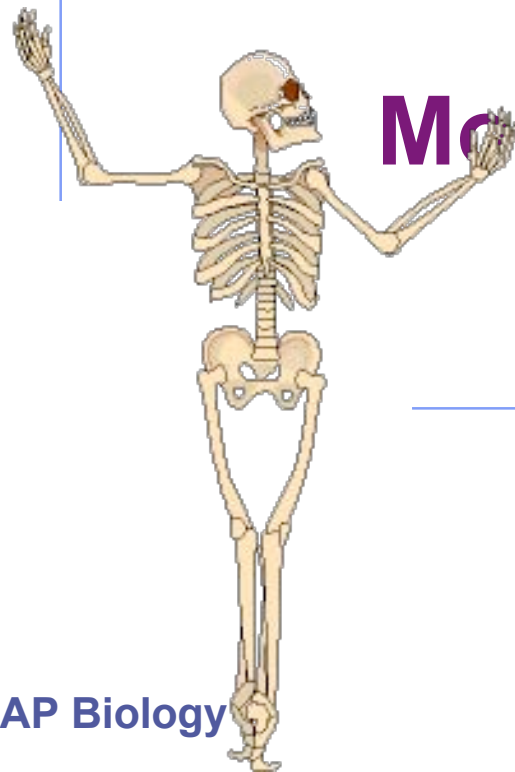


# Chapter 49.

## Muscles & Motor Locomotion



AP Biology



# Animal Locomotion

What are the advantages of locomotion?

**sessile**

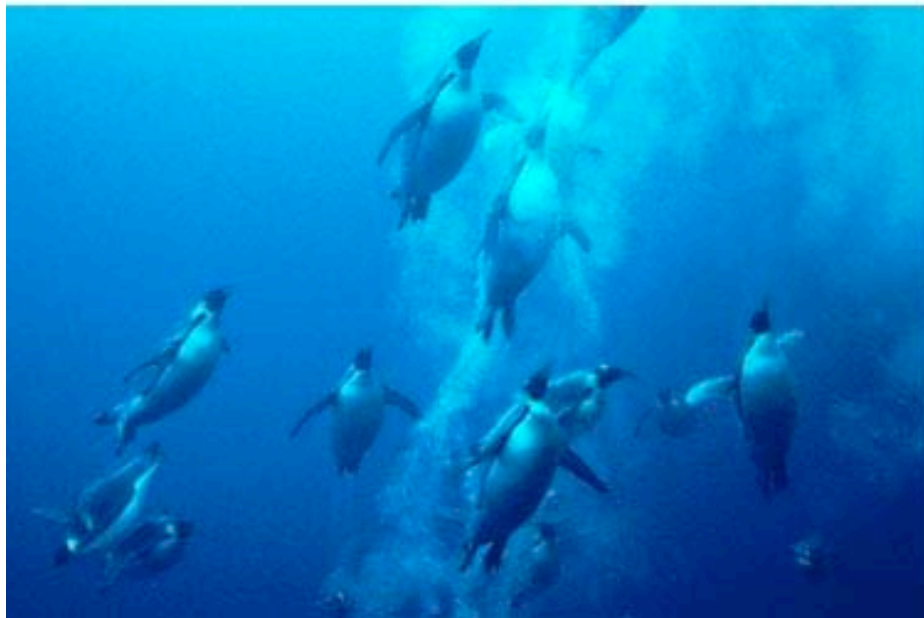


**motile**







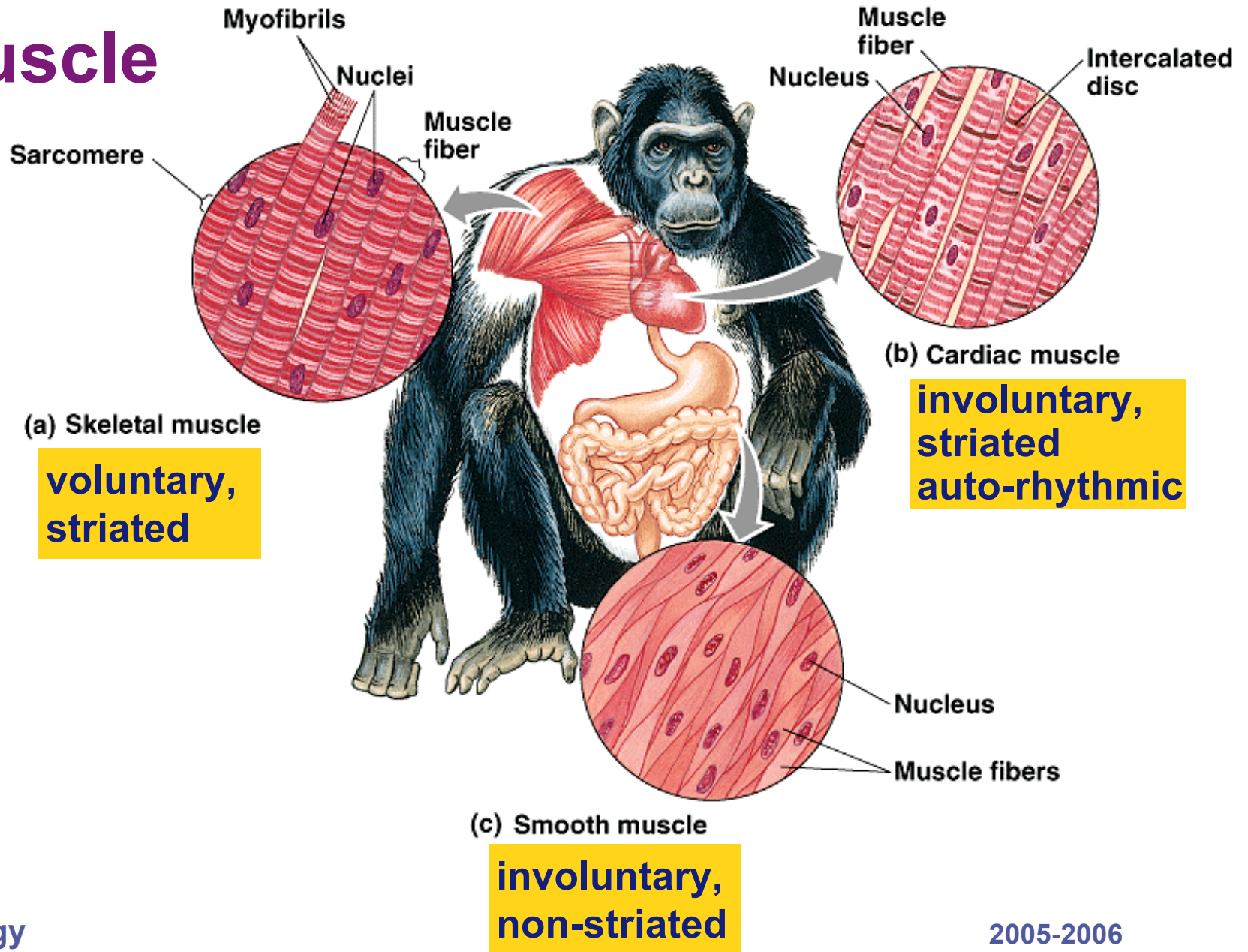


Benjamin  
Comings

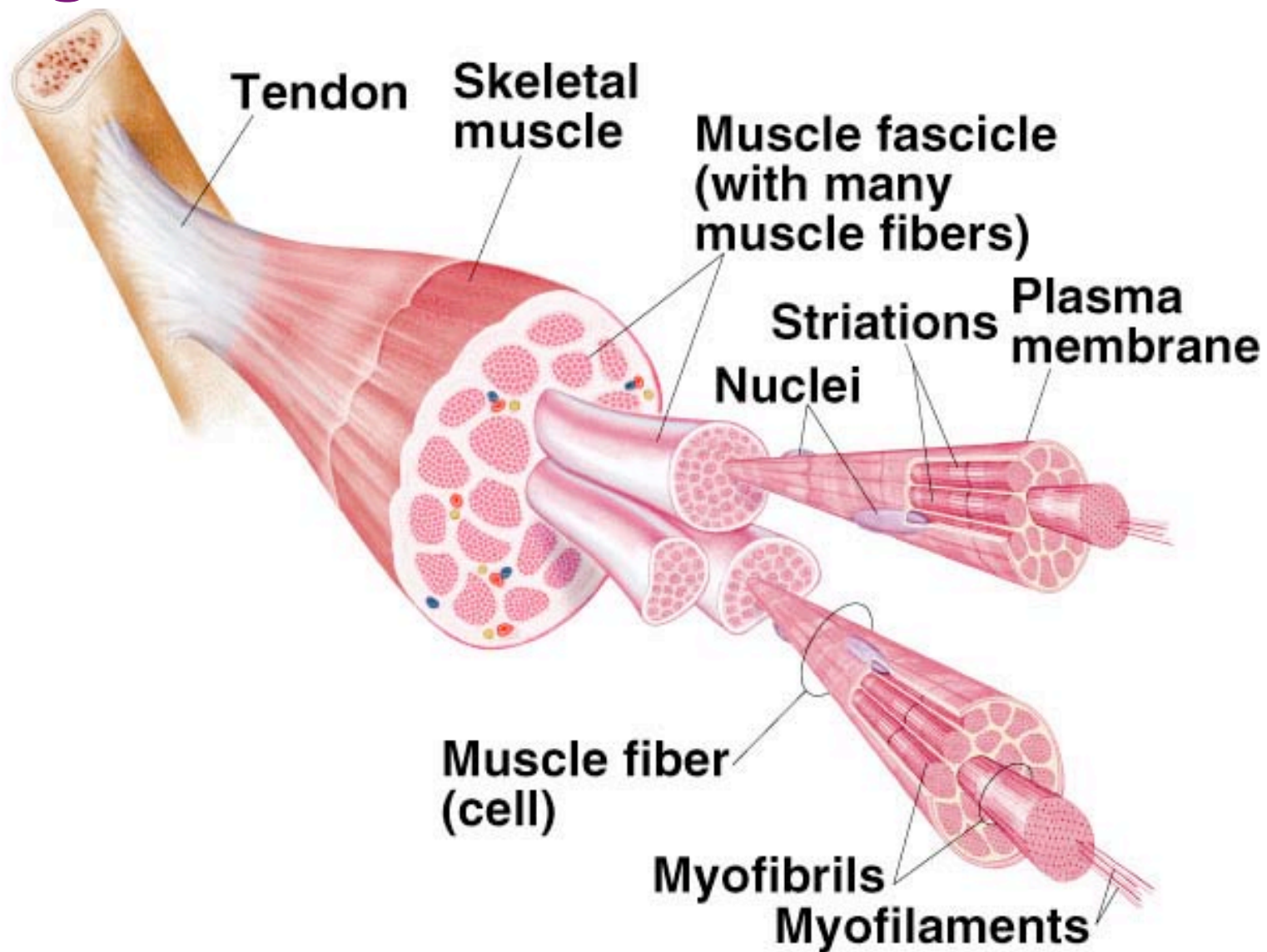




# Muscle

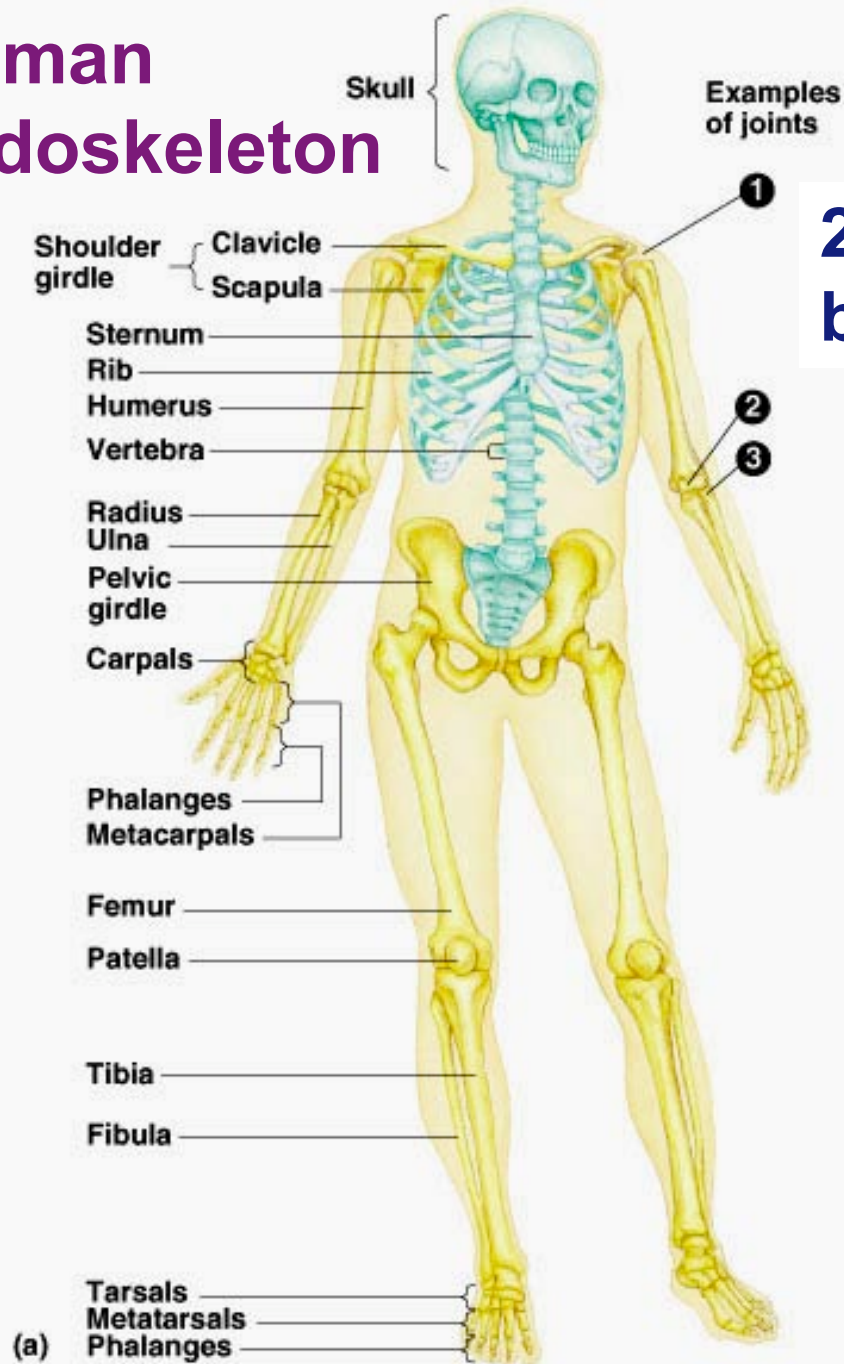


# Organization of Skeletal muscle

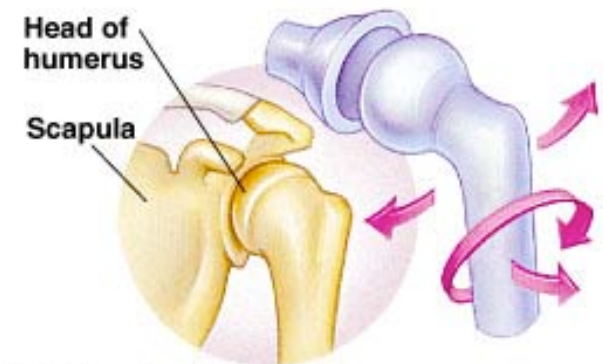




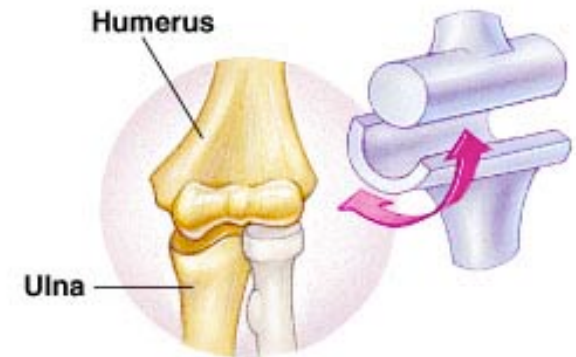
# Human endoskeleton



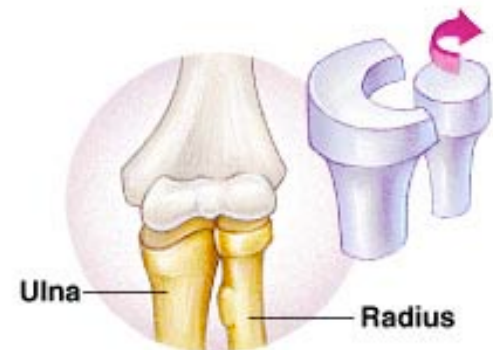
**206 bones**



**1 Ball-and-socket joint**



**2 Hinge joint**

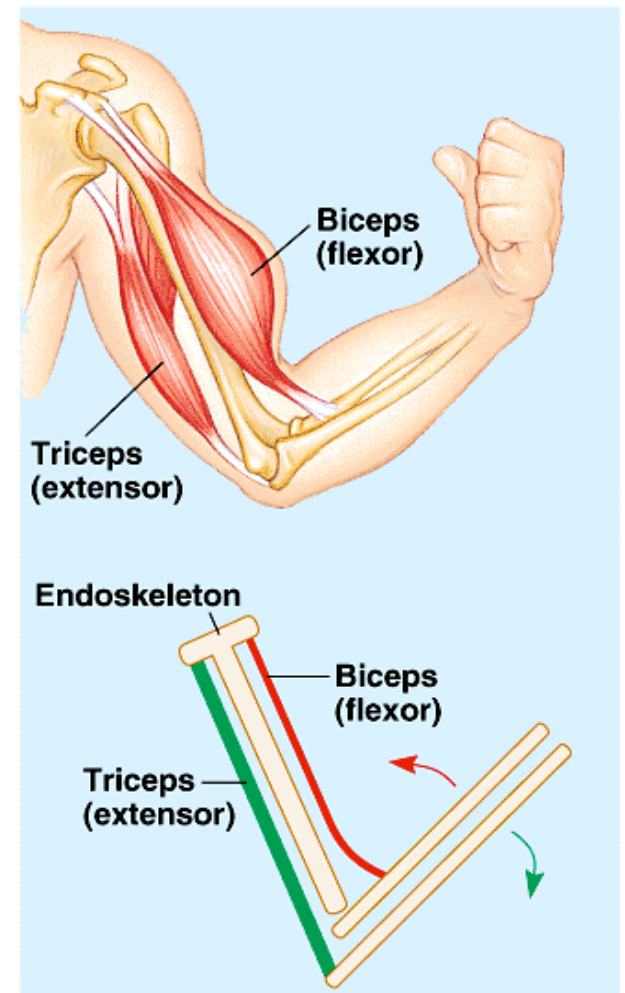


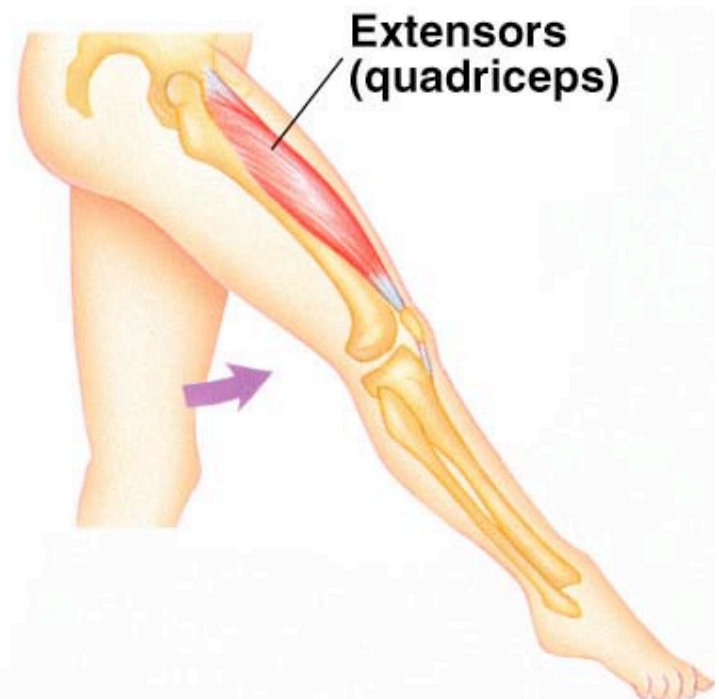
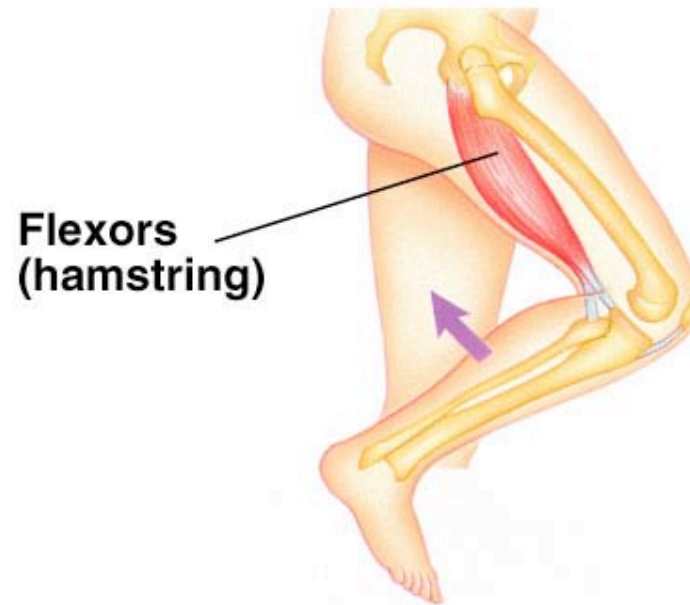
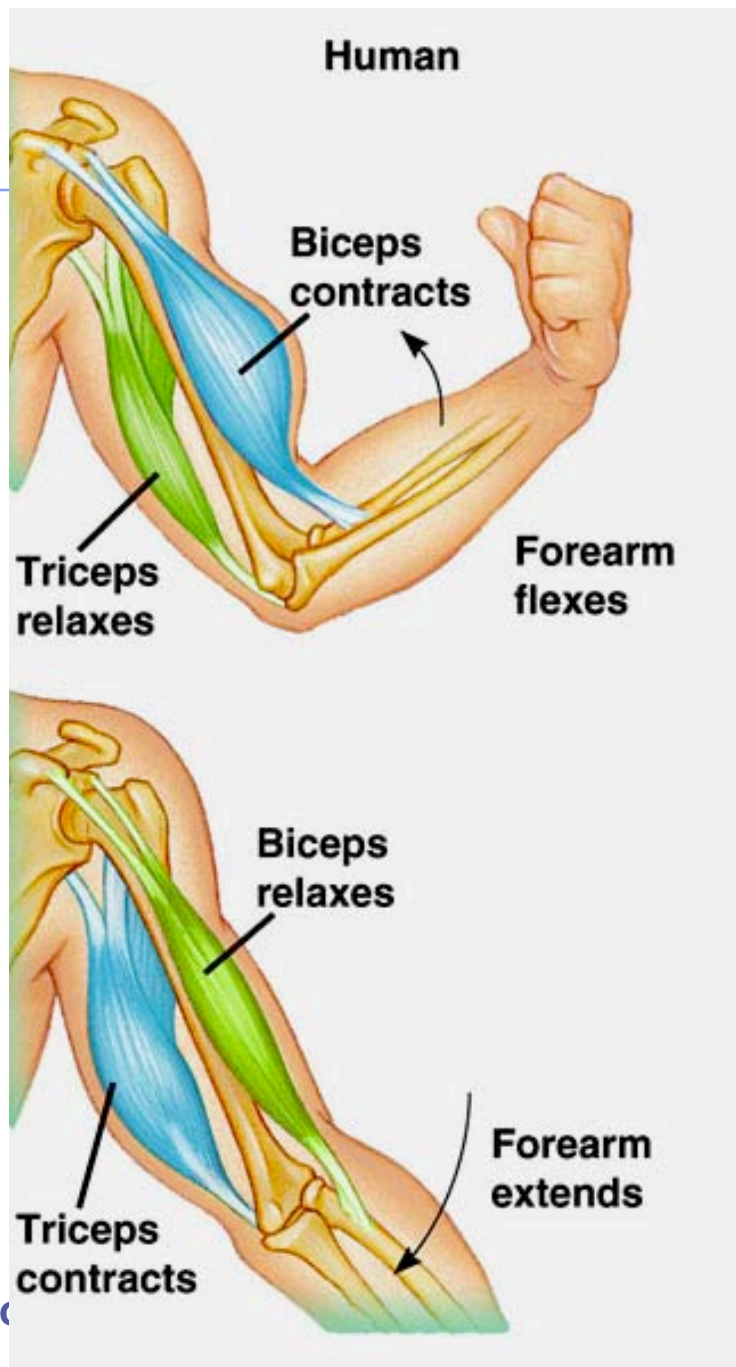
**3 Pivot joint**



# 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



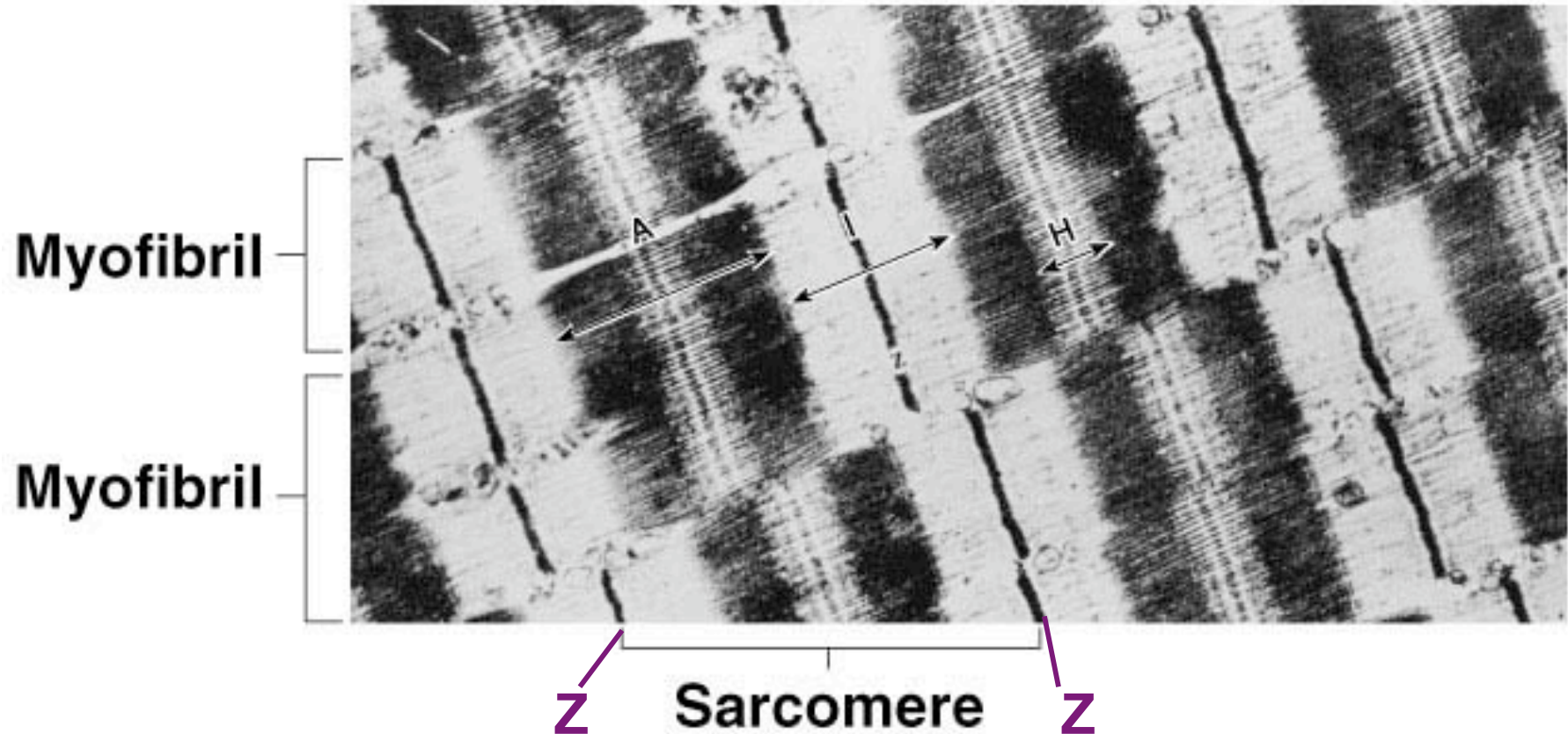




# Striated skeletal muscle

**A band = thick filaments = myosin**

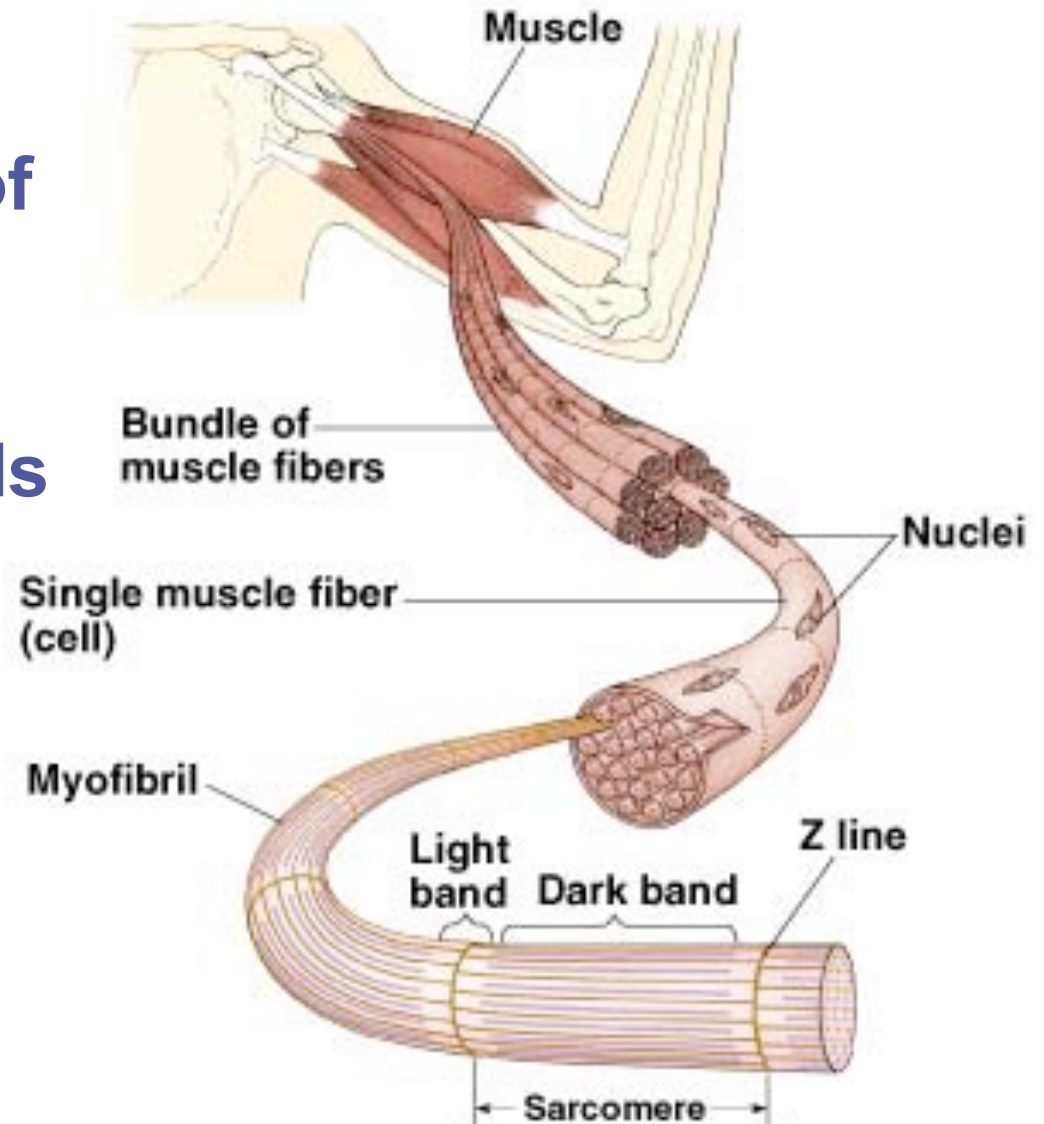
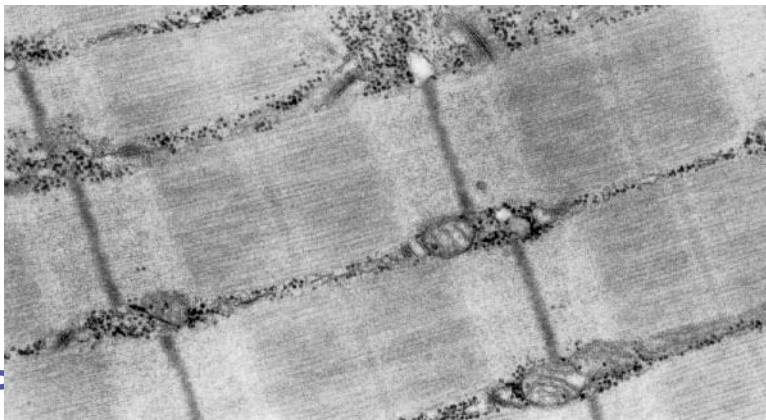
**I band = thin filaments = actin**



# Structure of skeletal muscle

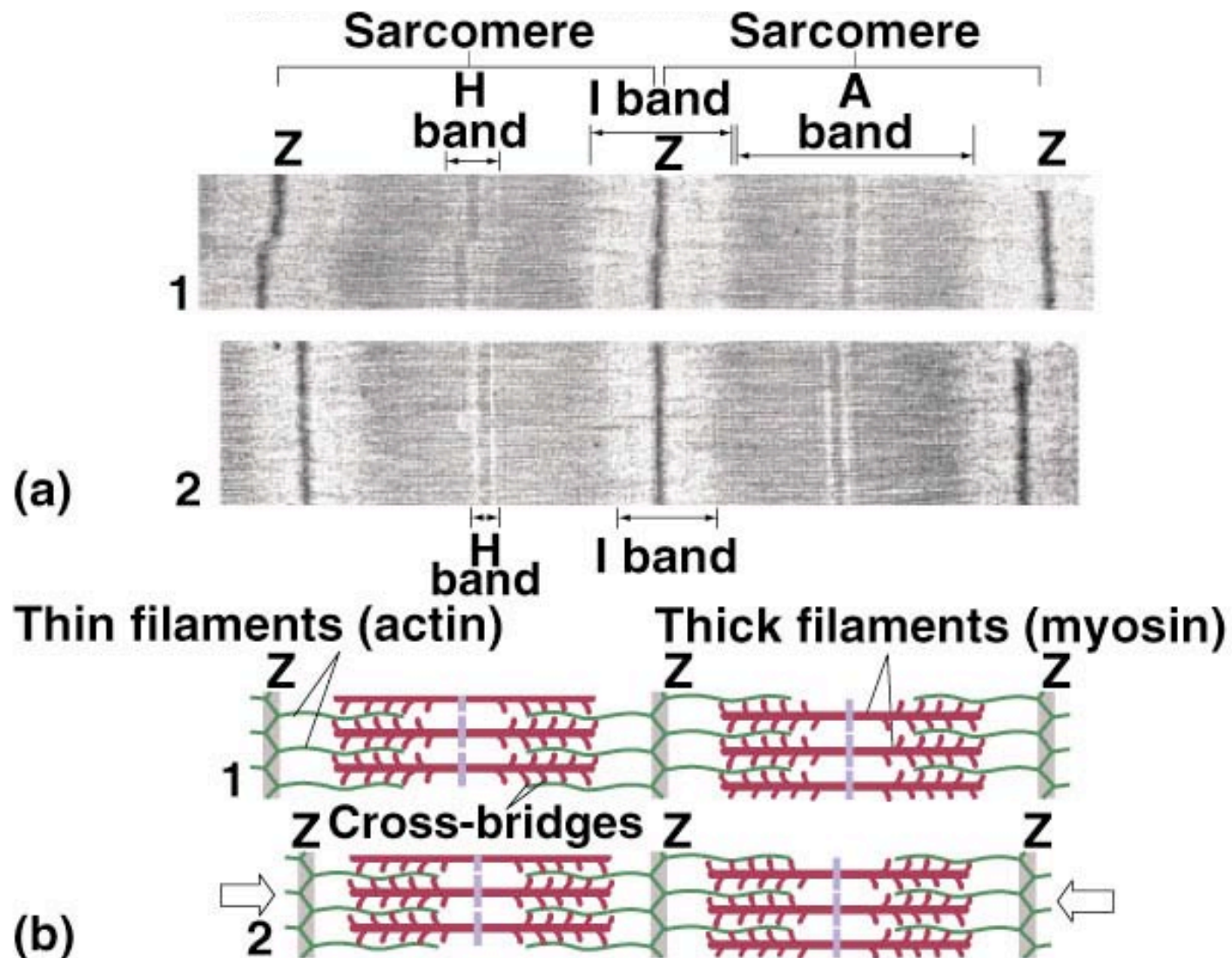
## ■ Sarcomere

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

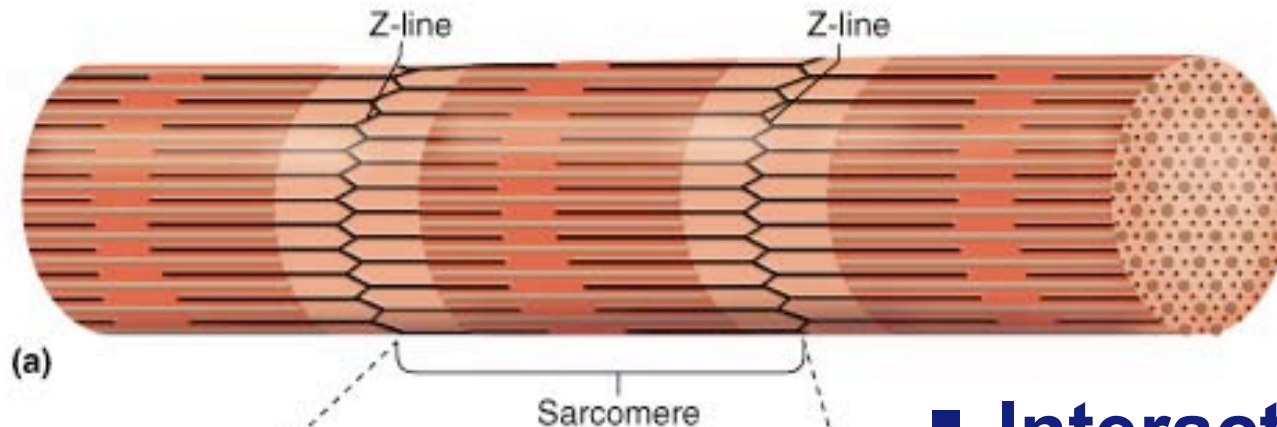




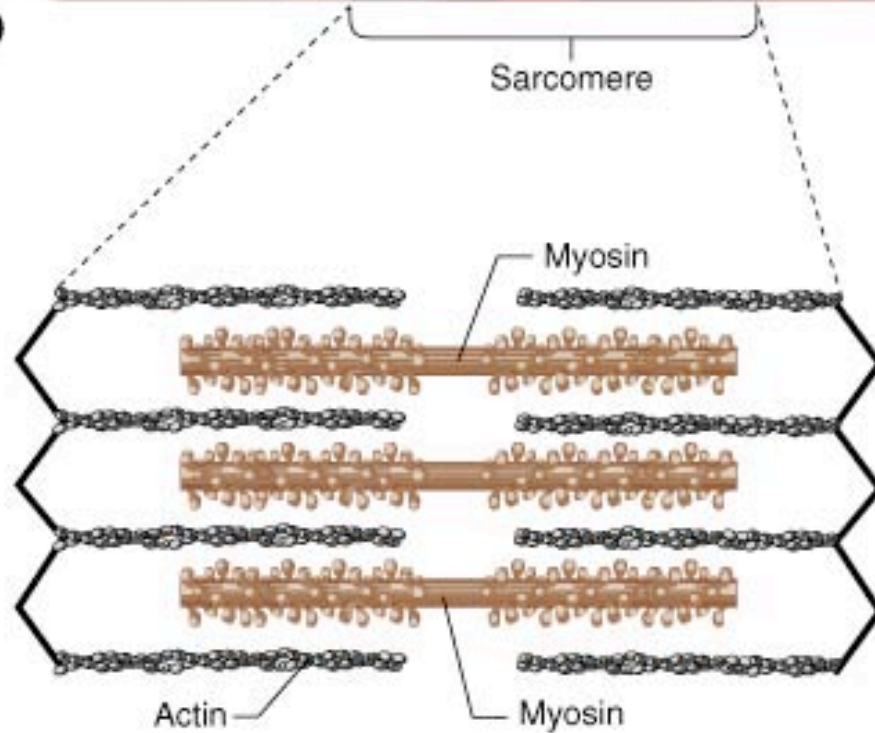
# Sliding Filament mechanism



# Muscle filaments & Sarcomere



(a)



(b)

## ■ Interacting proteins

### ◆ thin filaments

- braided strands of actin & tropomyosin coiled together

### ◆ thick filaments

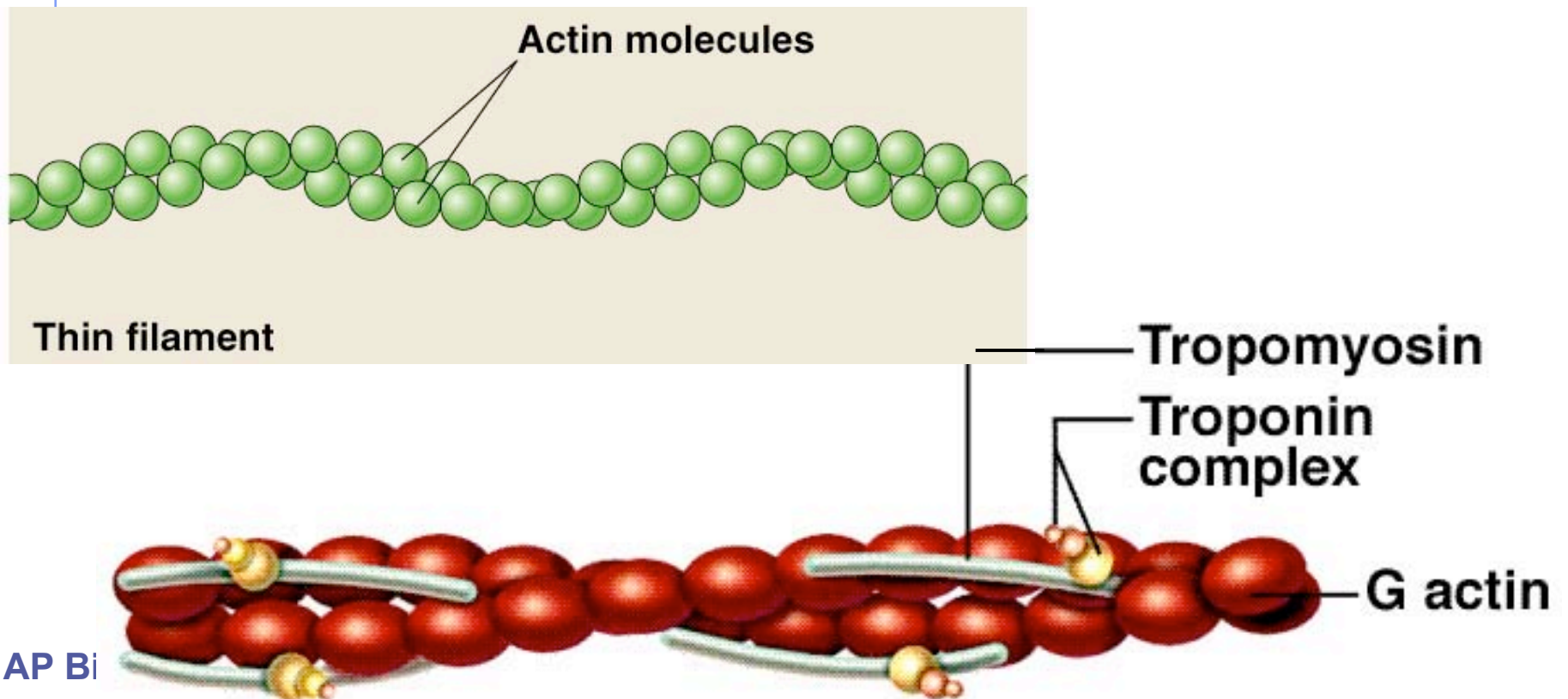
- myosin molecules



# Thin filaments: actin

## ■ Proteins

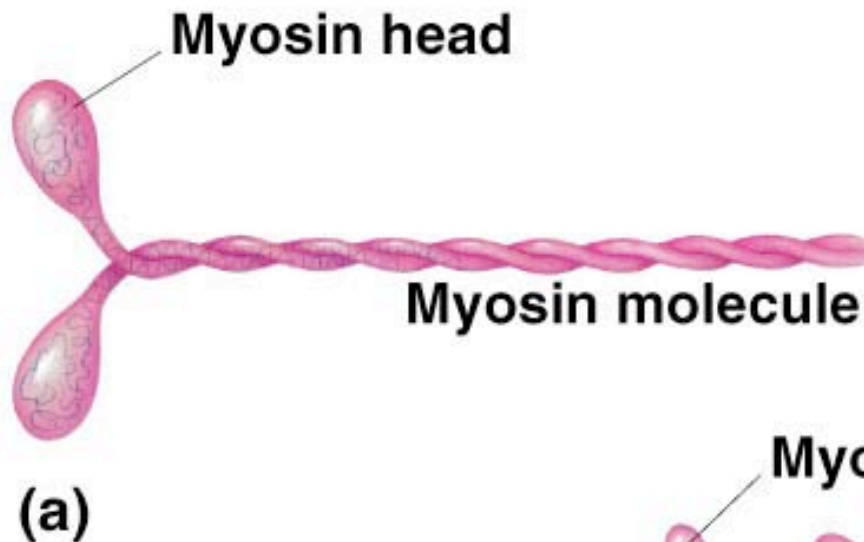
- ◆ braid of actin & tropomyosin molecules
- ◆ dotted with troponin molecules



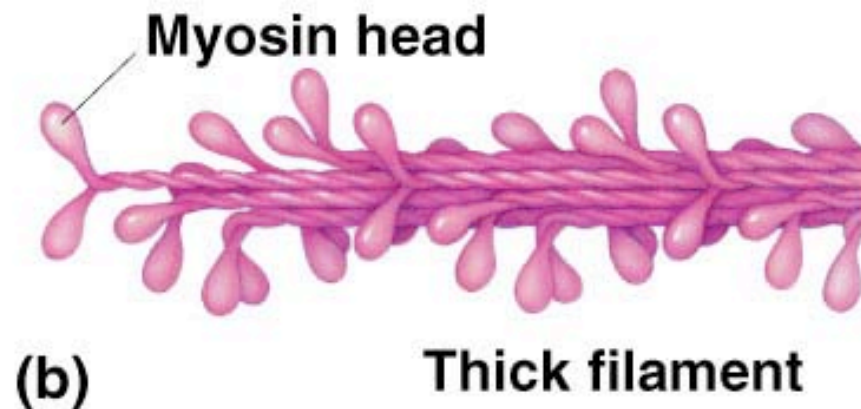
# Thick filaments: myosin

## ■ Protein

- ◆ myosin molecule
- ◆ long protein with globular head



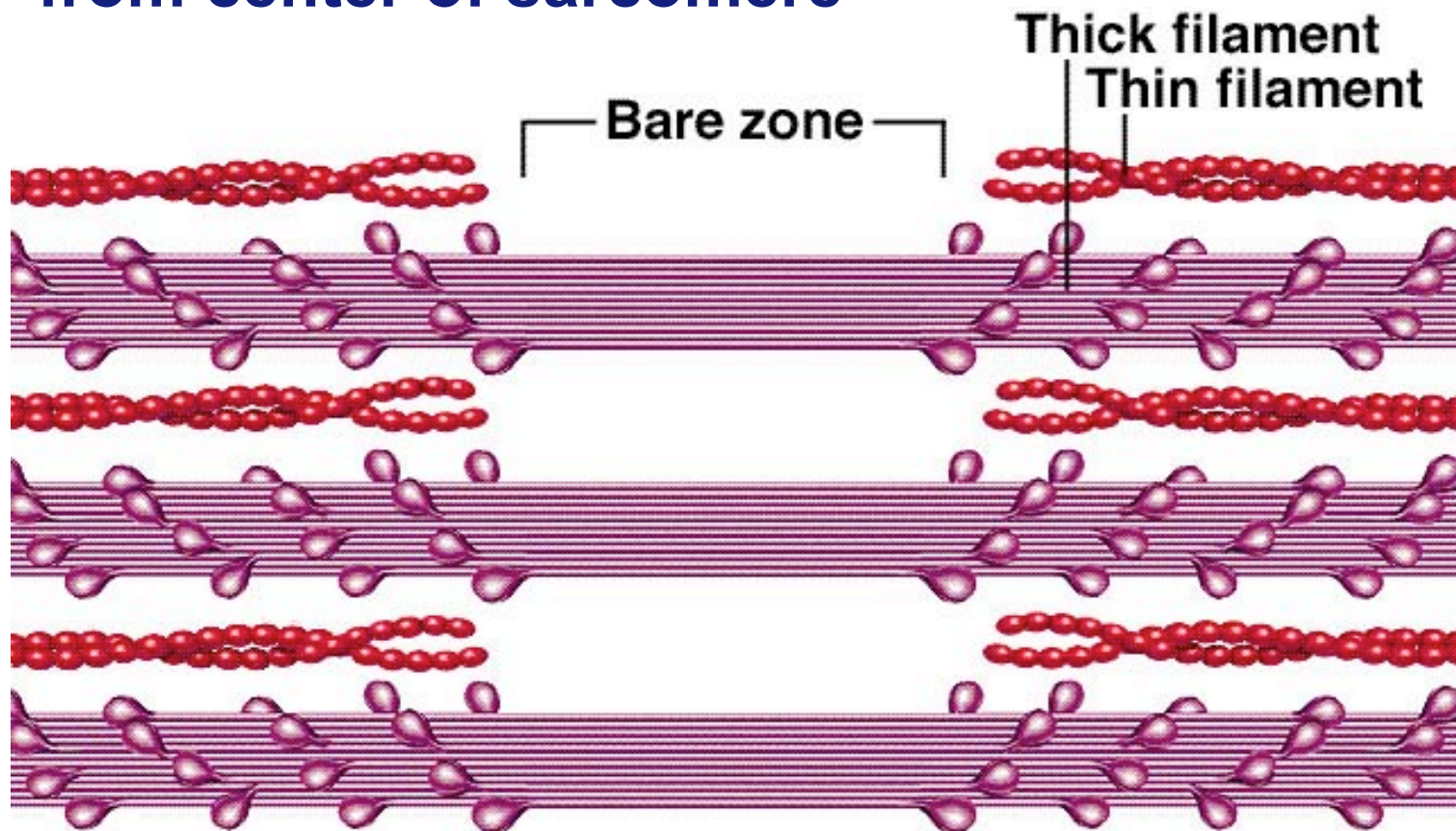
bundle of myosin proteins:  
globular heads aligned  
together





# Thick & thin filaments

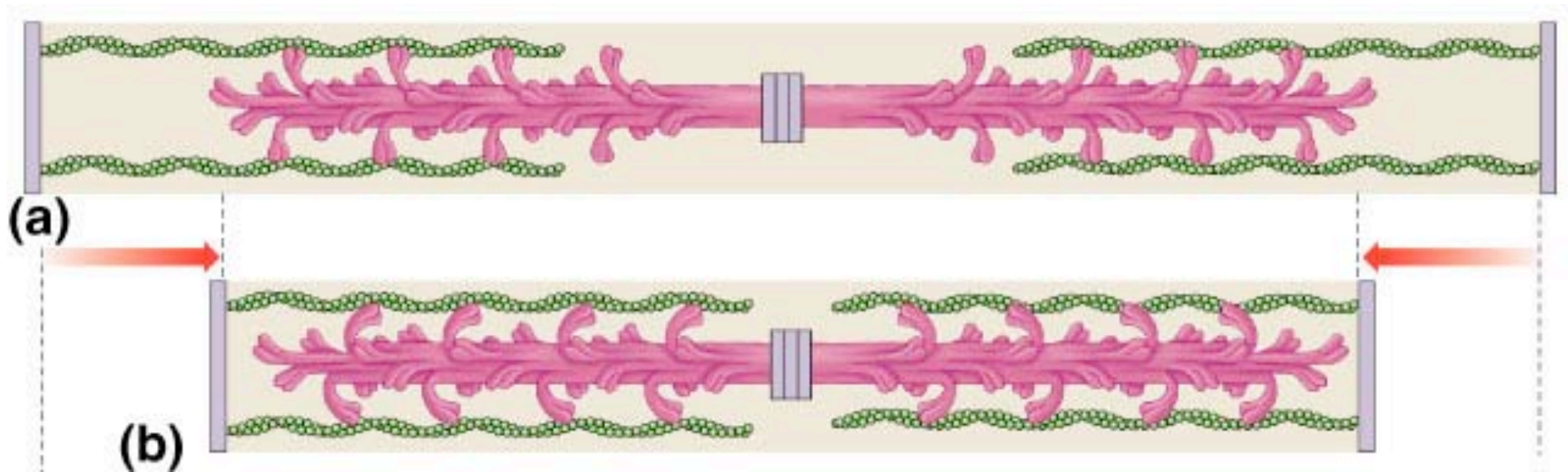
- Myosin tails together & heads pointed away from center of sarcomere



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

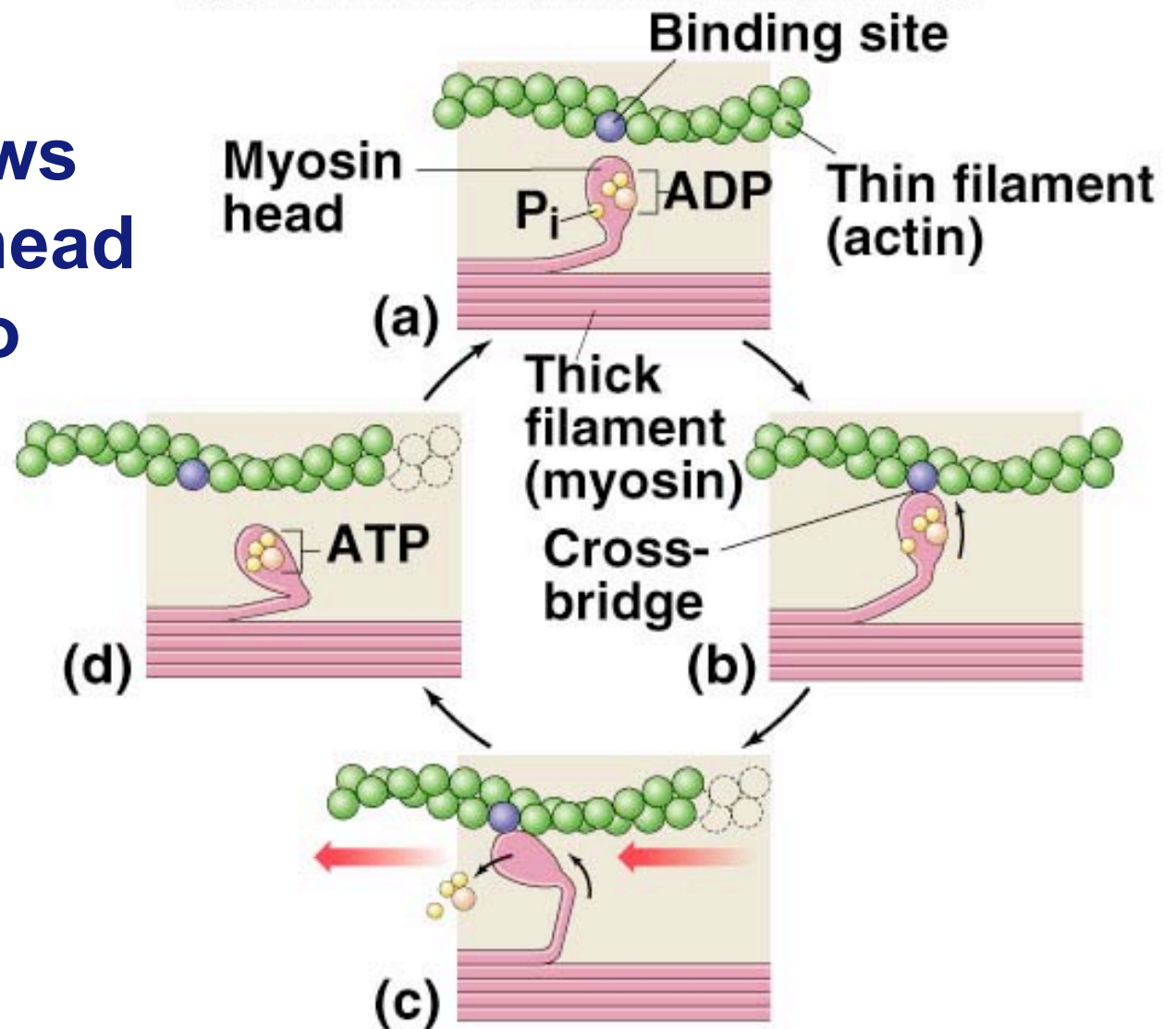
# Interaction of thick & thin filaments

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



# Cross bridge cycle

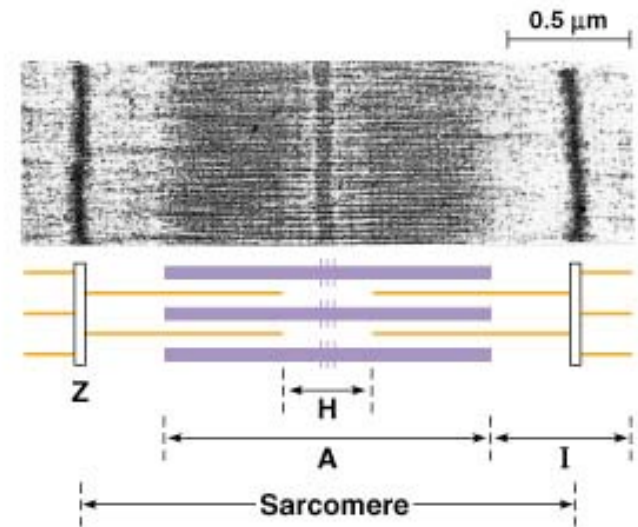
- **Cleaving ATP allows myosin head to bind to actin filament**



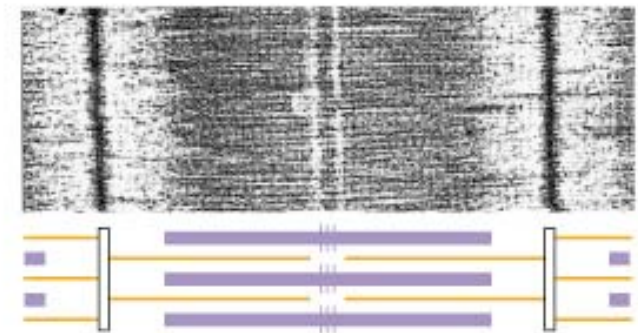


# How a muscle works

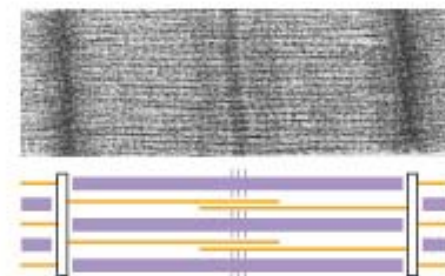
- Myosin pulls actin chain along toward center of sarcomere
- Sarcomere shortens (Z lines move closer together)
- Muscle contracts
  - ◆ energy from:
    - ATP
    - glycogen
    - creatine phosphate



(a) Muscle relaxed (extended)

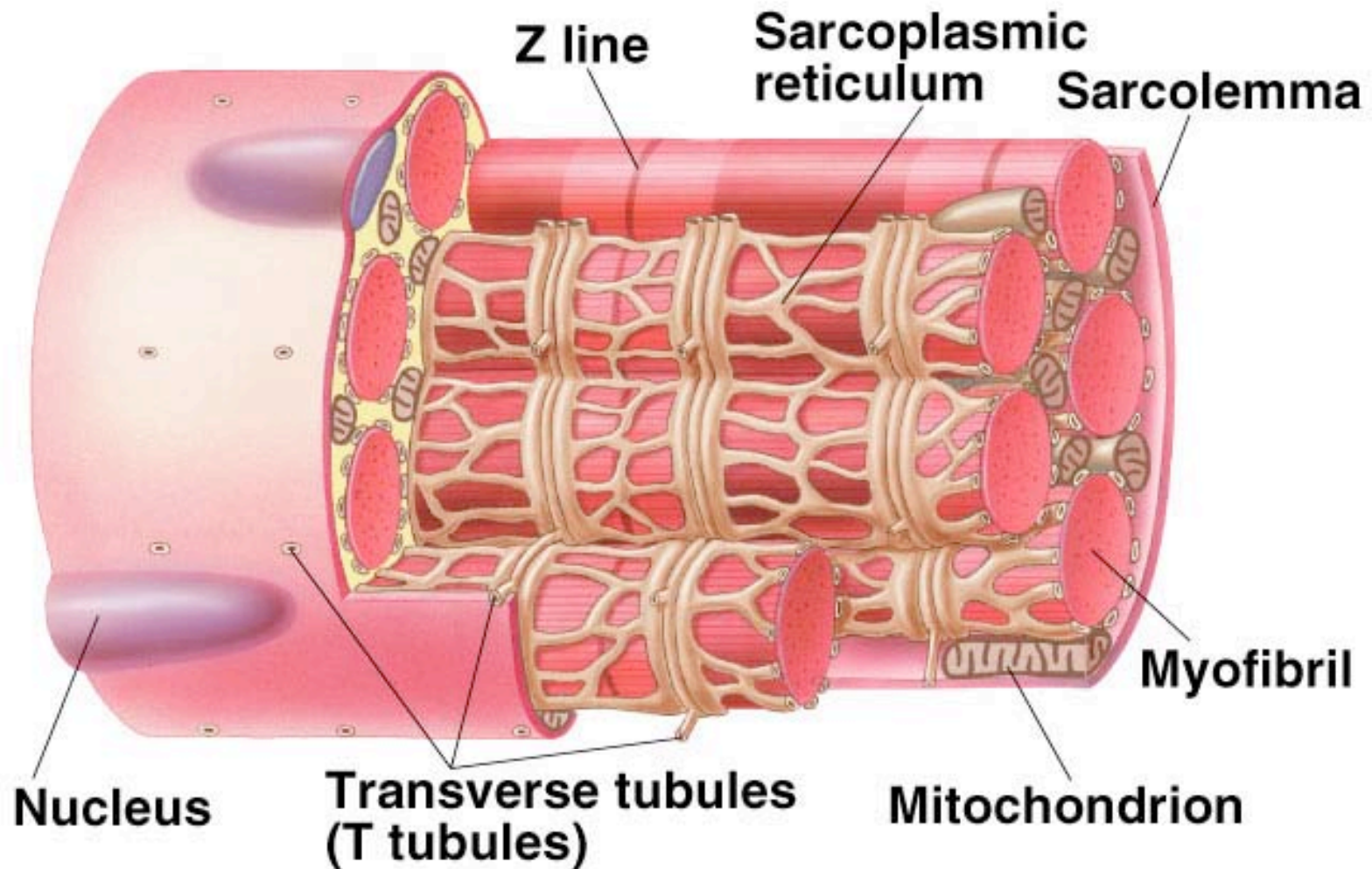


(b) Muscle contracting



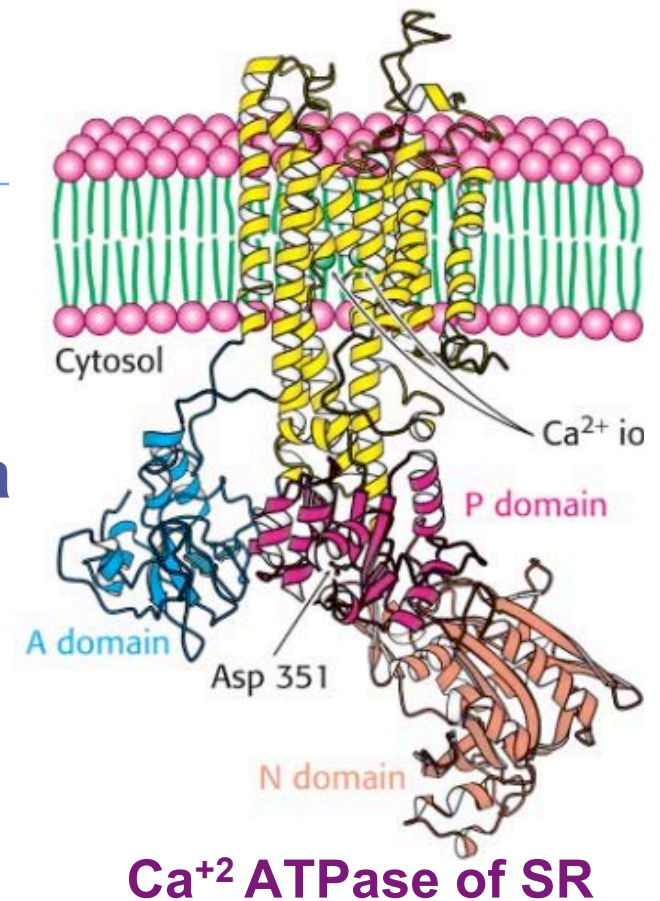
(c) Muscle contracted

# 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  $\text{Ca}^{+2}$ 
    - $\text{Ca}^{+2}$  released from SR through channels
    - $\text{Ca}^{+2}$  pumps then restore  $\text{Ca}^{+2}$  to SR
      - ◆ remove  $\text{Ca}^{+2}$  from cytosol
      - ◆ pumps use ATP

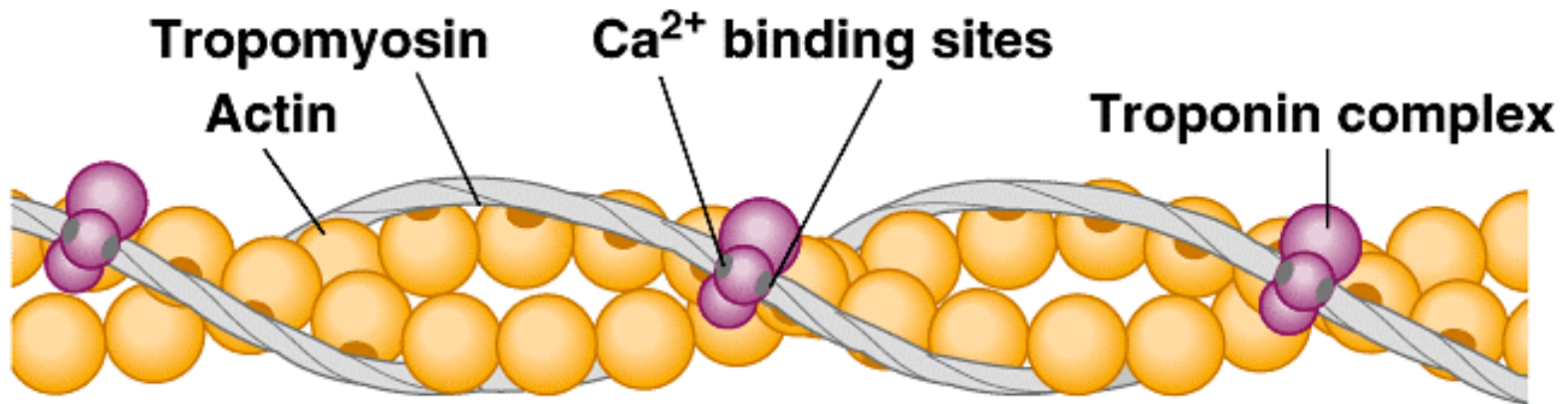




# Muscle at rest

- Interacting proteins

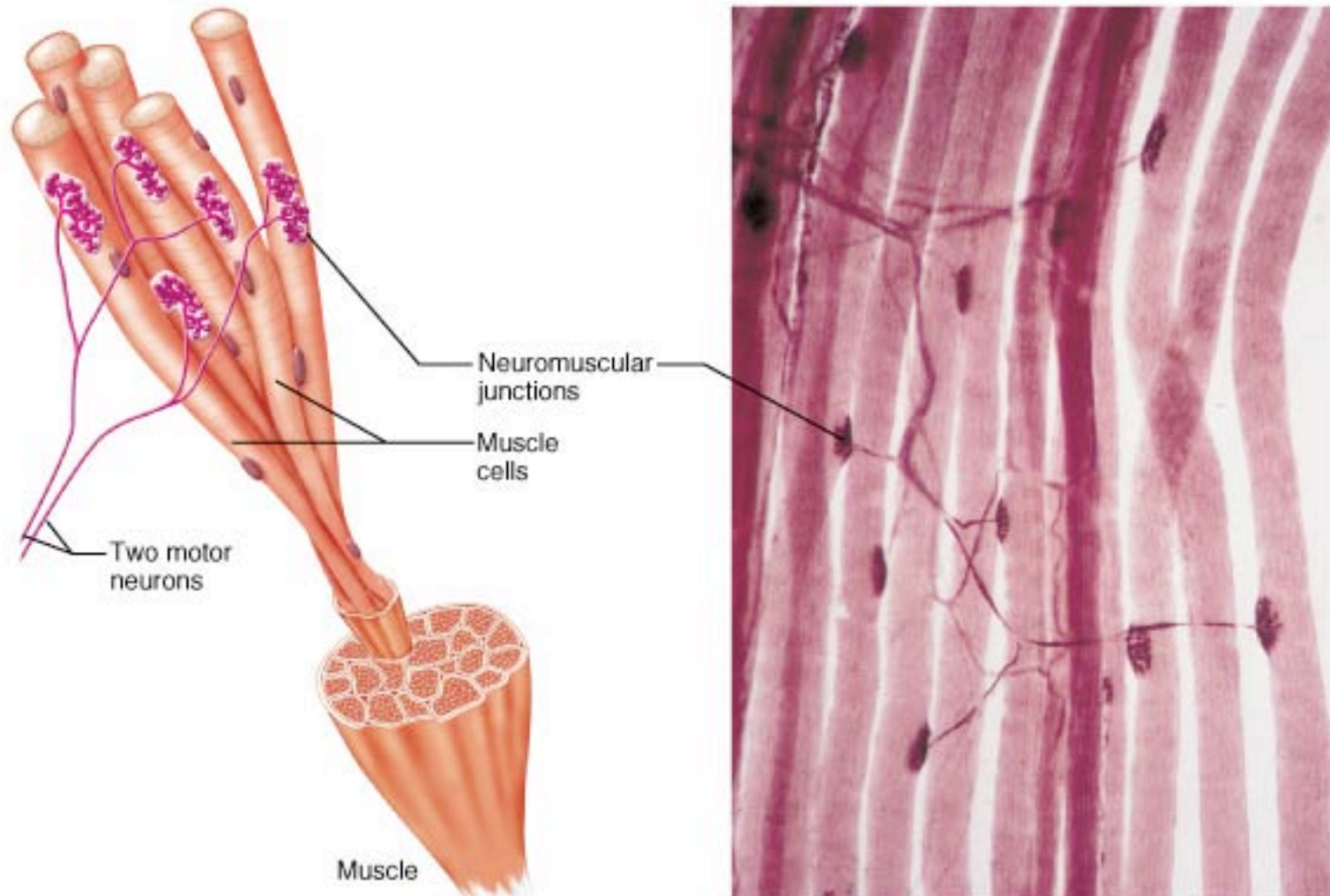
- ◆ at rest, troponin molecules hold tropomyosin molecules so that they cover the myosin-binding sites on actin



(a) Myosin binding sites blocked; muscle cannot contract

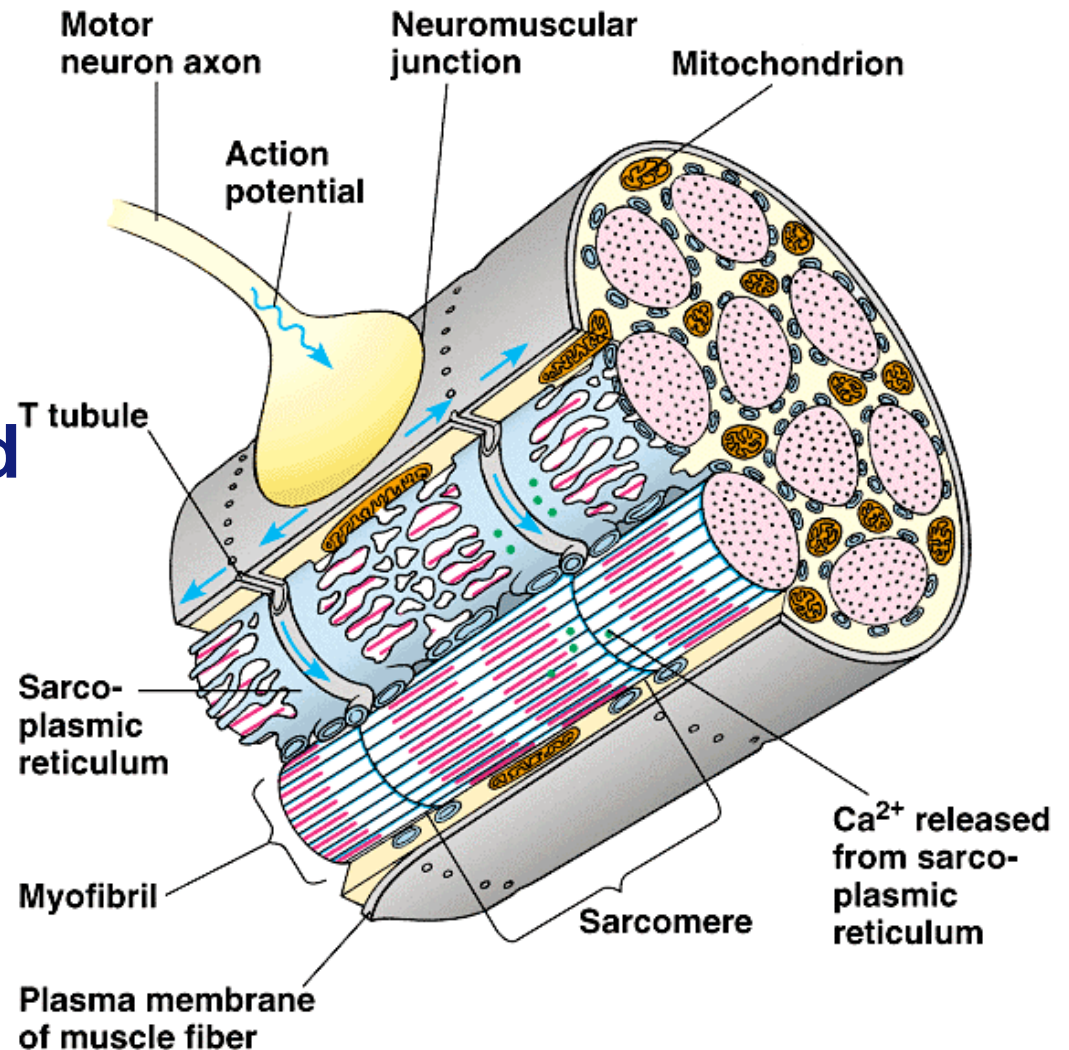
# The Trigger: motor neurons

- Motor neuron triggers muscle contraction



# Nerve trigger of muscle action

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

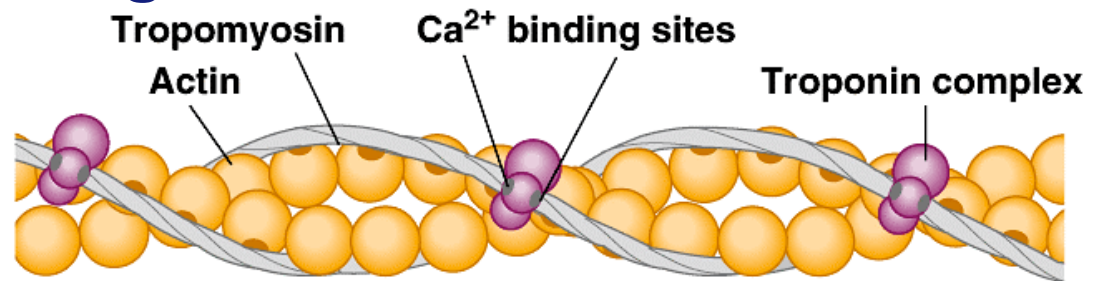




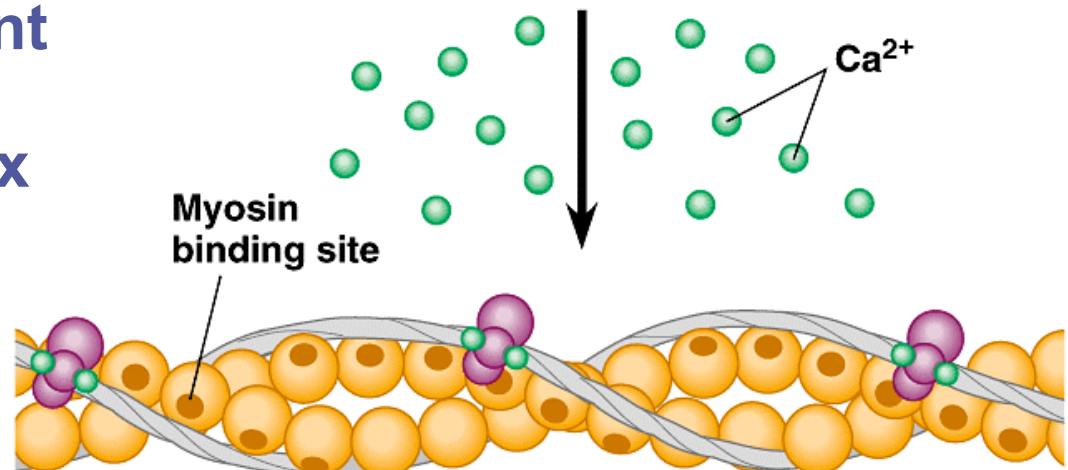
# Ca<sup>2+</sup> triggers muscle action

- At rest, tropomyosin blocks myosin-binding sites on actin
- Ca<sup>2+</sup> binds to troponin complex

- ◆ shape change causes movement of tropomyosin-troponin complex
- ◆ exposes actin's myosin-binding sites



(a) Myosin binding sites blocked; muscle cannot contract

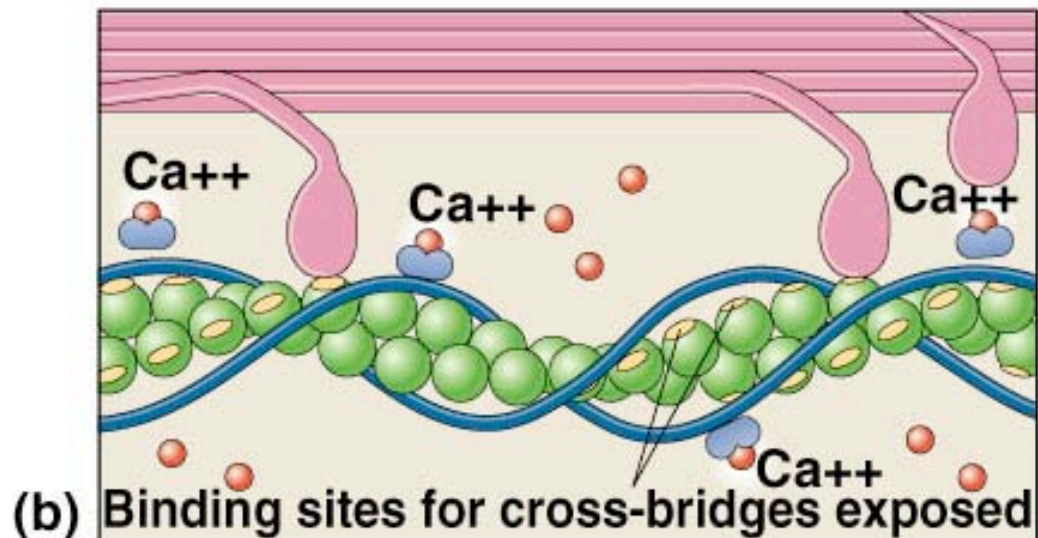
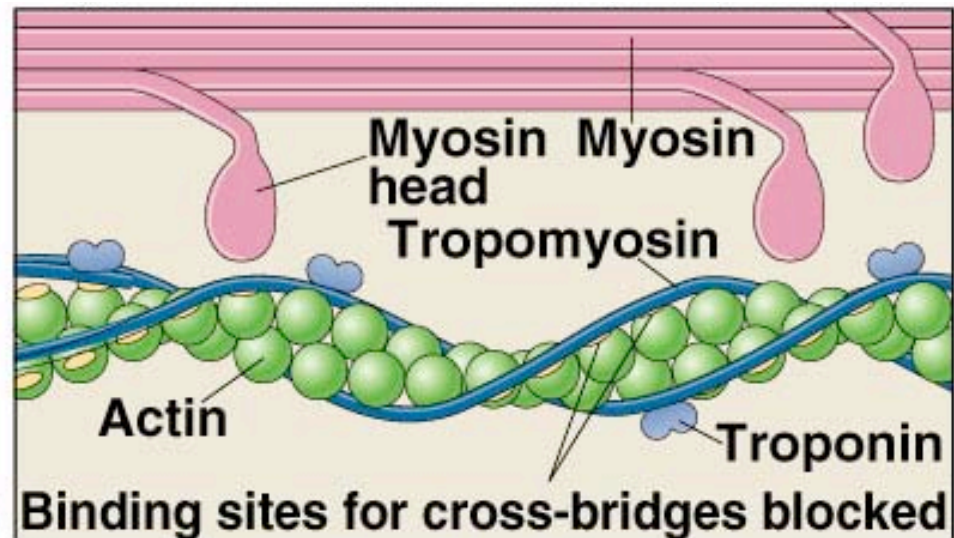


(b) Myosin binding sites exposed; muscle can contract

# How $\text{Ca}^{+2}$ controls muscle

- Sliding filament model
  - ◆ ratchet system

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

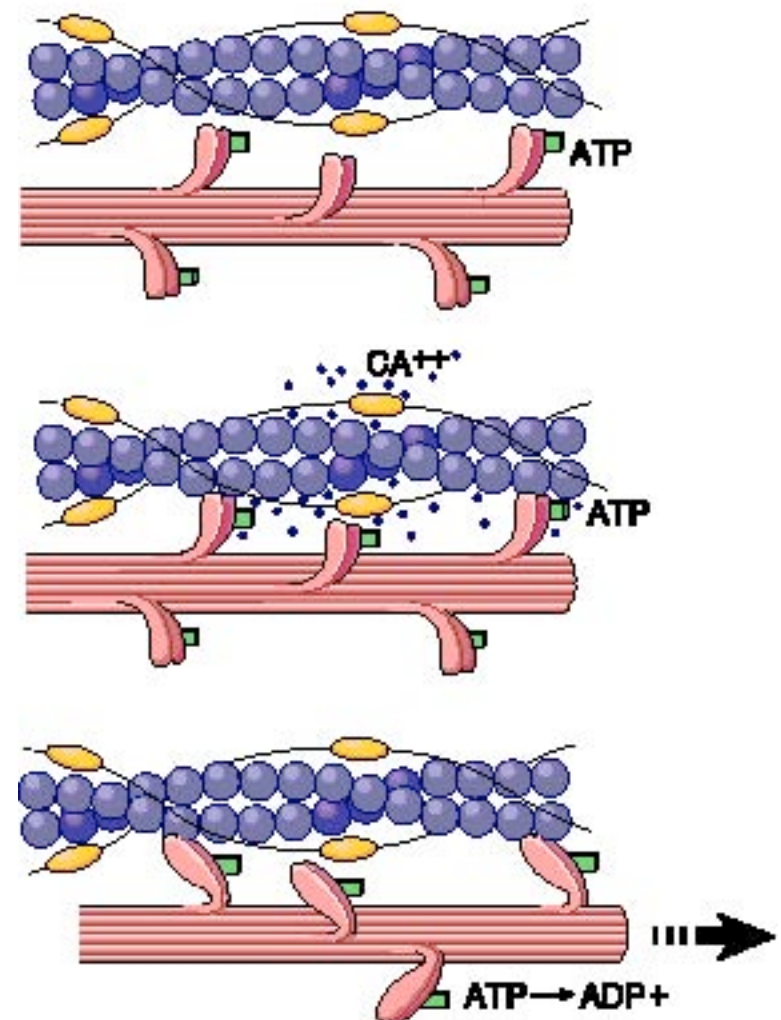


# Sliding filament model

## ■ Ratchet system

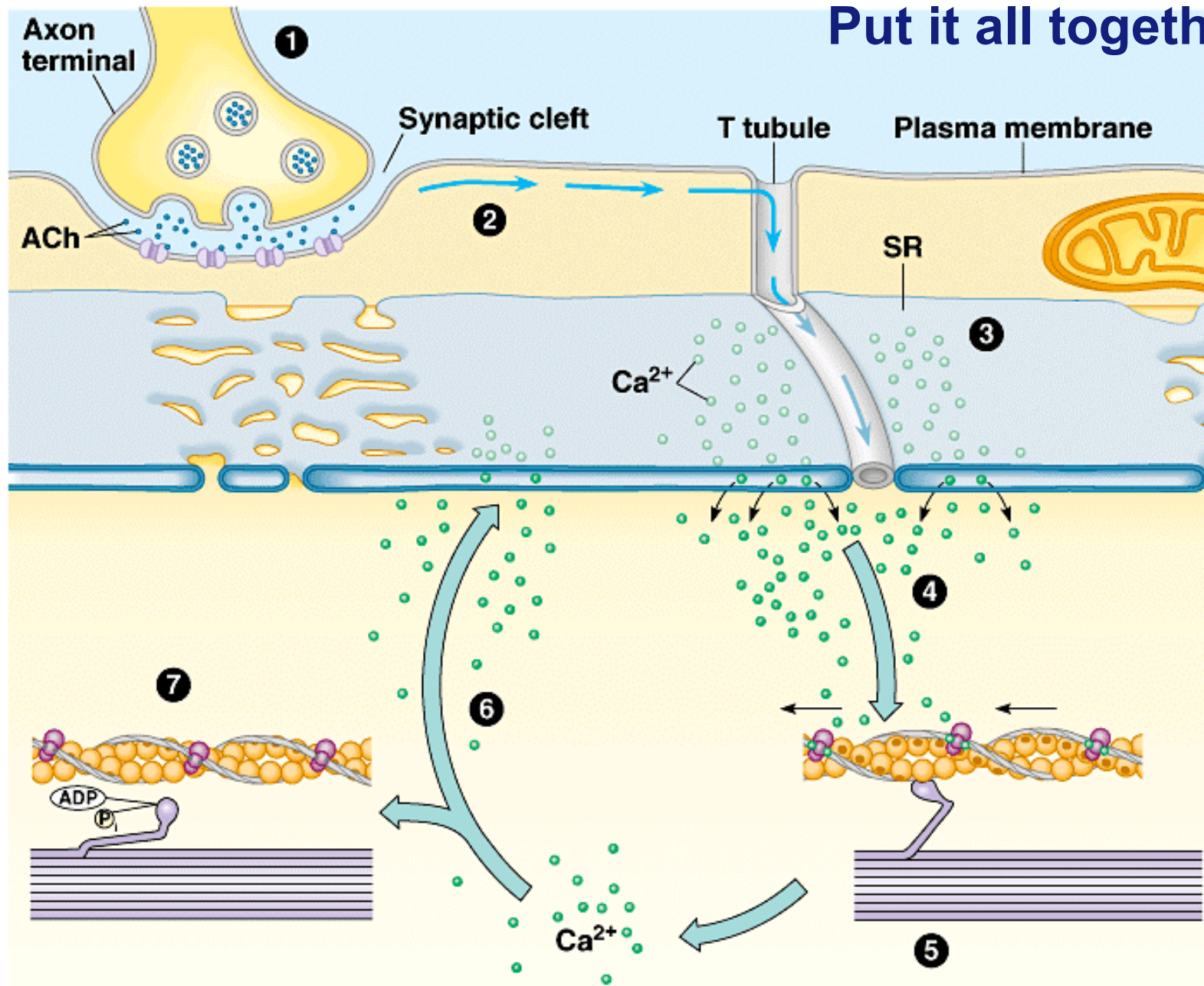
- ◆ myosin bonding with actin
- ◆ sliding thin & thick filaments past each other
- ◆ myosin head releases & binds to next active site on actin
- ◆ muscle doesn't relax until  $\text{Ca}^{+2}$  is pumped back into SR

"Walk-along" Mechanism for contraction of the muscle





Put it all together...



# How it all works...

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





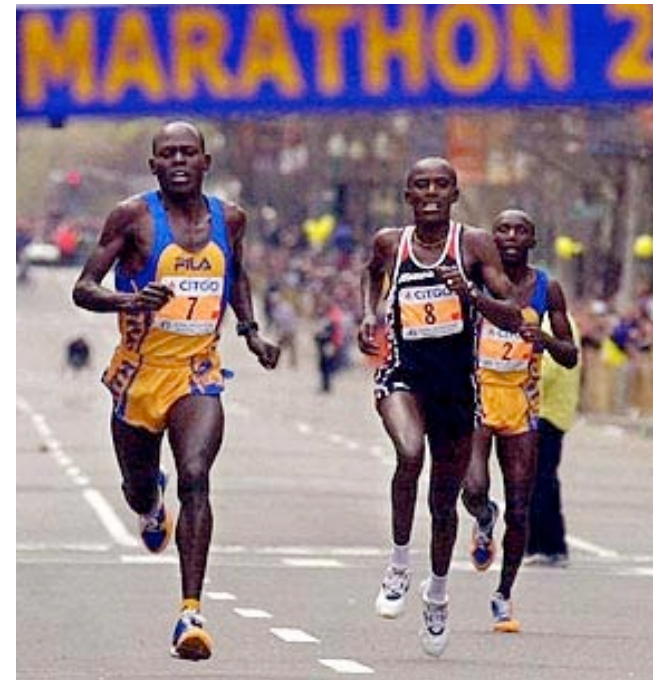
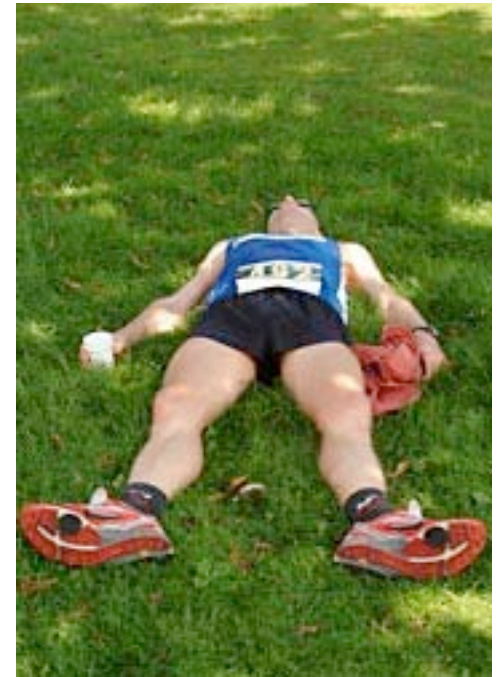
# Muscle fatigue

## ■ Muscle fatigue

- ◆ lack of sugar
  - lack of ATP to restore  $\text{Ca}^{+2}$  gradient
- ◆ low  $\text{O}_2$ 
  - 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**

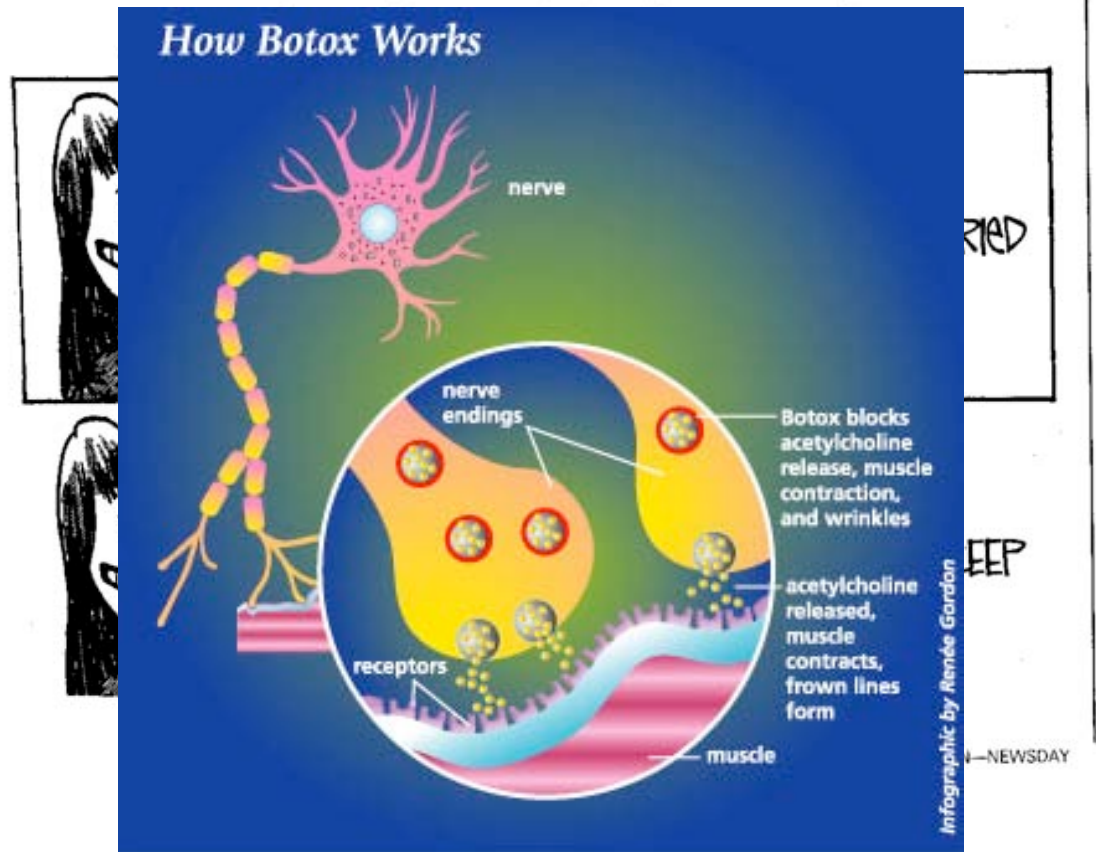
- ◆ auto-immune
- ◆ antibodies to acetylcholine receptors



2005-2006

# 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<sub>2</sub>
- ◆ no O<sub>2</sub>, no respiration
- ◆ no respiration, no ATP
- ◆ no ATP, no Ca<sup>+2</sup> pumps
- ◆ Ca<sup>+2</sup> cannot be removed
- ◆ continuous contraction
- ◆ muscles are tensed
  - muscles stiffen after death
- ◆ eventually tissues breakdown & relax





**Any Questions??**