

# Skeletal Muscle – Structure and Function

Human body: Has >400 voluntary sk. m., ~40-50% of total body wt.

It has 3 major functions: 1) Force generation for movement (most obvious function)

2) Force generation for postural support

3) Heat production during cold stress

Attached to bones: By tough connective tissue - **tendons**.

- One end attached to a bone that doesn't move (origin)
- Opposite end is fixed to a bone (insertion) that is moved in muscular contraction

Different movements are possible: depending on type of joint & muscles

- Muscles that ↓ joint angles = Flexors
- Muscles that ↑ joint angles = Extensors.

## Structure

**Different tissues**: Muscle, nerve, blood & dif connective tissues.

**Muscles are separated**: They're held in position by layers of connective tissue (**Fascia**)

**3 other layers (connective tissue)**

Epimysium: Tissue around each sk m, between its fascia & m. cells

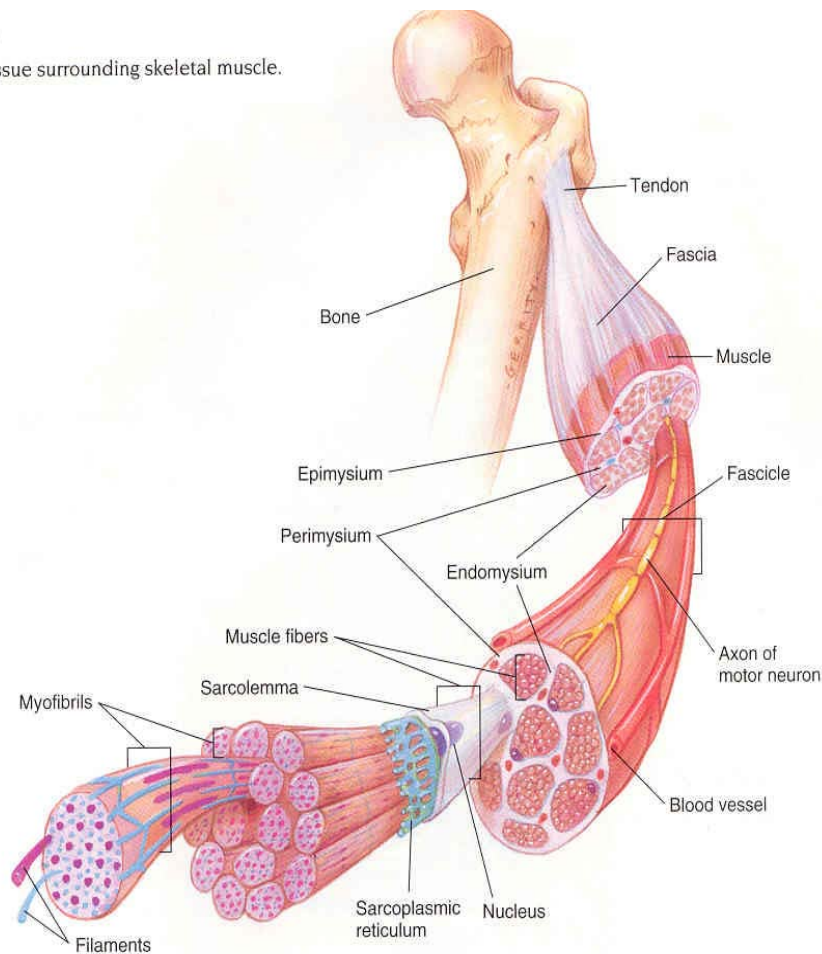
Perimysium: Surrounds each bundle of fibers (fasciculi)

Endomysium: Each fiber in fasciculus surrounded by this layer

Despite the shape, m. cells have same organelles like other cells, & they're **multinucleated**.

Figure 8.1

Connective tissue surrounding skeletal muscle.



## Unique microscopy

Striated: Alternating light & dark bands across the fiber

Thin, elongated cylinder: usually extends the length of the muscle

Cell membrane = **Sarcolemma**

Inside: **Sarcoplasm**, with cellular proteins, organelles, myofibrils.

Myofibrils: Threadlike structures with contractile proteins:

- Thick filaments composed of protein **myosin**
- Thin filaments composed of protein **actin**
- Arrangement of these 2 filaments gives skeletal muscle its striated look
- On the actin molecules there're 2 other proteins - **troponin** & **tropomyosin**. They're small part of the muscle, but play major role in regulation of the contractile process
- Can be further subdivided to individual segments (sarcomeres)

## Sarcomeres:

Divided from each other by a thin sheet of connective tissue = Z lines.

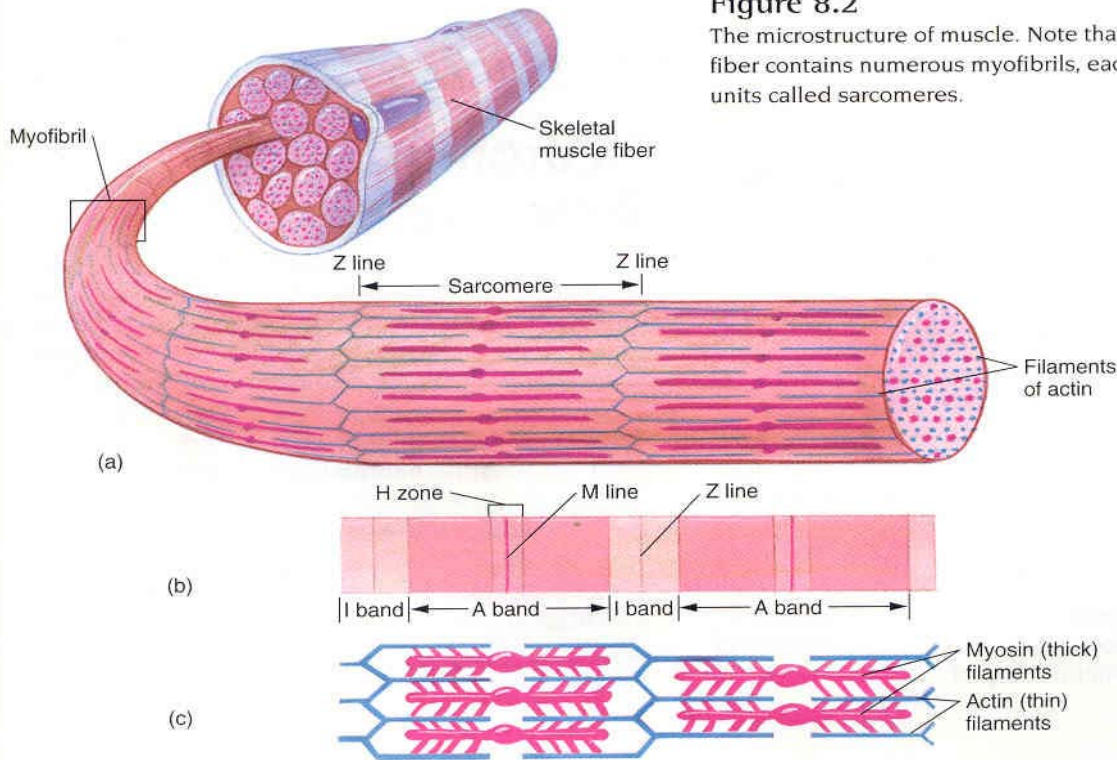
Myosin filaments- mainly in dark part of sarcomere (A band)

Actin filaments- mainly in lighter region (I band).

Actin overlap myosin filaments & also seen in A band.

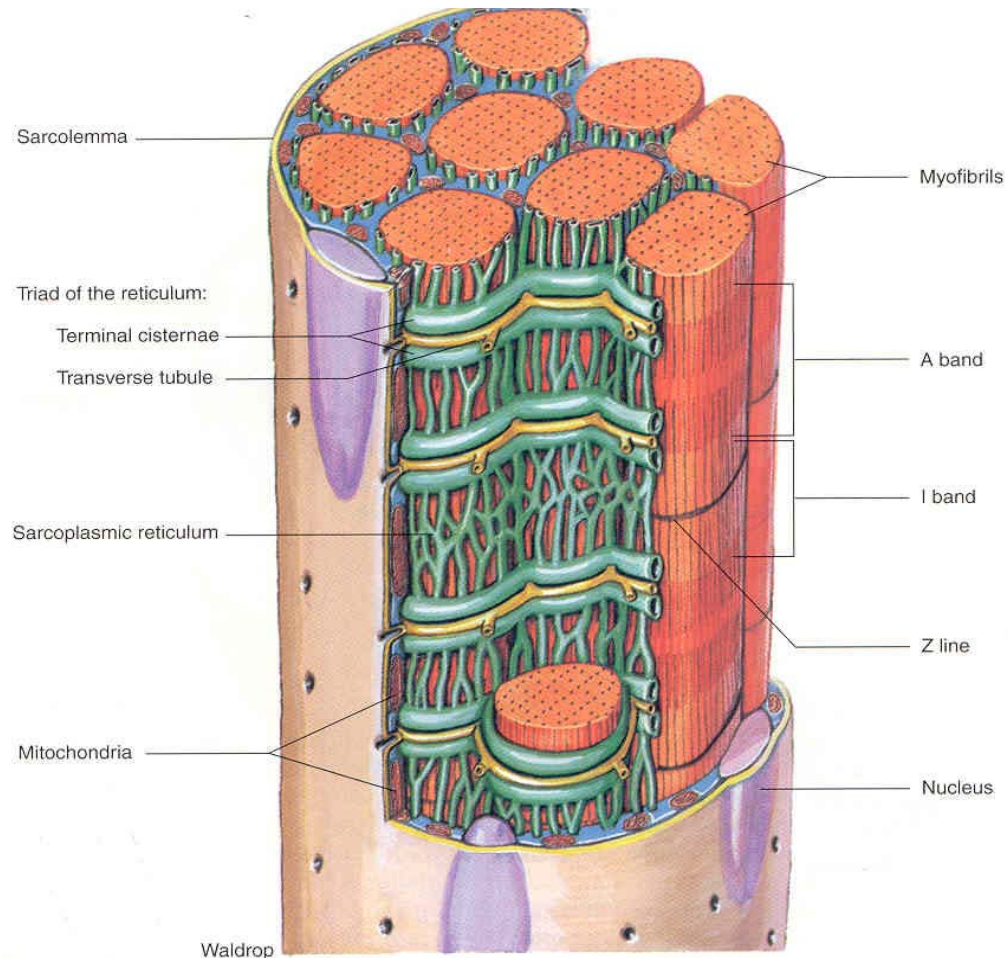
In sarcomere's center there is part of myosin filament with no overlap of actin (H zone)

In sarcoplasm: **Sarcoplasmic reticulum** (SR) is a network of mem. channels that surround each myofibril & run parallel with it. Act as storage sites for calcium (major role in m. contraction)



## Transverse tubules

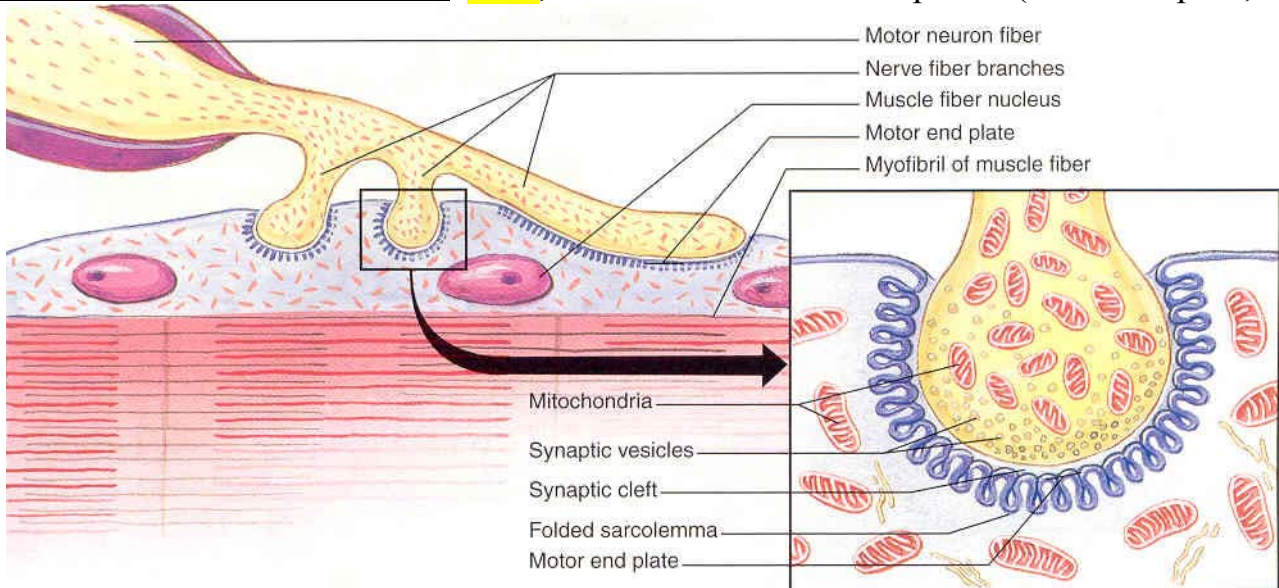
Set of membrane channels, from the sarcolemma inside, pass between 2 enlarged bags of SR = Terminal Cisternae, forming **triad** of reticulum (TT + 2•TC)



## Neuromuscular Junction (NMJ)

Each m. cell: Connected to nerve fiber branch (axon terminal) from nerve cell. These **Motor neurons** (MN) extend out of spinal cord, & with m. fibers they innervate, they're **motor units**

Site where MN & muscle cell meet: **NMJ**, where sarcolemma forms a pocket (motor end plate, **MEP**)

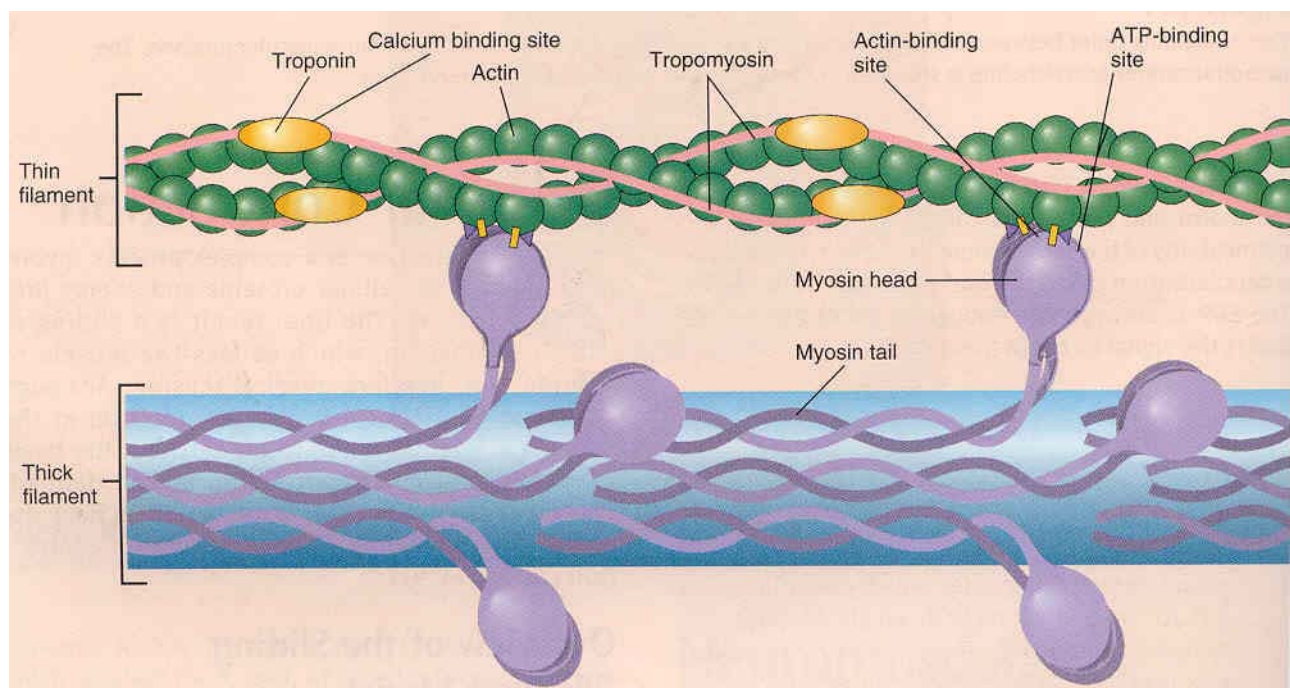


End of MN (axon terminal): separated from MEP by the NM cleft

- It  $\uparrow$  permeability of sarcolemma to  $\text{Na}^+$   $\rightarrow$  Depolarization (End Plate Potential (EPP))

## Contractile Process - Summary of Events

1. When AP reaches axon terminal, it releases **Ach** that cross the synaptic cleft & binds to its receptor
2. Accumulated Ach binding to MEP, produces EPP big enough to exceed threshold & leads to AP that are conducted down the transverse tubules, deep into the muscle fiber
3. When AP reaches TC,  $\text{Ca}^{+2}$  is released to sarcoplasm & binds to troponin. That causes a shift in position of tropomyosin to uncover "active sites" on actin.
4. The "cocked" myosin cross-bridge attaches to active site on actin & pulls it over the myosin
5. Attaching "fresh" ATP to myosin cross-bridges allows the cross-bridge to detach from actin
6. ATP is broken to  $\text{ADP} + \text{Pi} + \text{E}$ , & released E that is used to "cock" the myosin cross-bridges
7. This cycle is repeated as long as  $\text{Ca}^{+2}$  is present. When AP stops, SR removes  $\text{Ca}^{+2}$  from sarcoplasm & tropomyosin moves to its inhibitory position - covering active sites on actin.



**Excitation-contraction coupling** = Sequence where AP reaches m. membrane & leads to muscle shortening by cross-bridges activity.

**E of Contraction:** ATP hydrolysis by myosin ATPase (myosin head) energizes myosin cross-bridges, allowing the release of these heads & further pulling of actin over myosin, to shorten the muscle.

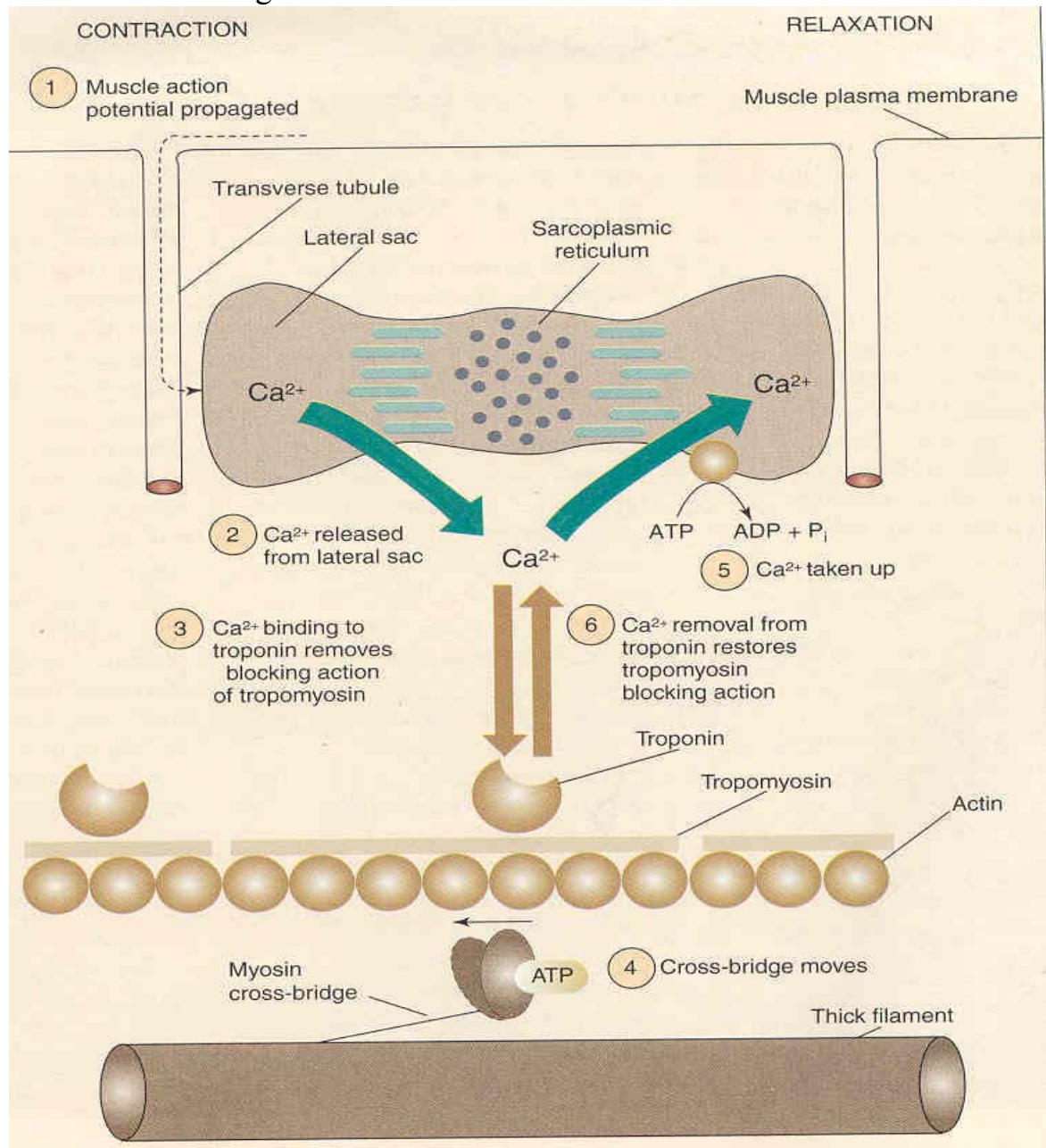
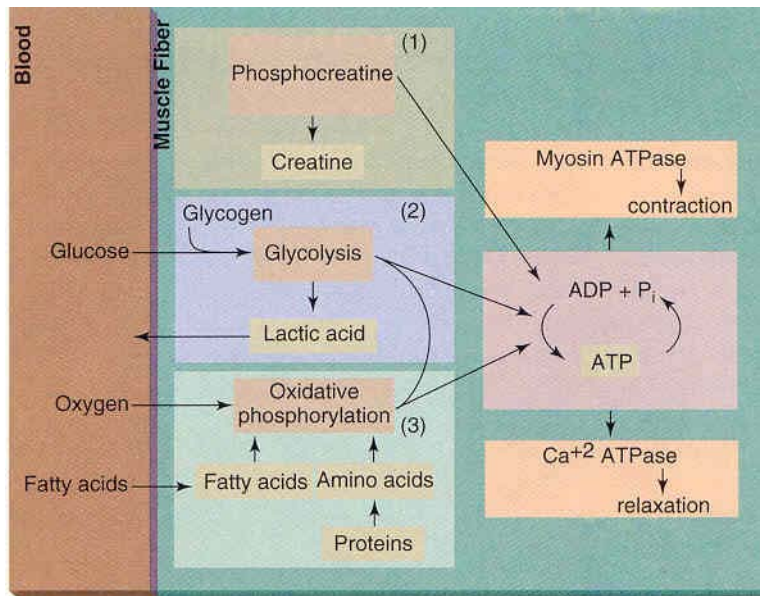
Fig 7: 3 sources of ATP in contraction

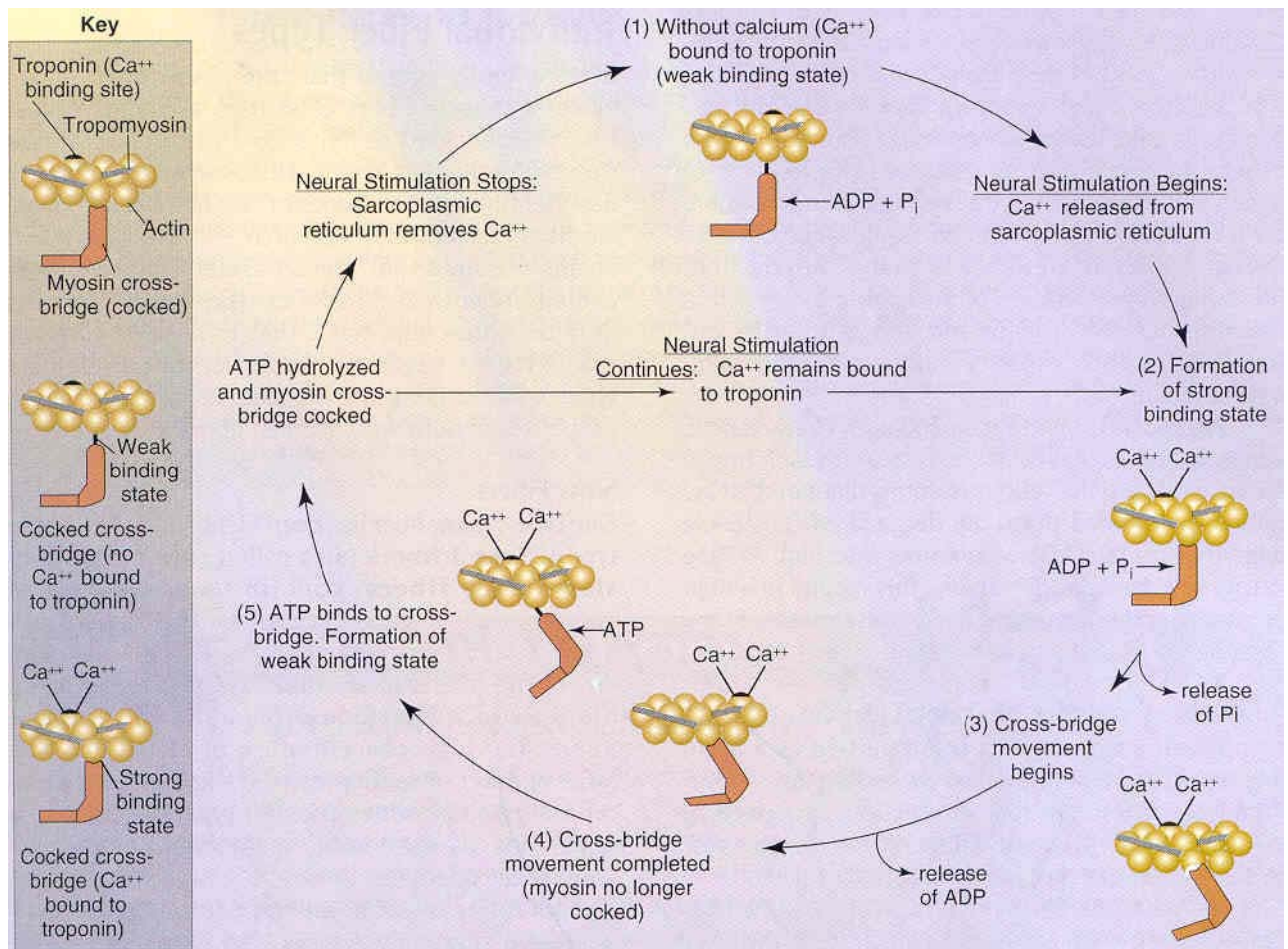
**Stopping the Contraction**

Signal: Lack of AP at the NMJ

E-dep. Ca<sup>+2</sup> pump (in SR): move it back to SR.

Ca<sup>+2</sup> is removed from troponin, allowing tropomyosin to move & cover binding sites.





## Muscle Fatigue

Short-term, high intensity or prolonged sub-max ex can induce  $\downarrow$  muscle force production  
 This fatigue is defined as  $\downarrow$  max force production  $\rightarrow$   $\downarrow$  ability to work

Cause of fatigue: Varies & dep on type of ex.

- **Fatigue D/T high intensity ex** (400 m' sprint)- D/T accumulated Inorganic P &  $\text{H}^+$  that interact with contractile proteins &  $\downarrow$  muscle force
- **Fatigue D/T prolonged ex** may involve failure of excitation-contraction coupling, by  $\downarrow$  release  $\text{Ca}^{+2}$  of SR  $\rightarrow$  Fewer cross-bridges in strong binding state (force generation) &  $\downarrow$  m. force production

