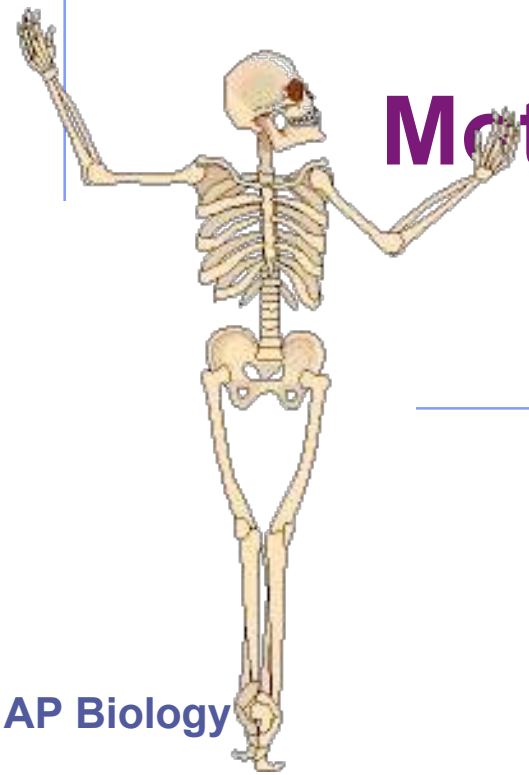
Chapter 49.

Muscles & Motor Locomotion



AP Biology

Animal Locomotion

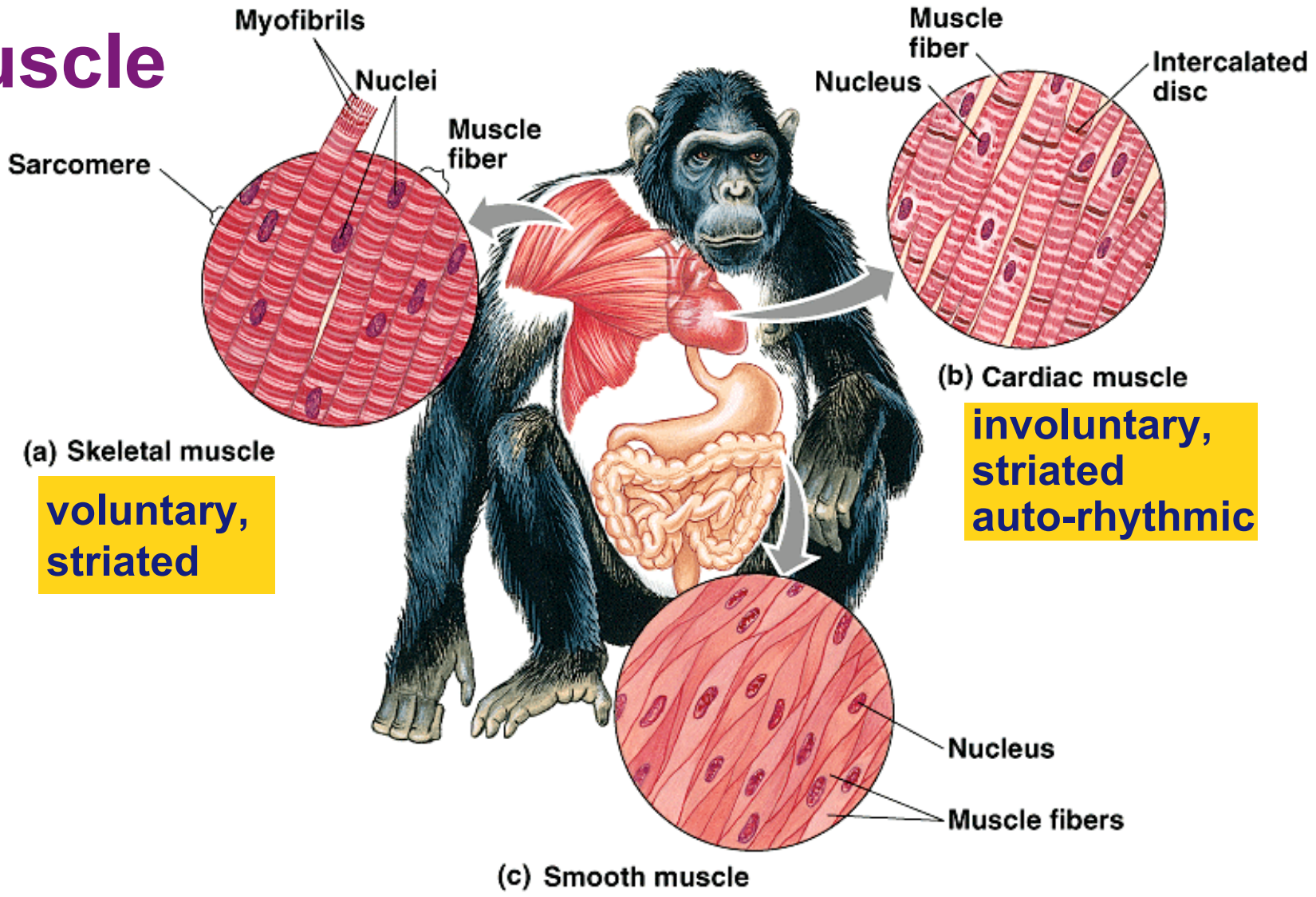
What are the advantages of locomotion?

sessile

motile


Benjamin
Cummings

Muscle

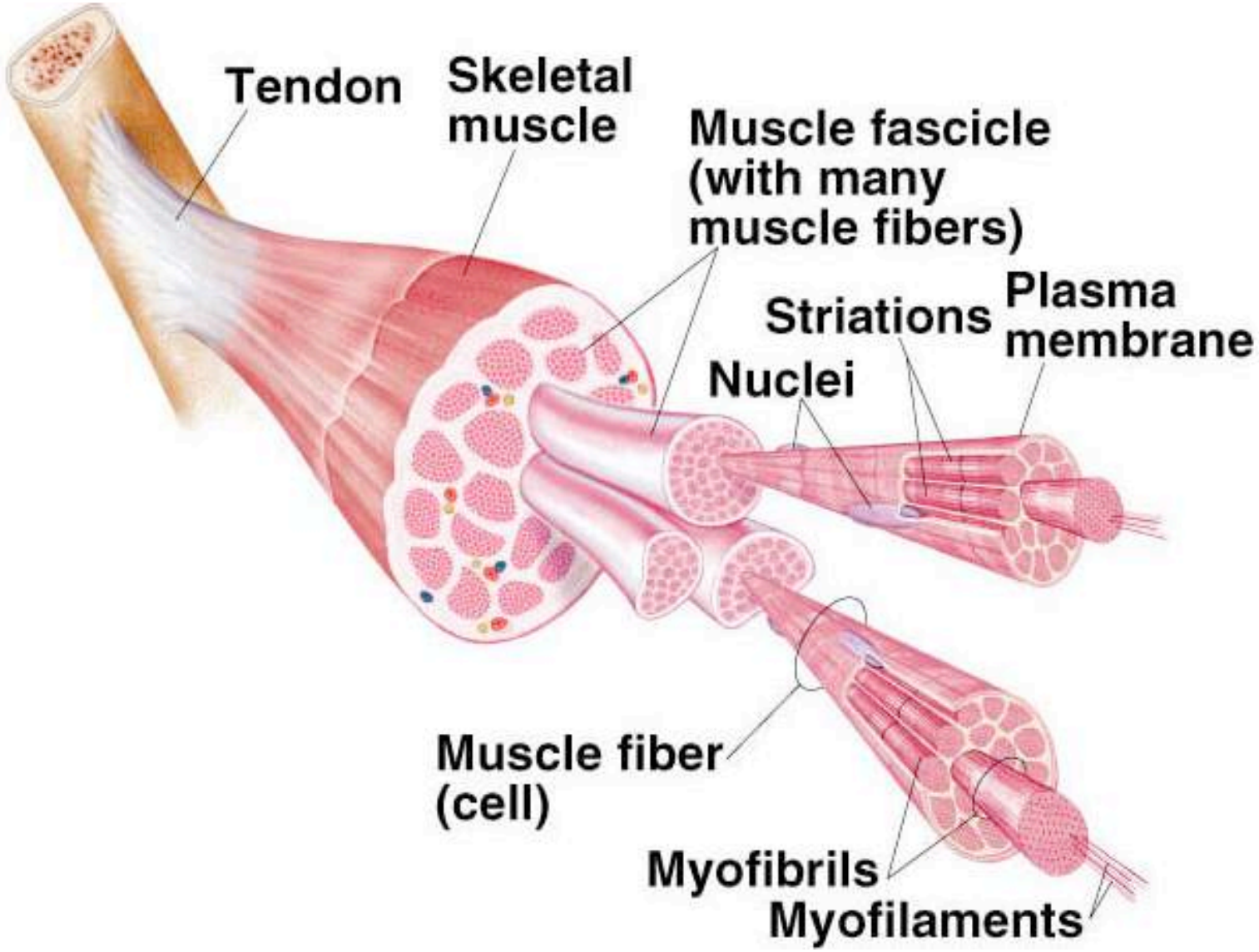


voluntary, striated

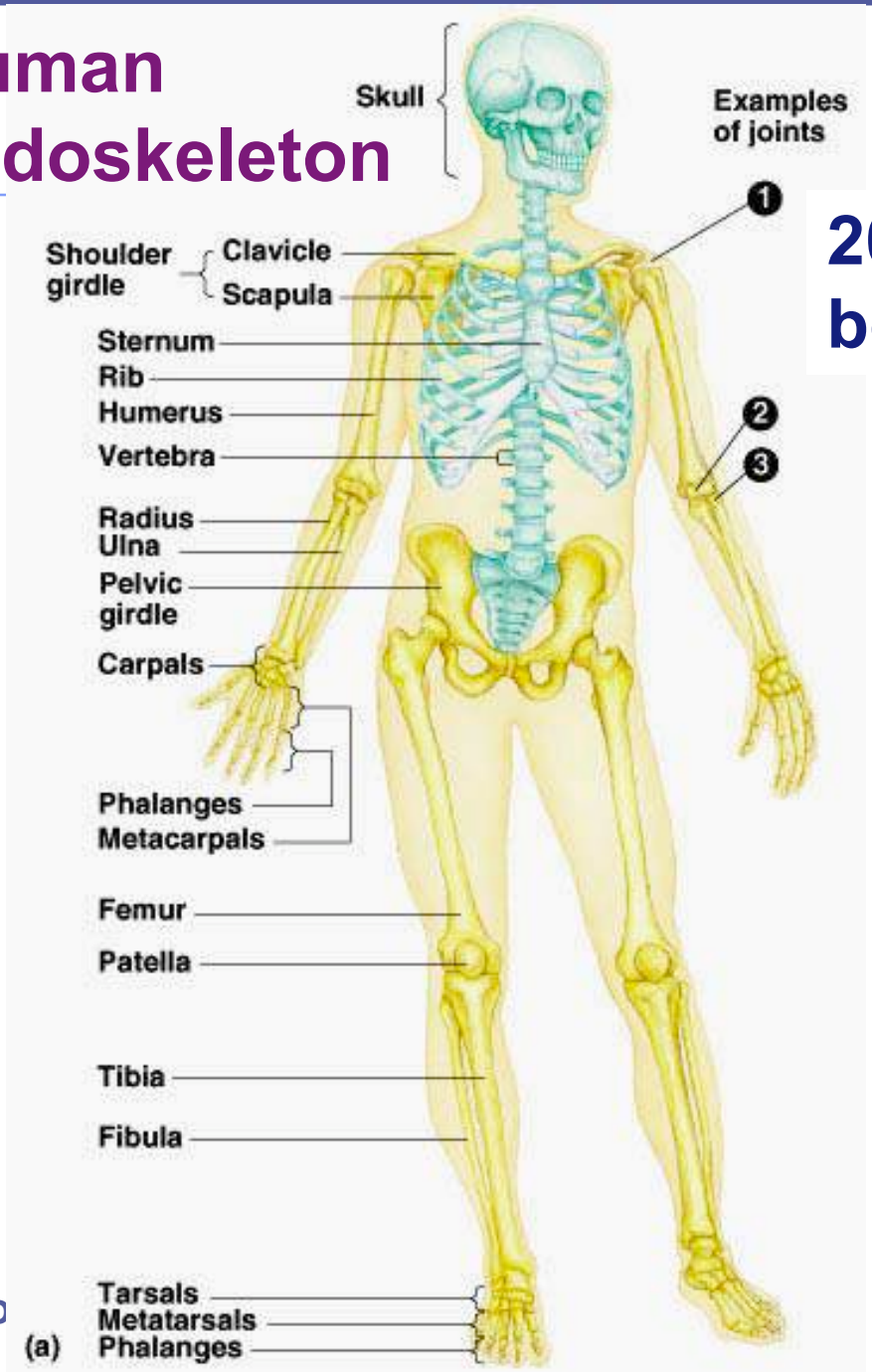
involuntary, striated auto-rhythmic

involuntary, non-striated

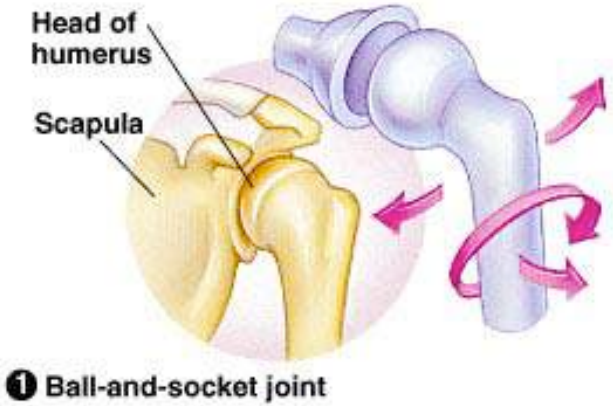
Organization of Skeletal muscle

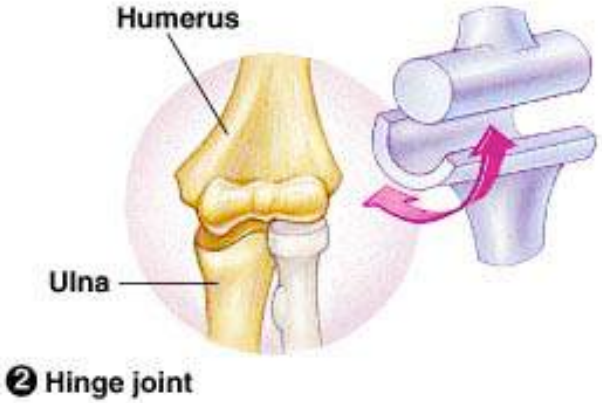


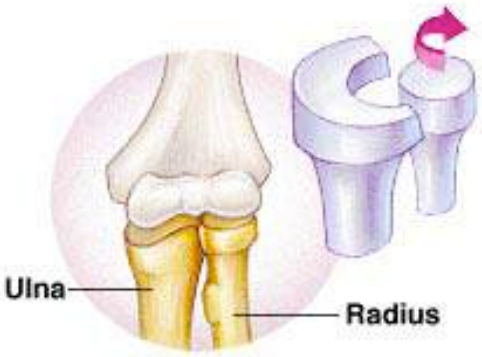
Human endoskeleton



206 bones



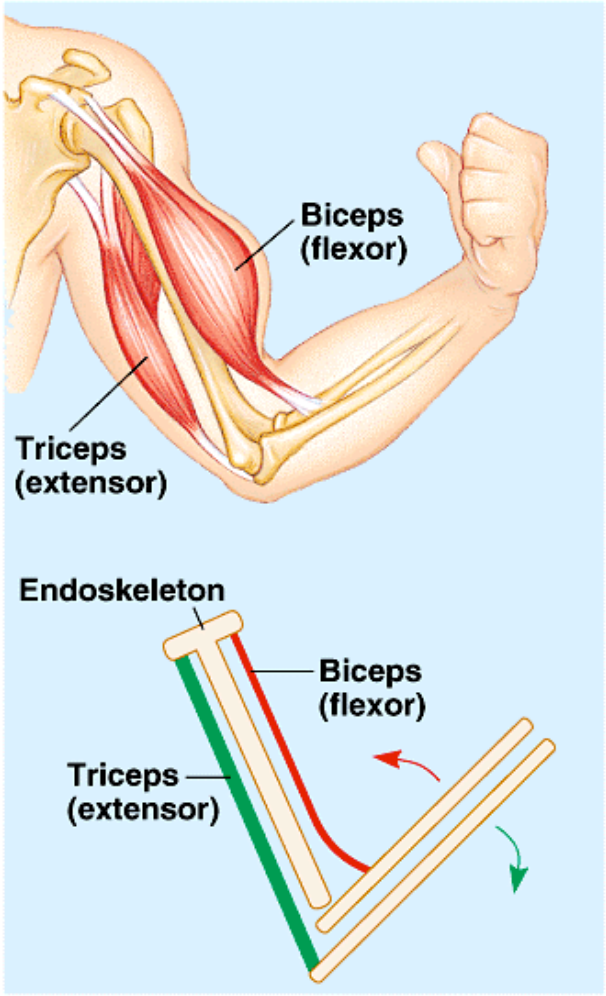


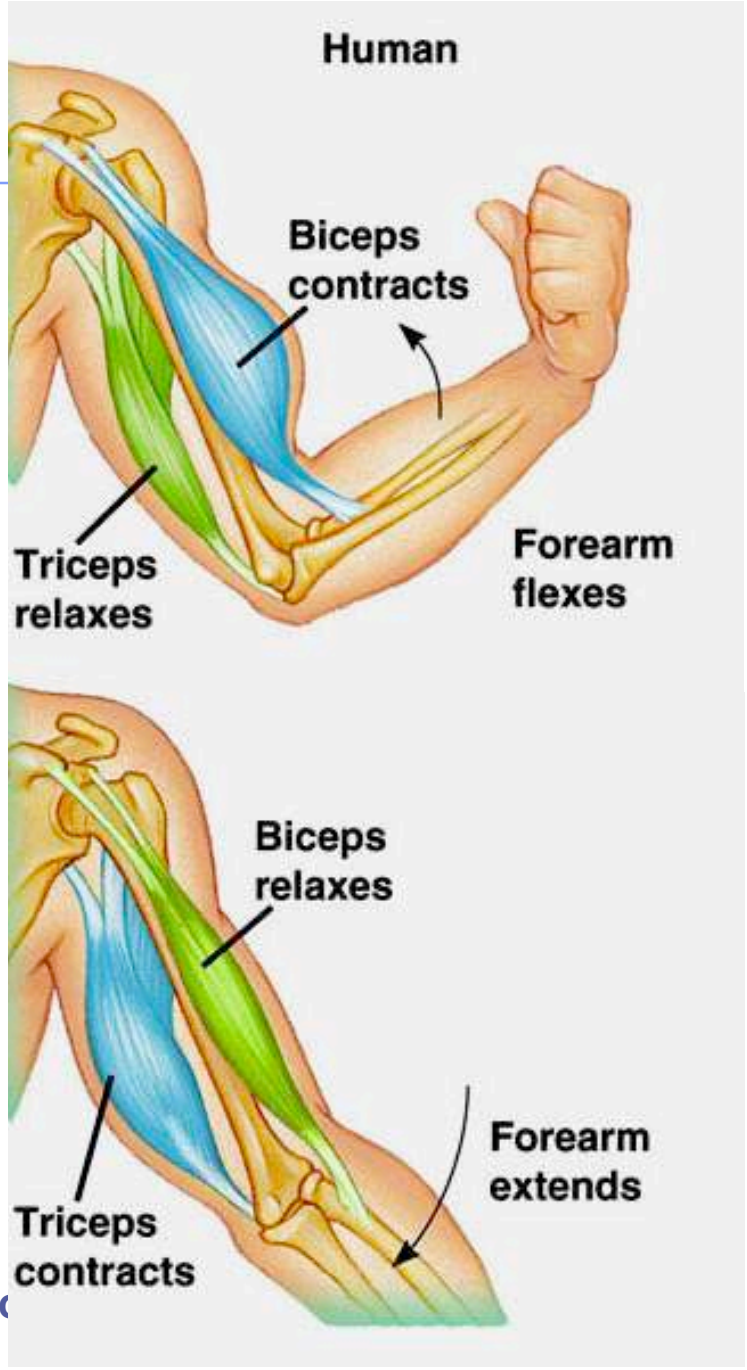


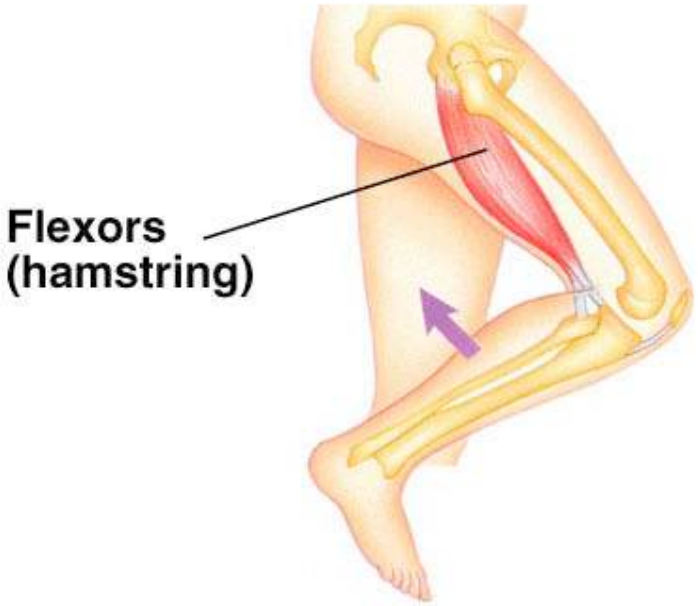
(b)

Muscles movement

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



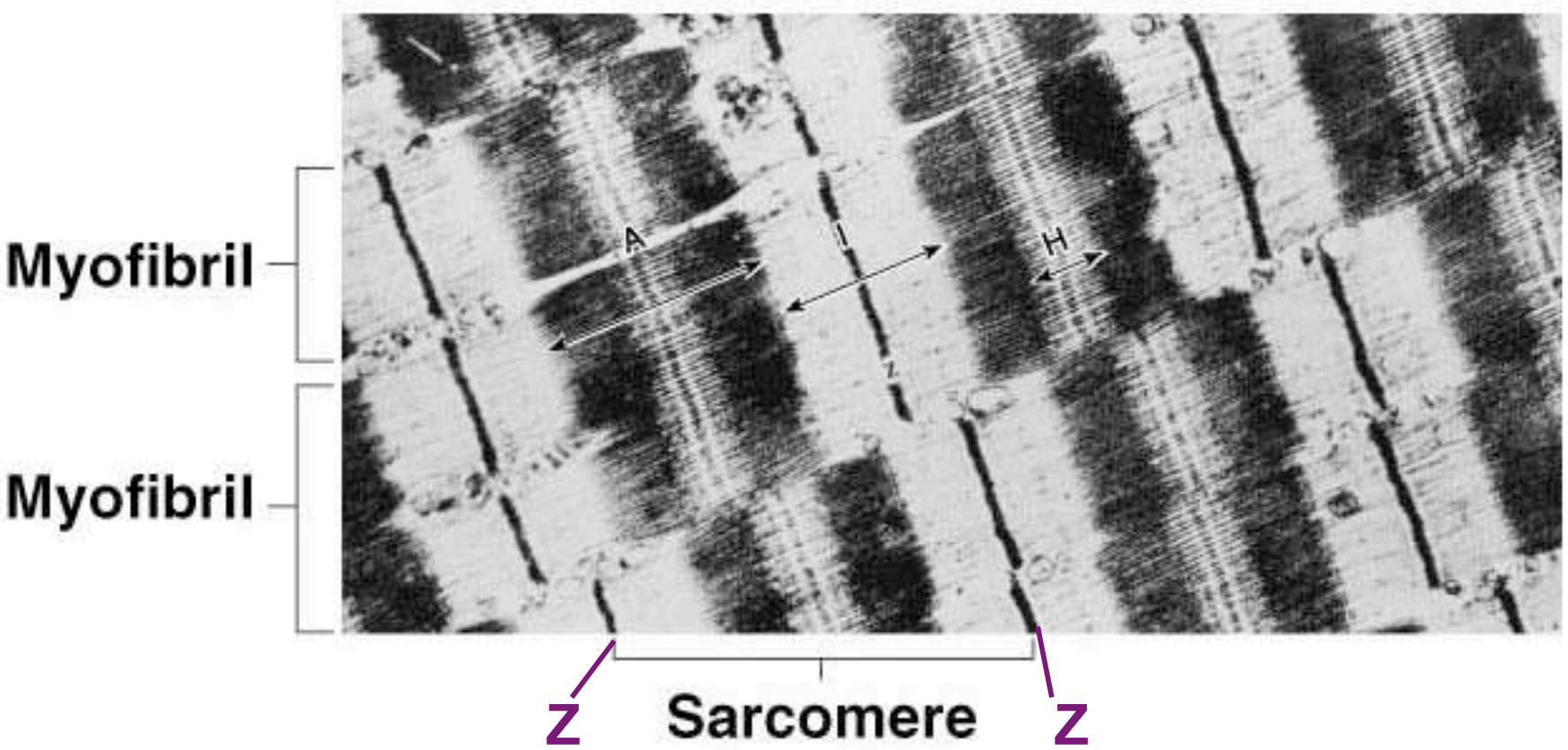

Human
Biceps contracts
Triceps relaxes
Forearm flexes
Biceps relaxes
Triceps contracts
Forearm extends


Flexors (hamstring)


Extensors (quadriceps)

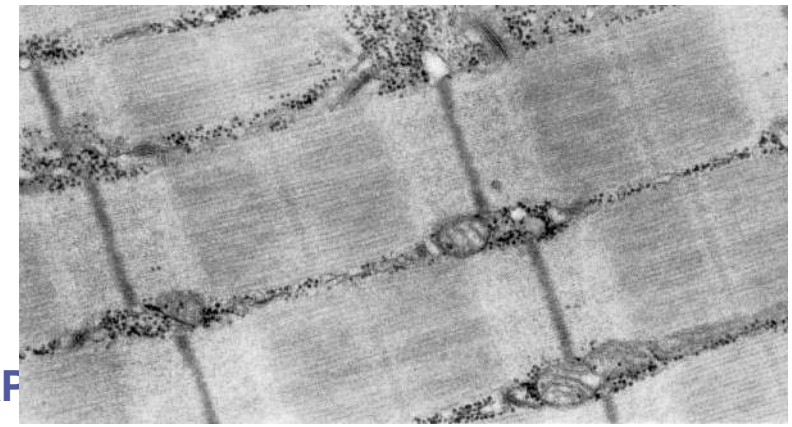
Striated skeletal muscle

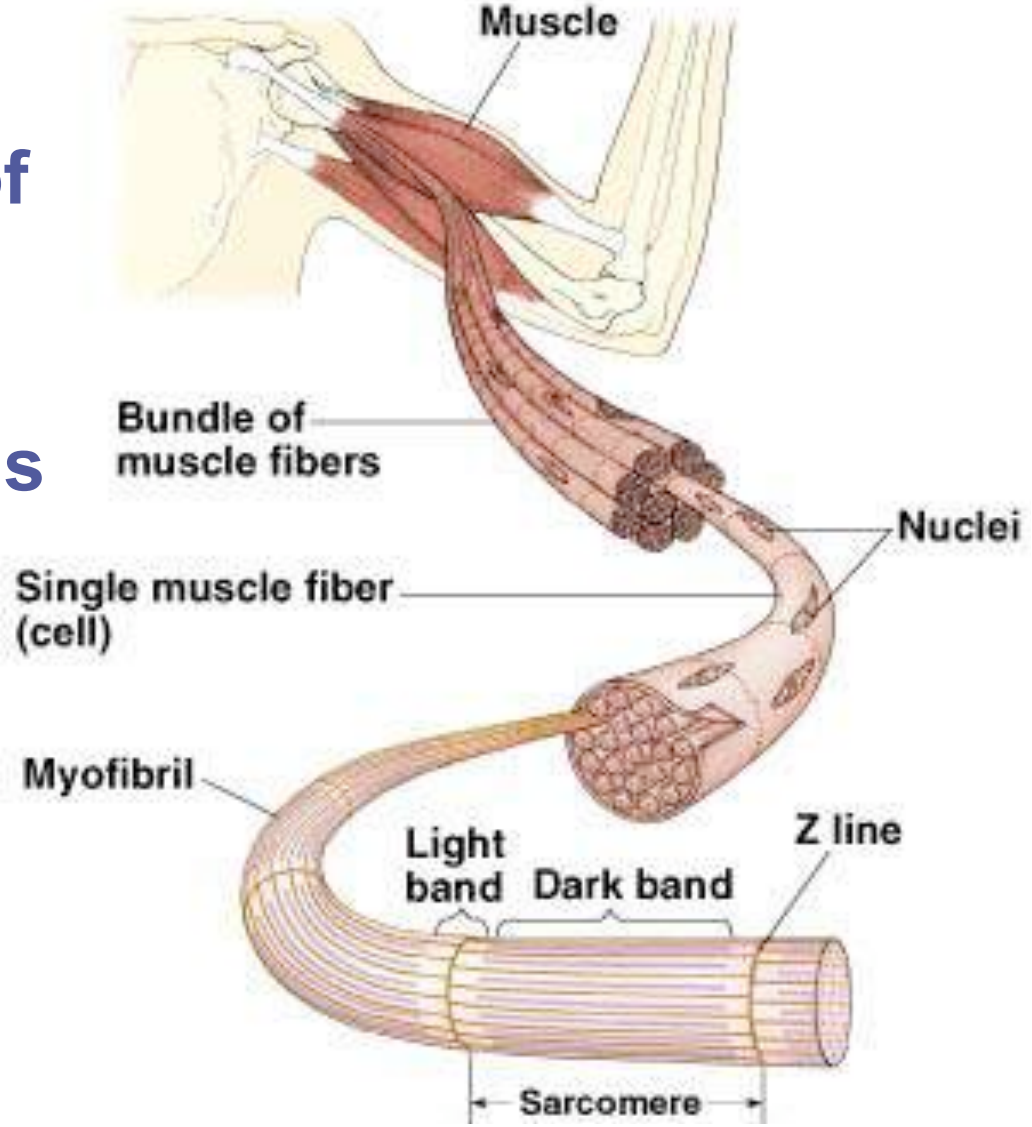
- A band = thick filaments = myosin
- I band = thin filaments = actin



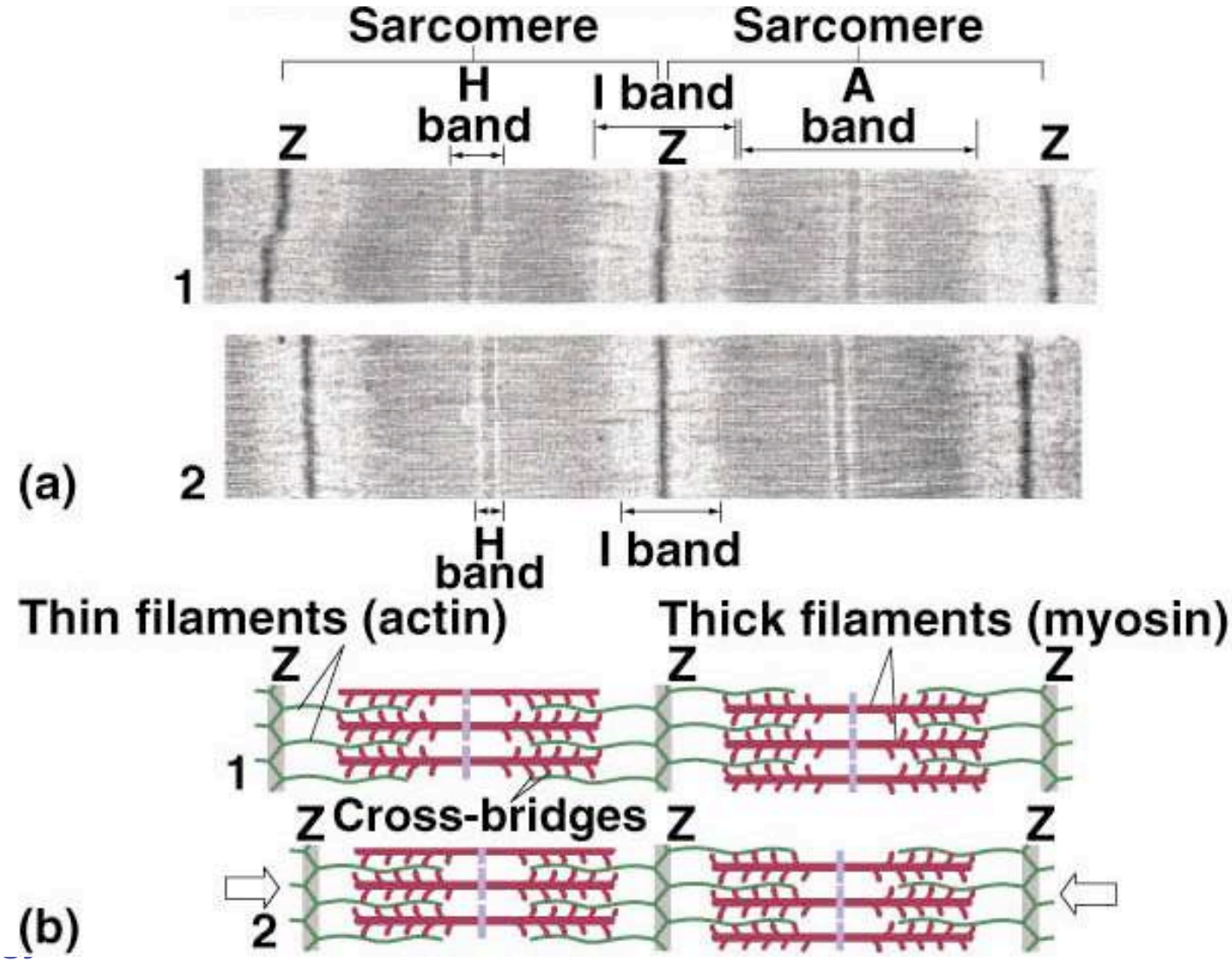
Structure of skeletal muscle

- **Sarcomere**
  - functional unit of muscle contraction
  - alternating bands of thin & thick filaments

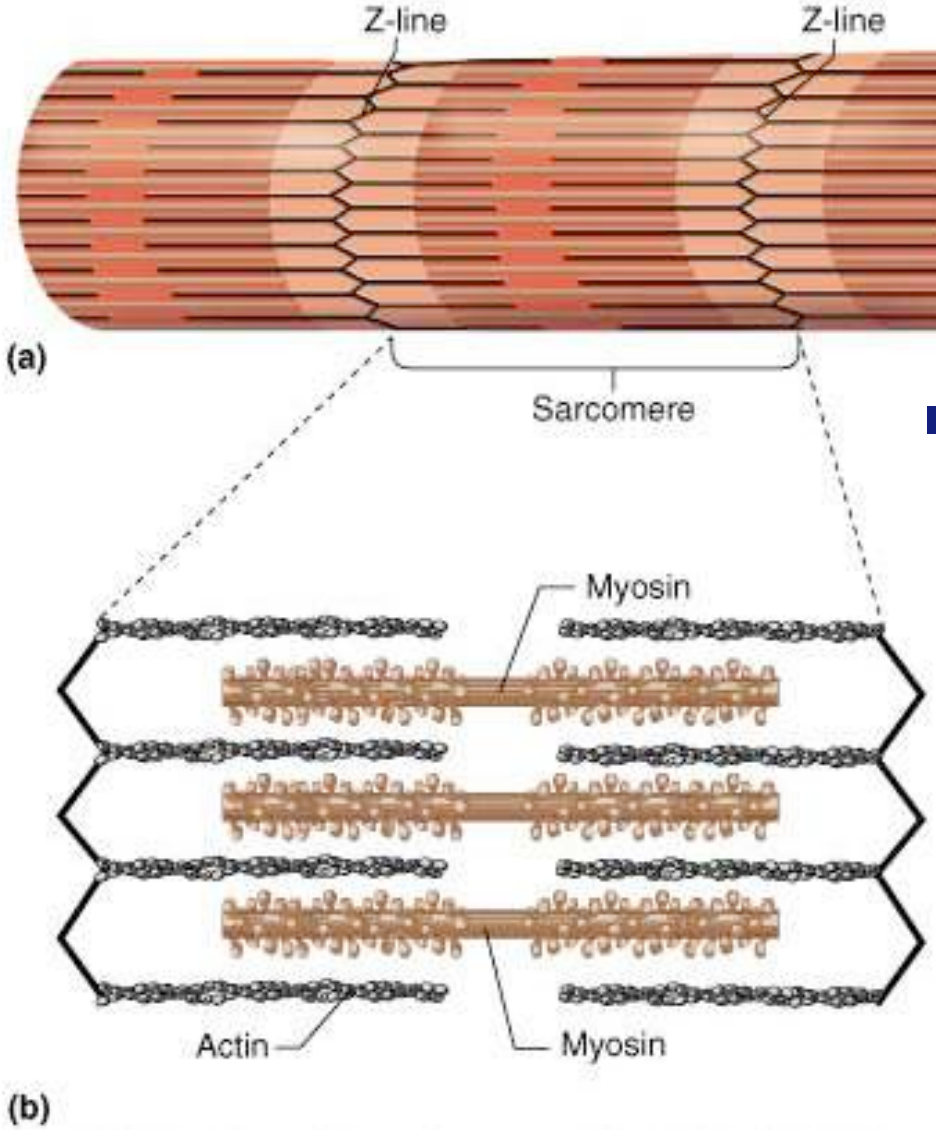




Sliding Filament mechanism



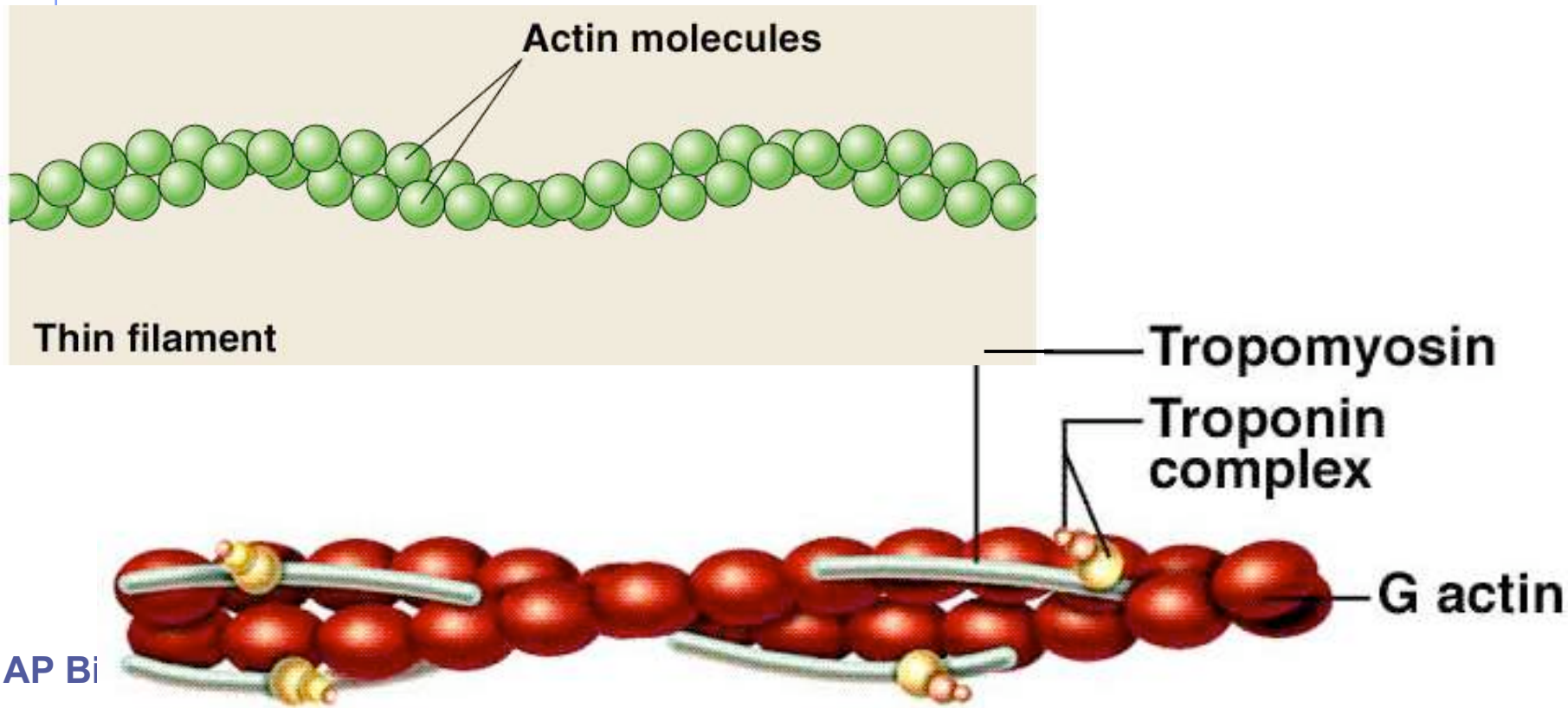
Muscle filaments & Sarcomere



- **Interacting proteins**
  - **thin filaments**
    - **braided strands of actin & tropomyosin coiled together**
  - **thick filaments**
    - **myosin molecules**

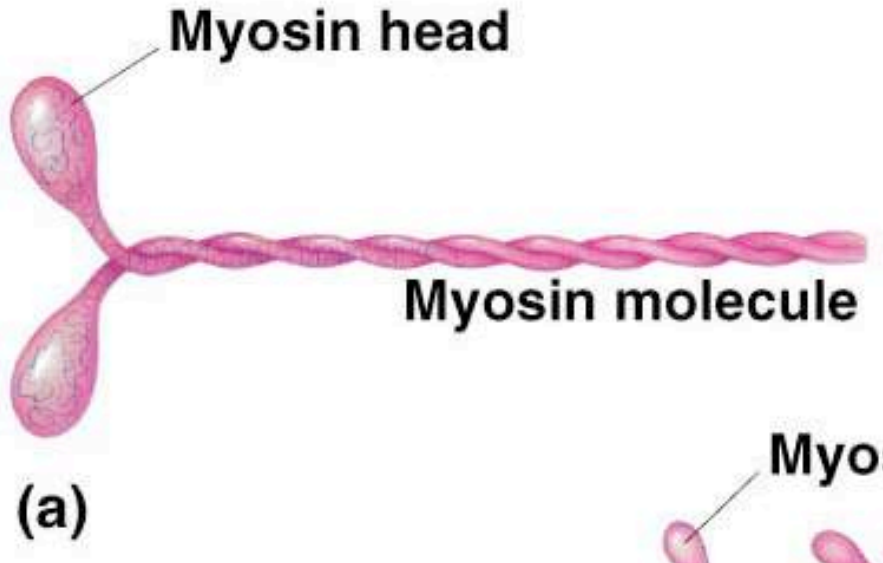
Thin filaments: actin

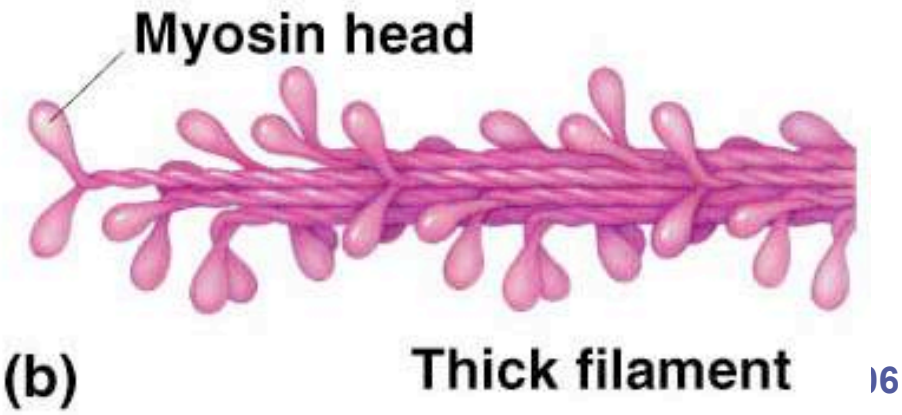
- **Proteins**
  - braid of actin & tropomyosin molecules
  - dotted with troponin molecules



Thick filaments: myosin

- **Protein**
  - myosin molecule
  - long protein with globular head

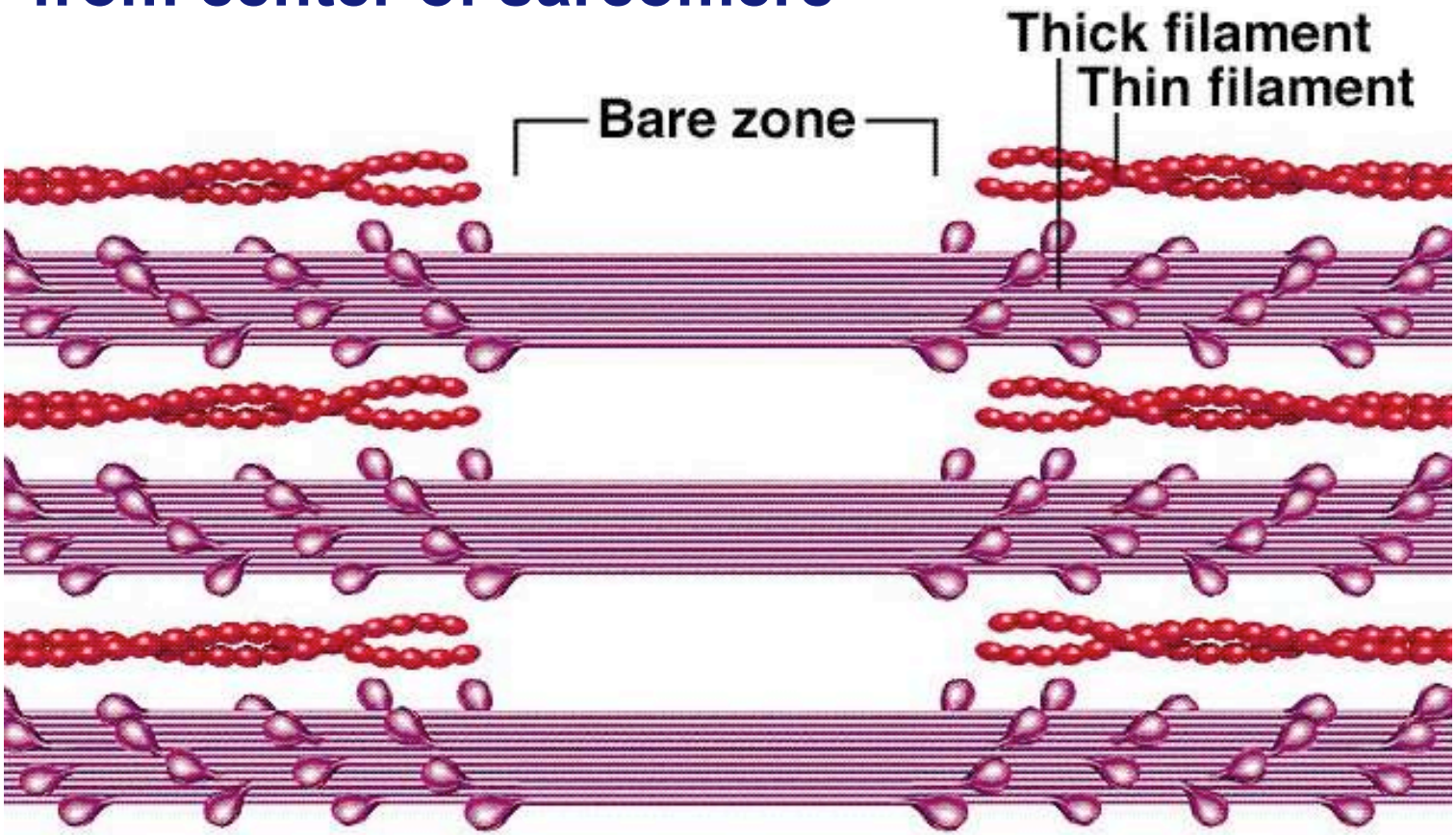




bundle of myosin proteins: globular heads aligned together

Thick & thin filaments

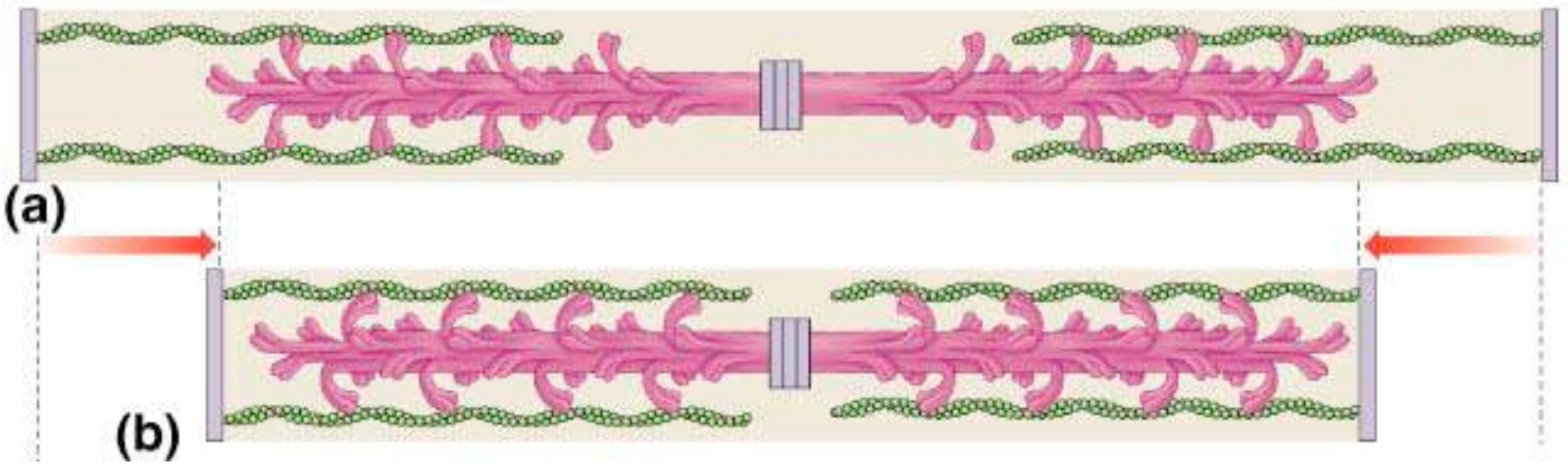
- Myosin tails together & heads pointed away from center of sarcomere



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

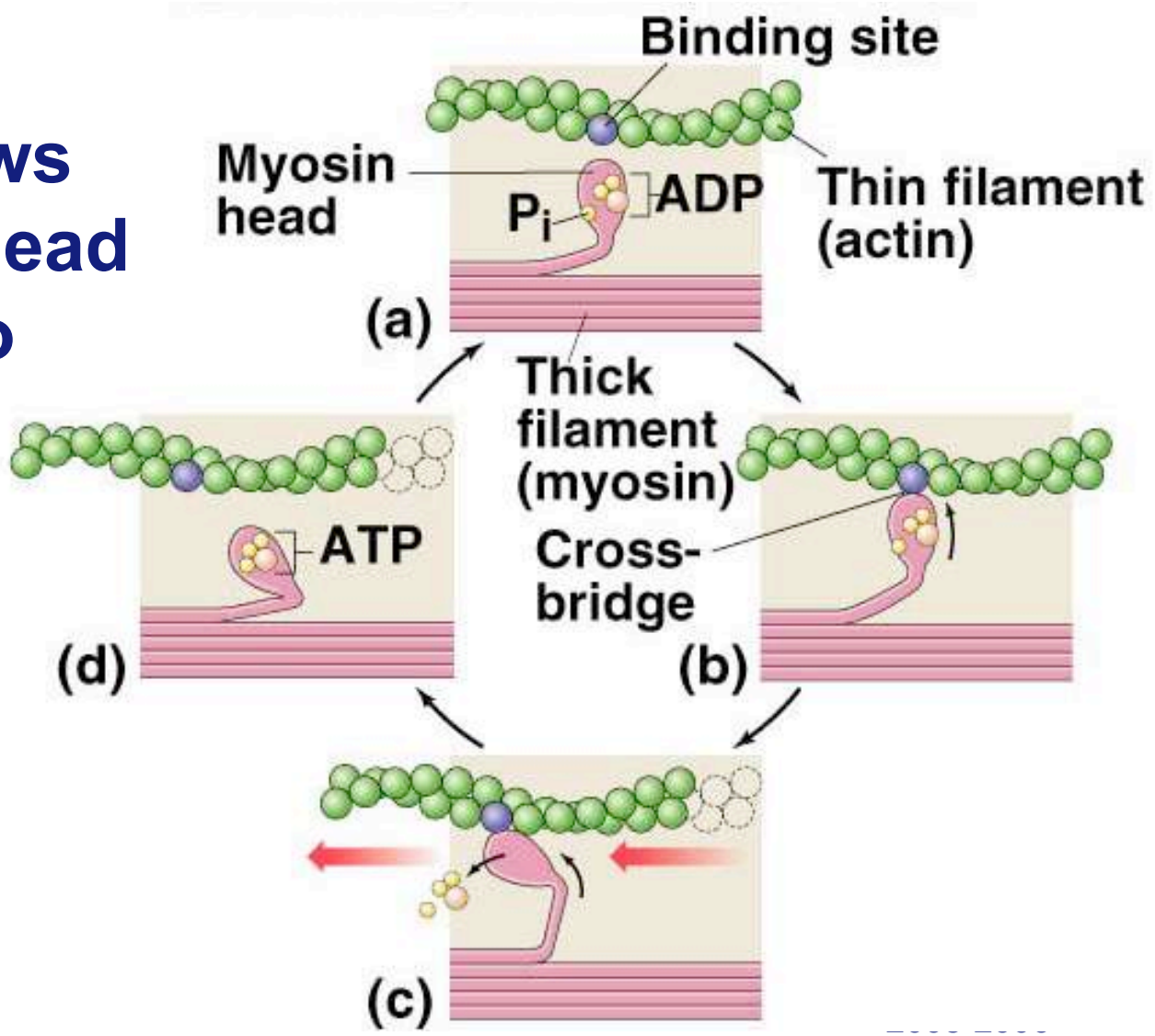
Interaction of thick & thin filaments

- Cross bridges formed between myosin heads (thick filaments) & actin (thin flaments) cause the muscle to shorten (contract)



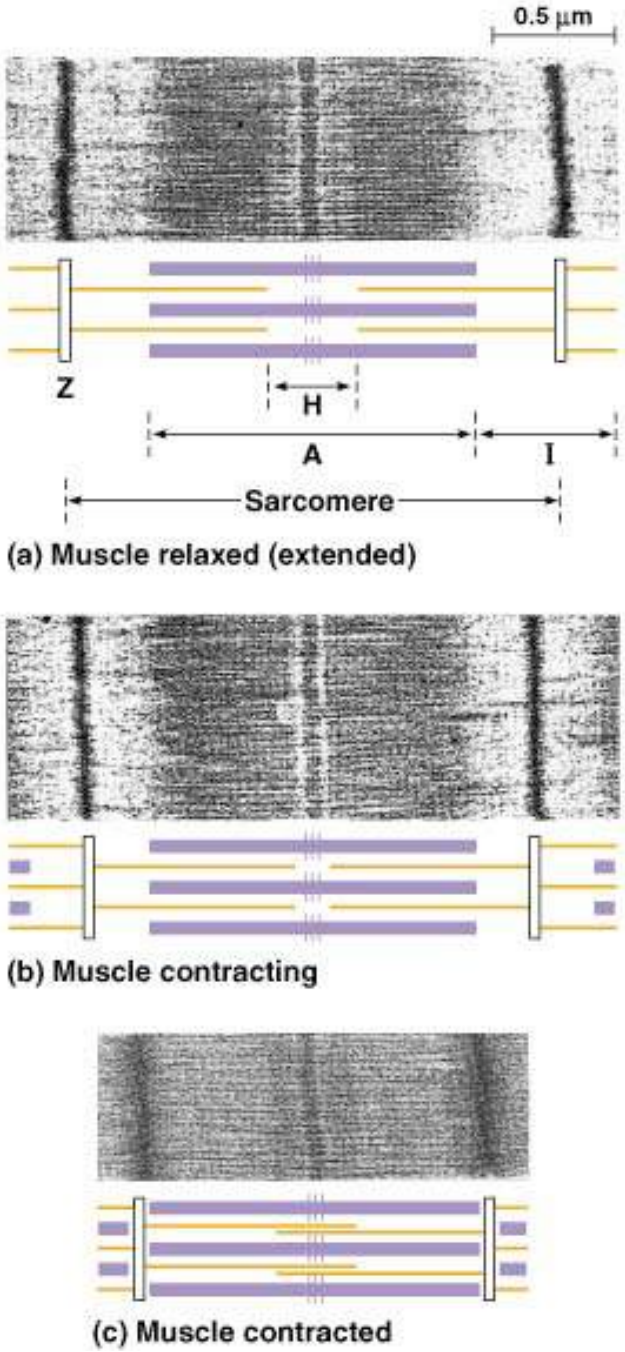
Cross bridge cycle

- Cleaving ATP allows myosin head to bind to actin filament

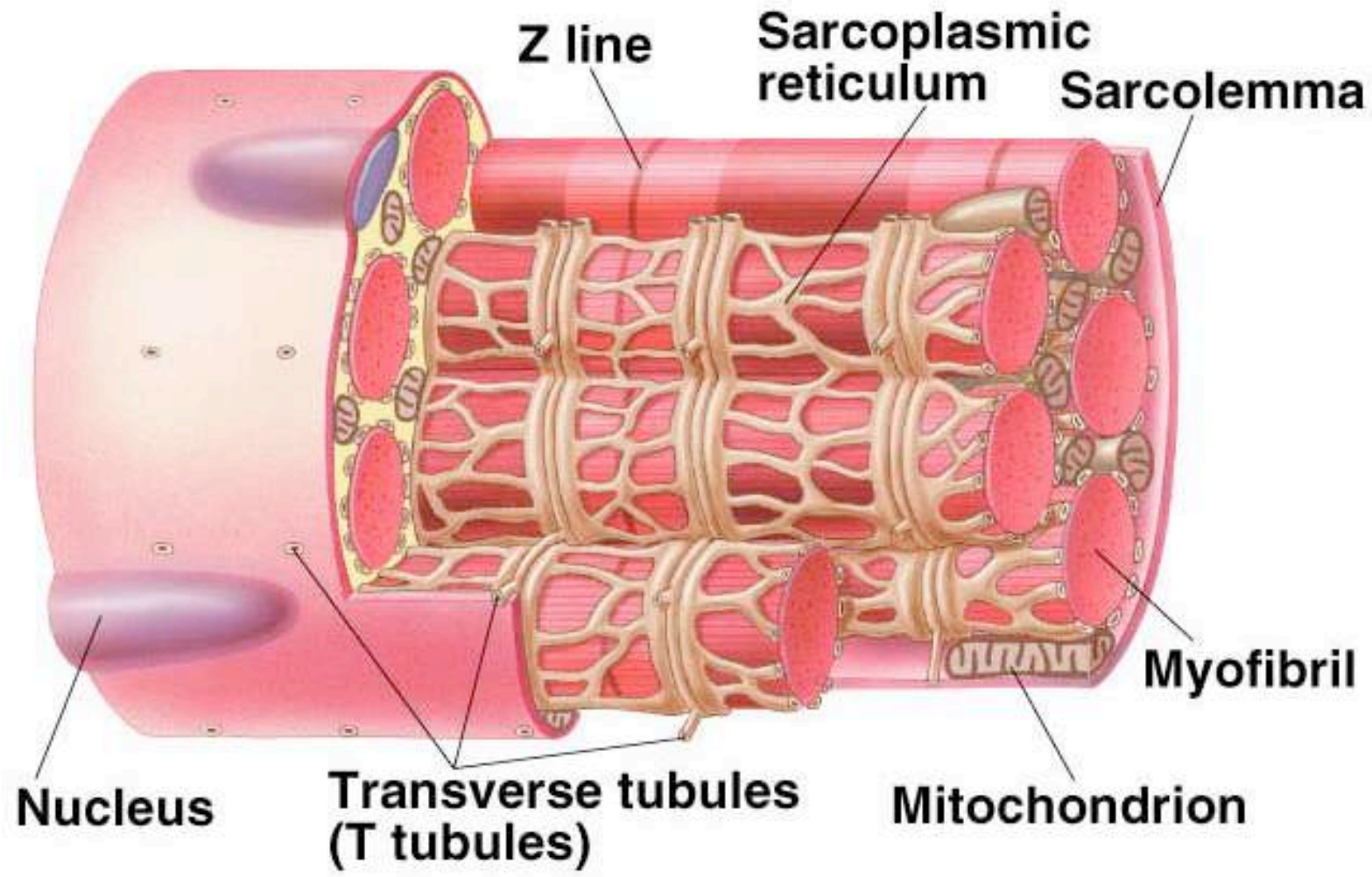


How a muscle works

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

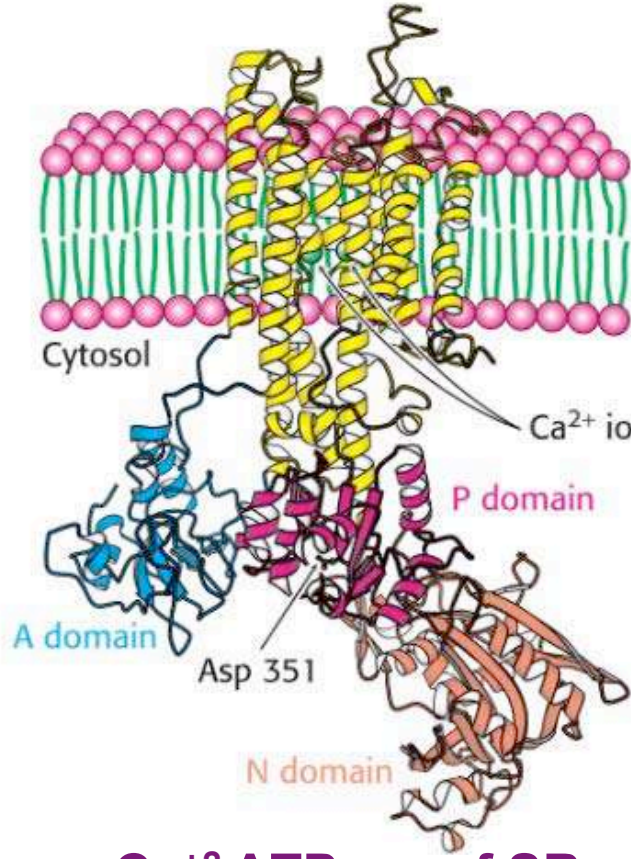


Closer look at muscle cell



Sarcoplasmic reticulum

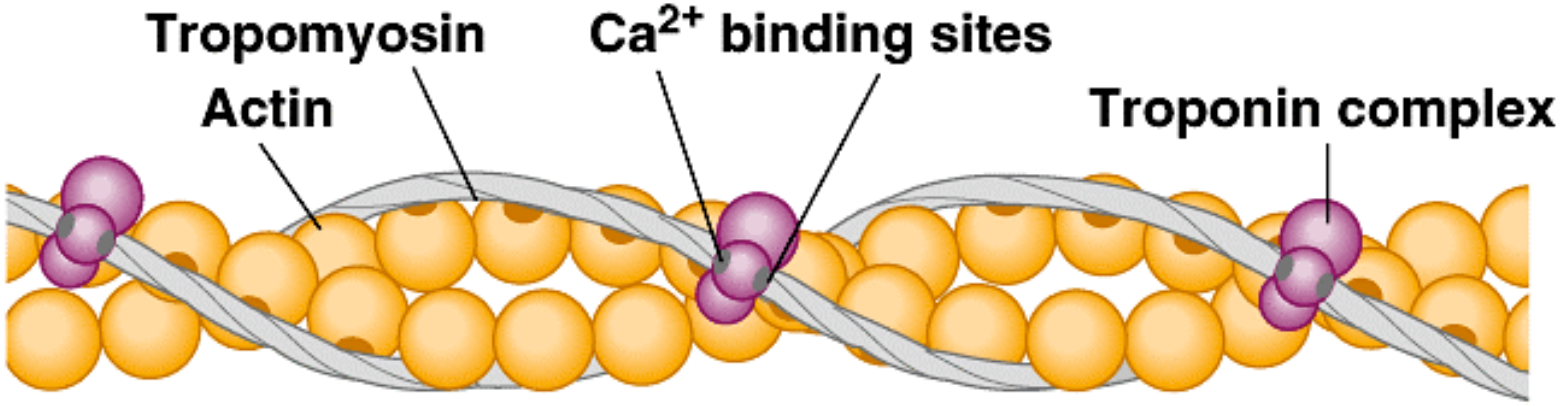
- Sarcoplasm
  - muscle cell cytoplasm
  - contains many mitochondria
- Sarcoplasmic reticulum (SR)
  - organelle similar to ER
    - network of tubes
  - stores Ca^{+2}
    - Ca^{+2} released from SR through channels
    - Ca^{+2} pumps then restore Ca^{+2} to SR
      - remove Ca^{+2} from cytosol
      - pumps use ATP



Ca^{+2} ATPase of SR

Muscle at rest

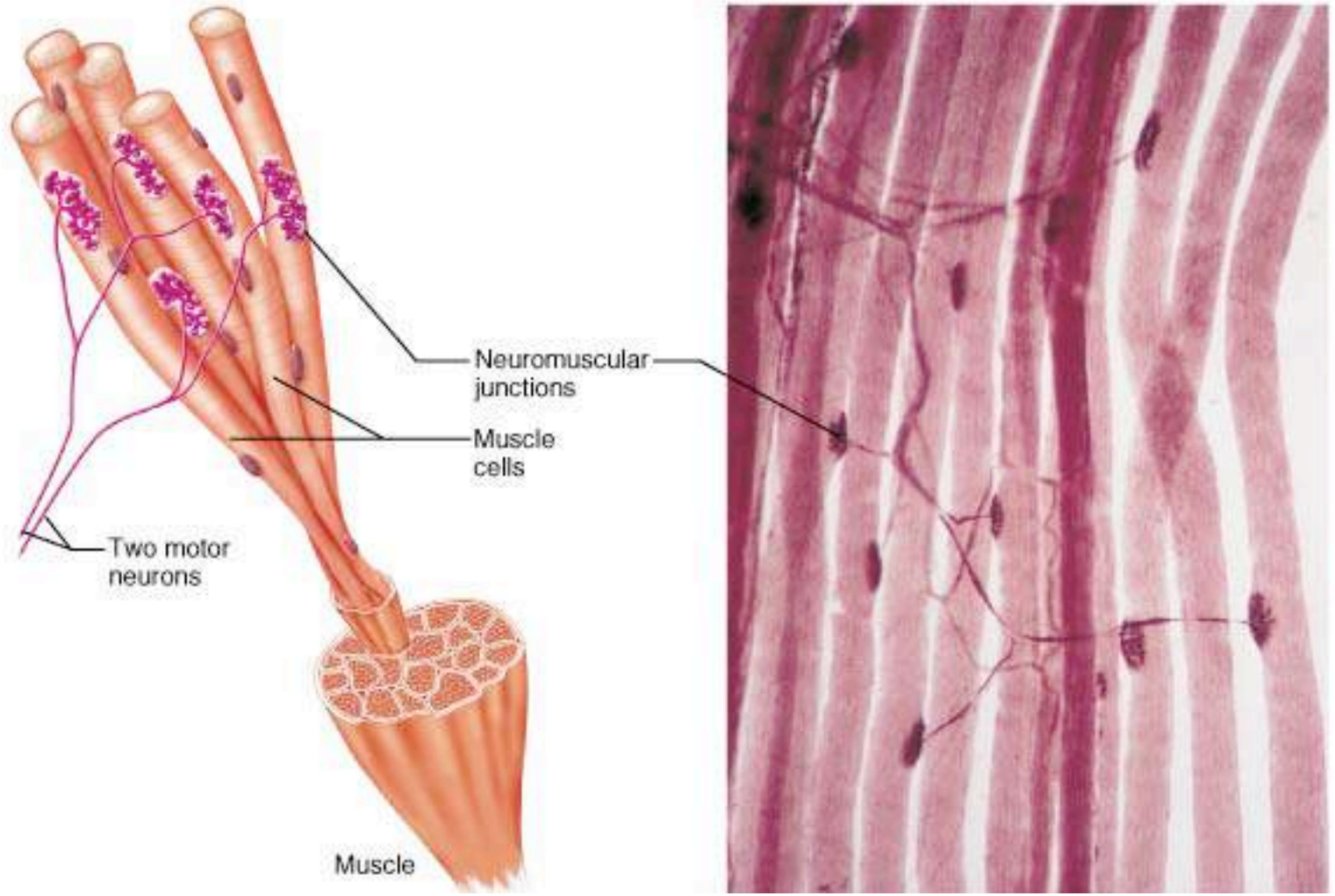
- **Interacting proteins**
  - at rest, troponin molecules hold tropomyosin molecules so that they cover the myosin-binding sites on actin



(a) Myosin binding sites blocked; muscle cannot contract

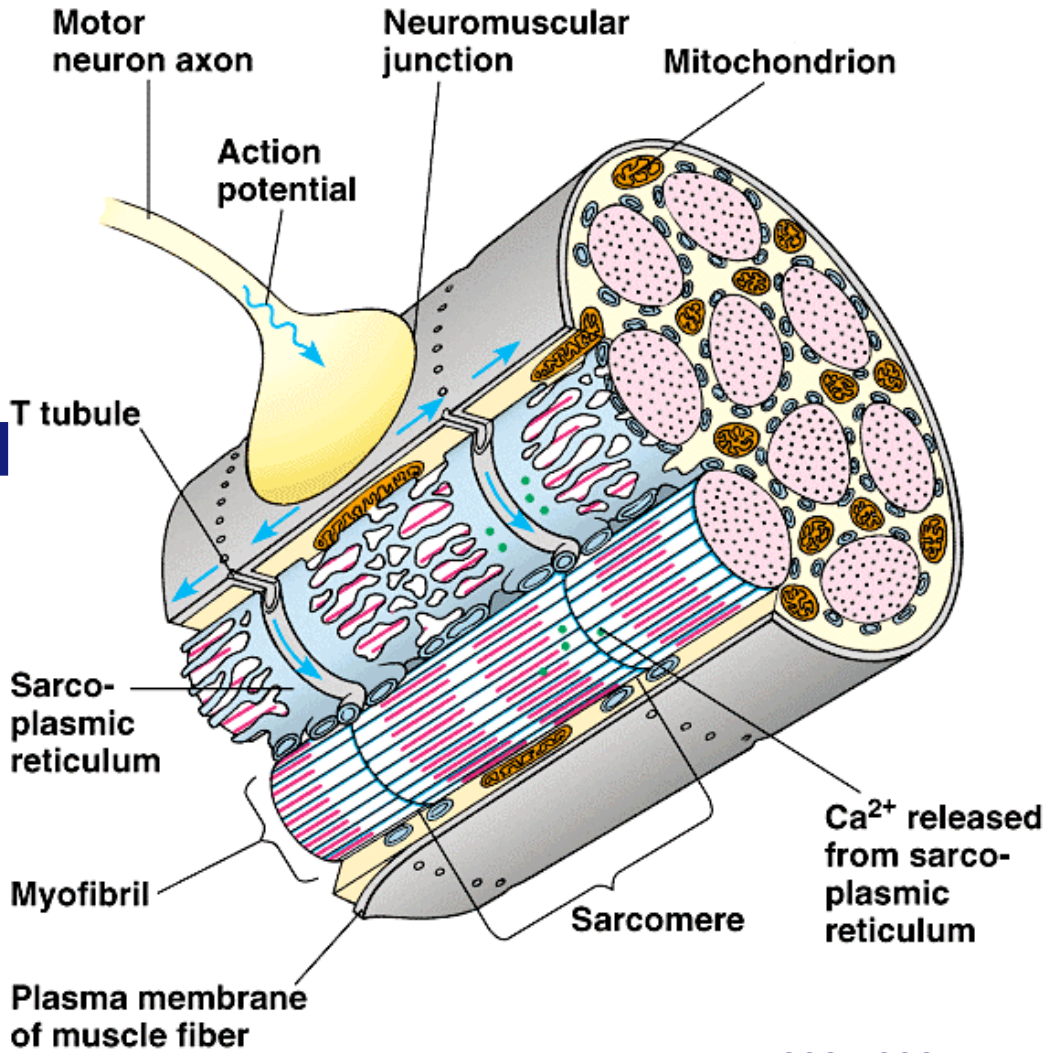
The Trigger: motor neurons

- **Motor neuron triggers muscle contraction**



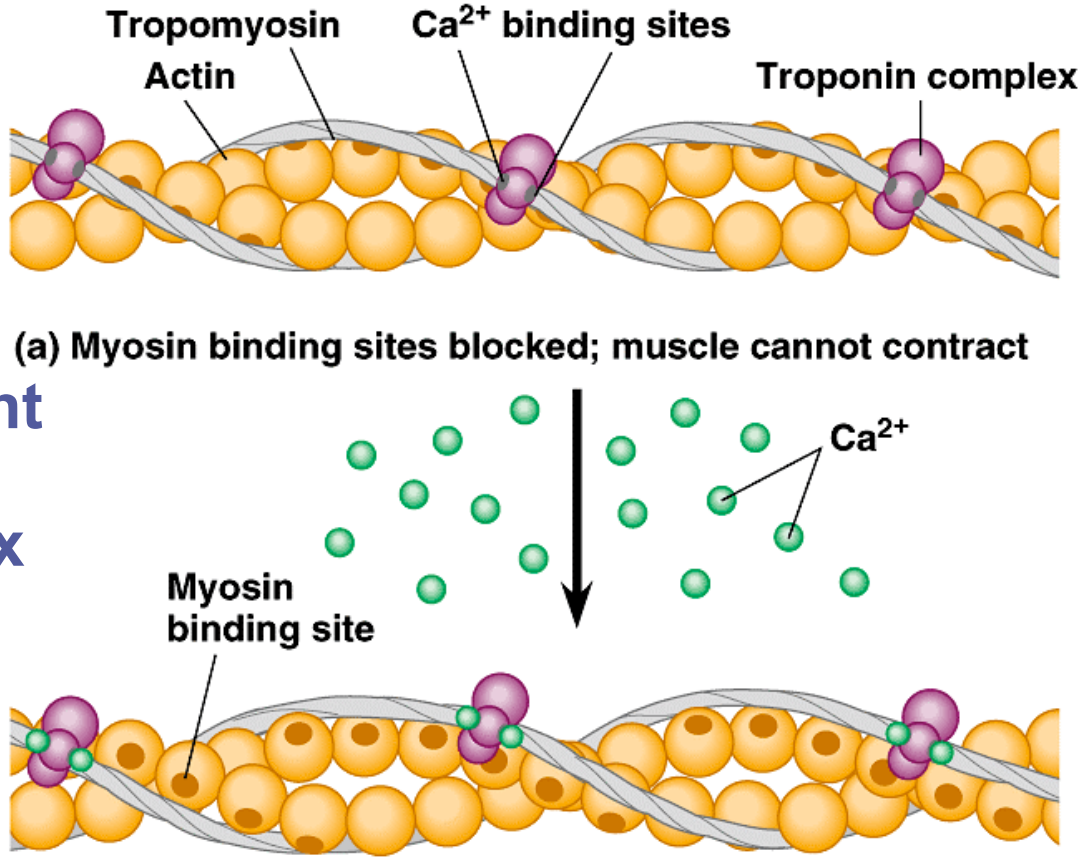
Nerve trigger of muscle action

- Nerve signal stimulates muscle cell's sarcoplasmic reticulum (SR) to release stored Ca^{+2}



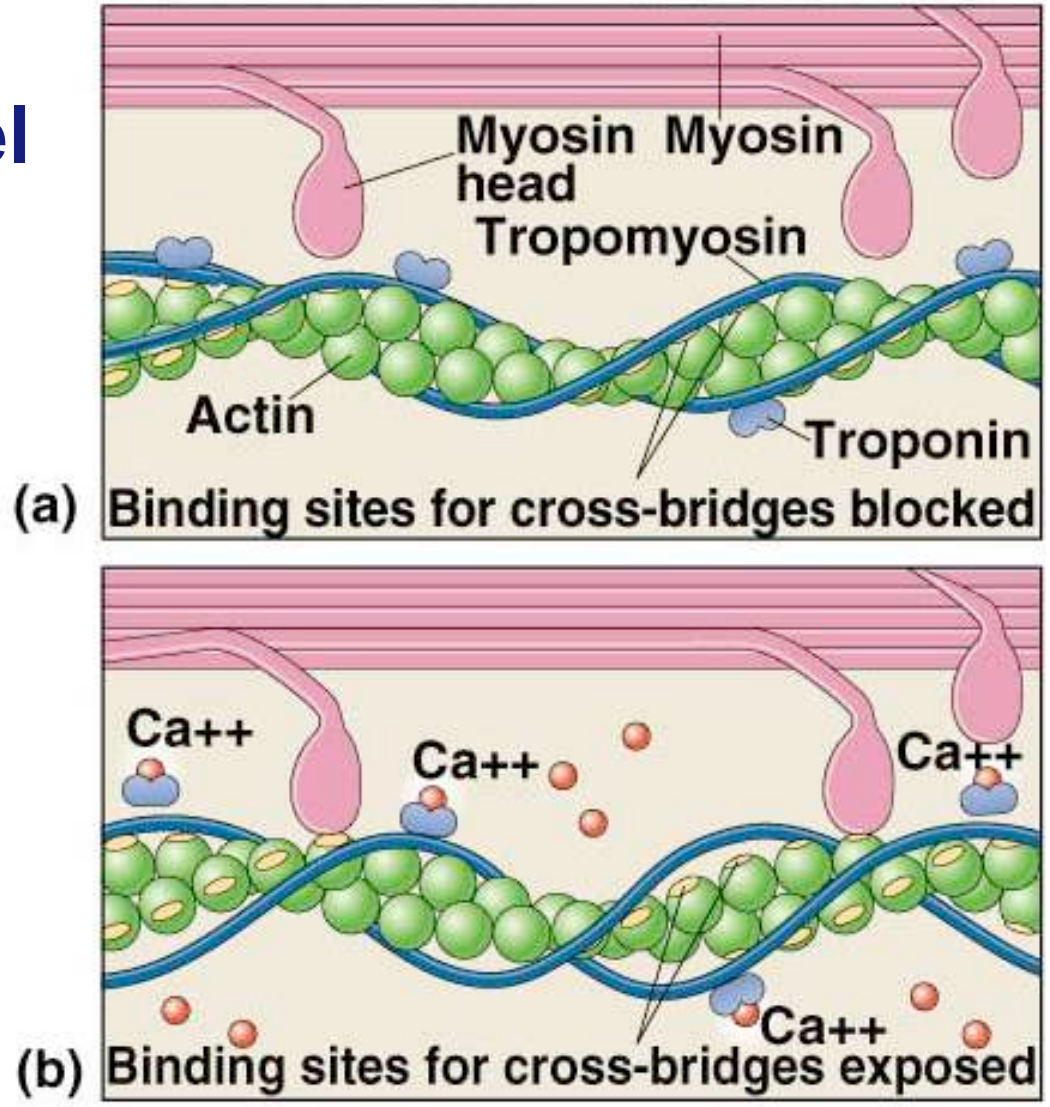
Ca^{+2} triggers muscle action

- At rest, tropomyosin blocks myosin-binding sites on actin
- Ca^{+2} binds to troponin complex
  - shape change causes movement of tropomyosin-troponin complex
  - exposes actin's myosin-binding sites



How Ca^{+2} controls muscle

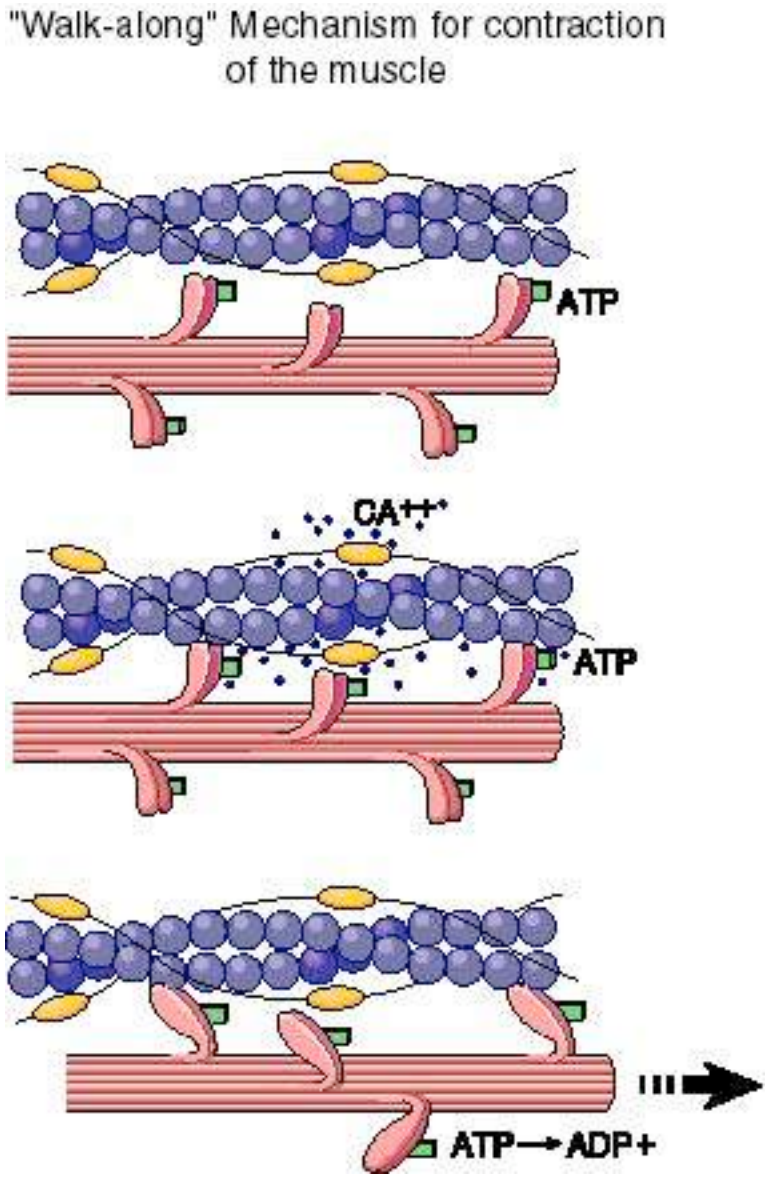
- Sliding filament model
  - ratchet system



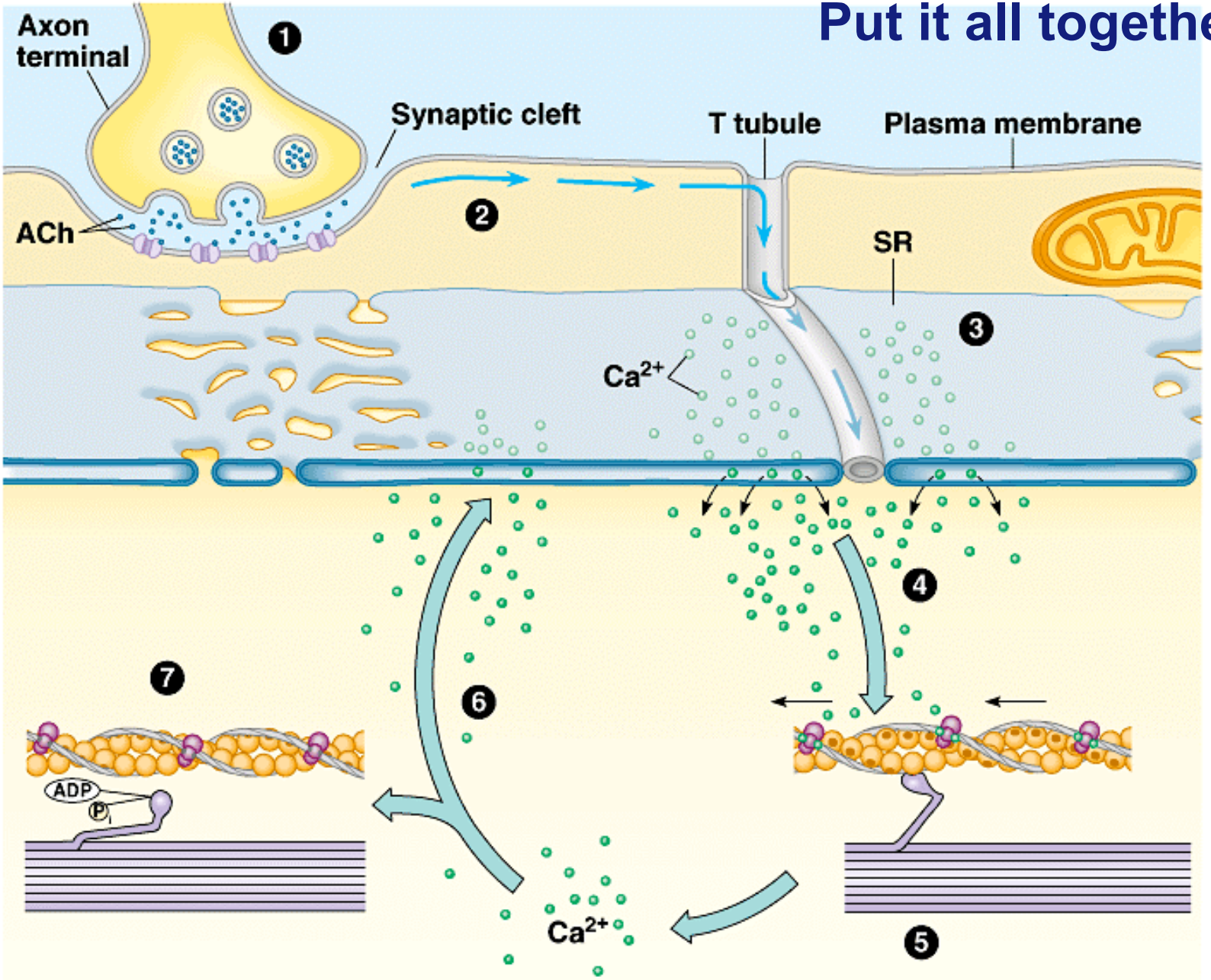
once myosin-binding sites on actin are uncovered, myosin heads bond to actin

Sliding filament model

- **Ratchet system**
  - myosin bonding with actin
  - sliding thin & thick filaments past each other
  - myosin head releases & binds to next active site on actin
  - muscle doesn't relax until Ca^{+2} is pumped back into SR



Put it all together…



How it all works…

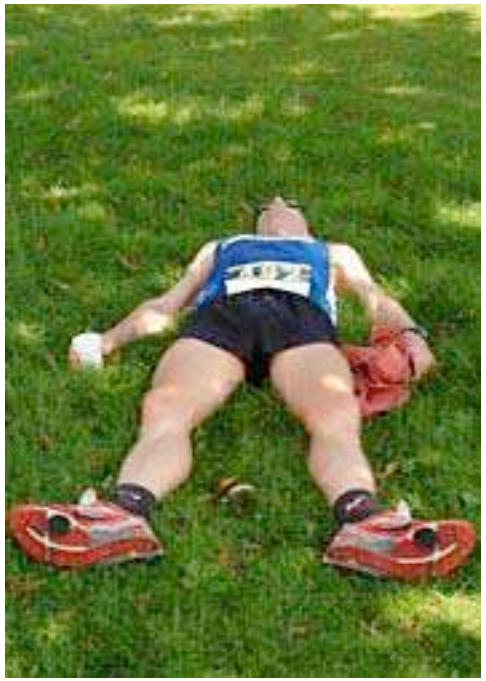
- Action potential causes Ca^{+2} release from SR
  - Ca^{+2} binds to troponin
- Troponin moves tropomyosin
- Tropomyosin uncovers myosin binding site on actin
- Myosin binds actin
  - uses ATP to "rachet" once
  - releases, "unratchets" & binds to next actin
- Myosin pulls actin chain along
- Sarcomere shortens
  - Z discs move closer together
- Whole fiber shortens → contraction!
- Ca^{+2} pumps restore Ca^{+2} to SR → relaxation!
  - pumps use ATP

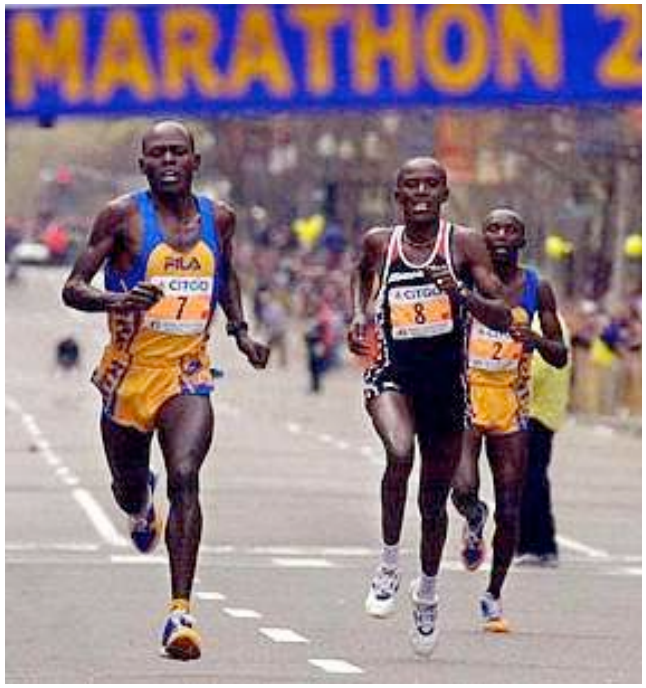
Fast twitch & slow twitch muscles

- **Slow twitch muscle fibers**
  - contract slowly, but keep going for a long time
    - more mitochondria for aerobic respiration
    - less SR → Ca^{+2} 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 fatigue

- **Muscle fatigue**
  - lack of sugar
    - lack of ATP to restore Ca^{+2} gradient
  - low O_2
    - lactic acid drops pH which interferes with protein function
  - synaptic fatigue
    - loss of acetylcholine
- **Muscle cramps**
  - ATP depletion
  - build up of lactic acid
  - ion imbalance
    - massage or stretching increases circulation



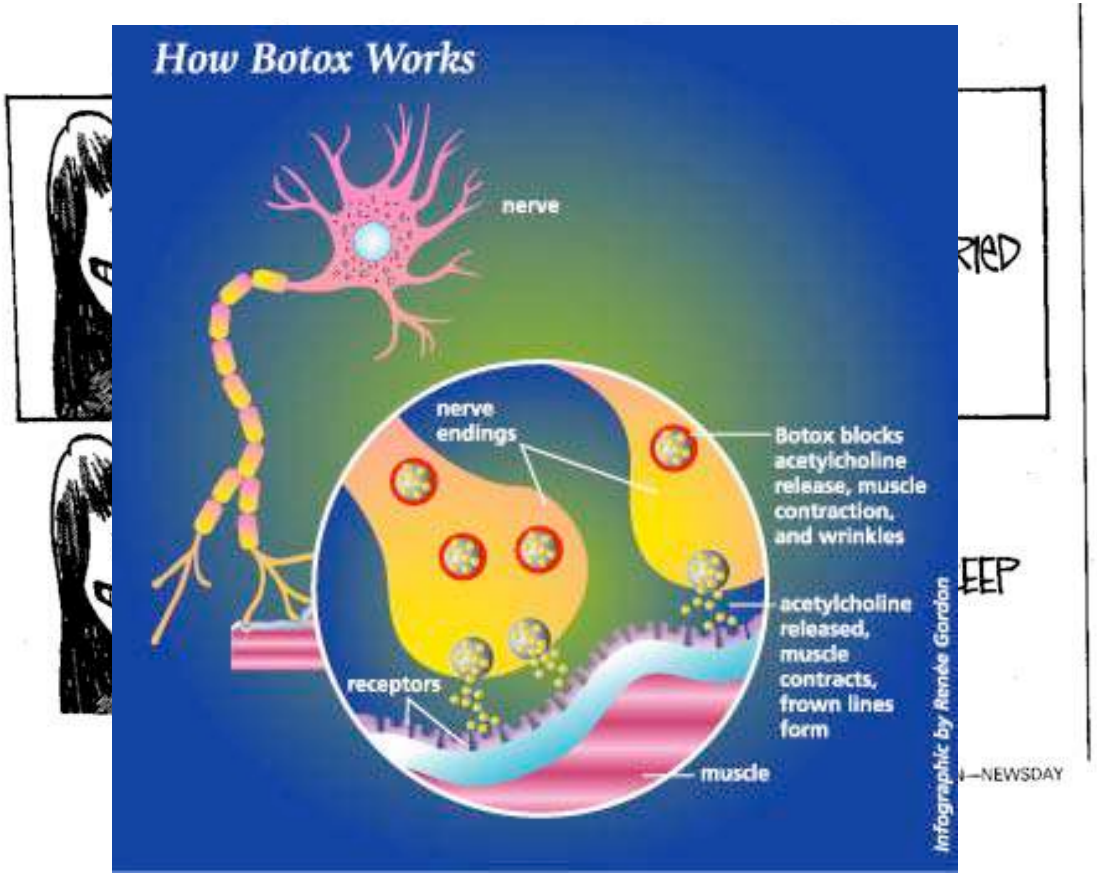


Diseases of Muscle tissue

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

Botox

- Bacteria *Clostridium botulinum* toxin
  - blocks release of acetylcholine



Rigor mortis

- So why are dead people "stiffs"?
  - no life, no breathing
  - no breathing, no O_2
  - no O_2, no respiration
  - no respiration, no ATP
  - no ATP, no Ca^{+2} pumps
  - Ca^{+2} cannot be removed
  - continuous contraction
  - muscles are tensed
    - muscles stiffen after death
  - eventually tissues breakdown & relax

Any Questions??