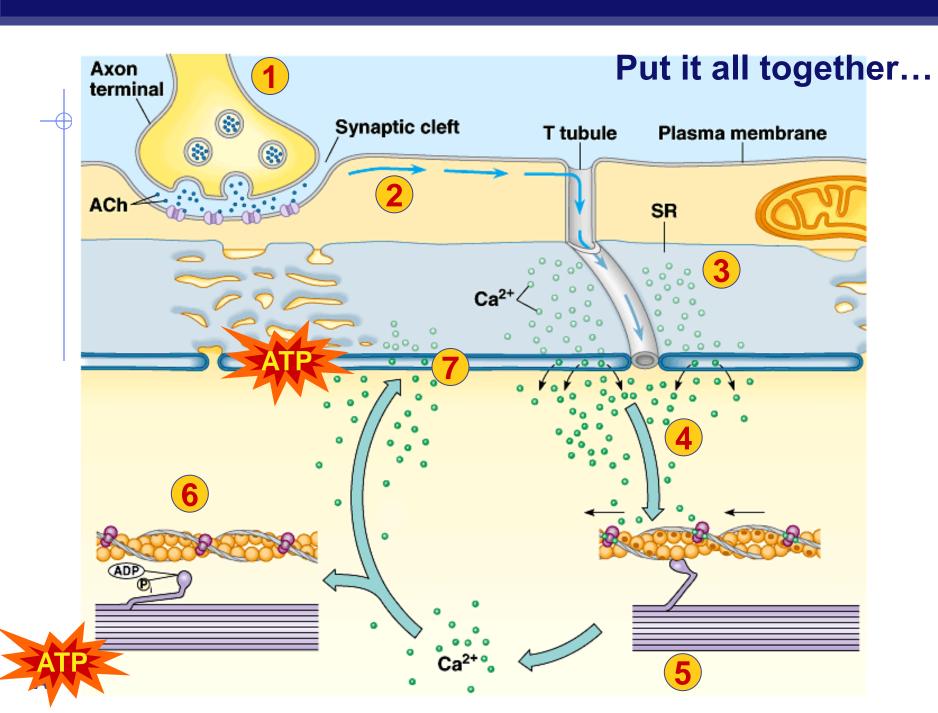
(b) Myosin binding sites exposed; muscle can contract

How Ca²⁺ controls muscle

- Sliding filament model
 - exposed actin binds to myosin
 - fibers slide past each other
 - ratchet system
 - shorten muscle cell
 - muscle contraction
 - muscle doesn't relax until Ca²⁺ is pumped back into SR
 - requires ATP







How it all works...

- Action potential causes Ca²⁺ release from SR
 - ◆ Ca²⁺ binds to <u>troponin</u>
- Troponin moves <u>tropomyosin</u> uncovering <u>myosin</u> <u>binding site</u> on actin
- Myosin binds <u>actin</u>
 - uses <u>ATP</u> to "ratchet" each time
 - releases, "unratchets" & binds to next actin
- Myosin pulls actin chain along
- Sarcomere <u>shortens</u>
 - Z discs move closer together
- Whole fiber shortens → contraction!
- Ca²⁺ pumps restore Ca²⁺ to SR → <u>relaxation</u>!
 - pumps use <u>ATP</u>

Fast twitch & slow twitch muscles

- Slow twitch muscle fibers
 - contract slowly, but keep going for a long time
 - more mitochondria for aerobic respiration
 - less SR → Ca²⁺ remains in cytosol longer
 - long distance runner
 - "dark" meat = more blood vessels
- Fast twitch muscle fibers
 - contract quickly, but get tired rapidly
 - store more glycogen for anaerobic respiration
 - sprinter
 - "white" meat



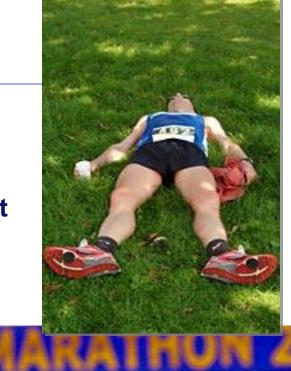
Muscle limits

Muscle fatigue

- lack of sugar
 - lack of ATP to restore Ca²⁺ gradient
- ◆ low O₂
 - lactic acid drops pH which interferes with protein function
- synaptic fatigue
 - loss of acetylcholine

Muscle cramps

- build up of lactic acid
- ATP depletion
- ion imbalance
 - massage or stretching increases circulation





Diseases of Muscle tissue

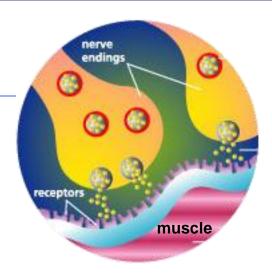
- ALS
 - amyotrophic lateral sclerosis
 - Lou Gehrig's disease
 - motor neurons degenerate
- Myasthenia gravis
 - auto-immune
 - antibodies to acetylcholine receptors





Botox

- Bacteria <u>Clostridium botulinum</u> toxin
 - blocks release of acetylcholine
 - botulism can be fatal







...THE MANY FACES OF THE BOTOX BABE ...















Rigor mortis

- So why are dead people "stiffs"?
 - no life, no breathing
 - ◆ no breathing, no O₂
 - ◆ no O₂, no aerobic respiration
 - no aerobic respiration, no ATP
 - ◆ no ATP, no Ca²⁺ pumps
 - ◆ Ca²⁺ stays in muscle cytoplasm
 - muscle fibers continually contract
 - tetany or rigor mortis
 - eventually tissues breakdown& relax
 - measure of time of death



So don't be a stiff! Ask Questions!!



Ghosts of Lectures Past (storage)

AP Biology 2006-2007

Shortening sarcomere

- Myosin pulls actin chain along toward center of sarcomere
- Sarcomere <u>shortens</u>
 (Z lines move closer together)
- Muscle contracts
 - energy from:
 - ATP
 - glycogen

