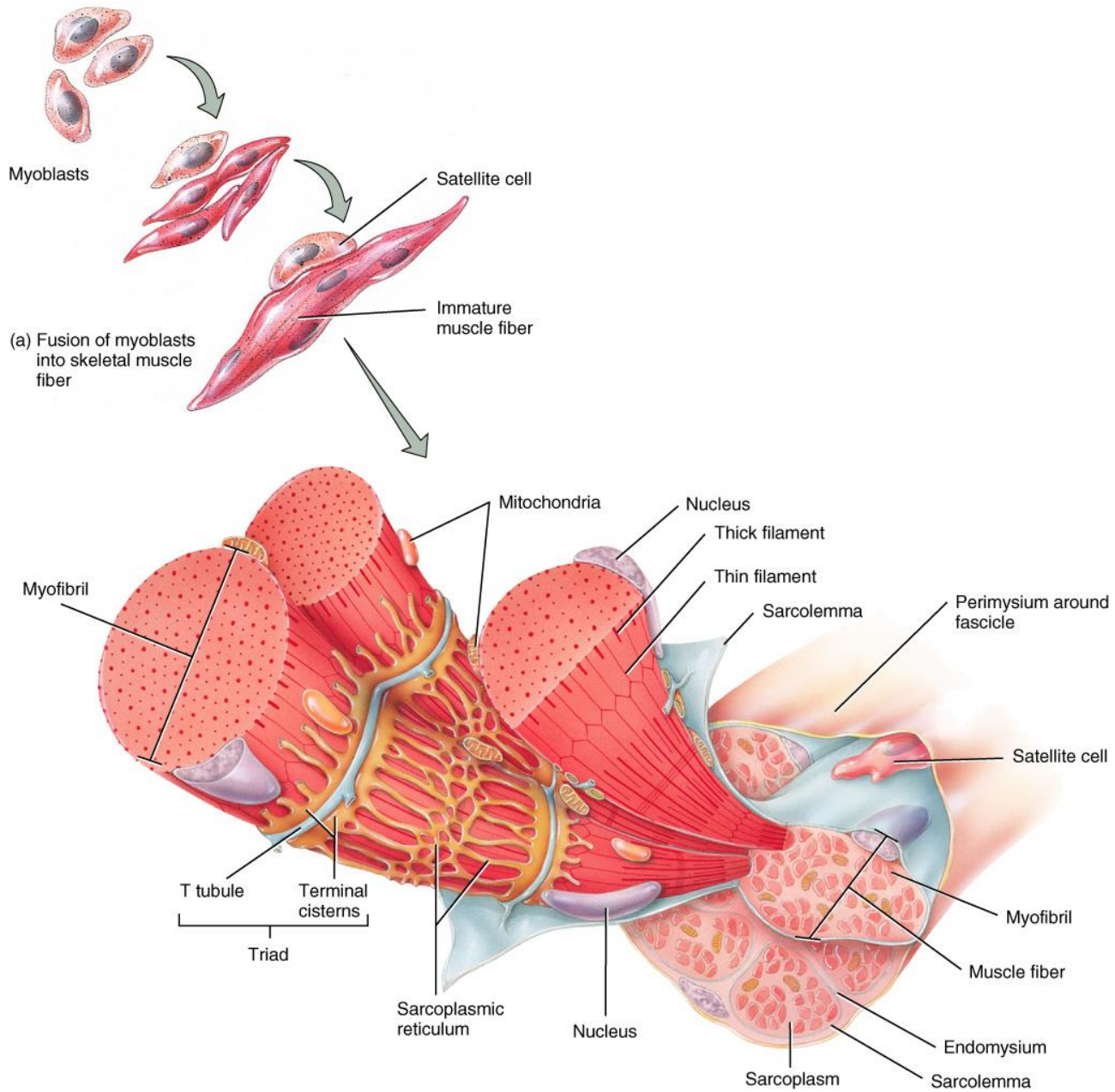


# **Muscle Physiology**

**Ref: Guyton,  
chapters: 6,7,8**

## **Types of Muscle**

**Fig. 10.03**



(a) Fusion of myoblasts into skeletal muscle fiber

(b) Details of several myofibrils

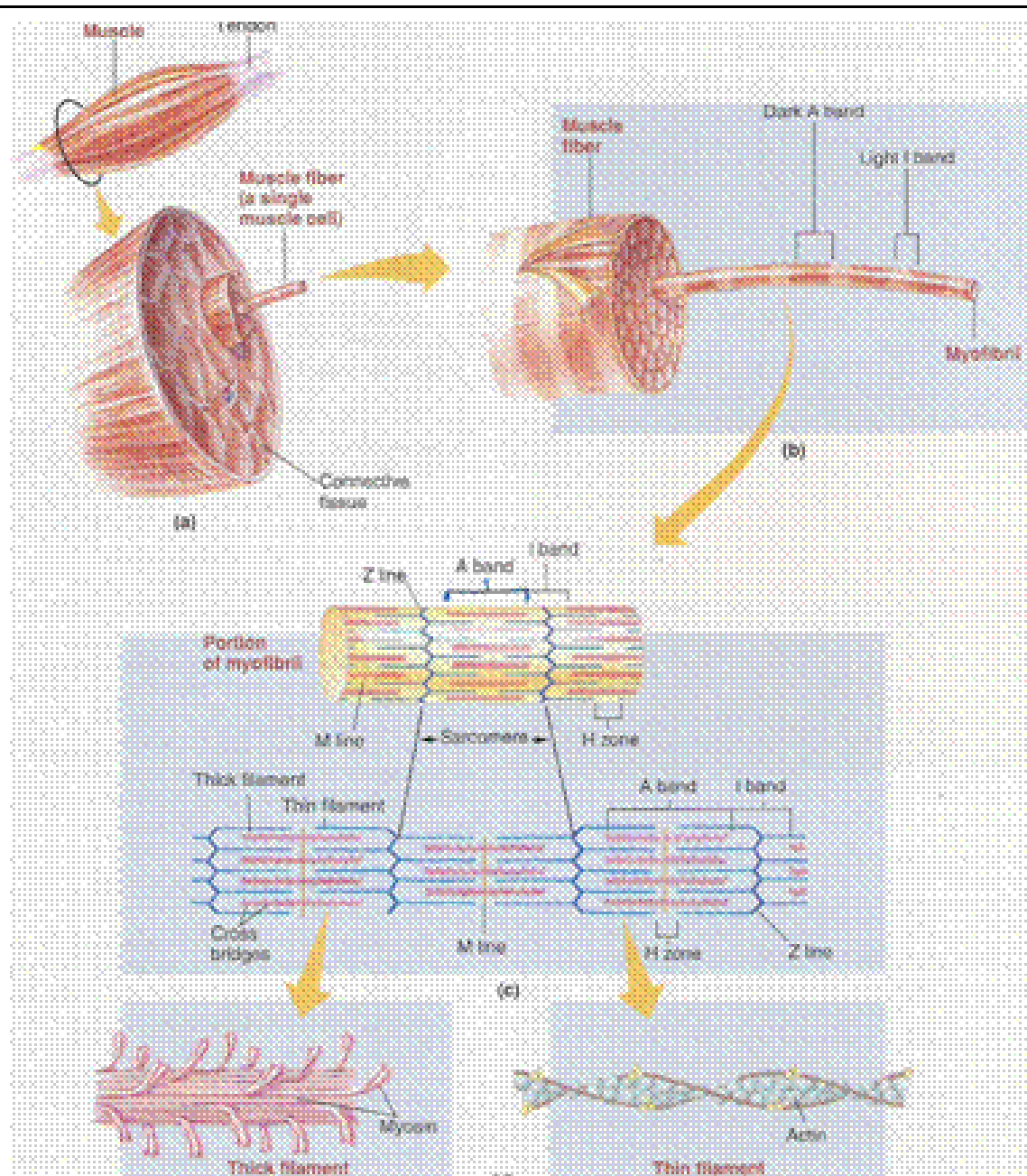


Figure 2. The components of a muscle.

Taken from Sherwood, 2004.

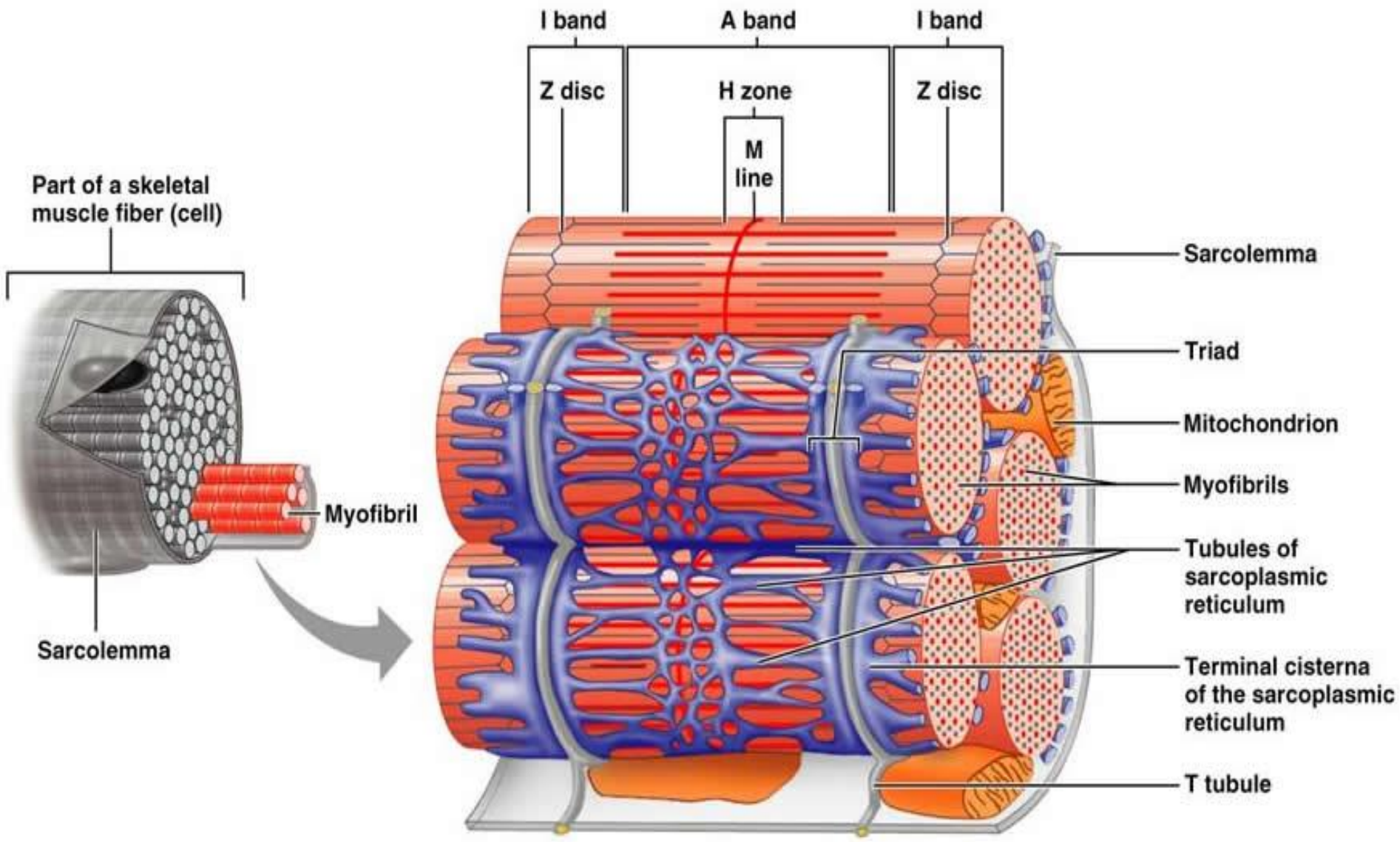
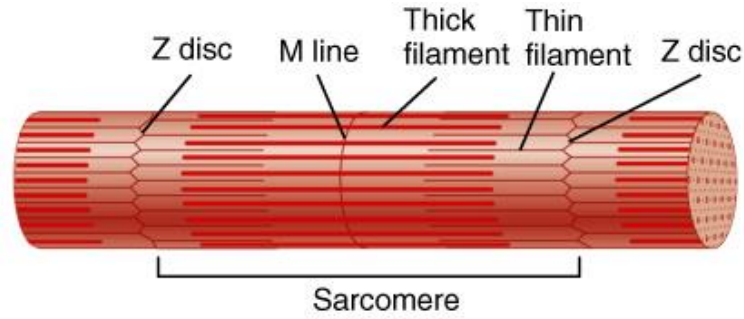
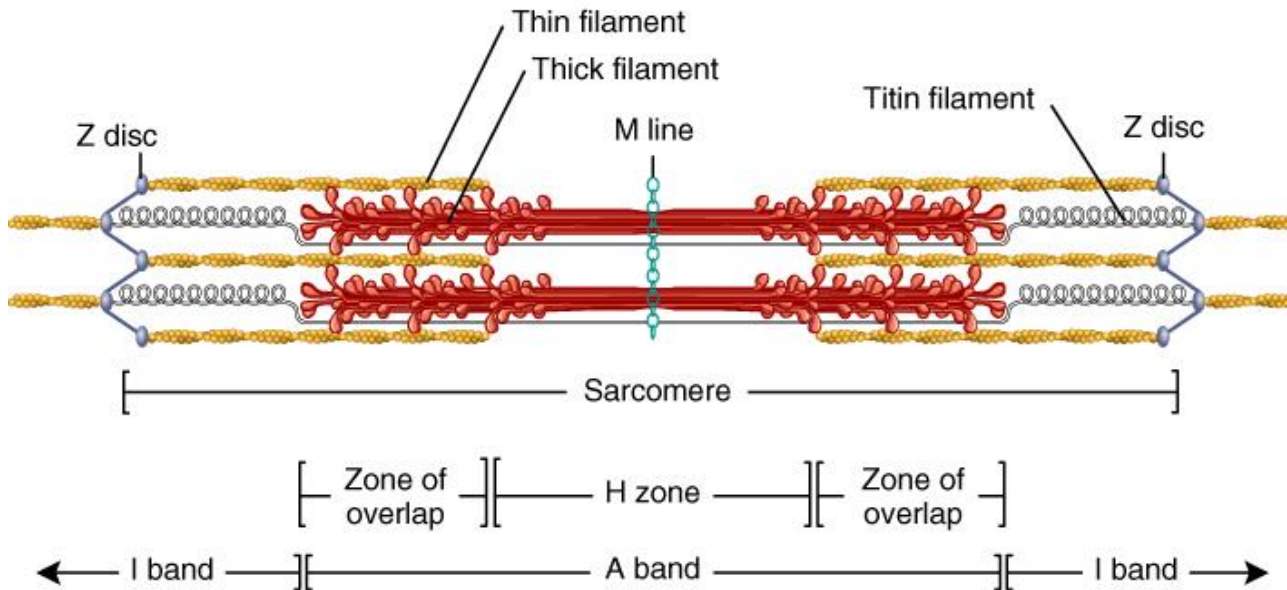


Fig. 10.04

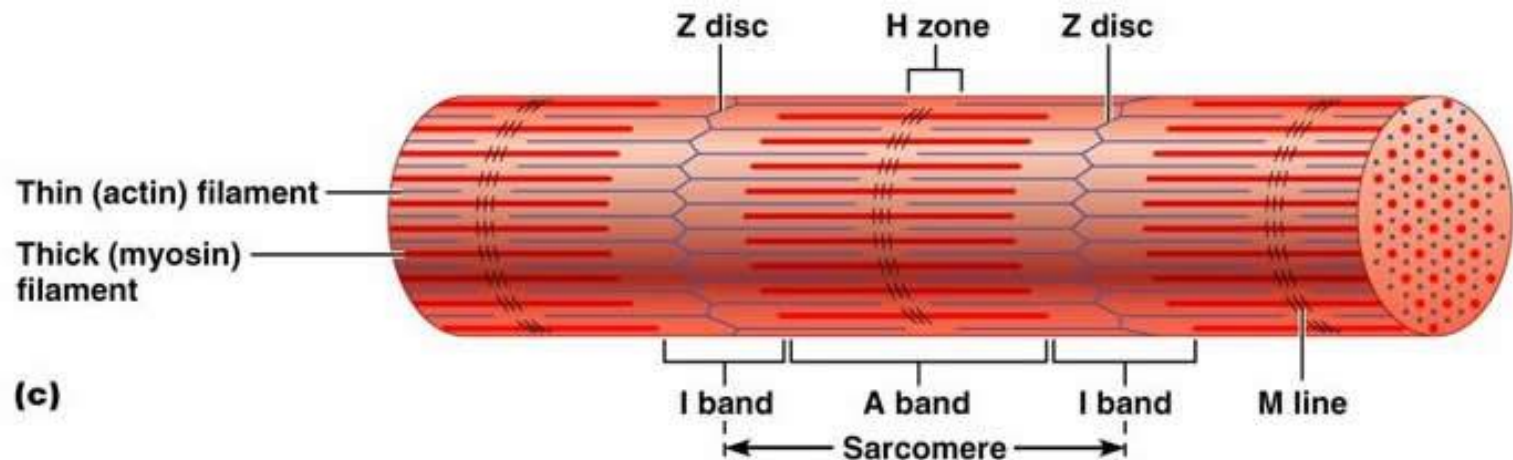


(a) Myofibril

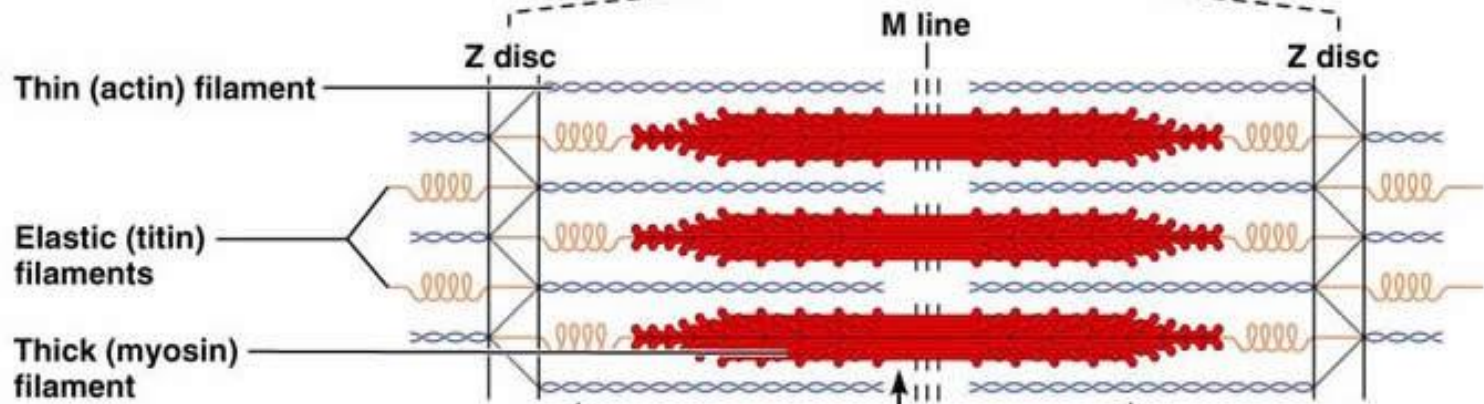


(b) Filaments

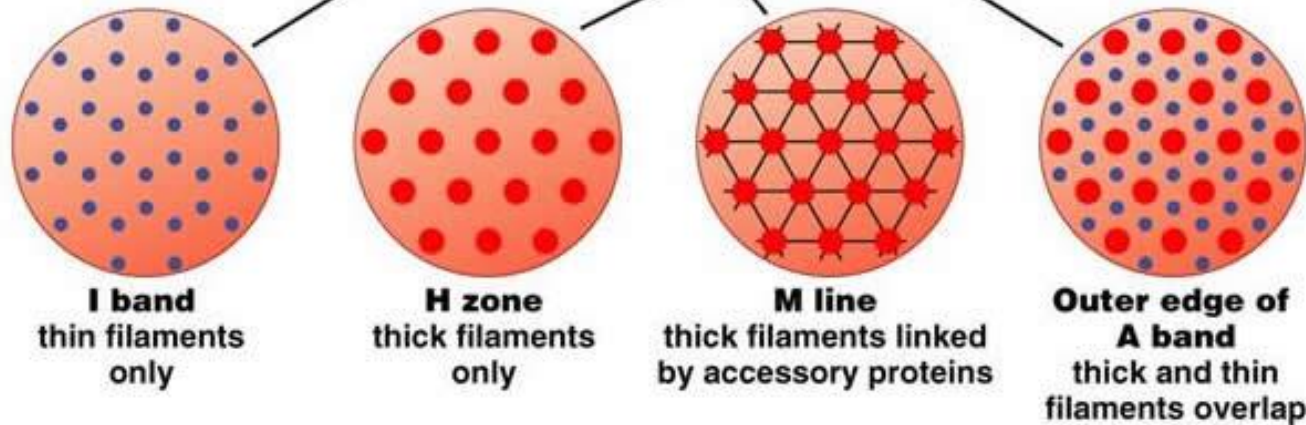




(c)



(d)



(e)

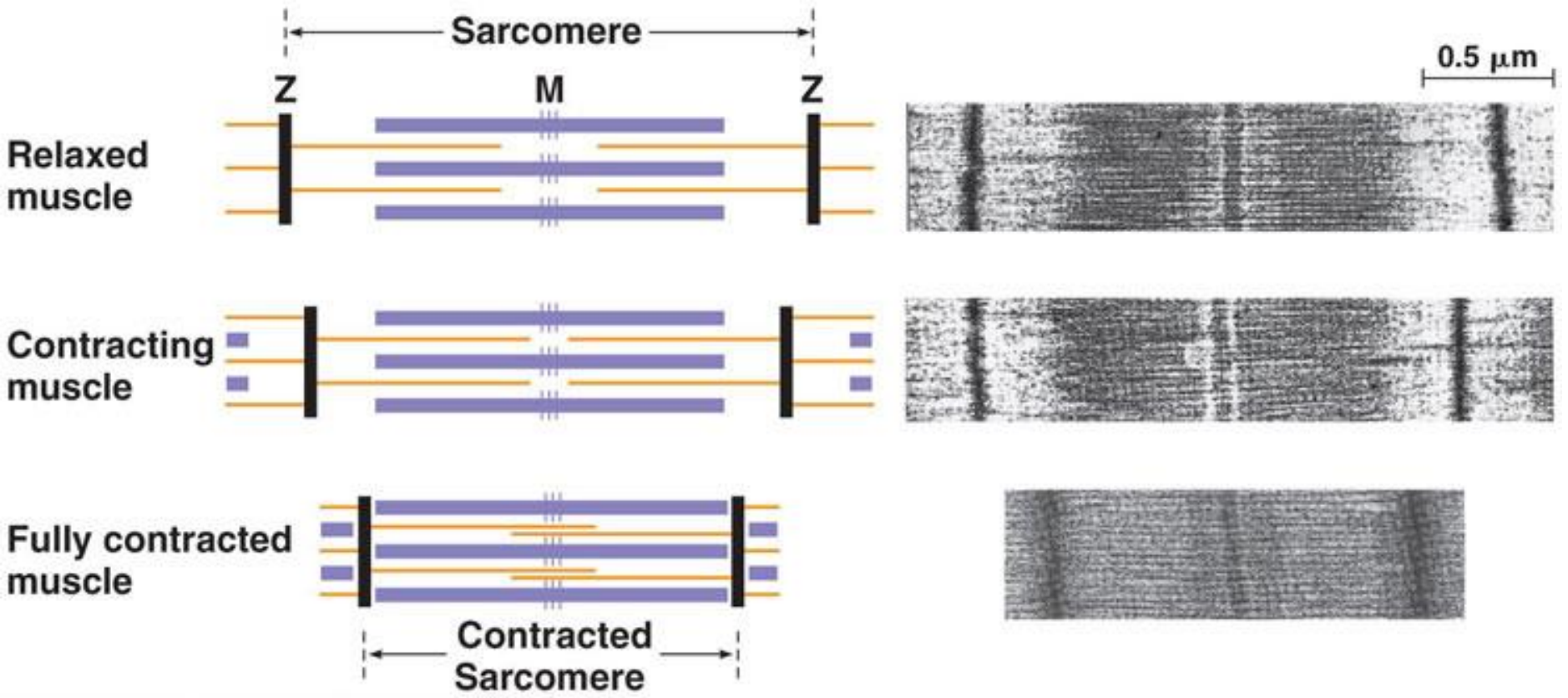
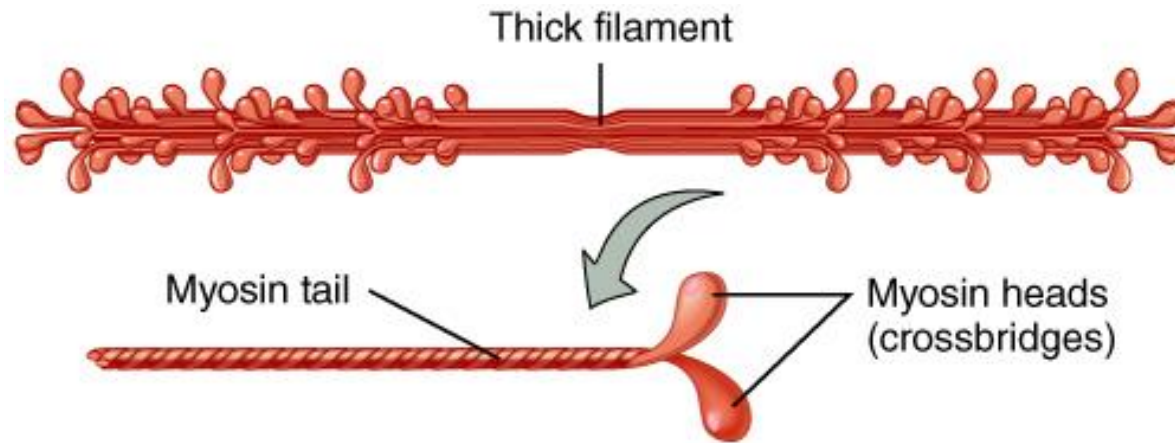
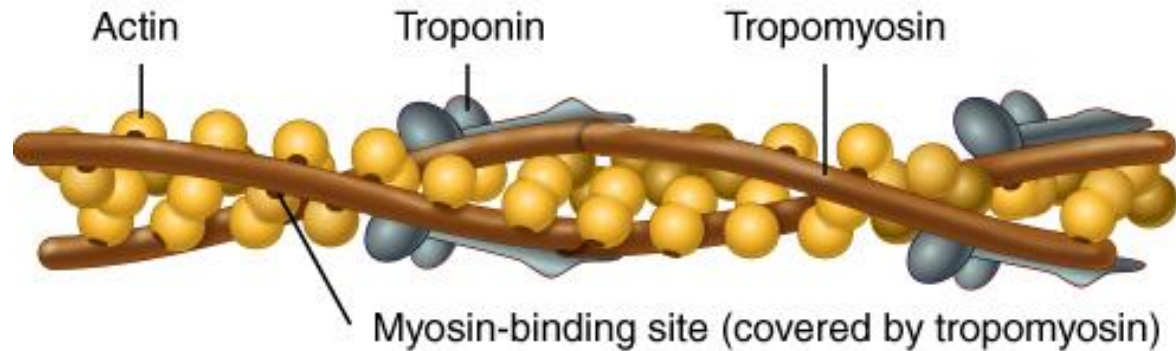


Fig. 10.06



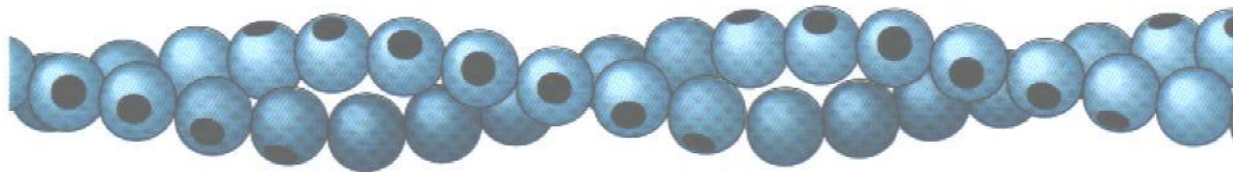
(a) One thick filament (above) and a myosin molecule (below)



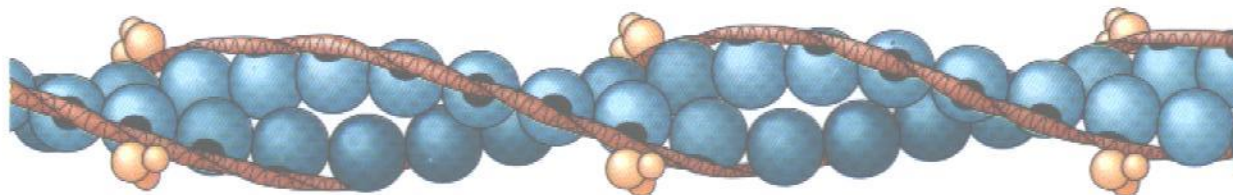
(b) Portion of a thin filament



### Composition of Thin Filaments

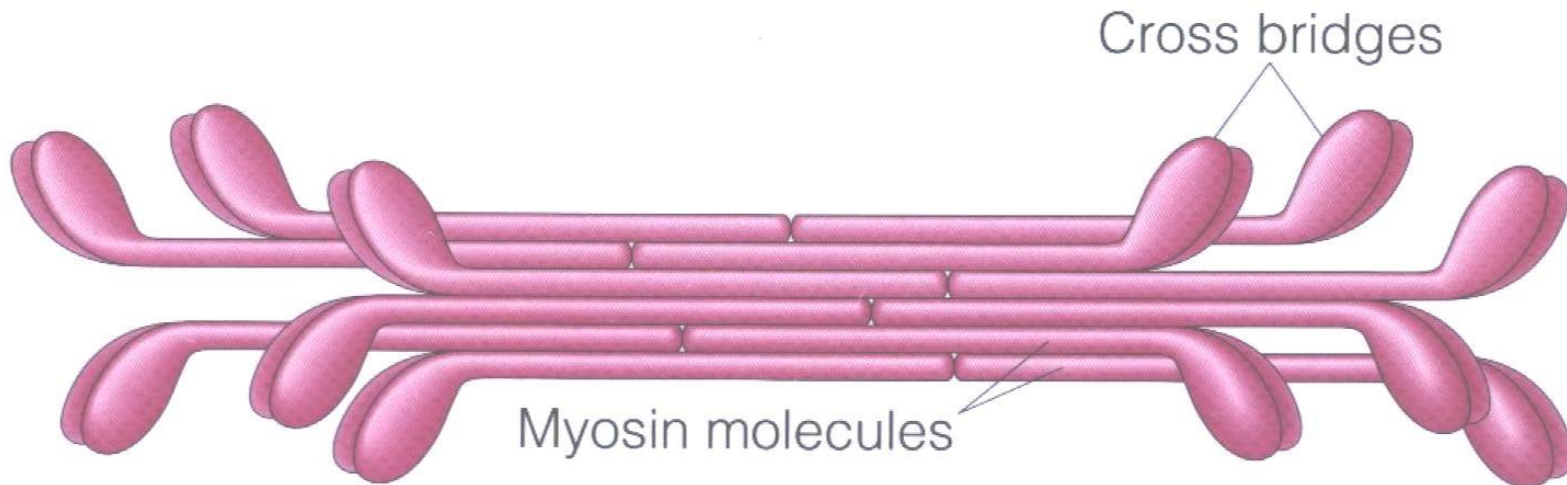


+

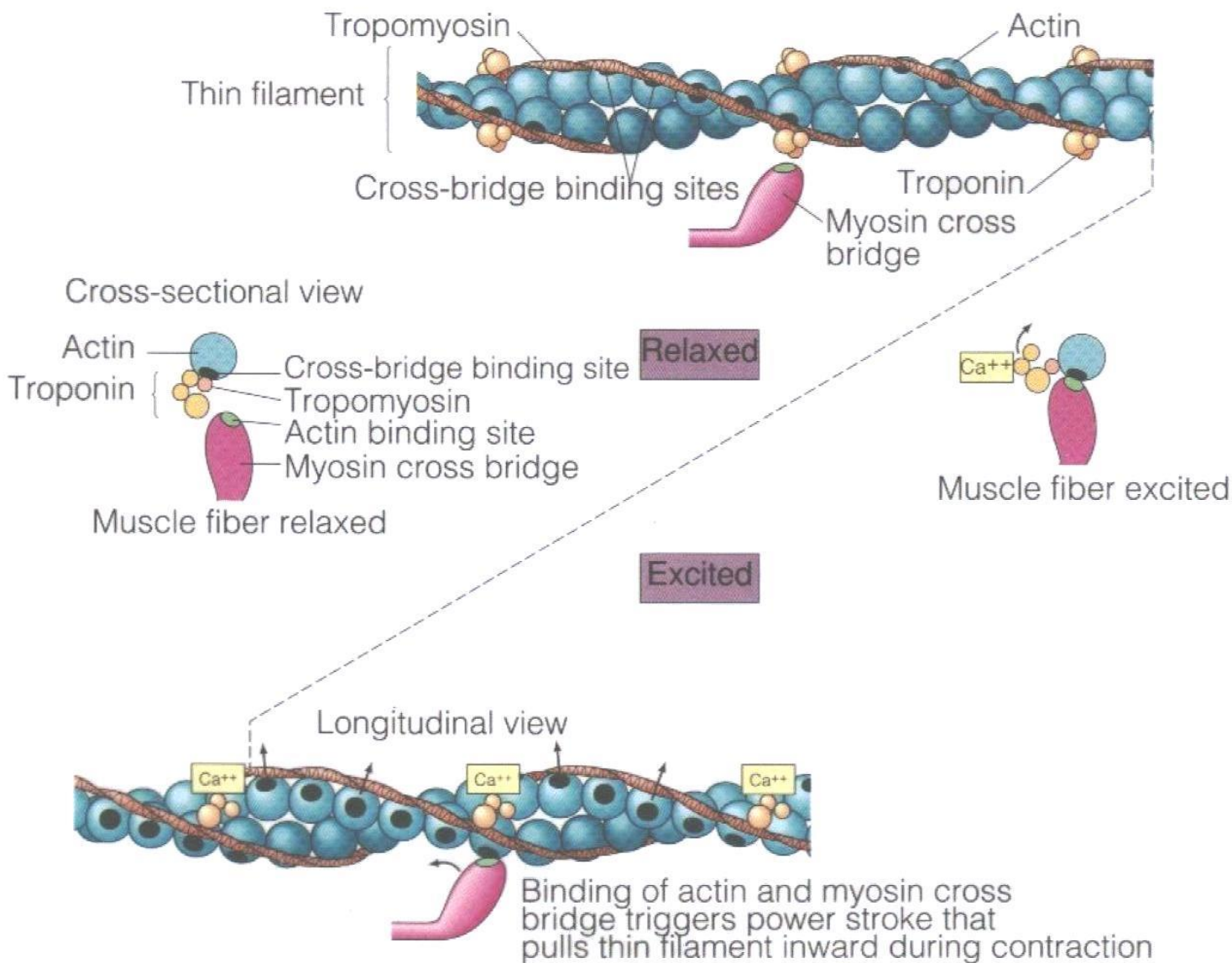


Thin filament

## Structure of Myosin Molecules and Their Organization within a Thick Filament

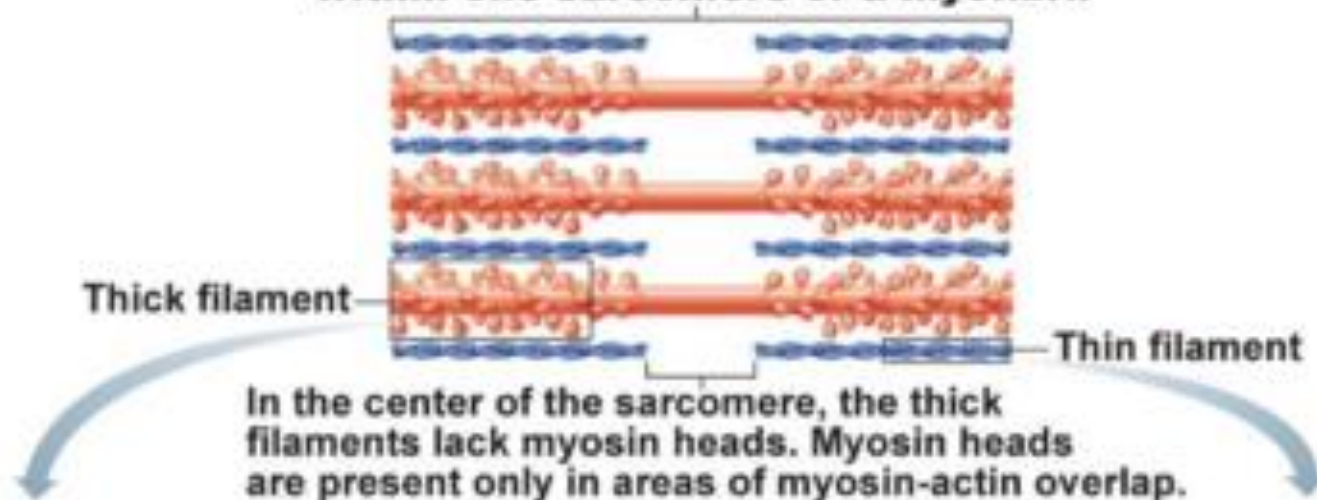


### Schematic Representation of Role of Calcium in Turning on Cross Bridges





## Longitudinal section of filaments within one sarcomere of a myofibril



### Thick filament

Each thick filament consists of many myosin molecules whose heads protrude at opposite ends of the filament.

#### Portion of a thick filament



Actin-binding sites

ATP-binding site

Heads

Tail

Flexible hinge region

Myosin molecule

### Thin filament

A thin filament consists of two strands of actin subunits twisted into a helix plus two types of regulatory proteins (troponin and tropomyosin).

#### Portion of a thin filament



Actin subunits

Actin subunits

Active sites for myosin attachment

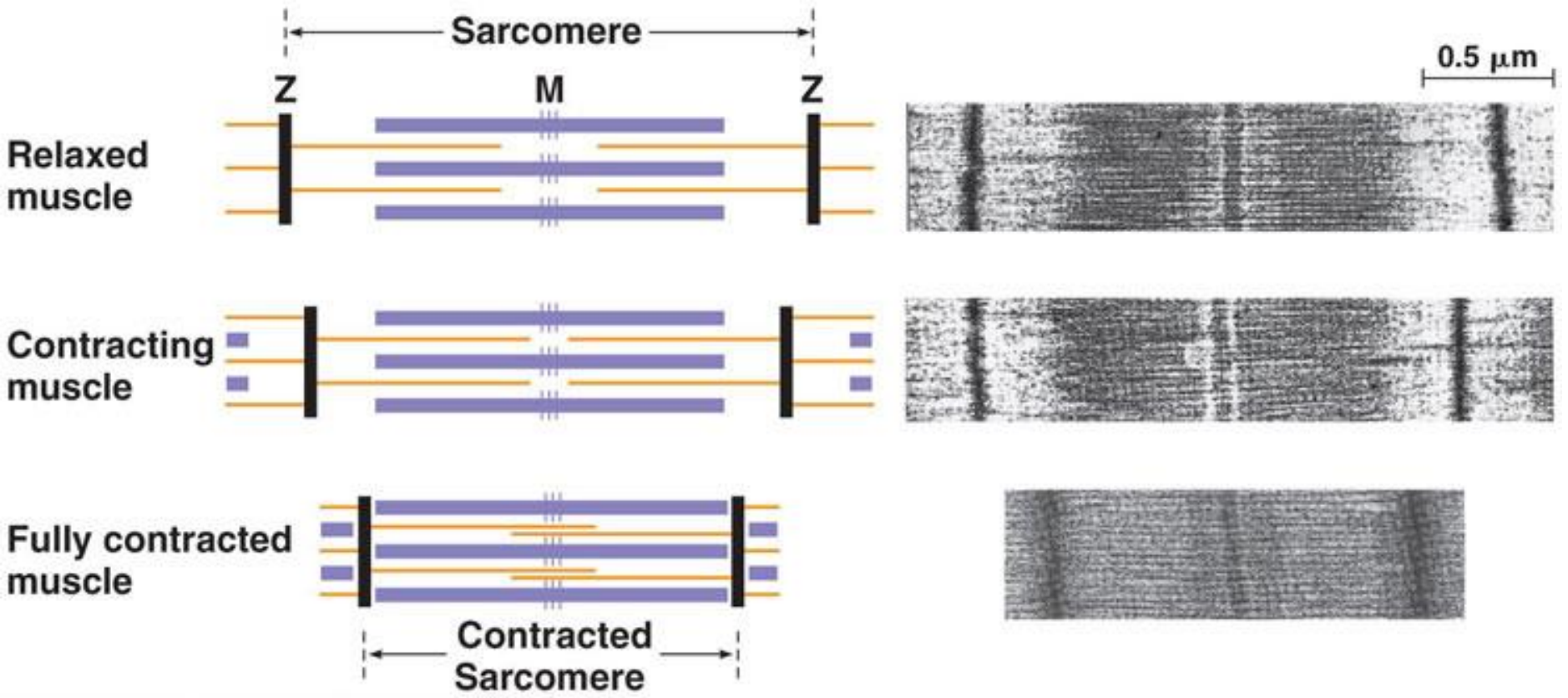




Fig. 10.07

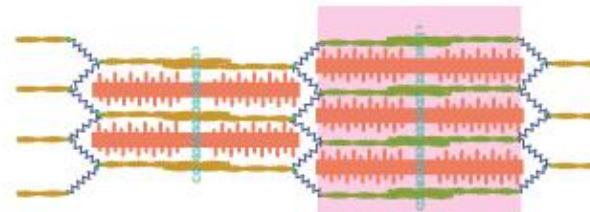
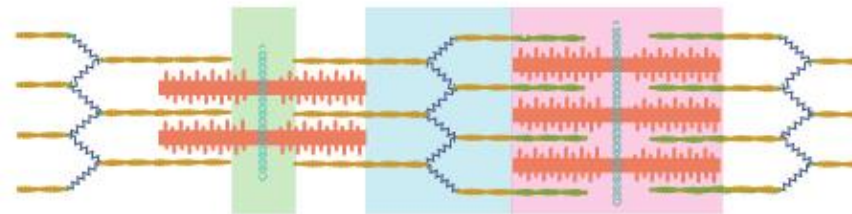
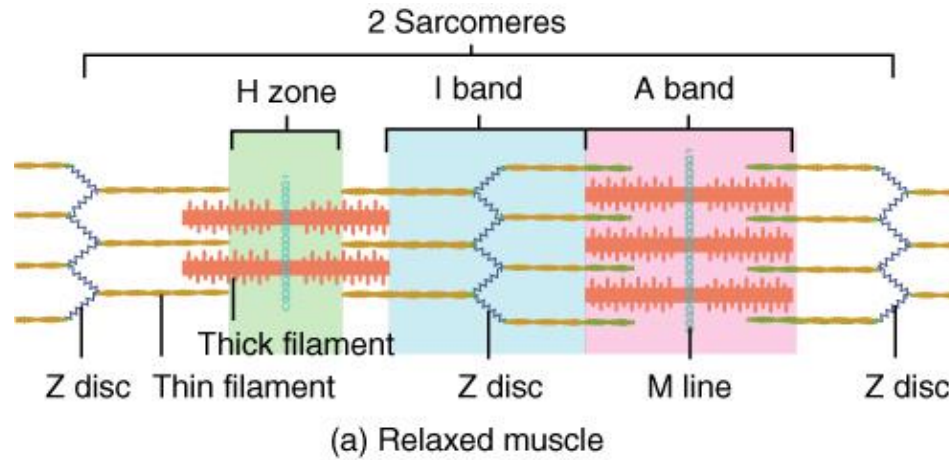
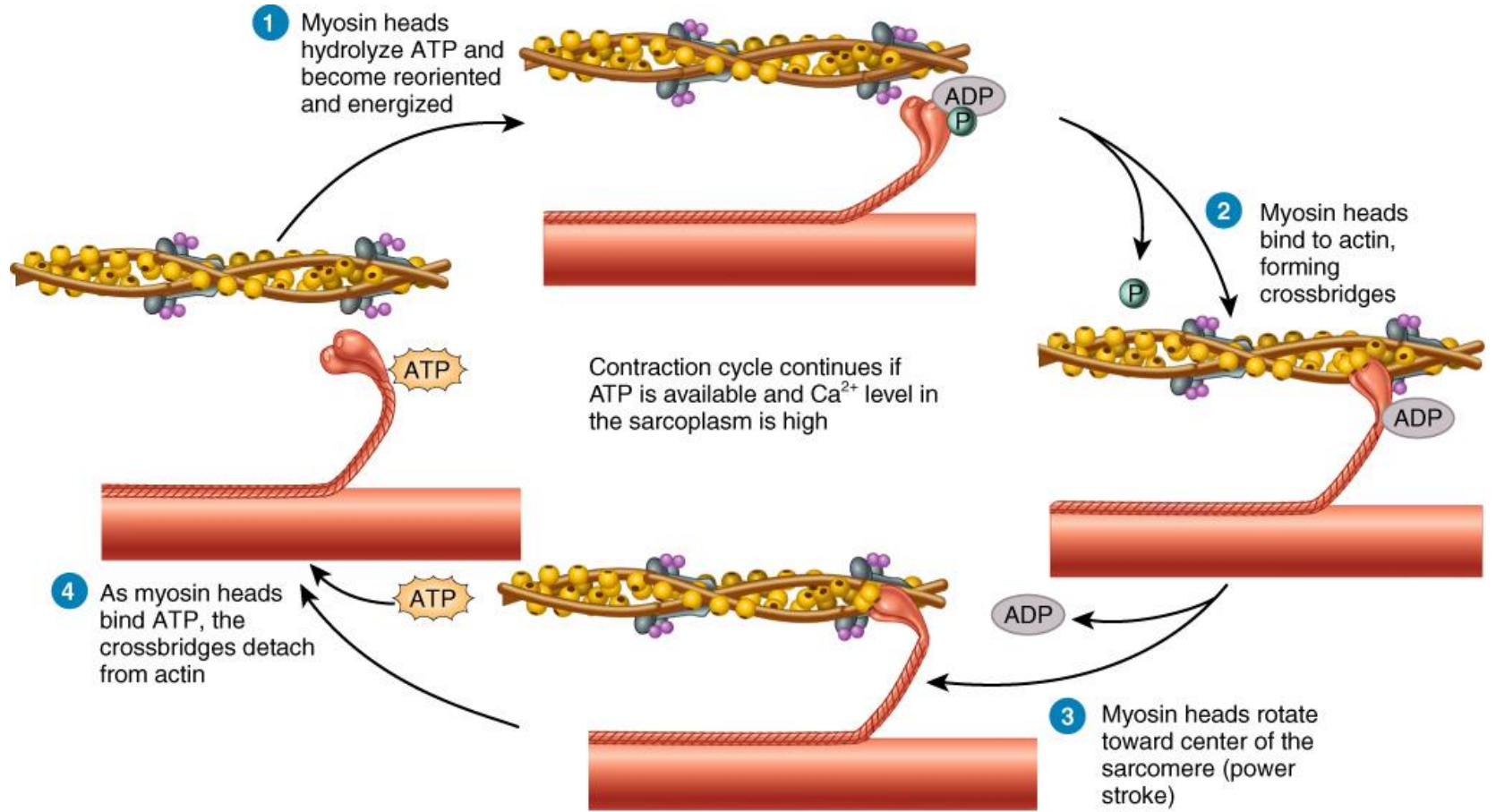
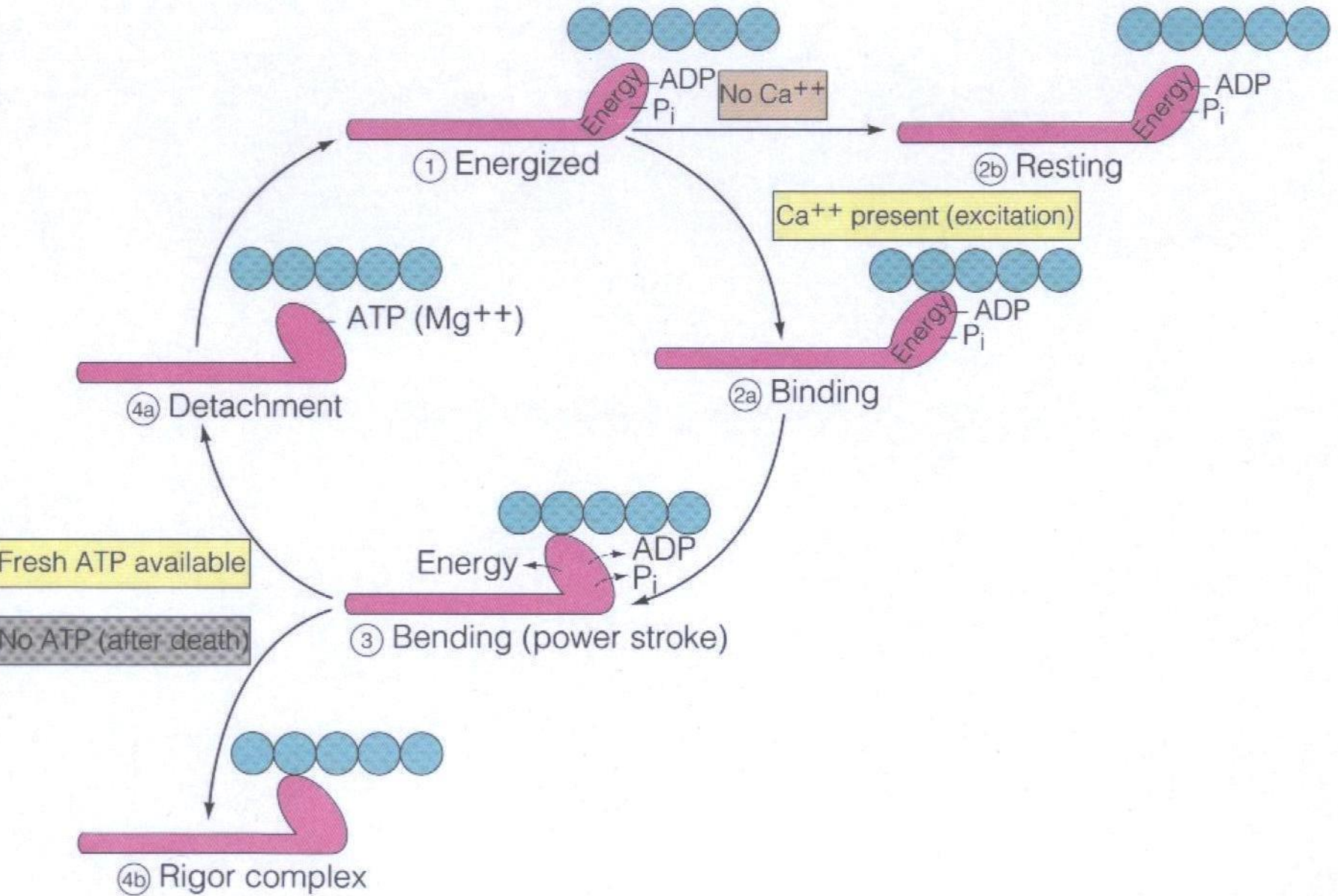


Fig. 10.08



# Cross-Bridge Cycle



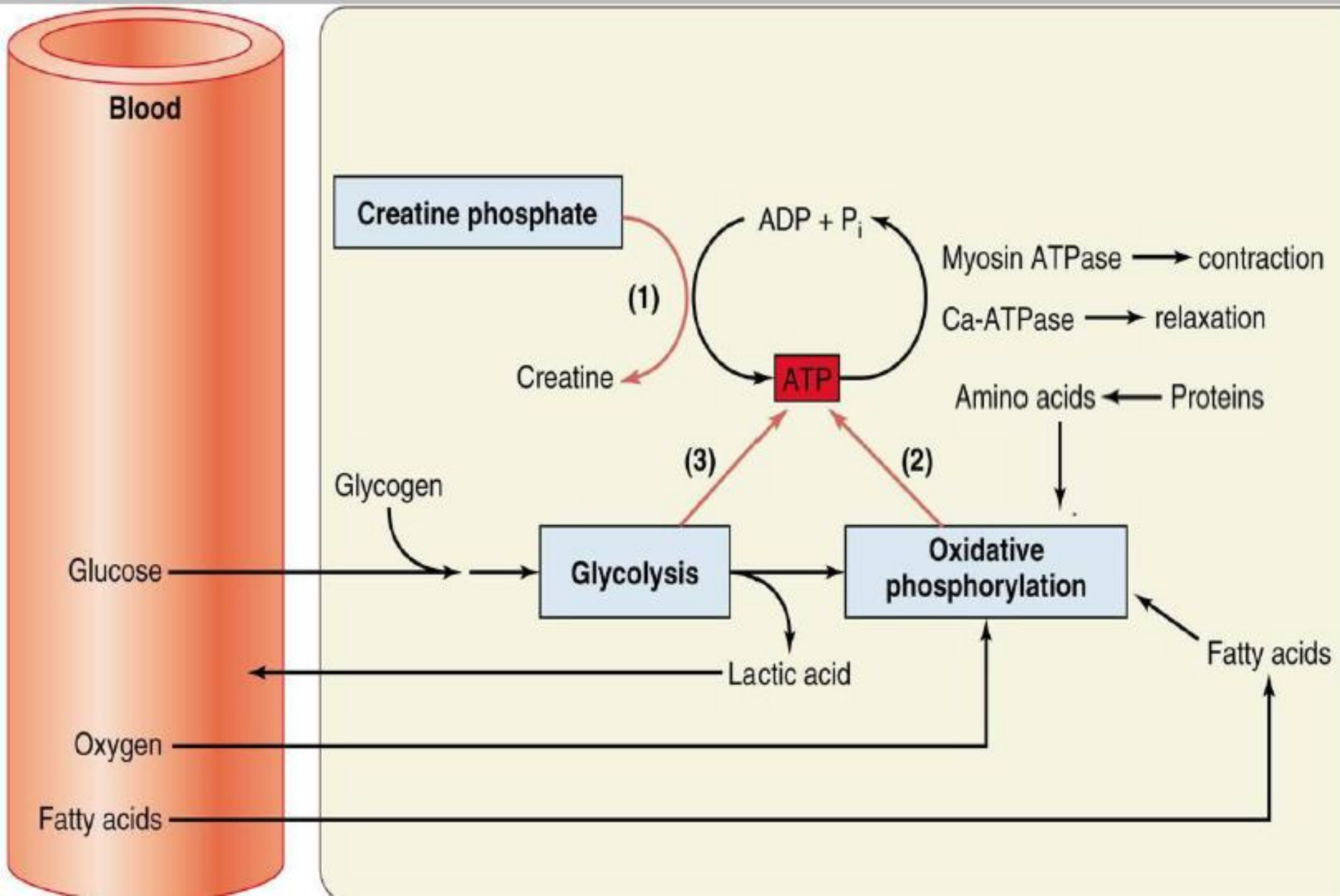
# Summary of Muscle Contraction

<https://www.youtube.com/watch?v=6YvdLWgT5mg>

# Muscle Energy

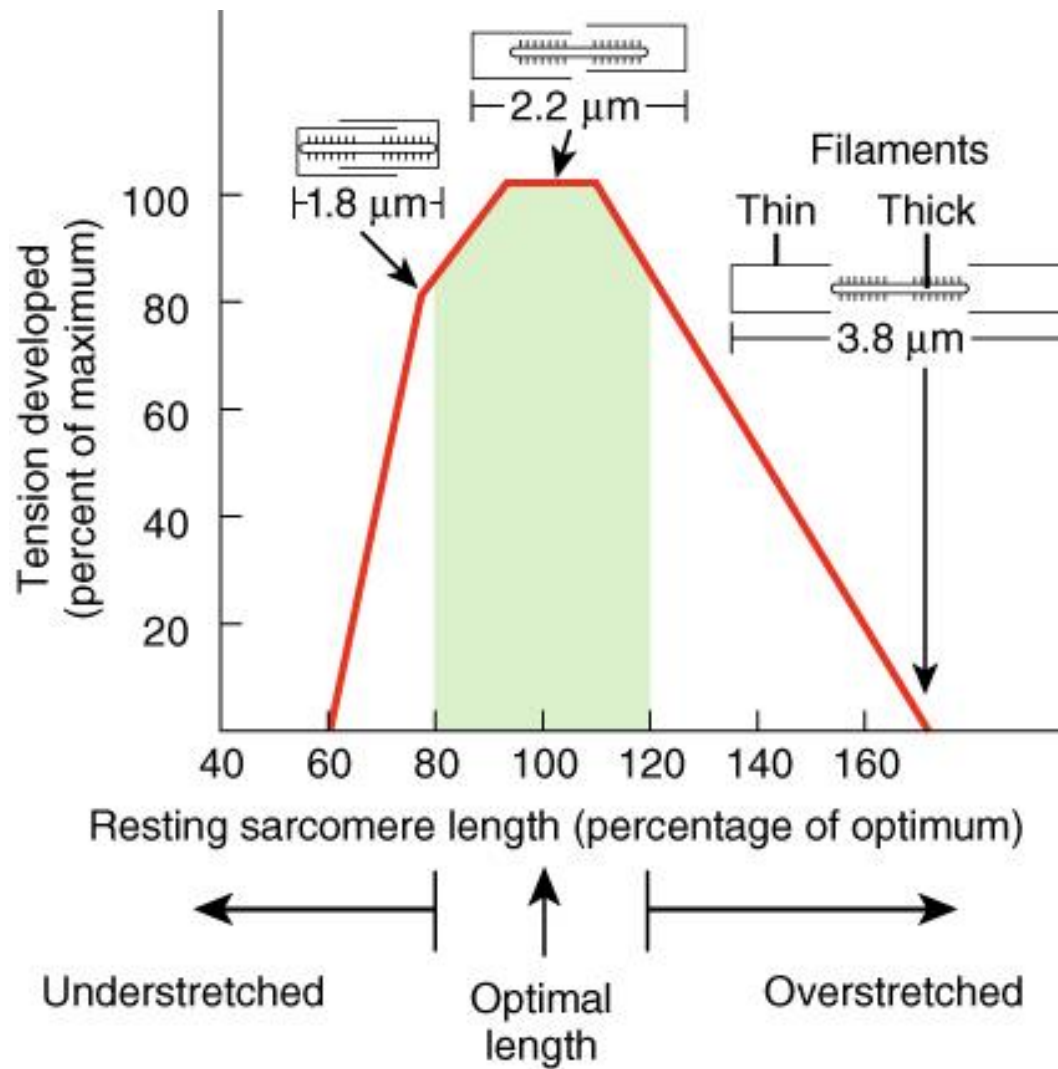


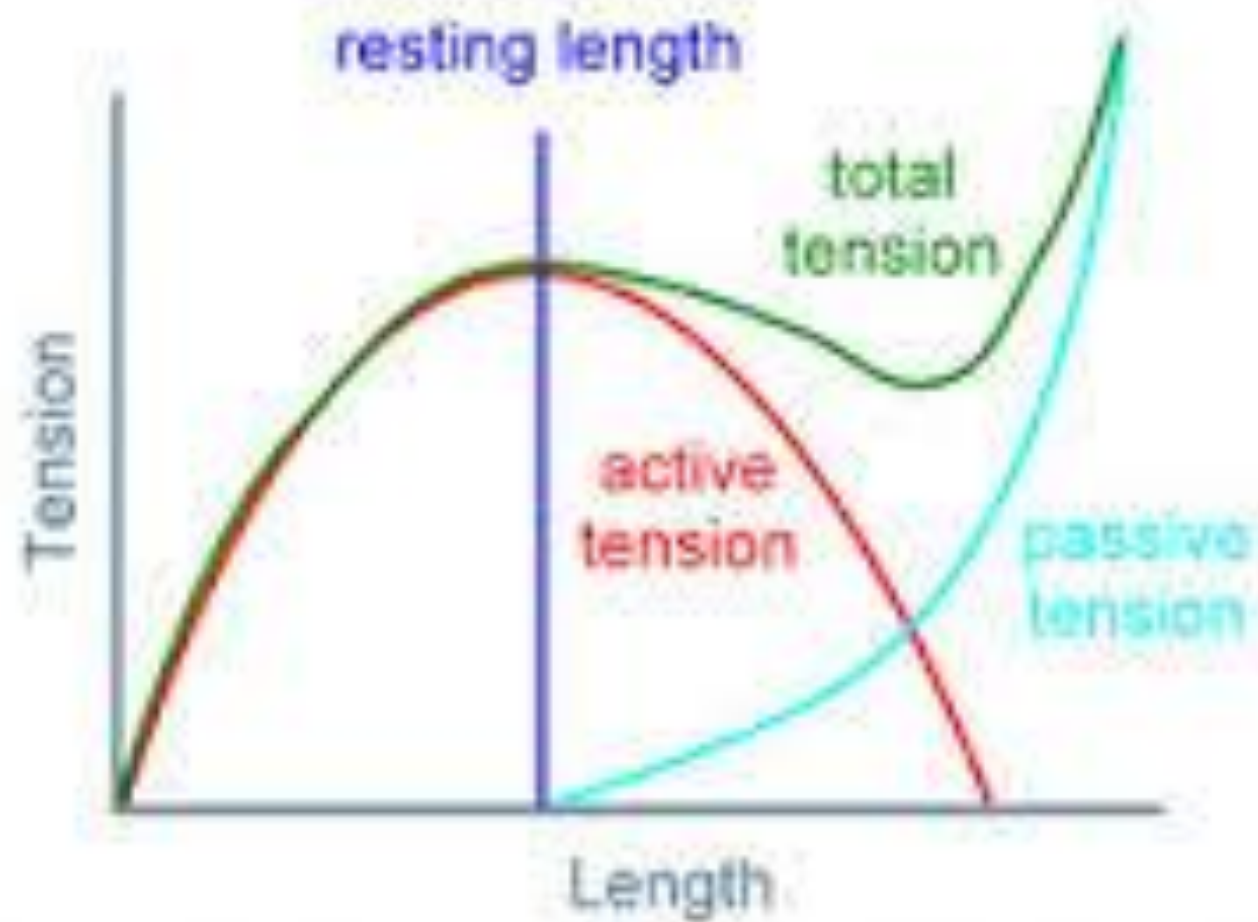
# Sources of energy for muscle contraction



# **Muscle Mechanics**

Fig. 10.10





Length-Tension Curve of a Muscle

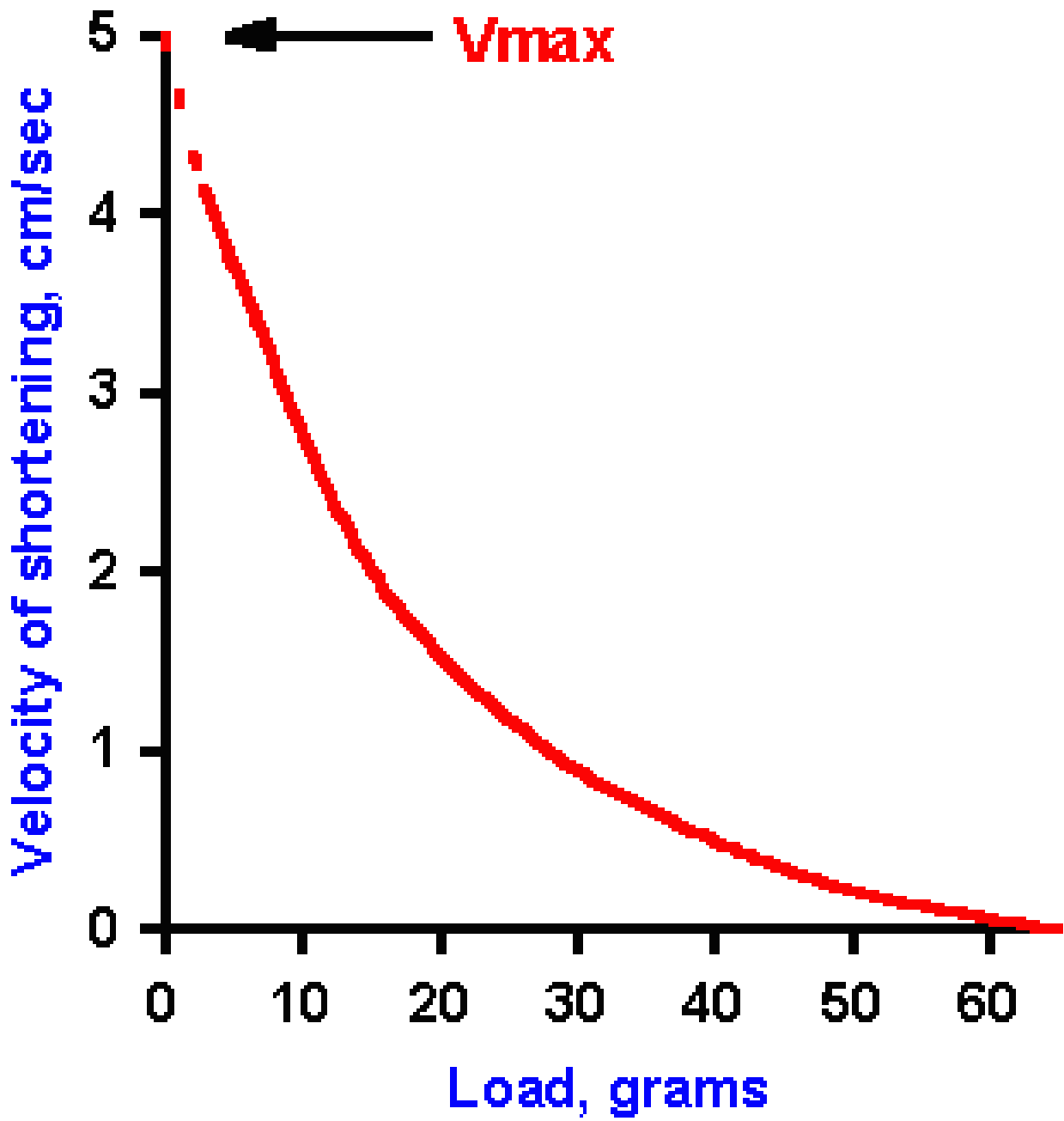
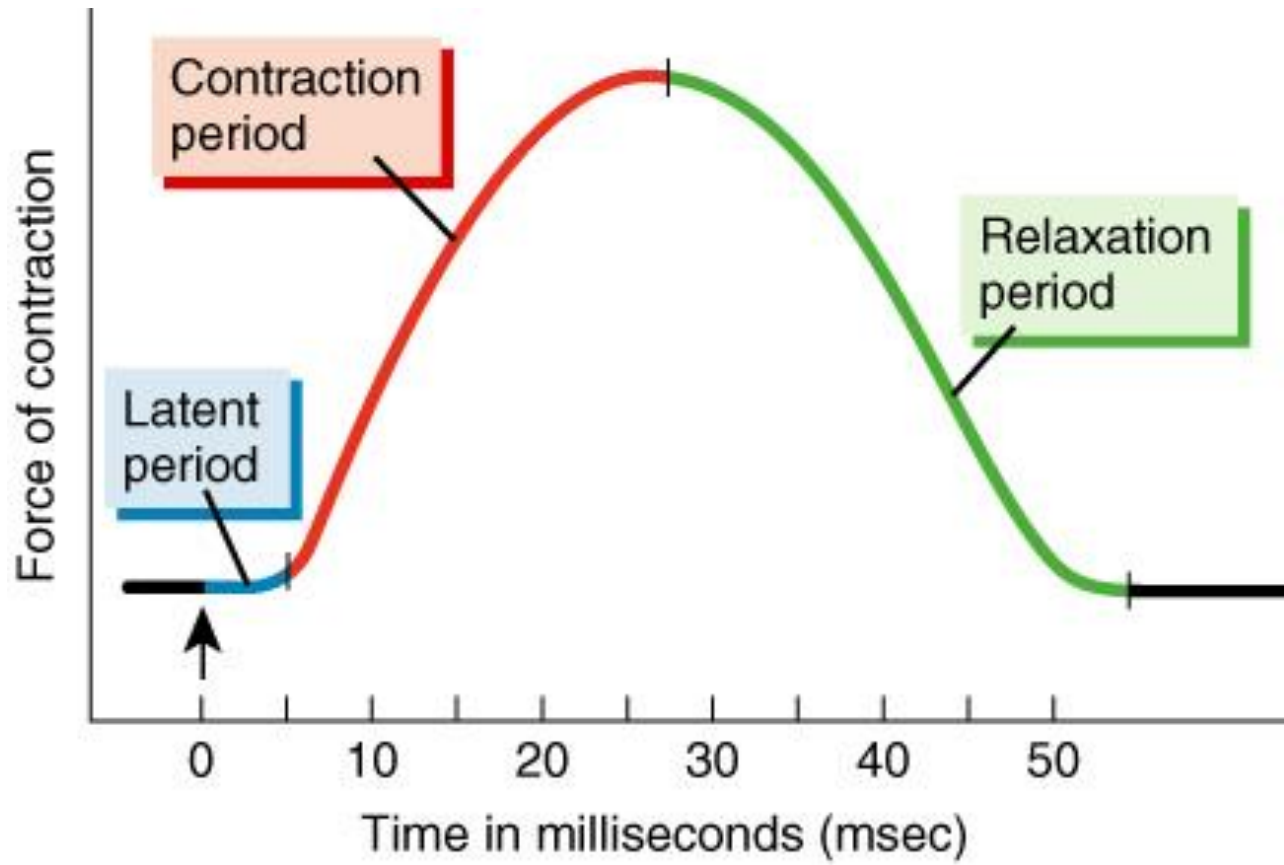


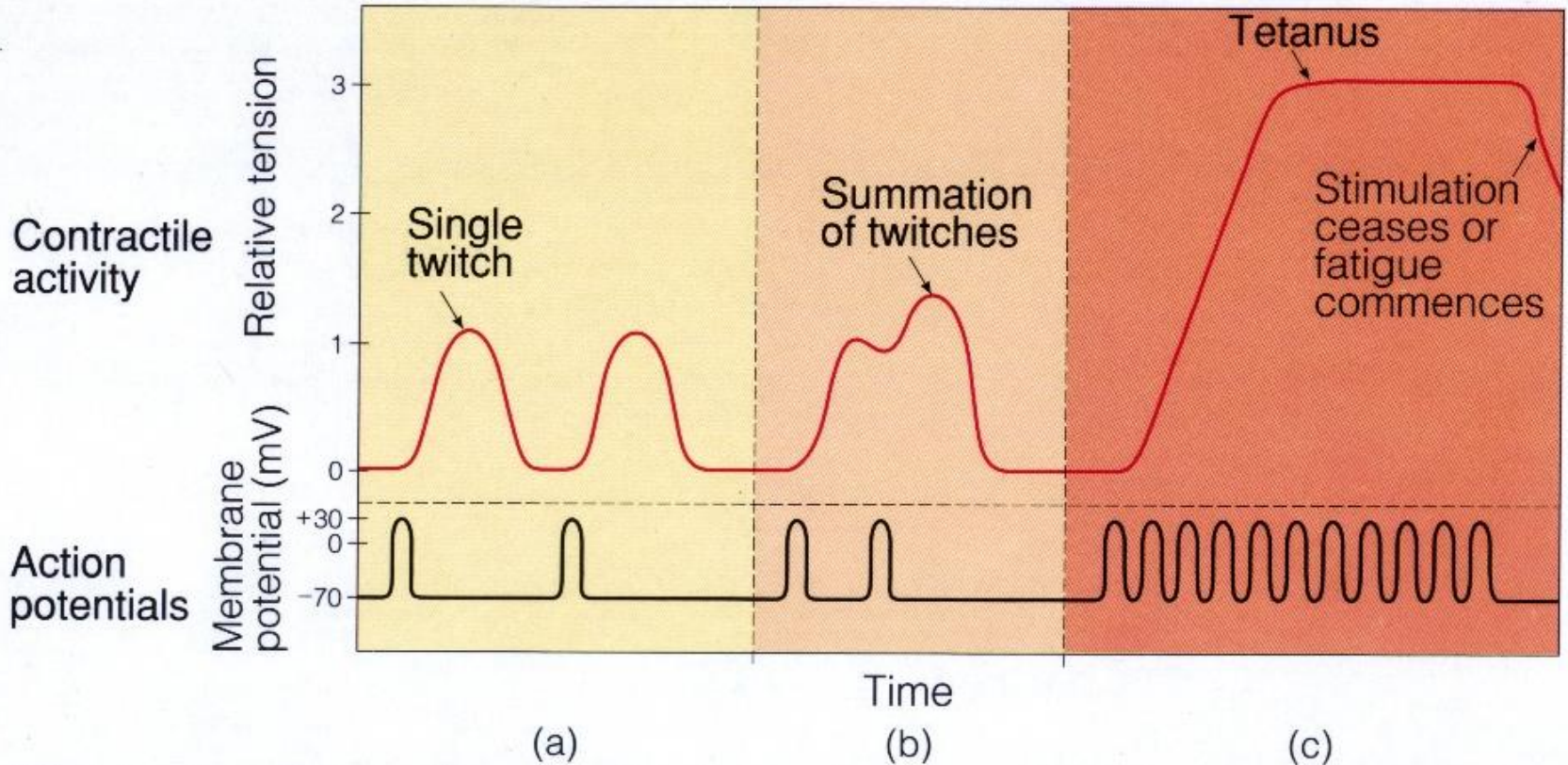


Fig. 10.15

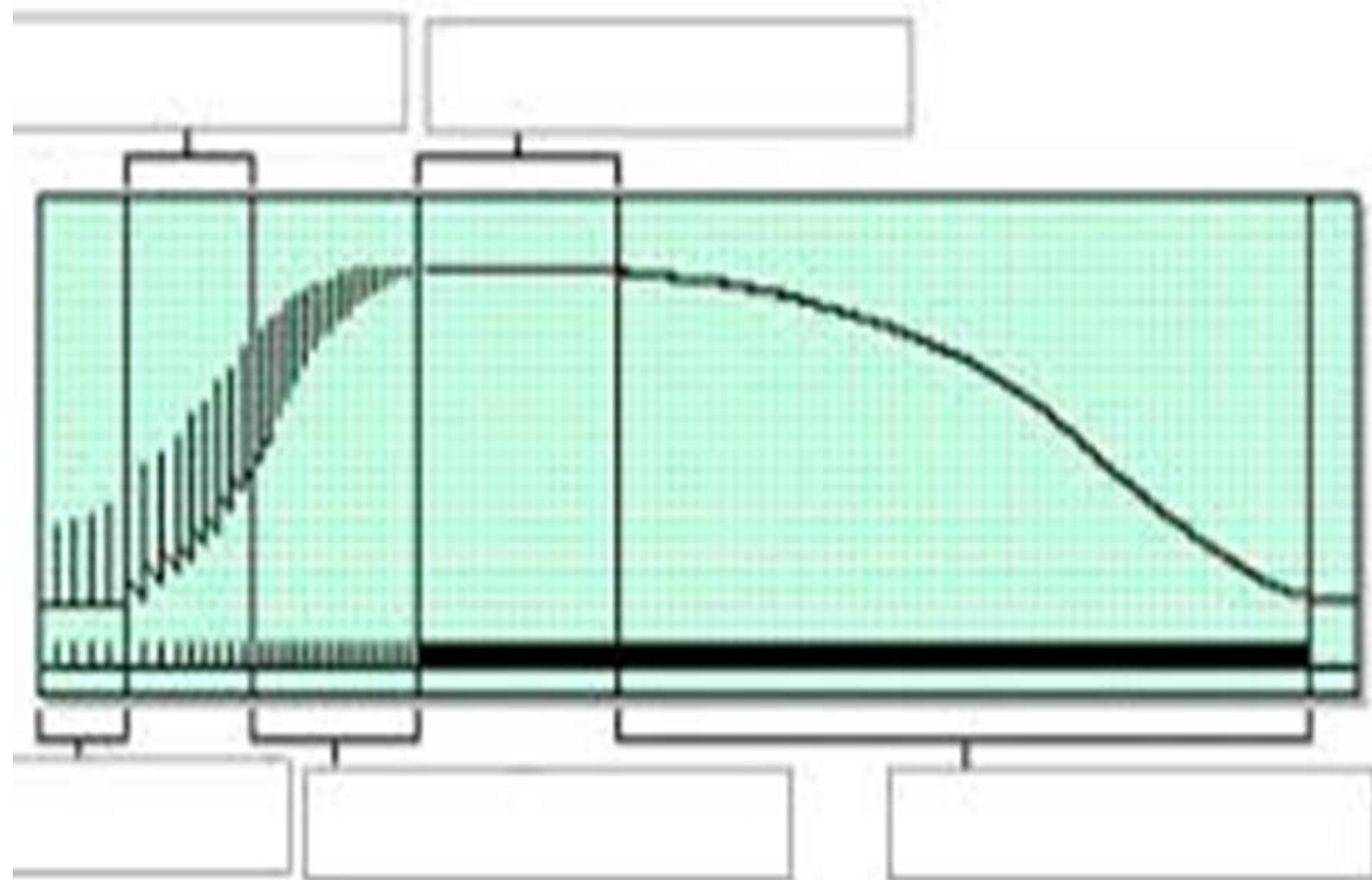


10.15

## Summation and Tetanus



The duration of the action potentials is not drawn to scale but is exaggerated.



● FIGURE 8-15

Schematic representation of motor units in a skeletal muscle

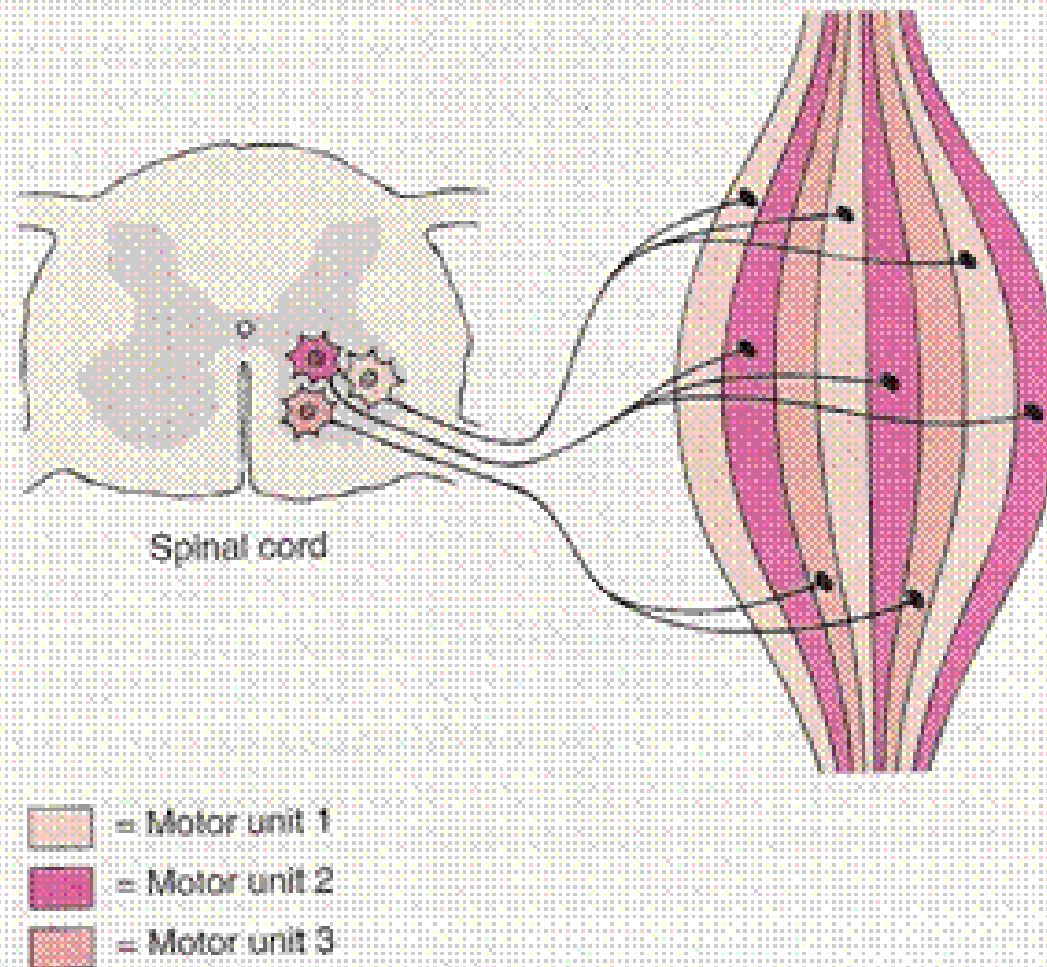


Figure 4. One motor unit can control multiple muscle cells.

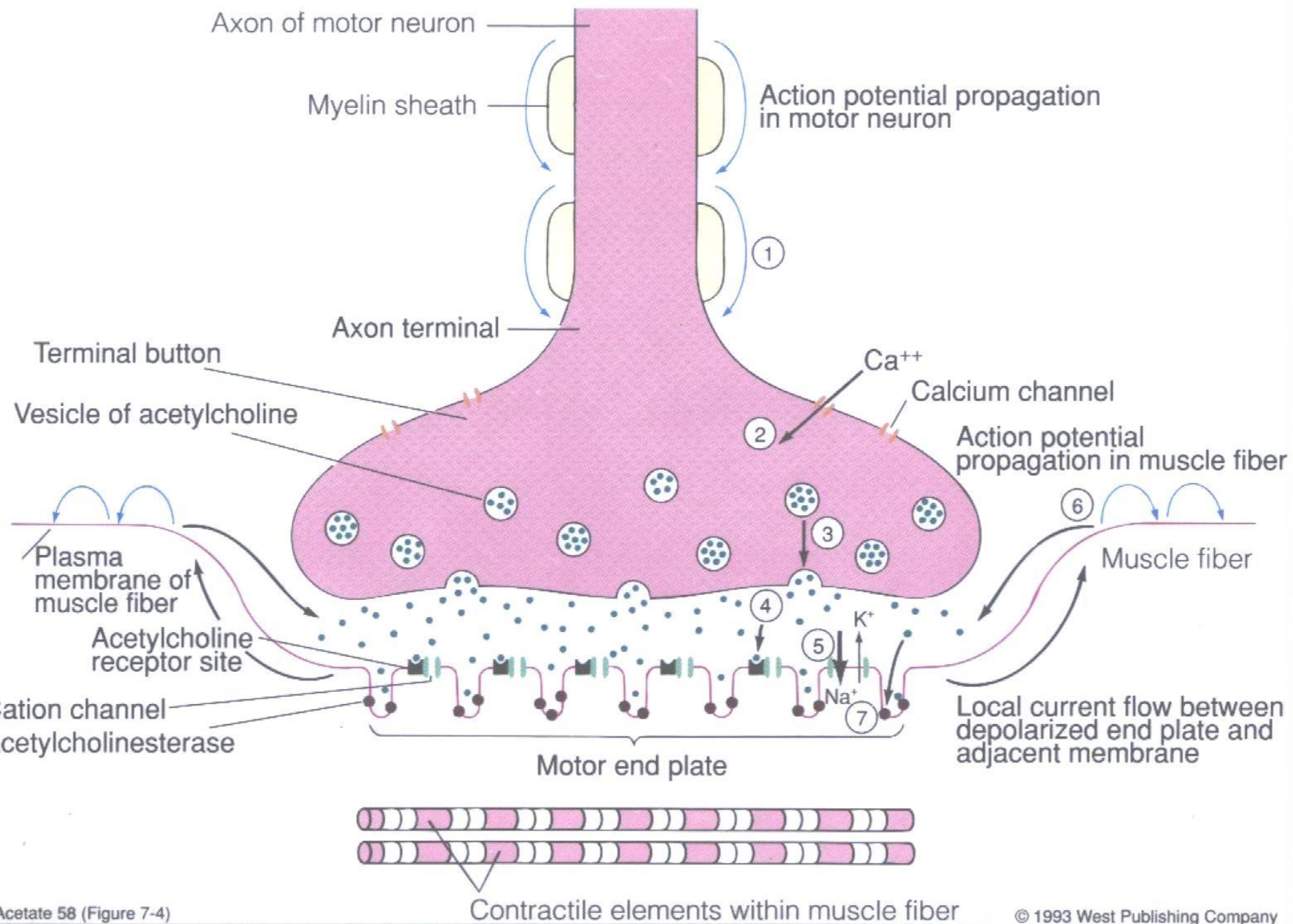
Taken from Sherwood, 2004

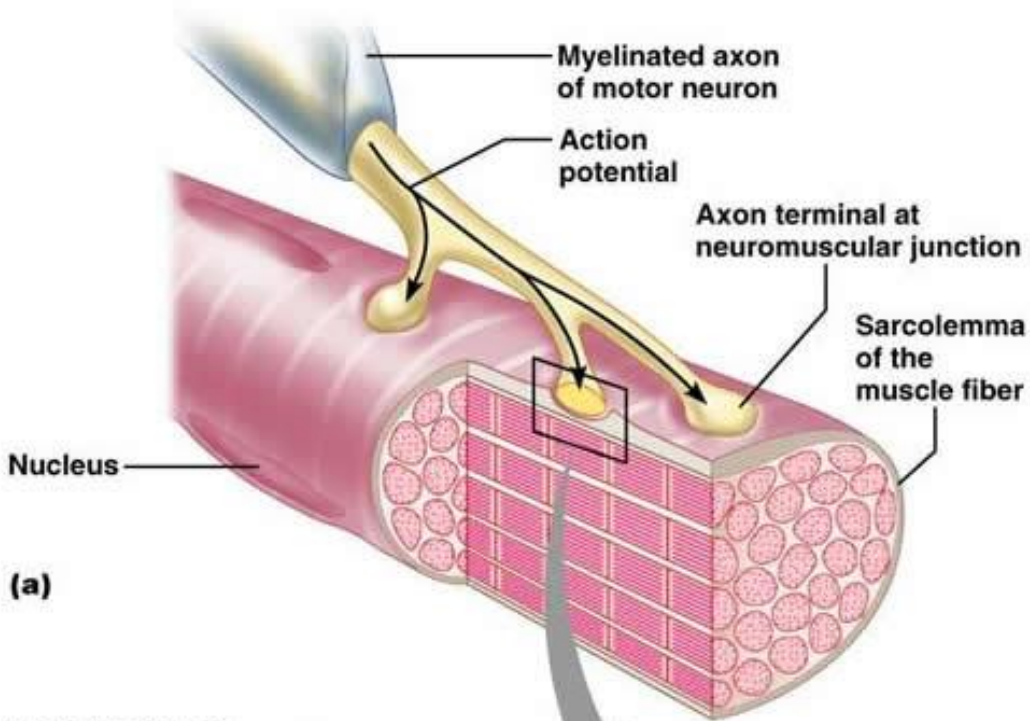
# Excitation – Contraction Coupling

- <https://www.youtube.com/watch?v=LlgaziPCFU0>

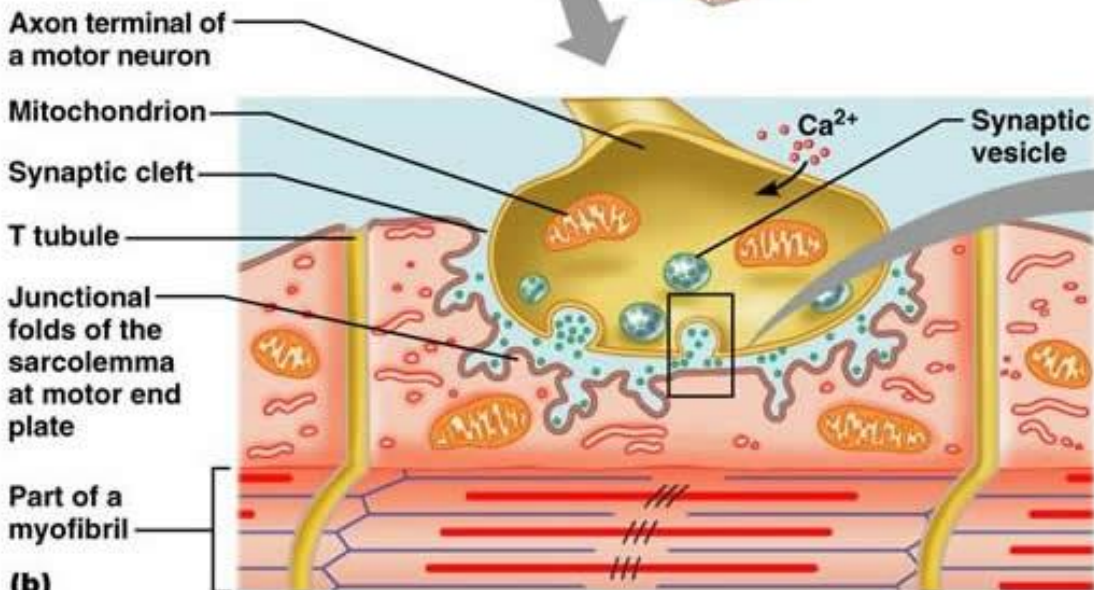


# Events at a Neuromuscular Junction

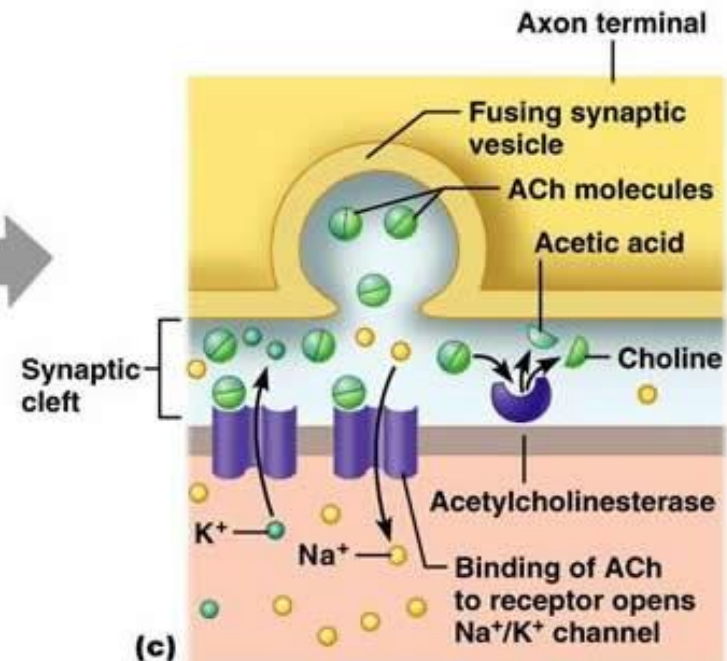




(a)



(b)



(c)

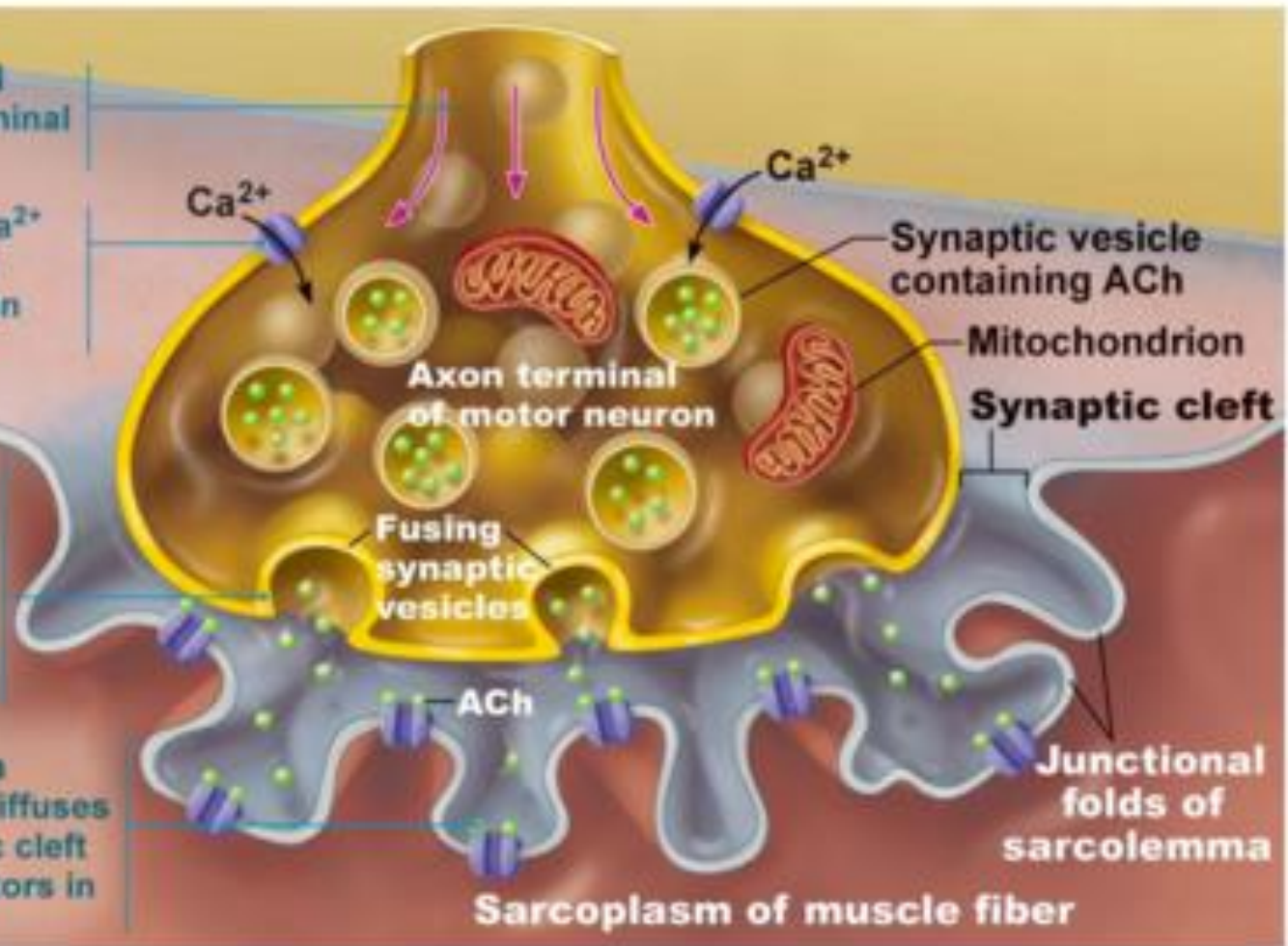


**1** Action potential arrives at axon terminal of motor neuron.

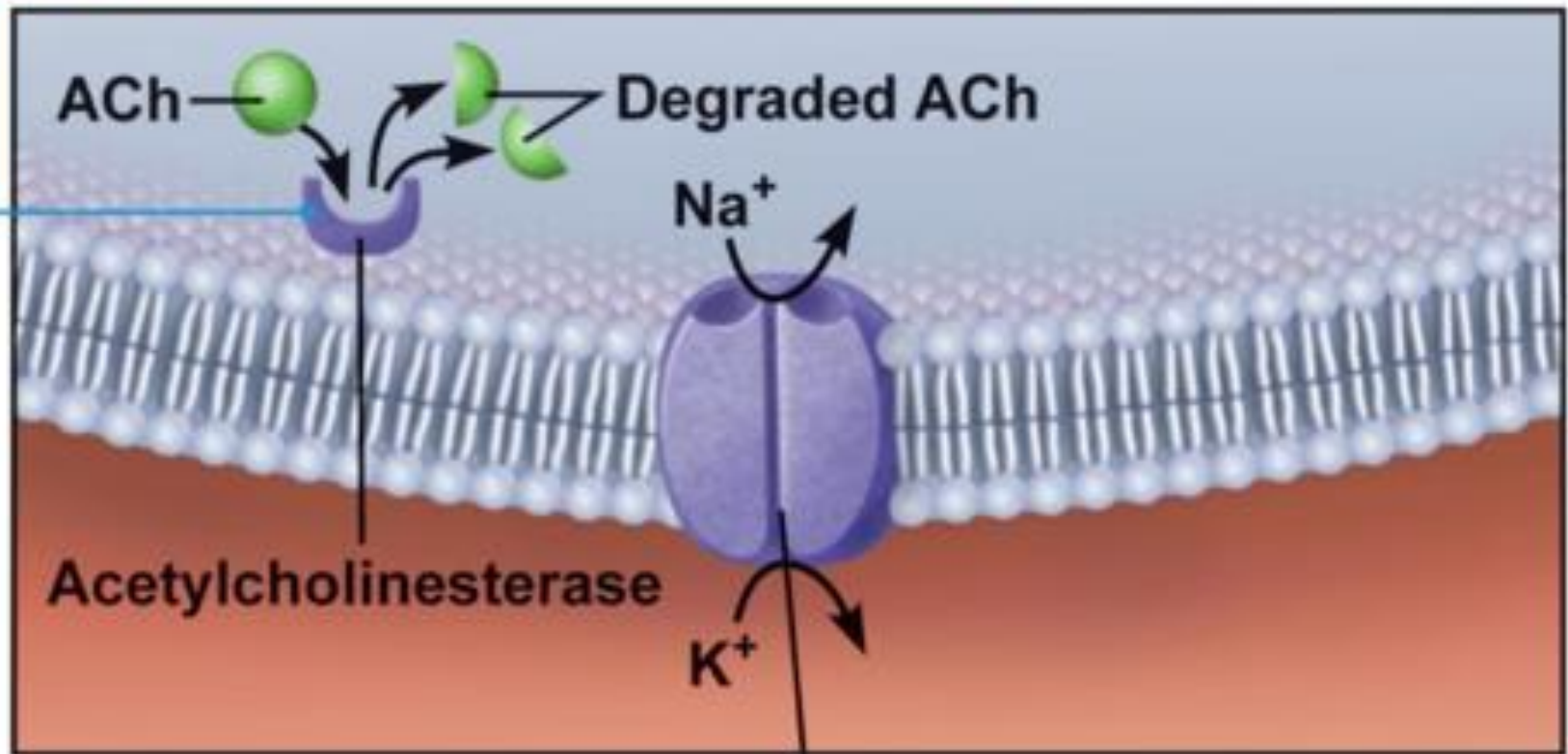
**2** Voltage-gated  $\text{Ca}^{2+}$  channels open and  $\text{Ca}^{2+}$  enters the axon terminal.

**3**  $\text{Ca}^{2+}$  entry causes some synaptic vesicles to release their contents (acetylcholine) by exocytosis.

**4** Acetylcholine, a neurotransmitter, diffuses across the synaptic cleft and binds to receptors in the sarcolemma.



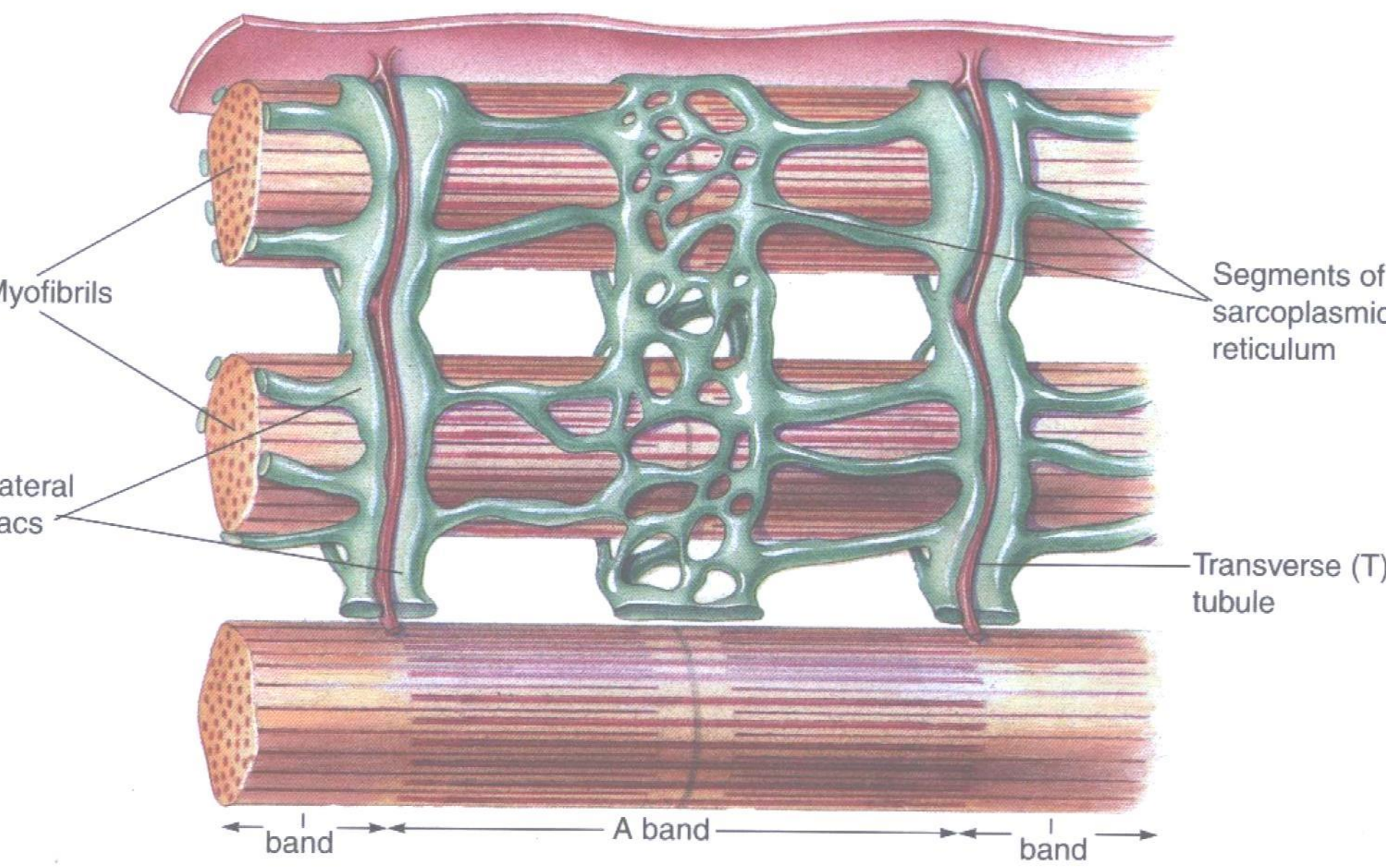
⑥ ACh effects are terminated by its enzymatic breakdown in the synaptic cleft by acetylcholinesterase.



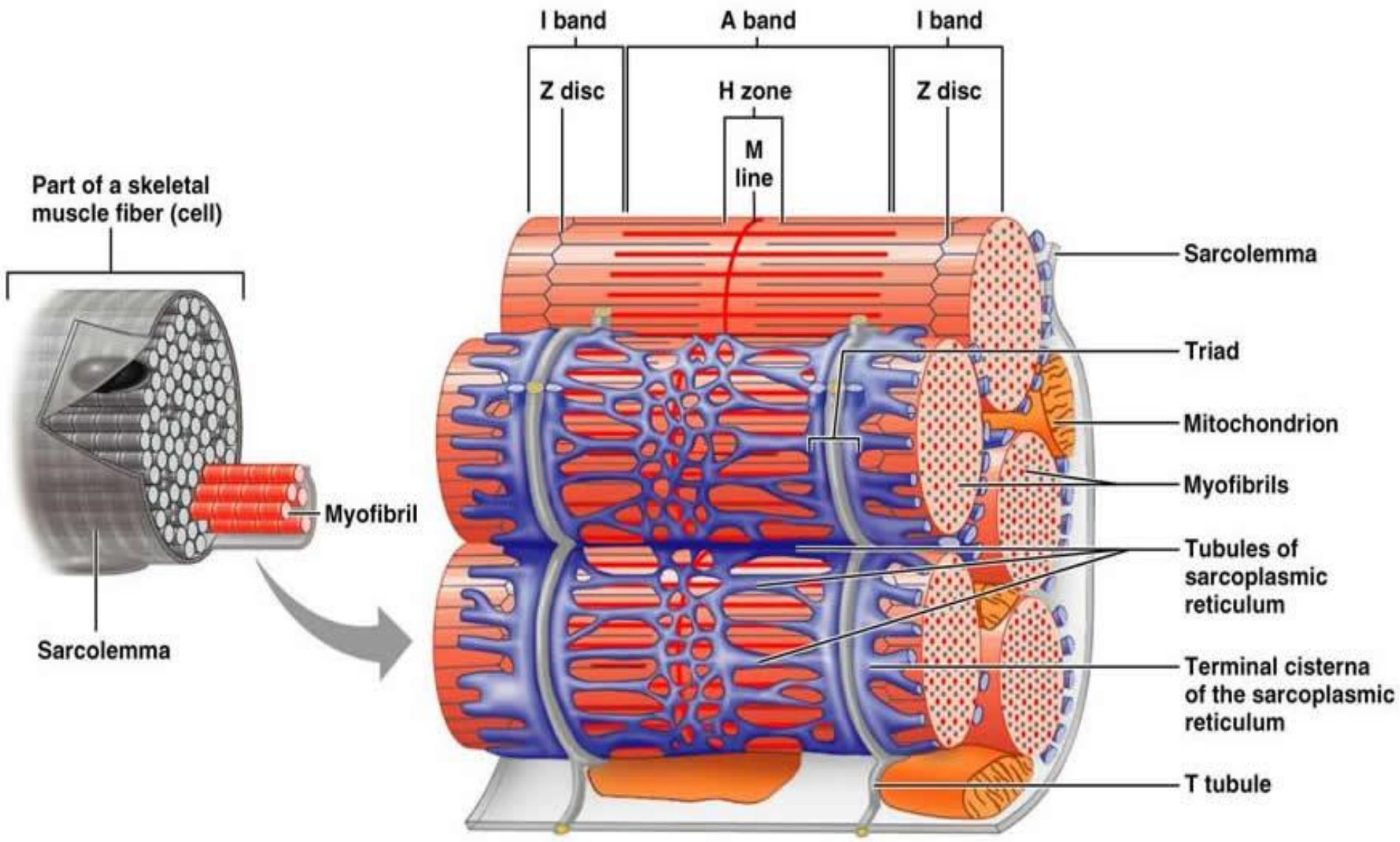
Postsynaptic membrane ion channel closed; ions cannot pass.

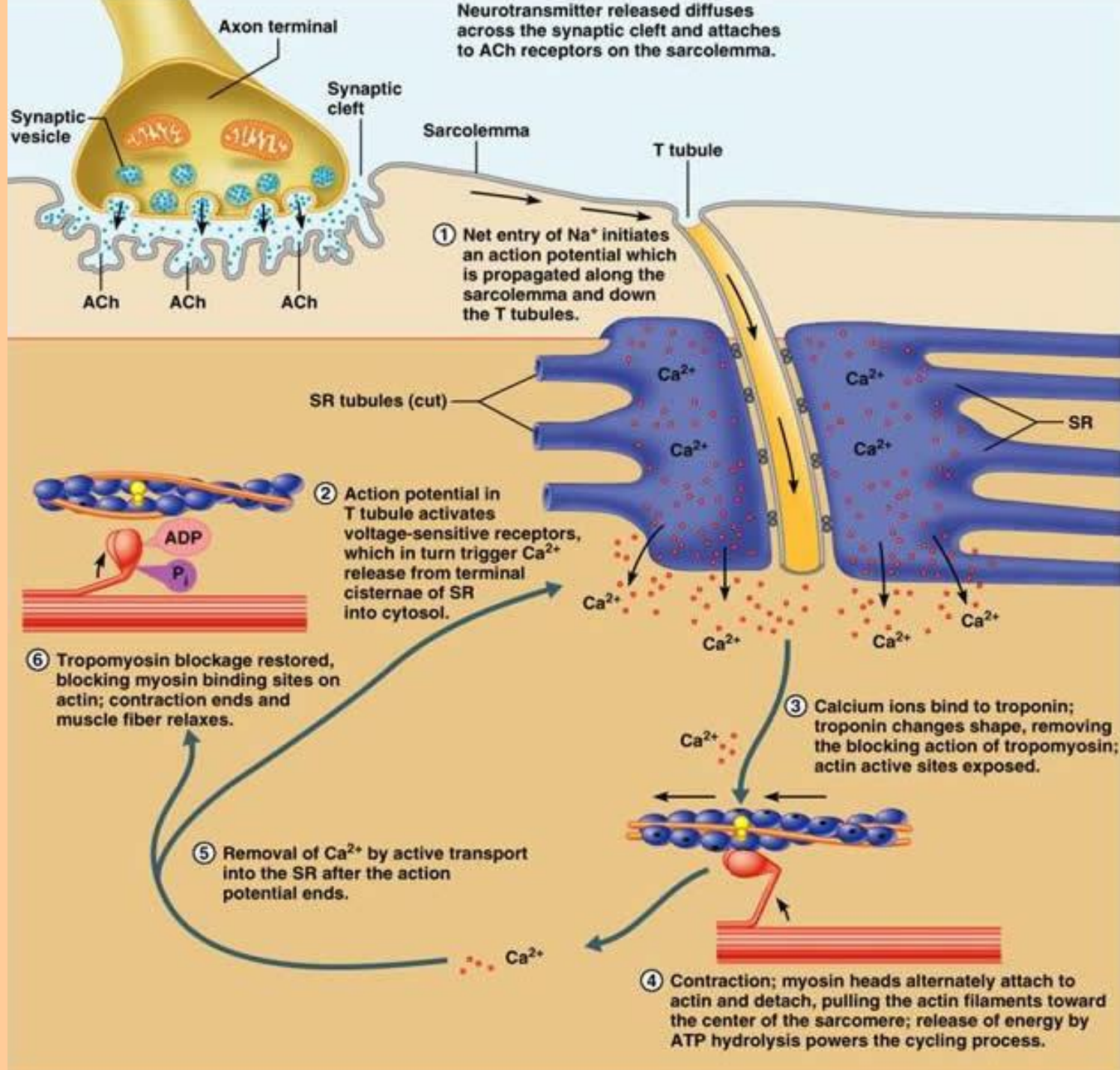


Surface membrane of muscle fiber

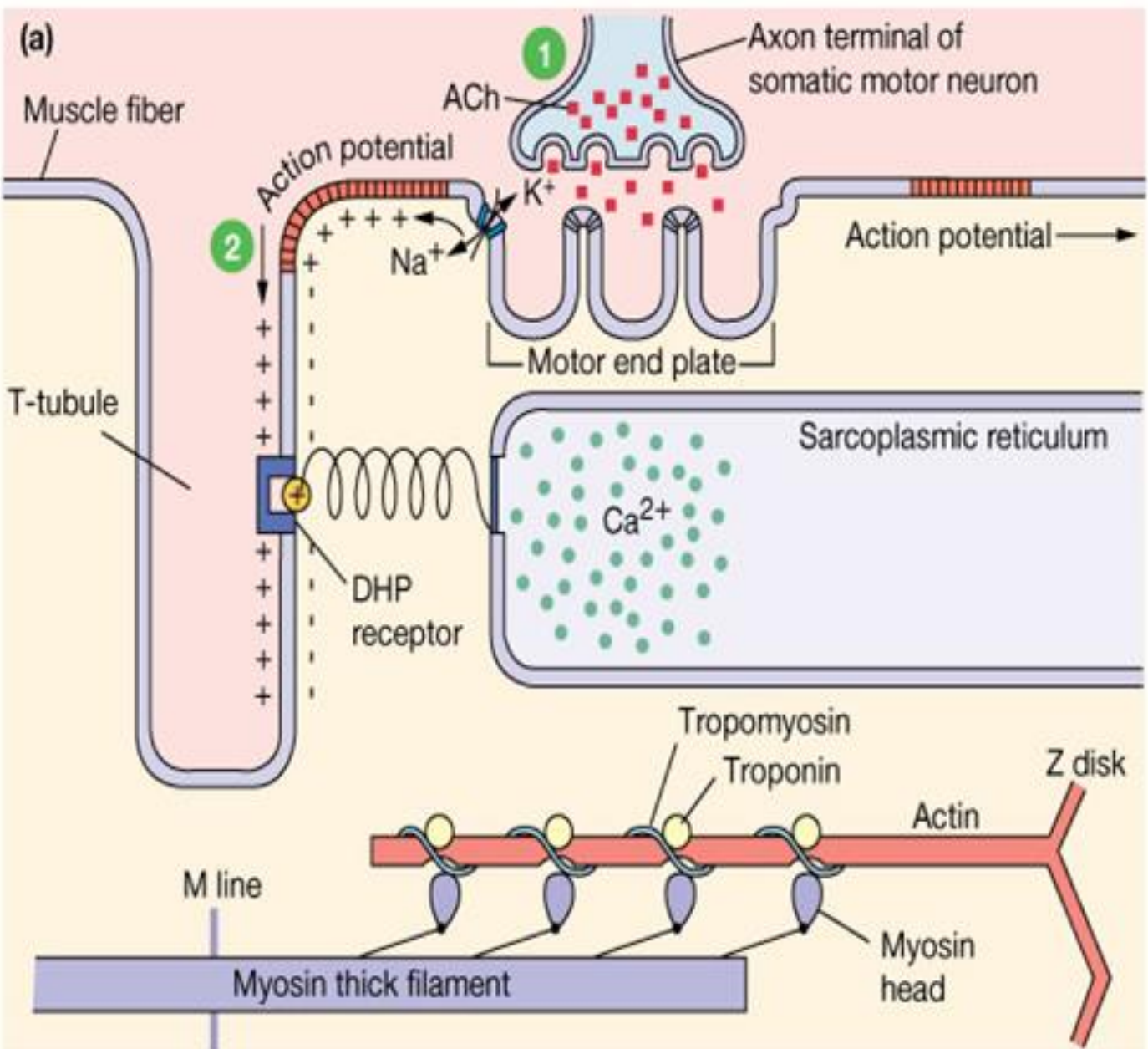






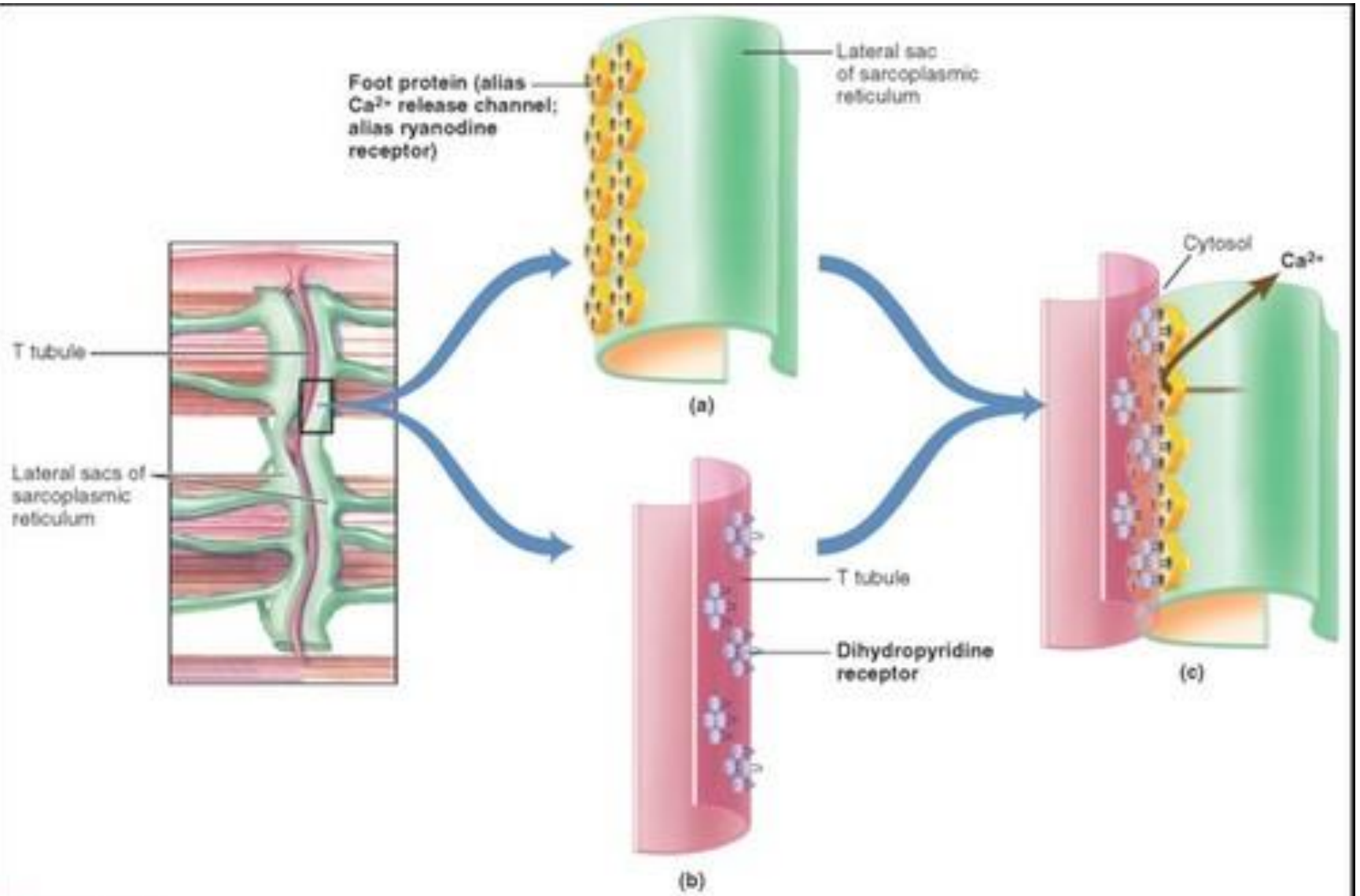






1 Somatic motor neuron releases ACh at neuromuscular junction.

2 Net entry of Na<sup>+</sup> through ACh receptor-channel initiates a muscle action potential.



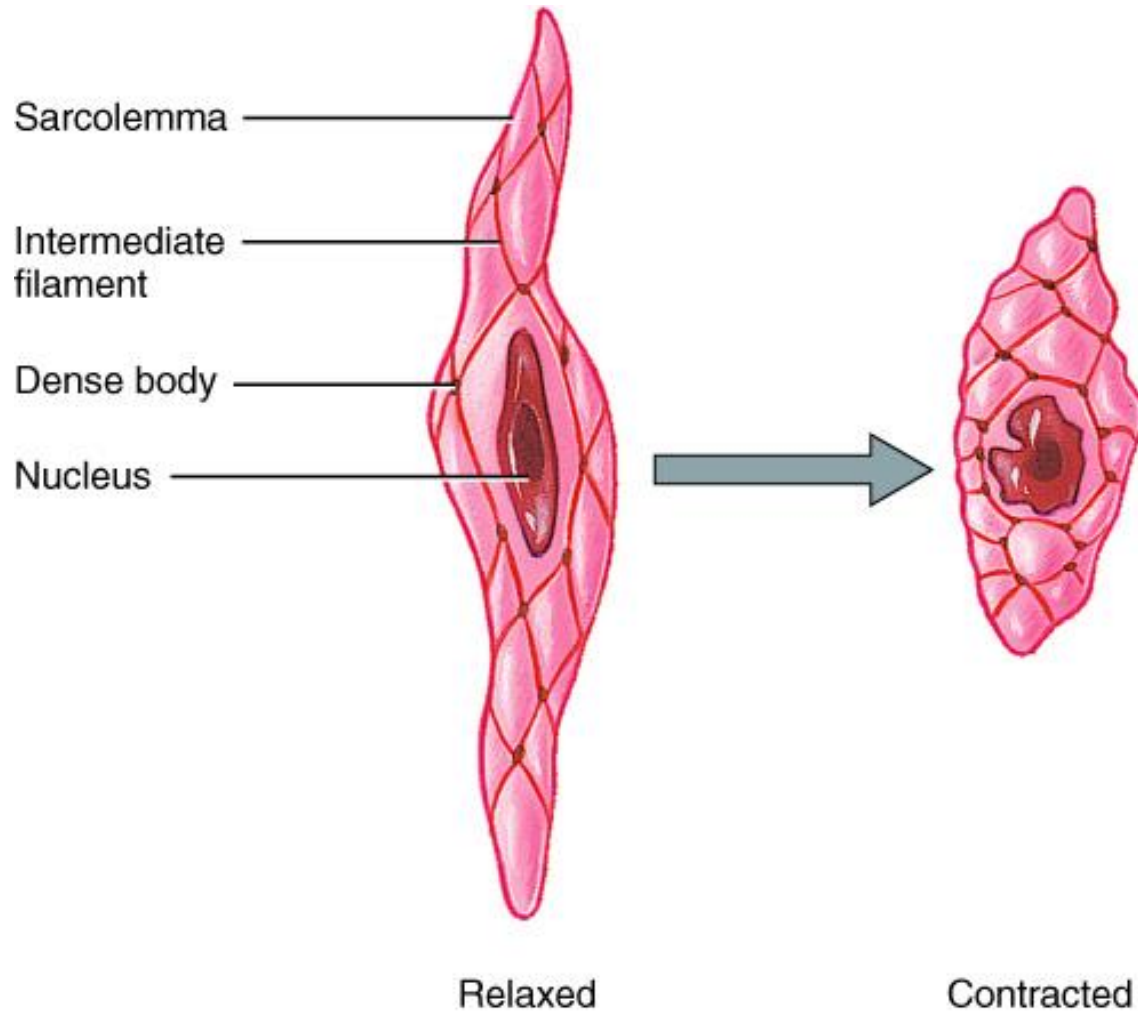
● **FIGURE 8-11**

Relationship between a T tubule and the adjacent lateral sacs of the sarcoplasmic reticulum

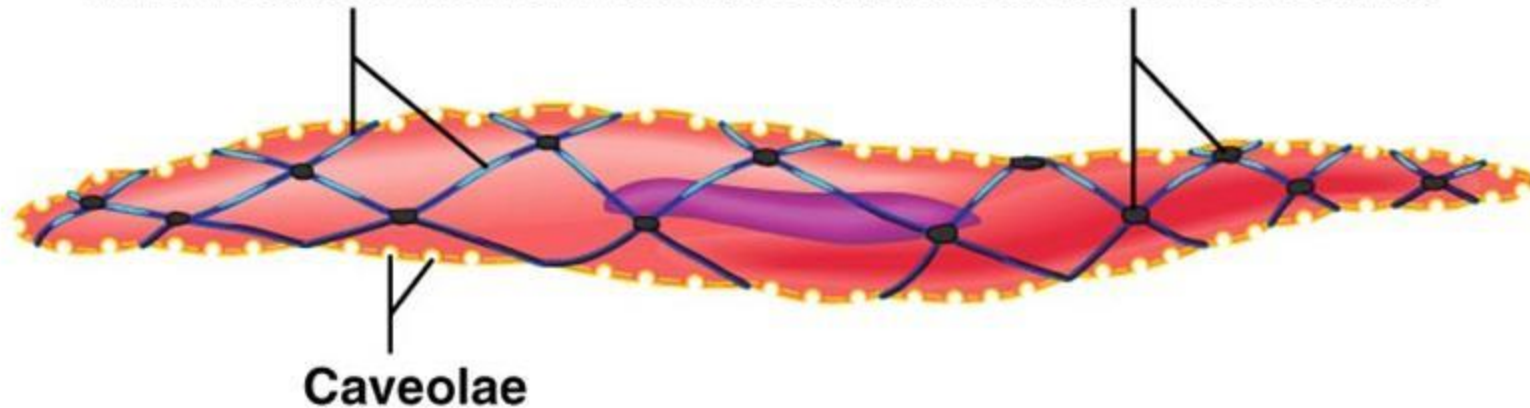
# **SMOOTH MUSCLE CELLS**



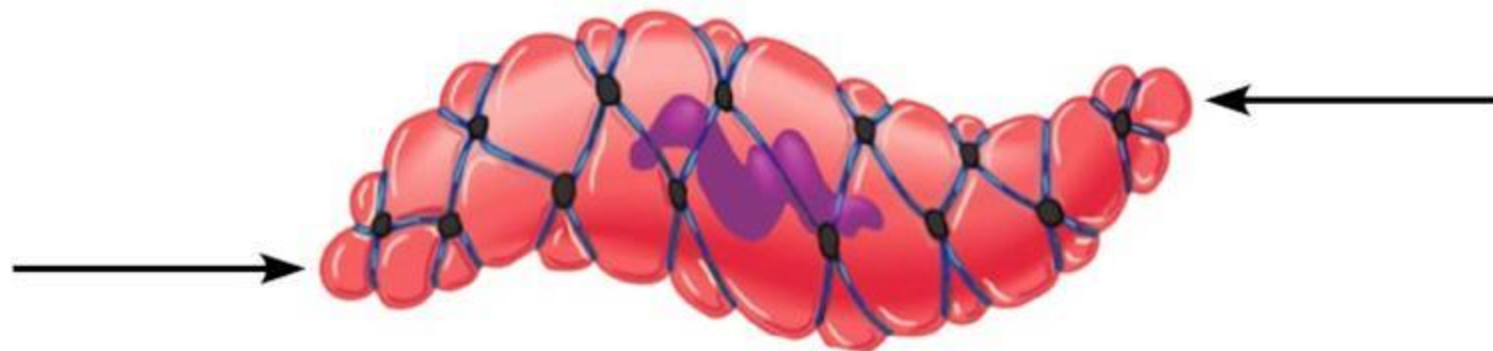
Fig. 10.19



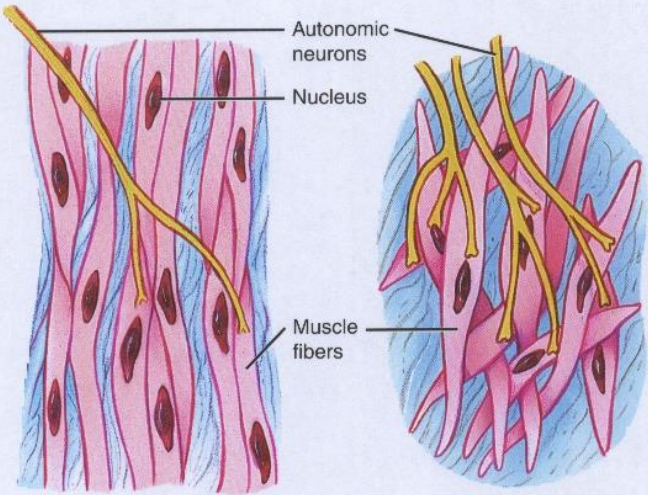
Intermediate filament bundles attached to dense bodies



**(a) Relaxed smooth muscle cell**

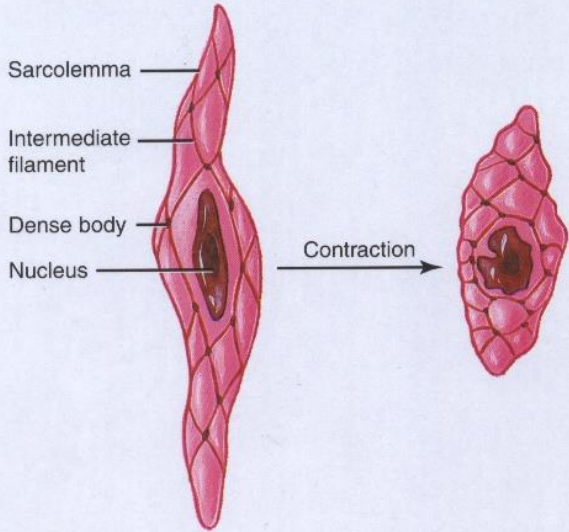


**(b) Contracted smooth muscle cell**

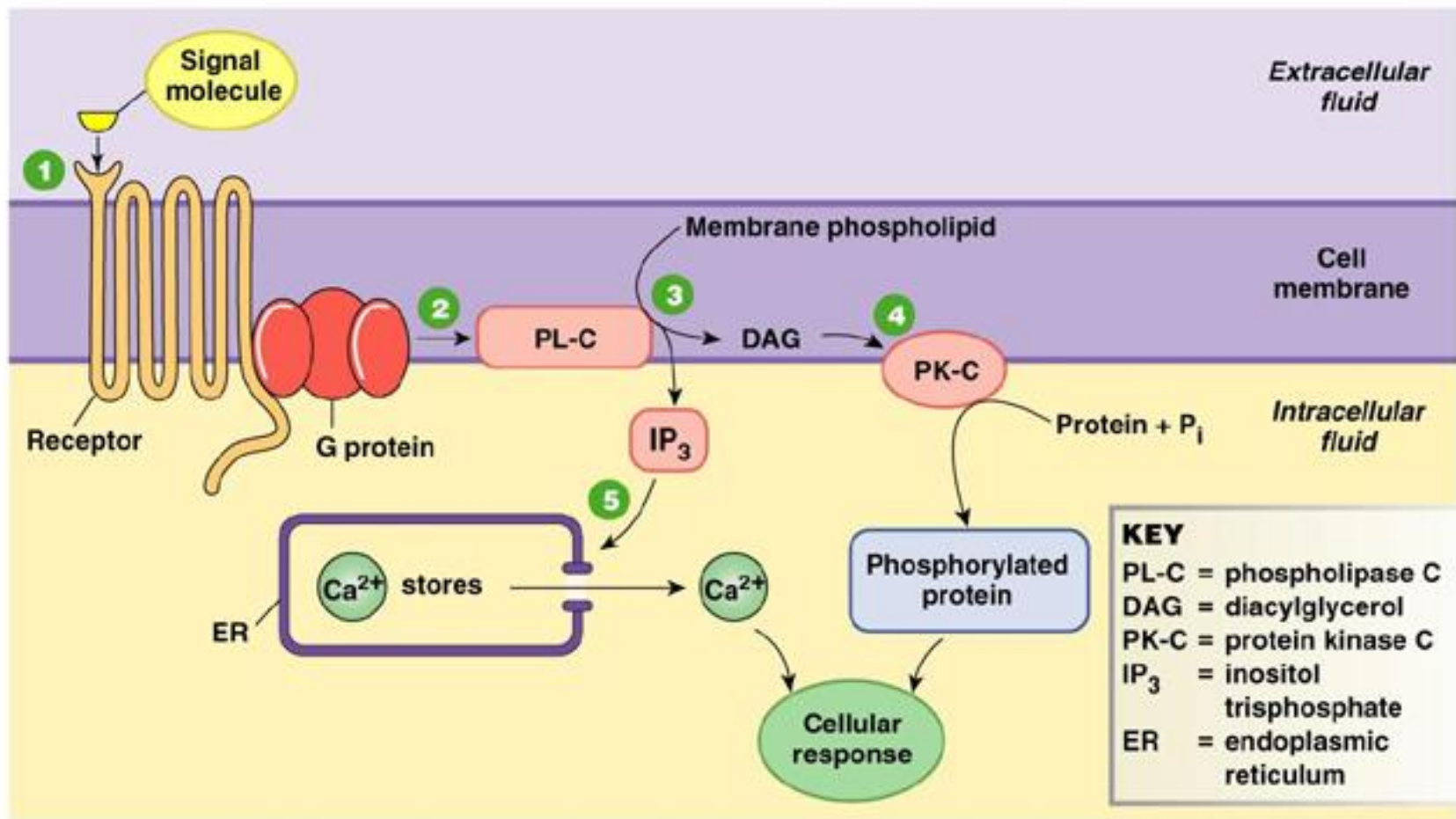


(a) Visceral (single-unit) smooth muscle tissue

(b) Multiunit smooth muscle tissue



(c) Details of a smooth muscle fiber

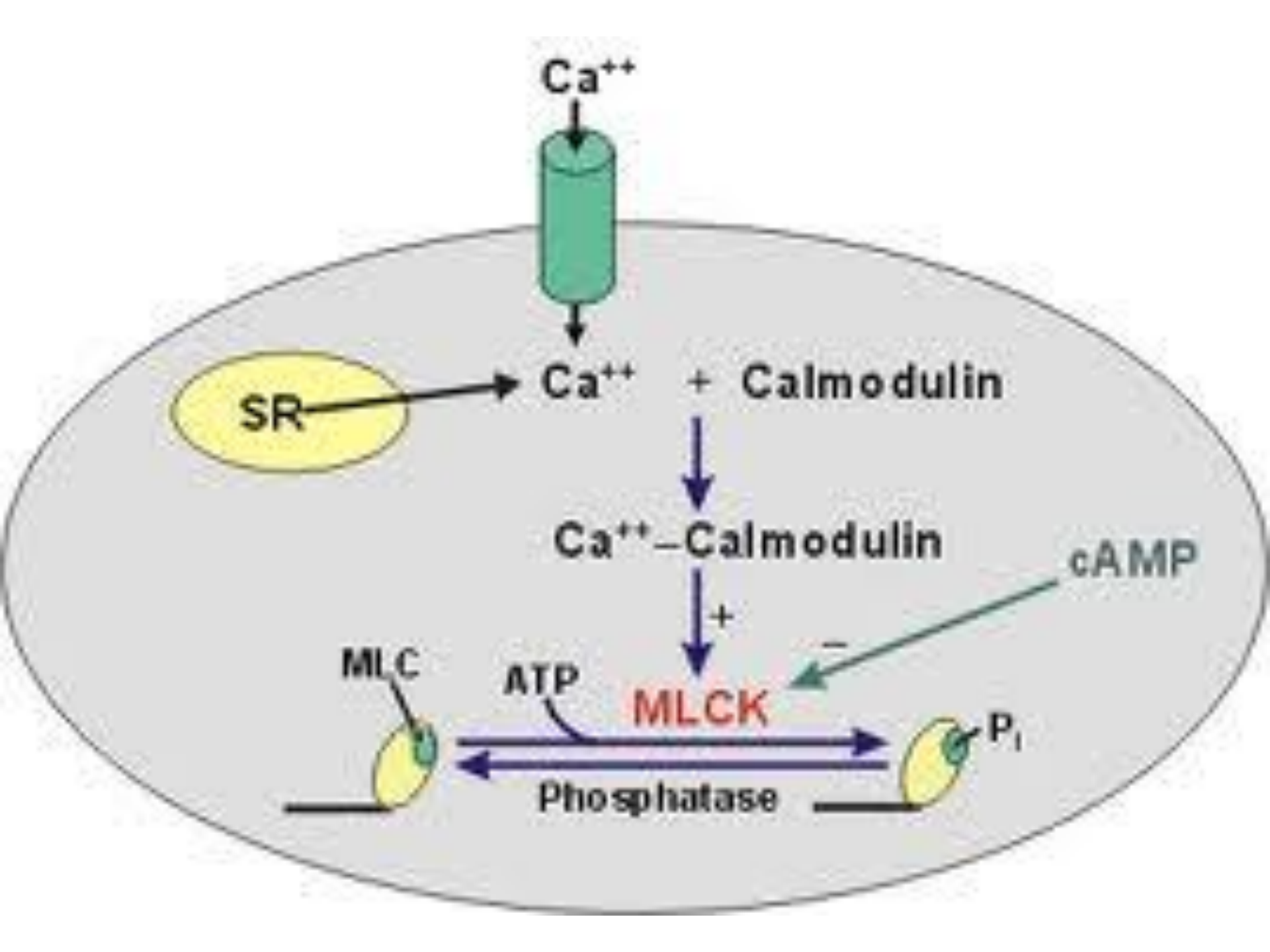


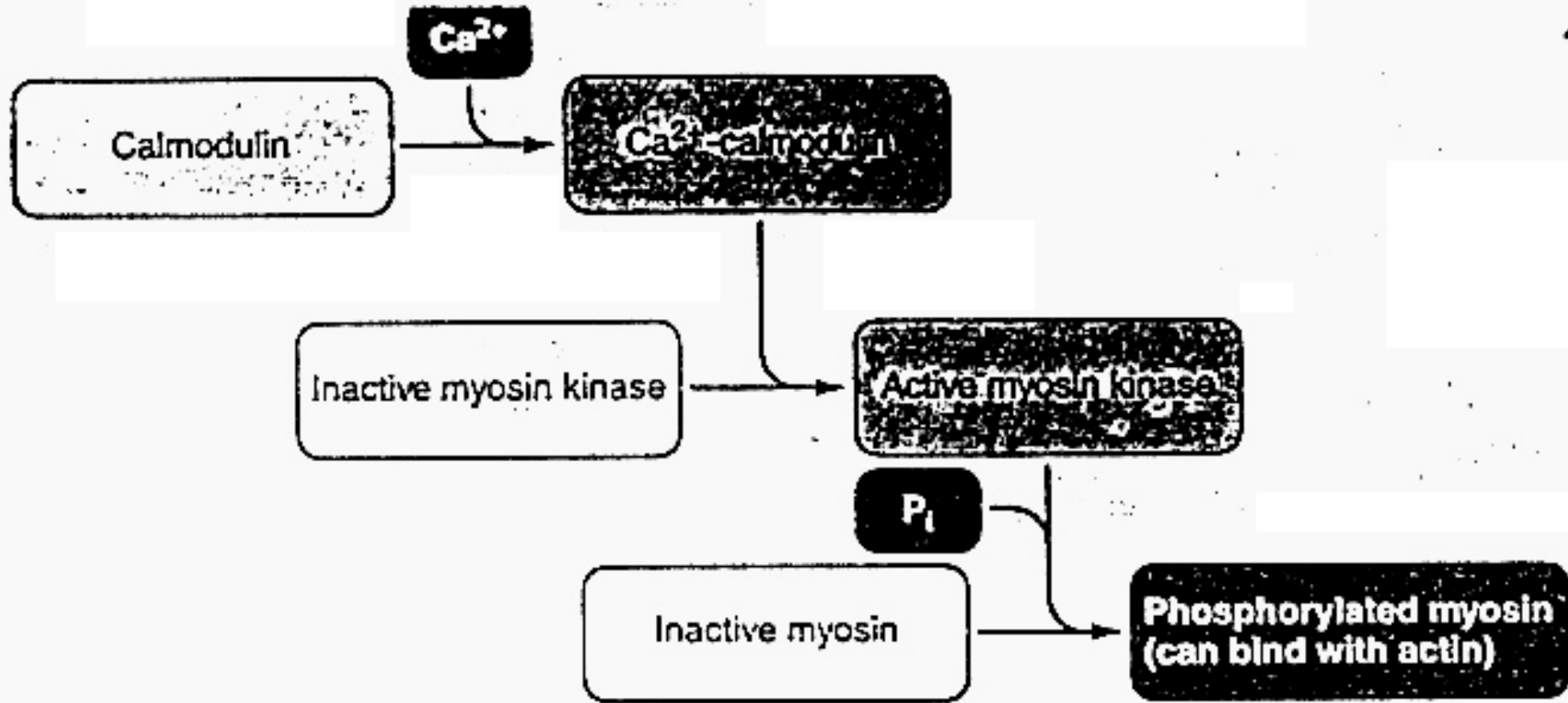
- 1** Signal molecule activates receptor and associated G protein.
- 2** G protein activates phospholipase C (PL-C), an amplifier enzyme.
- 3** PL-C converts membrane phospholipids into diacylglycerol (DAG), which remains in the membrane, and IP<sub>3</sub>, which diffuses into the cytoplasm.
- 4** DAG activates protein kinase C (PK-C), which phosphorylates proteins.
- 5** IP<sub>3</sub> causes release of Ca<sup>2+</sup> from organelles, creating a Ca<sup>2+</sup> signal.











**Table 12.8 | Comparison of Skeletal, Cardiac, and Smooth Muscle**

<b>Skeletal Muscle</b>	<b>Cardiac Muscle</b>	<b>Smooth Muscle</b>
Striated; actin and myosin arranged in sarcomeres	Striated; actin and myosin arranged in sarcomeres	Not striated; more actin than myosin; actin inserts into dense bodies and cell membrane
Well-developed sarcoplasmic reticulum and transverse tubules	Moderately developed sarcoplasmic reticulum and transverse tubules	Poorly developed sarcoplasmic reticulum; no transverse tubules
Contains troponin in the thin filaments	Contains troponin in the thin filaments	Contains calmodulin, a protein that, when bound to $\text{Ca}^{2+}$ , activates the enzyme myosin light-chain kinase
$\text{Ca}^{2+}$ released into cytoplasm from sarcoplasmic reticulum	$\text{Ca}^{2+}$ enters cytoplasm from sarcoplasmic reticulum and extracellular fluid	$\text{Ca}^{2+}$ enters cytoplasm from extracellular fluid, sarcoplasmic reticulum, and perhaps mitochondria
Cannot contract without nerve stimulation; denervation results in muscle atrophy	Can contract without nerve stimulation; action potentials originate in pacemaker cells of heart	Maintains tone in absence of nerve stimulation; visceral smooth muscle produces pacemaker potentials; denervation results in hypersensitivity to stimulation
Muscle fibers stimulated independently; no gap junctions	Gap junctions present as intercalated discs	Gap junctions generally present