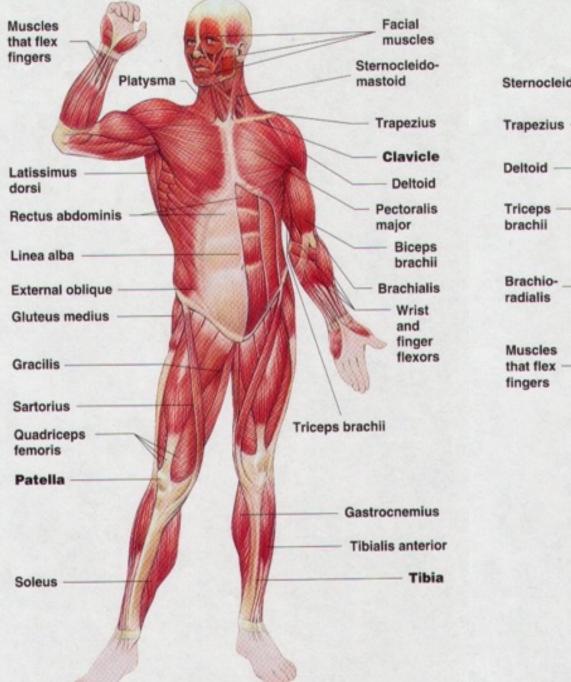
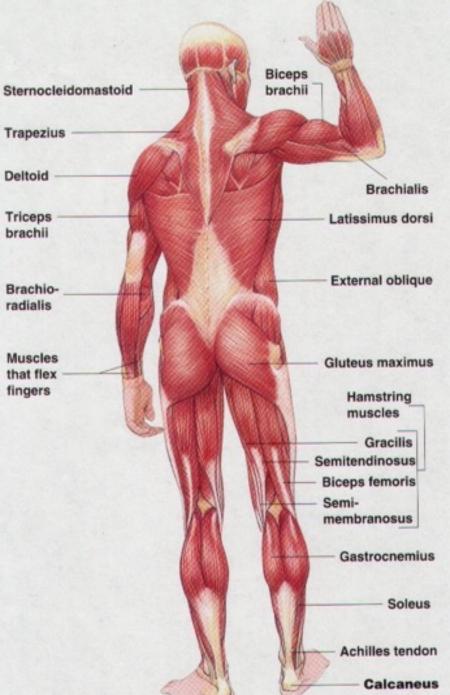
The Muscular System

- What do skeletal muscles do?
- How do muscles work?







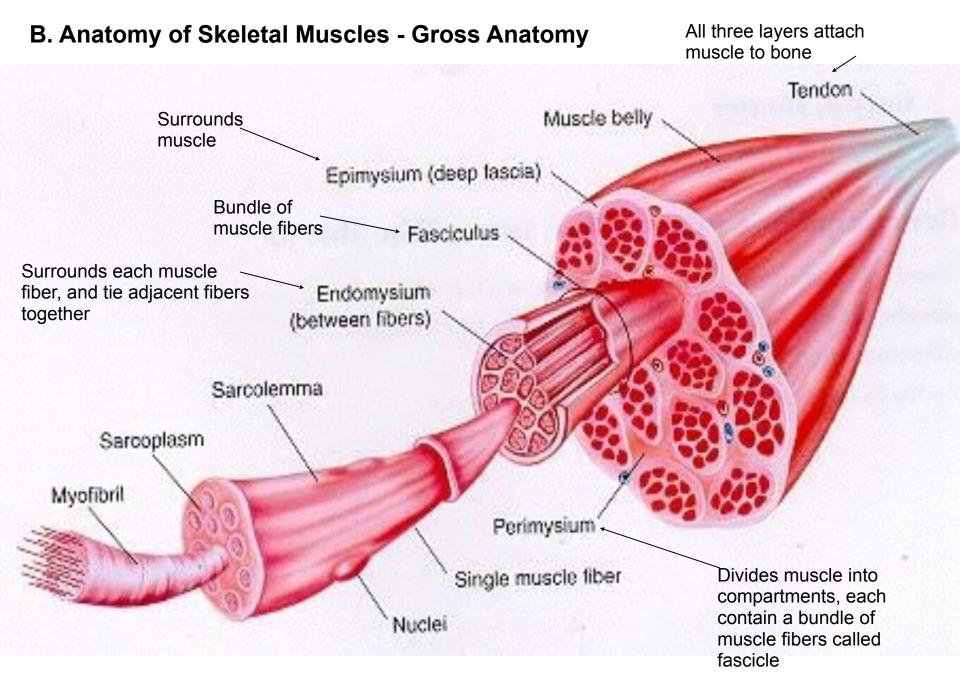
A. Function of Skeletal Muscles

- Produce movement

 Muscle pulls tendons to move the skeleton
- Maintain posture and body position

 Continuous muscle contraction
- Support soft tissue

 Support weight of visceral organs
- Guard entrances and exits
 - Encircle openings to digestive and urinary tracts.
 Control swallowing, defecation and urination
- Maintain body temperature
 - Energy from contraction is converted to heat

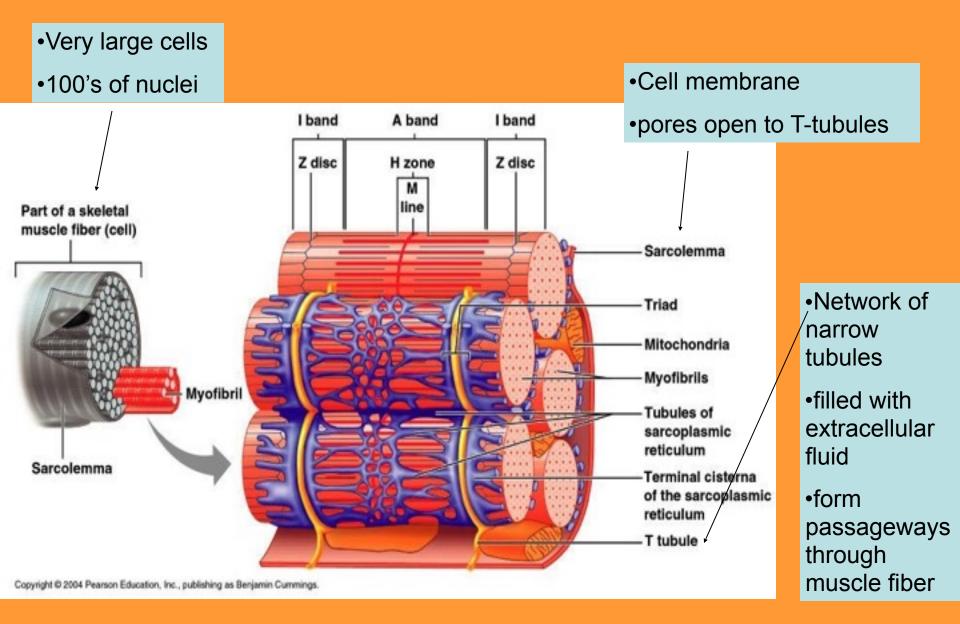


B. Anatomy of a Skeletal Muscle – Blood Vessels and Nerves

- Muscle contractions require energy

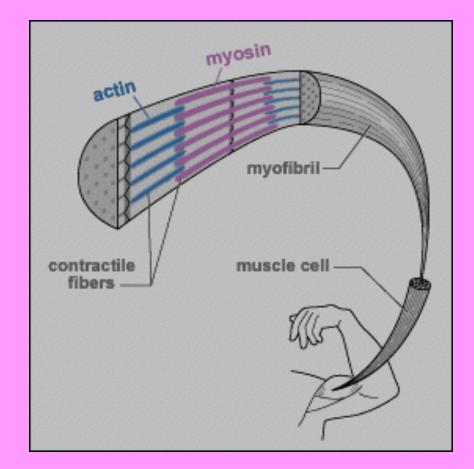
 Blood vessels deliver oxygen and nutrients to produce ATP
- Muscle contractions are under stimulation from the CNS
 - Voluntary control
 - Axons connect to individual muscle fibers

Microanatomy – Sarcolemma and T-Tubules



Myofibrils

- Cylinder as long as entire muscle fiber
- Each fiber contains 100s to 1000s
- Responsible for contraction
- When myofibrils contract the whole cell contracts
- Consist of proteins
 - Actin thin filaments
 - Myosin thick filaments



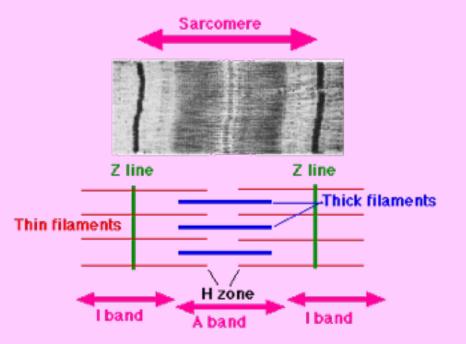
Sarcoplasmic Reticulum

- Specialized form of SER
- Tubular network around each myofibril
- In contact with T-Tubule
- Cisternae expanded chambers of SR, store Calcium



Sarcomere

- Smallest functional unit of muscle fiber
- Each myofibril contains 10,000 sarcomeres end to end
- Interaction between thick and thin filaments cause contraction
- Banded appearance



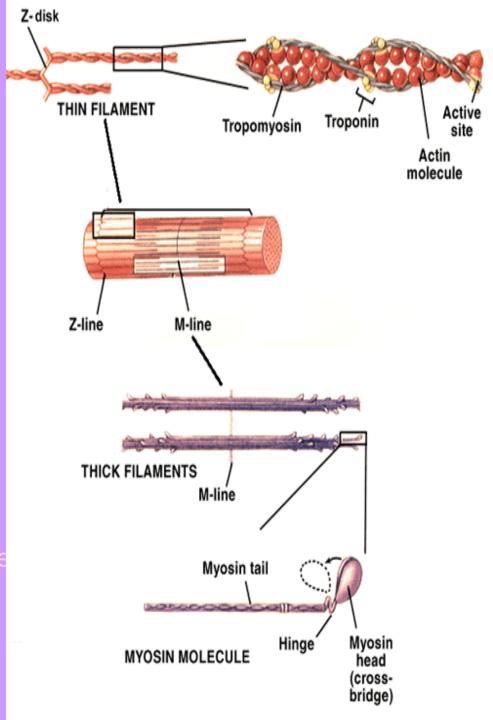
Thick and Thin Filaments

• Thin

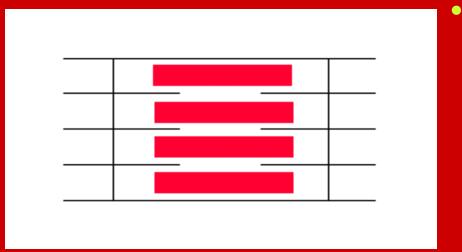
- twisted actin molecules
- Each has an active site where they interact with myosin
- Resting active site covered by tropomyosin which is held in place by troponin

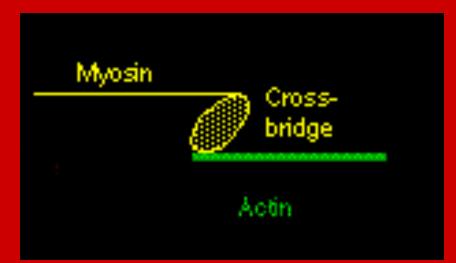
• Thick

- Myosin
- Head attaches to actin during contraction
- Can only happen if troponin changes position, moving tropomyosin to expose active site



Sliding Filaments and Cross Bridges





Sarcomere contraction – Sliding Filament Theory

- Thin filaments slide toward center of sarcomere
- Thick filaments are stationary
- Myosin head attaches to active site on actin (cross bridge)
- Pull actin towards center, then detaches

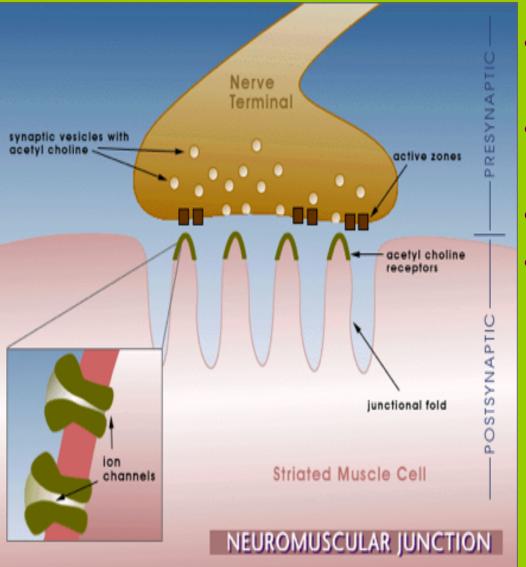
Questions

- How would severing the tendon attached to a muscle affect the ability of the muscle to move a body part?
- Why does skeletal muscle appear striated when viewed through a microscope?
- Where would you expect the greatest concentration of calcium ions in resting skeletal muscles to be?

Control of Muscle Fiber Contraction

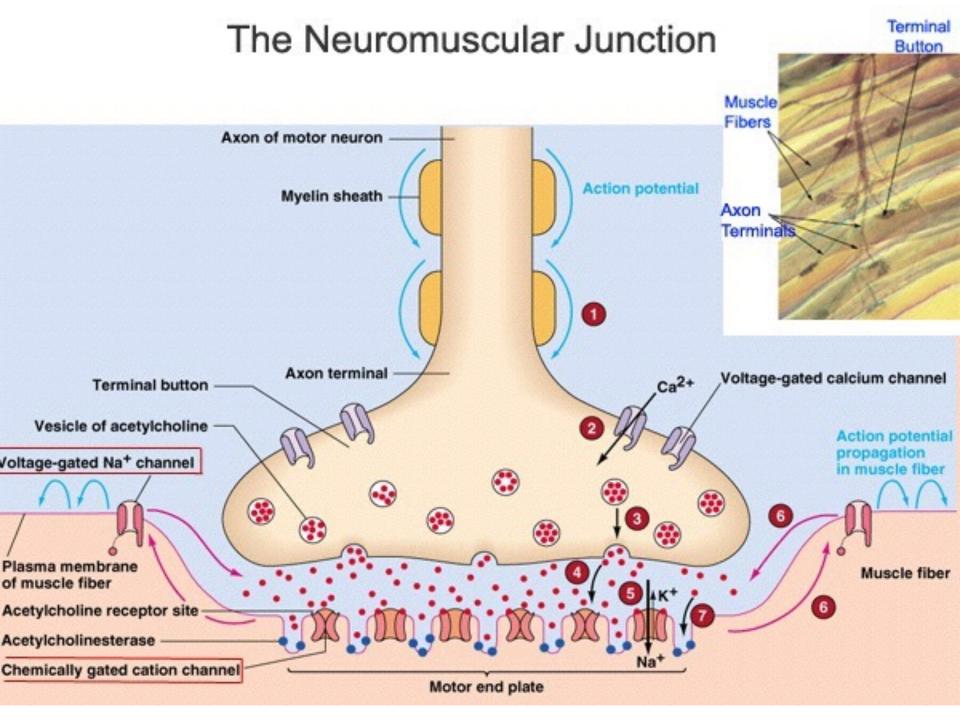
Under control of the nervous system

Neuromuscular Junction

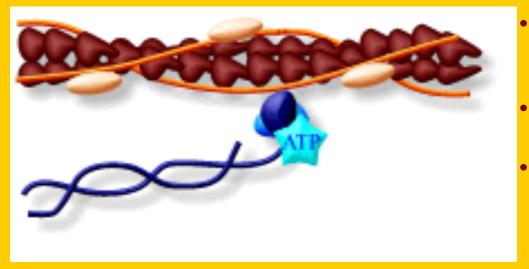


- Link between NS and muscle
- Motor neuron control skeletal muscle fibers
- Synaptic terminal
- Acetylcholine (Ach) chemical released by neuron to communicate with other cells
 - Triggers change in sarcolemma which triggers contraction

- 1. Action potential travels to axon of motor neuron
- 2. Ach is released into synaptic cleft
- 3. Ach diffuses across synaptic cleft & binds to Ach receptors on sarcolemma
 - 1. This changes permeability to sodium
 - 2. Sudden rush of sodium into sarcolemma
 - 3. Causes action potential sarcolemma
- 4. Action potential spreads over entire sarcolemma, down t-tubules to cisternae
- 5. Cisternae release massive amounts of calcium
- 6. Increase in calcium sarcomeres contract
- 7. Ach broken down by AchE



The Contraction Cycle



- Resting sarcomere
 - ADP + P attached to myosin head (stored energy)
- Step 1
 - Ca+ binds to troponin exposing active site on actin
- Step 2
 - Myosin head attaches to actin
- Step 3
 - Pulling of crossbridge towards center of sarcomere
 - ADP + P released (energy used)
- Step 4
 - Myosin head binds another ATP
 - Detachment of cross bridge
- Step 5
 - ATP ADP + P, reactivation of myosin head

Summary of Muscle Contraction

- 1. Brain spinal cord motor nerve neuromuscular junction
- 2. Acetylcholine(ACH) released by synaptic vesicles, crosses synaptic cleft -Acetylcholinesterase enzyme breaks down ACH, binds to receptors
- Sodium ions "leak" into muscle cell initiating action potential which travels T-tubules to sarcoplasmic reticulum (SR)
- 4. Calcium ions (hifg affinity for troponin) released from SR
- 5. Calcium binds with troponin
- 6. Shift of tropomyosin, make sites available for myosin
- 7. With ATP present, ATPase splits ATP to ADP + P + Energy
- 8. Myosin combines with actin
- 9. Sliding action of actin over myosin (Sliding filament theory)
- 10. Impulse stops, calcium or ATP depleted, calcium ions pumped to SR
- Tropomyosin returns over active sites on actin, myosin no longer bound

Questions

How would a drug that interferes with cross-bridge formation affect muscle contraction?

What would you expect to happen to a resting skeletal muscle if the sarcolemma suddenly became very permeable to calcium ions?

Predict what would happen to a muscle if the motor end plate did not contain acetylcholinesterase.

