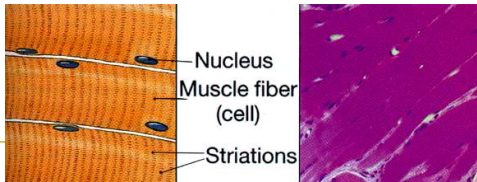


Chapter 12: Muscles

Review muscle anatomy (esp. microanatomy of skeletal muscle)



Terminology:

- sarcolemma
- t-tubules
- sarcoplasmic reticulum
- myofibers, myofibrils, myofilaments
- sarcomere

More Terminology:

- Tension
- Contraction
- Load
- Excitation-contraction coupling
- Rigor
- Relaxation

Anatomy

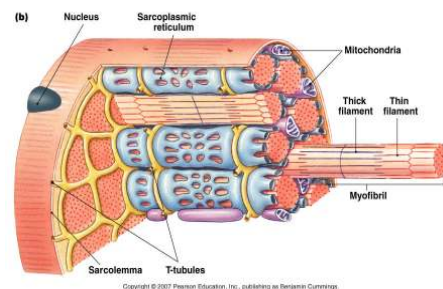


Fig 12-3

More Anatomy

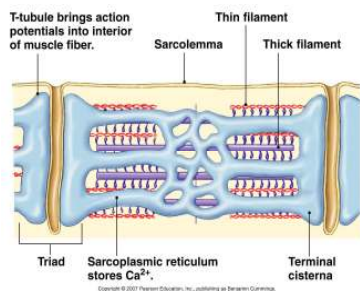


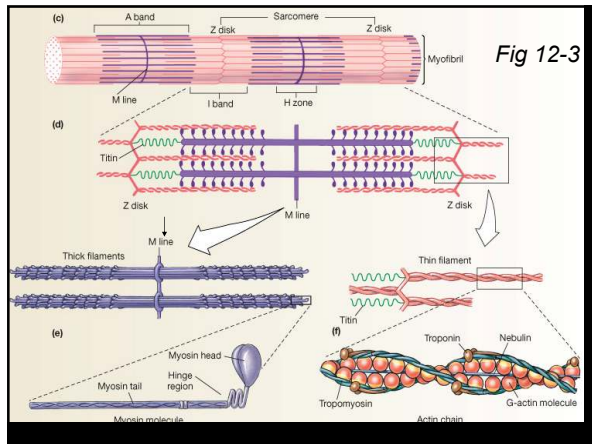
Fig 12-3

Myofibrils = Contractile Organelles of Myofiber

Contain 6 types of protein:

- Actin
 - Myosin
 - Tropomyosin
 - Troponin
 - Titin
 - Nebulin
- Contractile**
Regulatory
Accessory

Fig 12-3 c-f



Titin and Nebulin

- **Titin:** biggest protein known (25,000 aa); elastic!
 - Stabilizes position of contractile filaments
 - Return to relaxed location
- **Nebulin:** inelastic giant protein
 - Alignment of A & M

Fig 12-6

Sliding Filament Theory p 403

- Sarcomere = unit of contraction
- Myosin "walks down" an actin fiber towards Z-line
 - ? - band shortens
 - ? - band does not shorten
- Myosin = motor protein: chemical energy → mechanical energy of motion

Fig 12-8

Changes in a Sarcomere during Contraction

Fig 12-8

The Molecular Basis of Contraction

Rigor State

Compare to Fig 12-9

1. Tight binding in the rigor state. The crossbridge is at a 45° angle relative to the filaments.

- Tight binding between G-actin and myosin
- No nucleotide bound

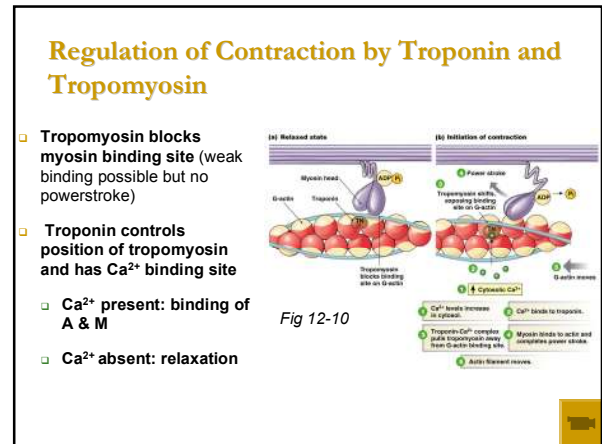
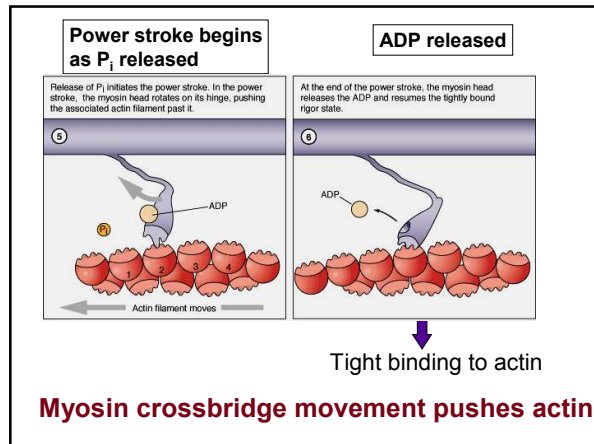
2. ATP binds to nucleotide-binding site on myosin. Myosin then dissociates from actin.

ATP binds ⇒ dissociation

3. The ATPase activity of myosin hydrolyzes the ATP to ADP and inorganic phosphate. Both products remain bound to myosin.

4. The myosin head swings over and binds weakly to a new actin molecule. The cross bridge is now at 90° relative to the filaments.

Relaxed muscle state when sufficient ATP



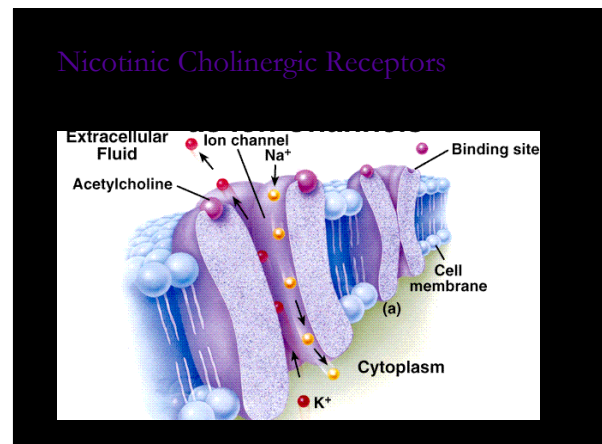
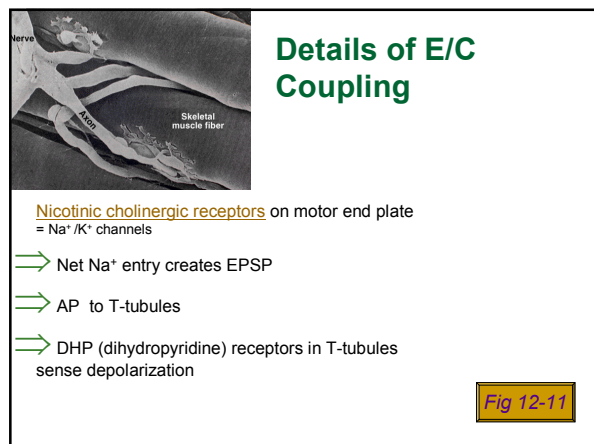
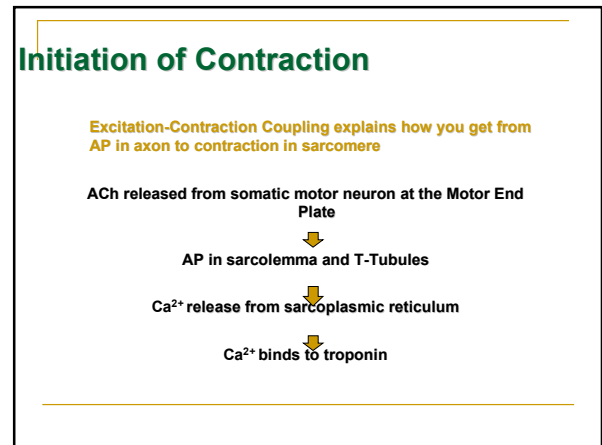
Rigor mortis

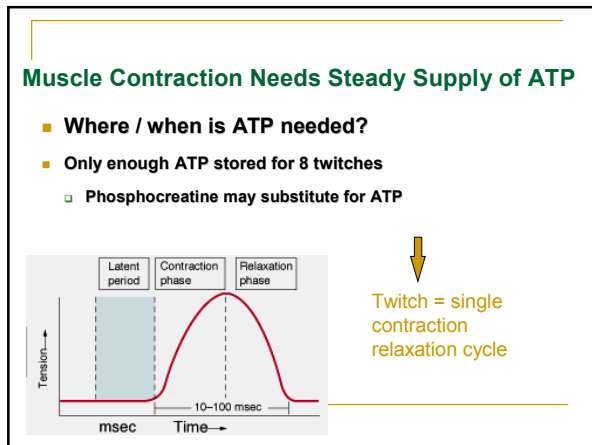
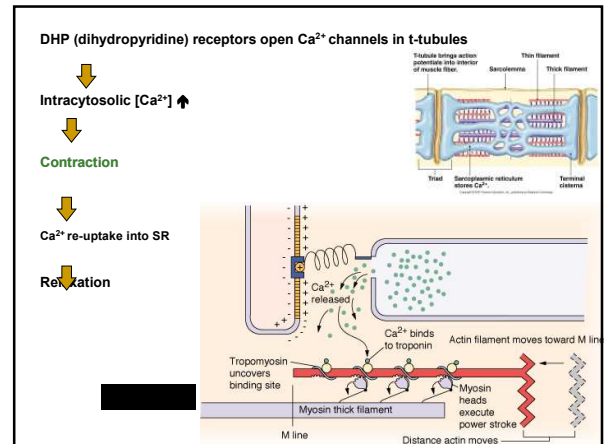
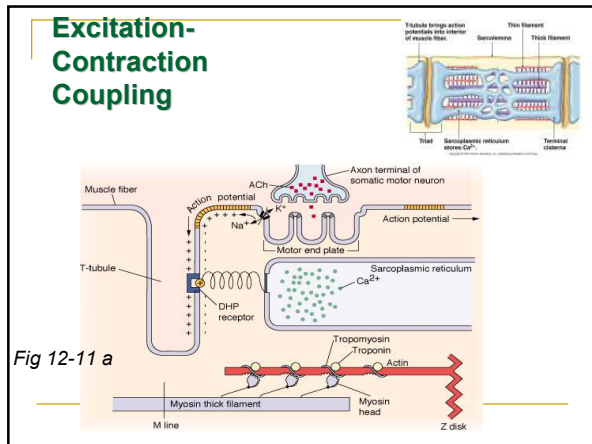
Joint stiffness and muscular rigidity of dead body

Begins 2 – 4 h post mortem. Can last up to 4 days depending on temperature and other conditions

Caused by leakage of Ca^{2+} ions into cell and ATP depletion

Maximum stiffness ~ 12-24 h post mortem, then?

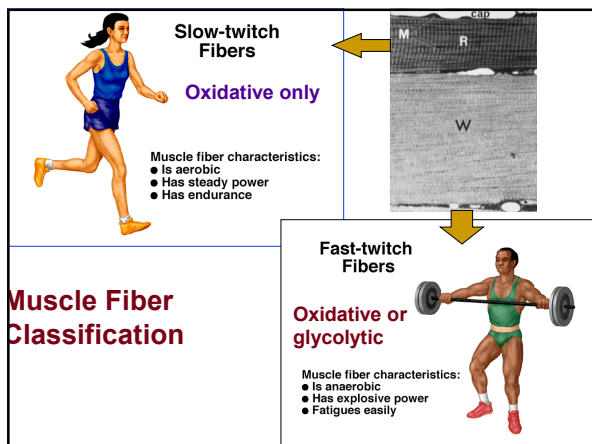




Where does all this ATP come from?

- Phosphocreatine:** backup energy source

$$\text{phosphocreatine} + \text{ADP} \xrightarrow{\text{C(P)K}} \text{creatine} + \text{ATP}$$
- CHO:** aerobic and anaerobic resp.
- Fatty acid breakdown** always requires O_2 – is too slow for heavy exercise
 - Some intracellular FA



Muscle Adaptation to Exercise

(not in book)

Endurance training:

- More & bigger mitochondria
- More enzymes for aerobic respiration
- More myoglobin

no hypertrophy

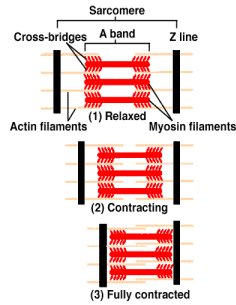
Resistance training:

- More actin & myosin proteins & more sarcomeres
- More myofibrils

muscle hypertrophy

Muscle Tension is Function of Fiber Length

- Sarcomere length reflects thick, thin filament overlap
- Long Sarcomere: little overlap, few crossbridges \Rightarrow weak tension generation
- Short Sarcomere: Too much overlap limited crossbridge formation \Rightarrow tension decreases rapidly

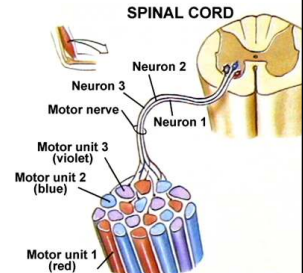


Force of Contraction (all-or-none)

- Increases With
 - muscle-twitch summation
 - recruitment of motor units

Mechanics of body movement covered in lab only

Fig 12-17



Smooth muscle

- A few differences
 - Innervation by varicosities
 - Smaller cells
 - Longer myofilaments
 - Myofilaments arranged in periphery of cell
- Cardiac muscle contraction covered later

