# Muscle tissue

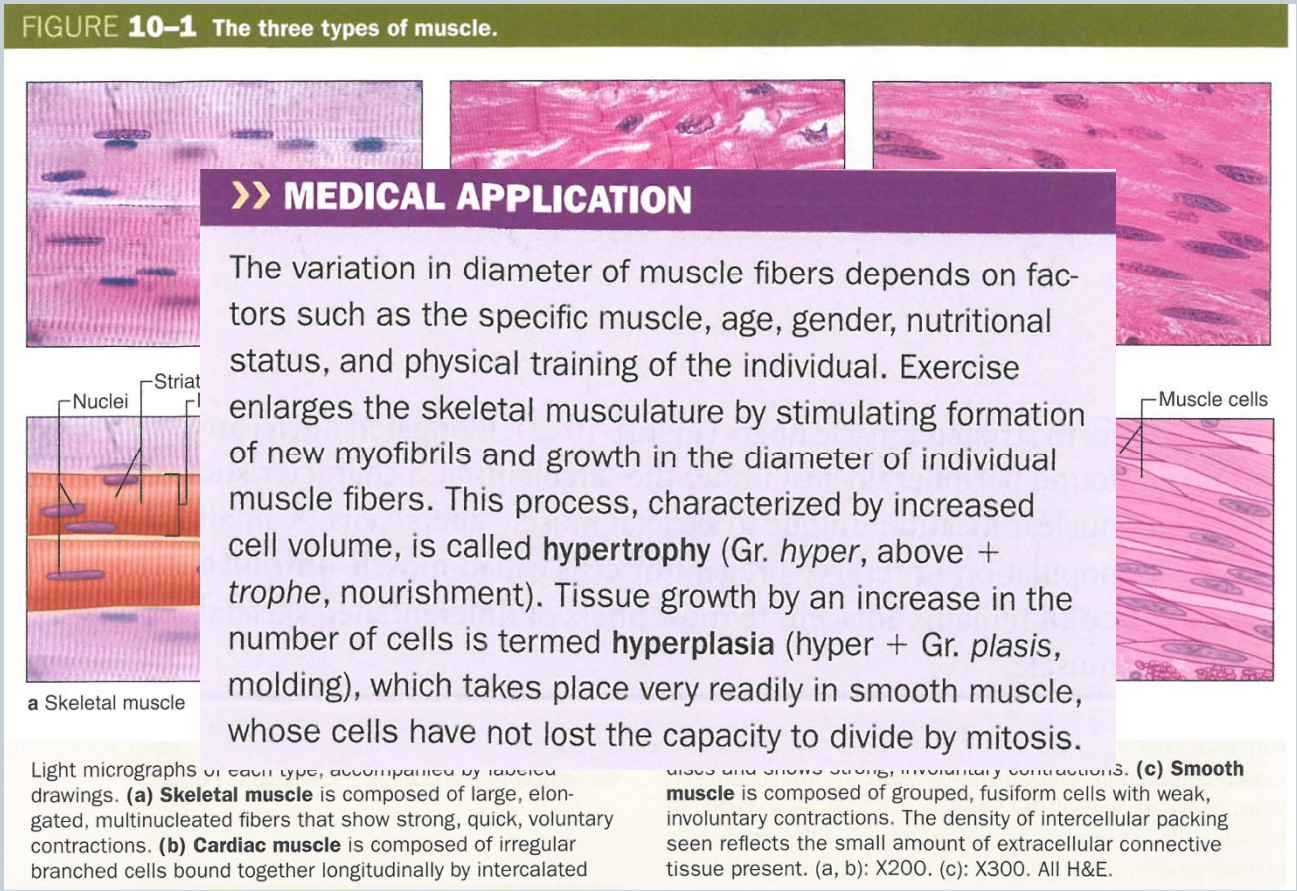
# Muscle tissue

- Contractility (Actin filaments & Myosin)
- Organ, blood & body movement
- Mesoderm origin

Types:

1. Skeletal muscle
2. Cardiac muscle
3. Smooth muscle

- Myofibroblast
- Pericyte
- Myoepithelial cell

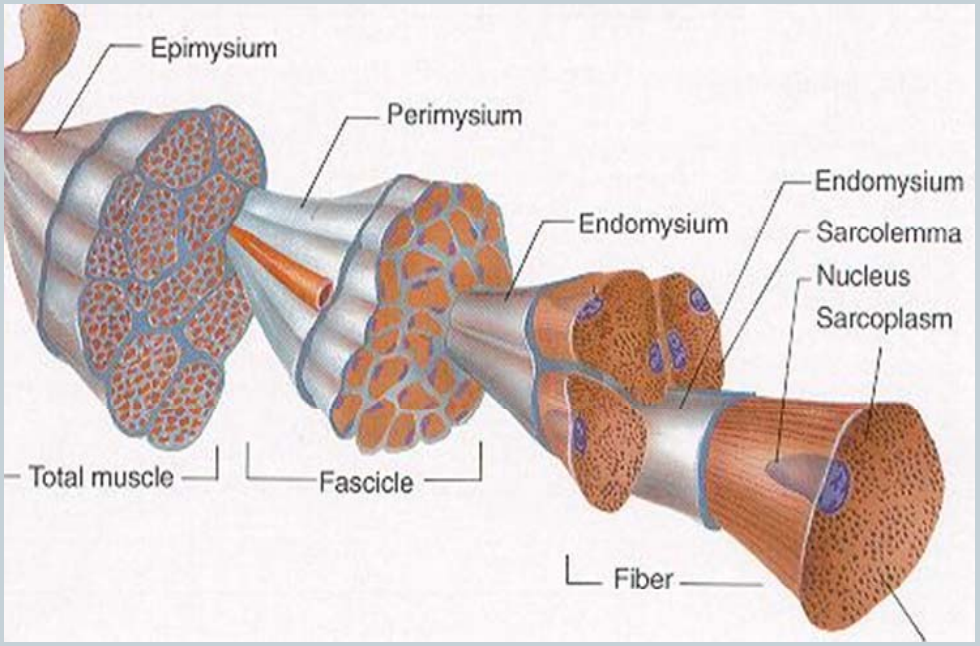



**» MEDICAL APPLICATION**

The variation in diameter of muscle fibers depends on factors such as the specific muscle, age, gender, nutritional status, and physical training of the individual. Exercise enlarges the skeletal musculature by stimulating formation of new myofibrils and growth in the diameter of individual muscle fibers. This process, characterized by increased cell volume, is called **hypertrophy** (Gr. *hyper*, above + *trophe*, nourishment). Tissue growth by an increase in the number of cells is termed **hyperplasia** (hyper + Gr. *plasis*, molding), which takes place very readily in smooth muscle, whose cells have not lost the capacity to divide by mitosis.

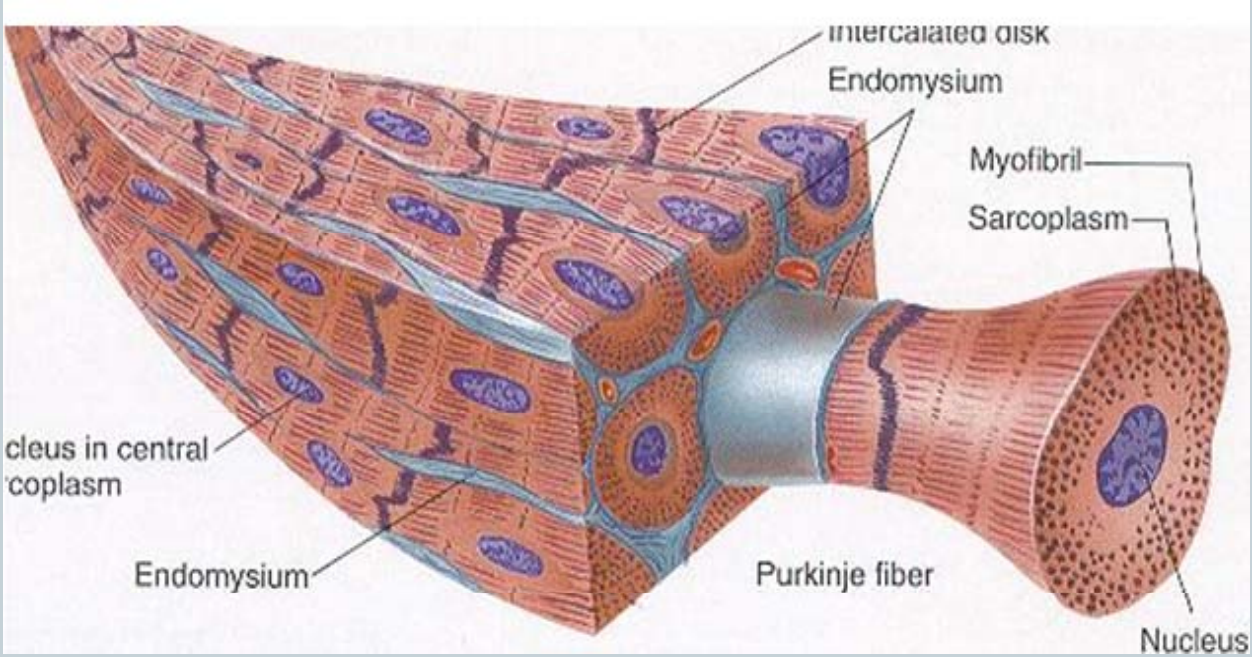
# Skeletal muscle

- Skeletal (striated): long, cylindrical multinucleated cells with cross-striations in register; peripheral nuclei, quick & forceful contraction; voluntary control



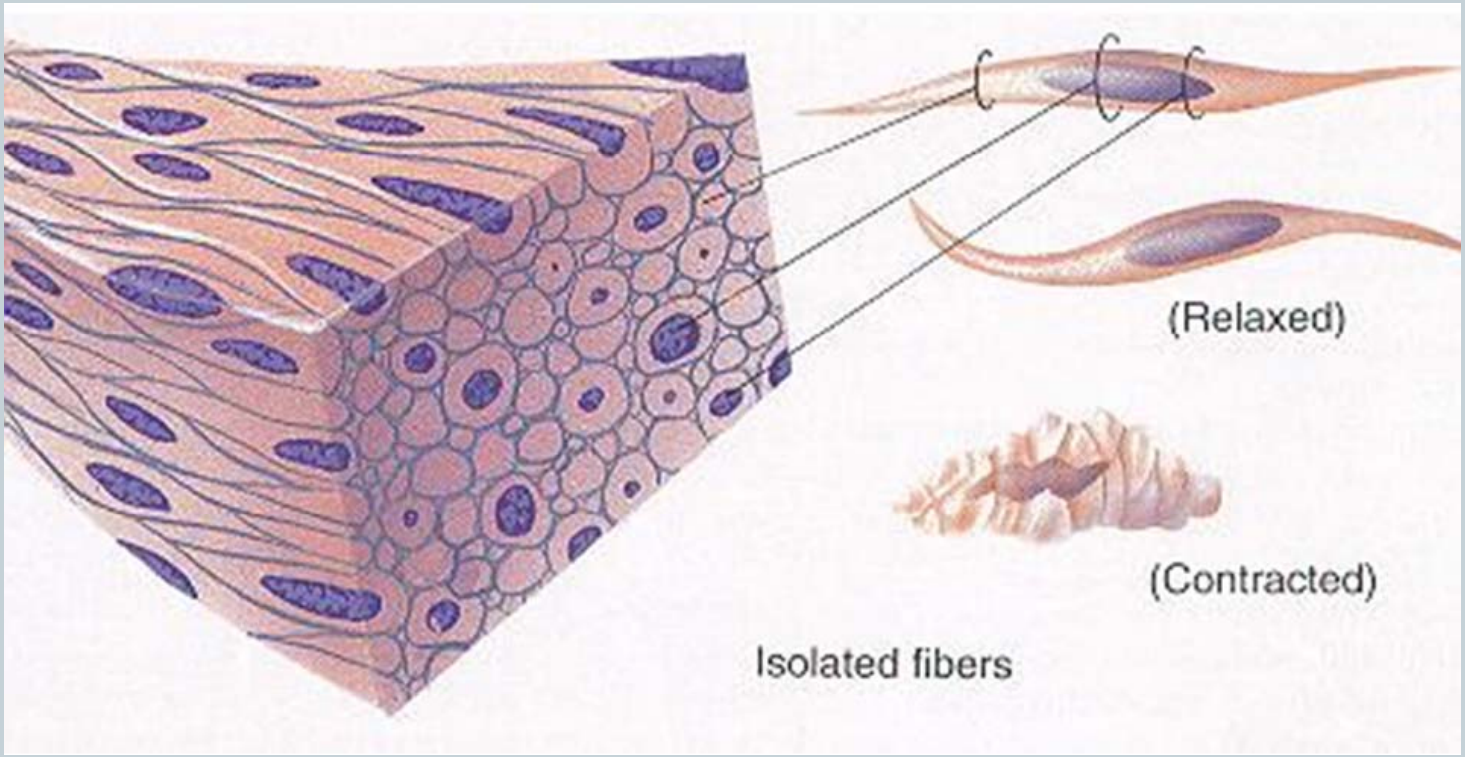
# Cardiac muscle

- Cardiac: cross-striations not in register, nucleus central, elongated and branched cells joined by intercalated disks; involuntary, rhythmic and forceful



# Smooth muscle

- Smooth (visceral): fusiform cells, central nucleus, no striations, slow contraction, involuntary

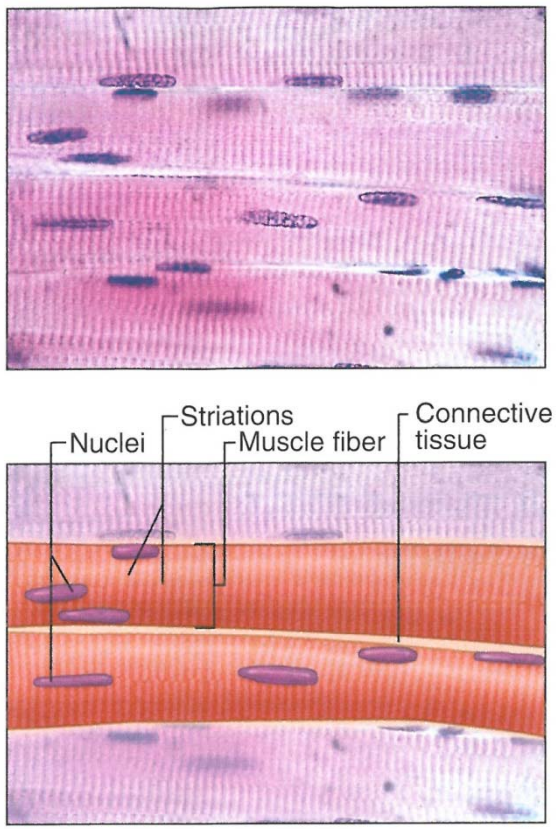


# Skeletal (striated) muscle

- Long & cylindrical multinucleated
- 10-100 µ

1. Mesenchymal myoblasts
2. Multinucleated myotubes
3. Striated muscular fibers

Satellite cells (progenitor)



**a** Skeletal muscle

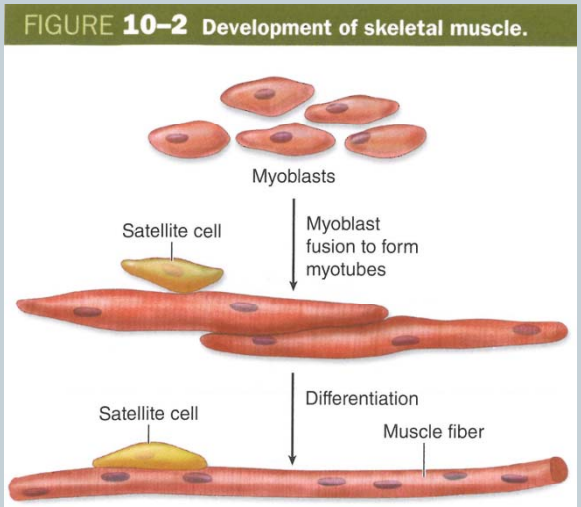


FIGURE **10–2** **Development of skeletal muscle.**

Skeletal muscle begins to differentiate when mesenchymal cells, called **myoblasts**, align and fuse together to make longer, multinucleated tubes called **myotubes**. Myotubes synthesize the proteins to make up myofilaments and gradually begin to show cross-striations by light microscopy. Myotubes continue differentiating to form functional myofilaments, and the nuclei are displaced against the sarcolemma.

Part of the myoblast population does not fuse and differentiate but remains as a group of mesenchymal cells called muscle **satellite cells** located on the external surface of muscle fibers inside the developing external lamina. Satellite cells proliferate and produce new muscle fibers following muscle injury.

# Skeletal muscle organization

- Thin layer of connective tissue

1. Epimysium (dens connective T.)
2. Perimysium (fascicle)
3. Endomysium (reticular fibers & fibroblasts)

Collagen transmit mechanical force

Myotendinous junctions

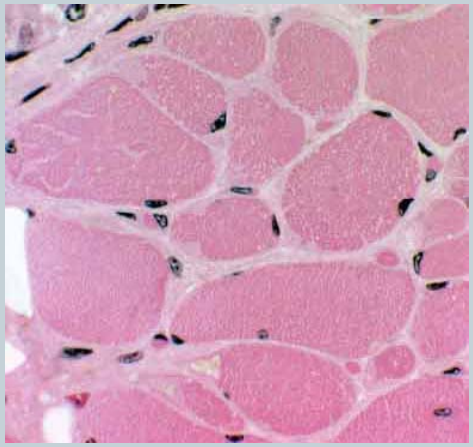
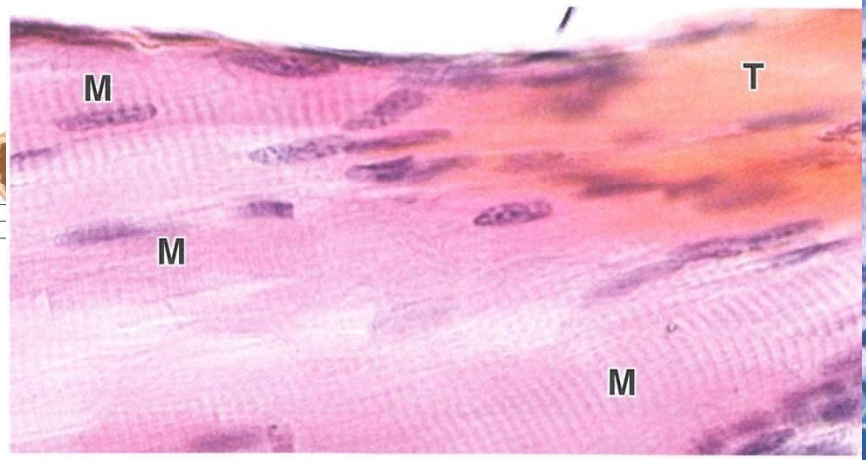


FIGURE 10–5 Capillaries of skeletal muscle.

FIGURE 10–6 Myotendinous junction.



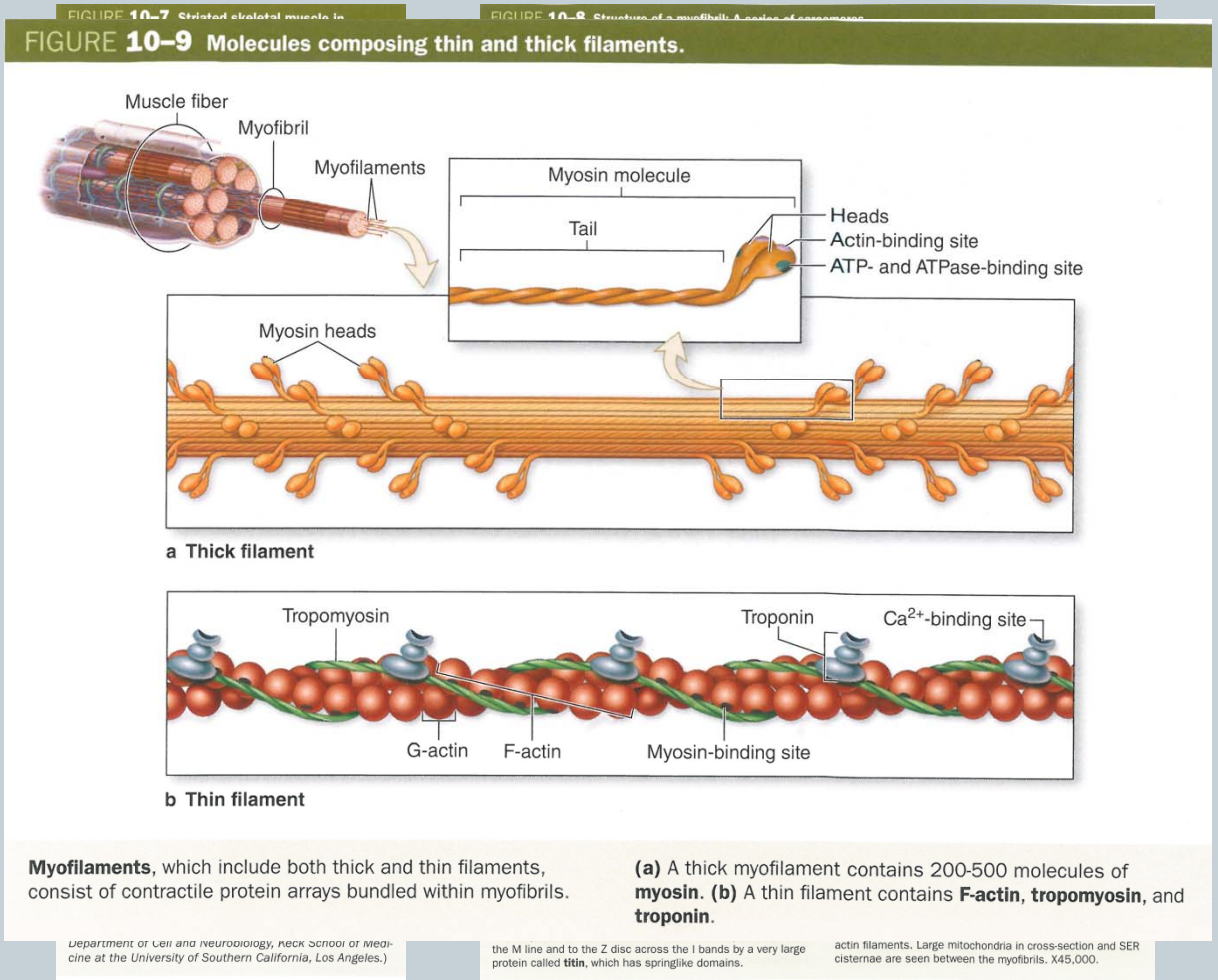

Tendons develop together with skeletal muscles and join muscles to the periosteum of bones. The collagen fibers of a tendon (**T**) are continuous with those in the connective tissue layers around muscle fibers (**M**), forming a strong unit that allows muscle contraction to move the skeleton. X400. H&E.

# Muscular fibers organization

- Striated
- A band
- I band
- Z line
- Sarcomere
- 2.5 µ

- Myofibrils
- myofilaments
- Myosin structure
- Actomyosin ATPase activity

- Actin filament
- α actinin (actin-z line binding)
- Tropomyosin
- Troponin (Tn T, Tn C, Tn I)



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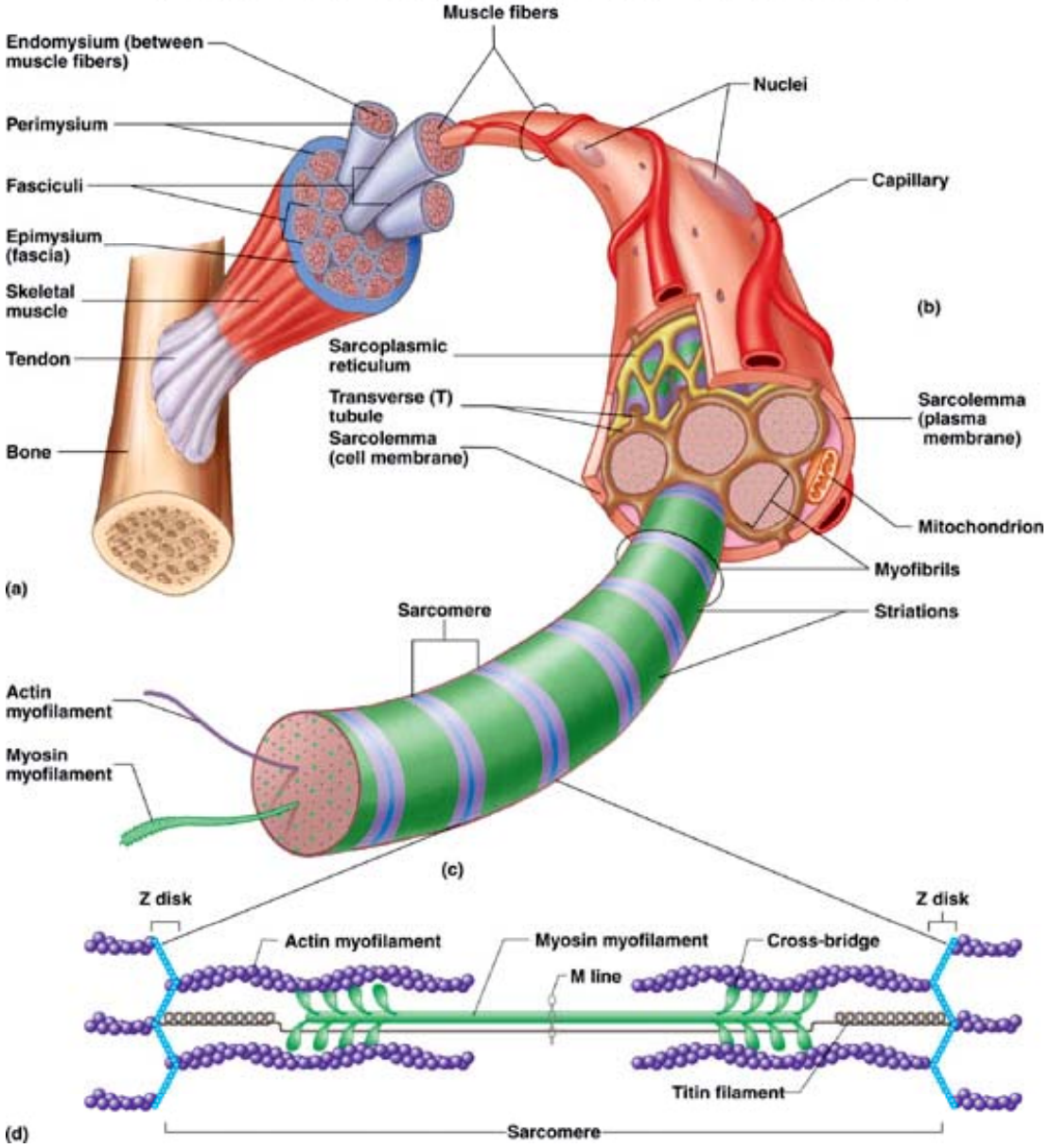


# I band

- Thin filaments
- Actin

1.0 long μ & 8 nm wide

- Tropomyosin

40 nm long coil of 2 poly peptide chains

- troponin

Complex of 3 subunits

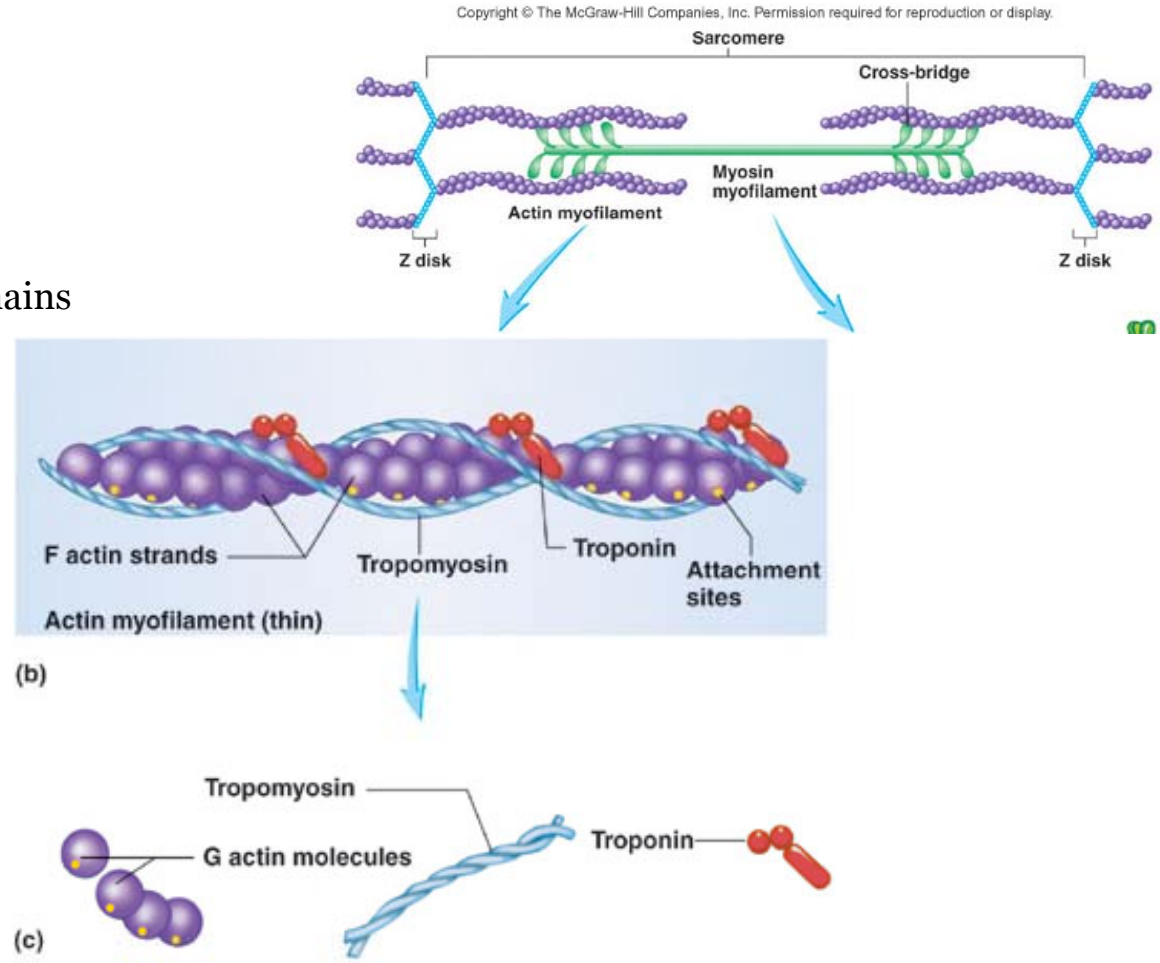
- Titin

Largest pr in body

Bind thick filaments to Z line

- Nebulin

Bind thin myofilament to α actinin



# A band

- Thin & thick filaments

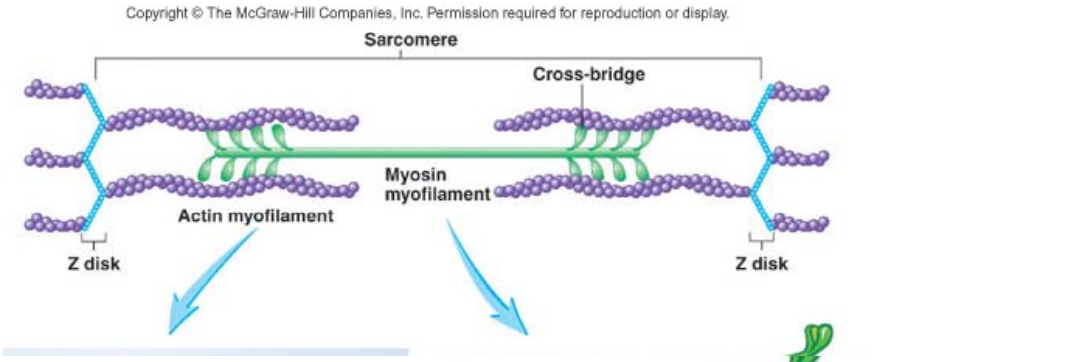
- H zone

Rodlike portion of myosin

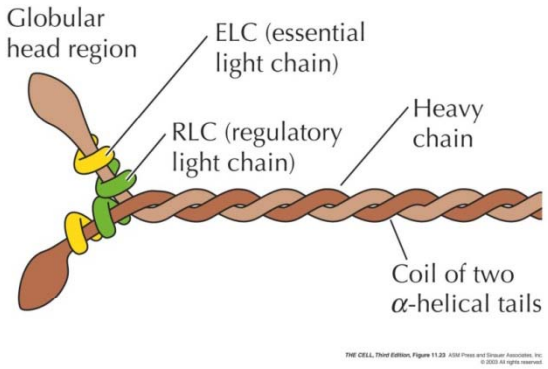
- M line

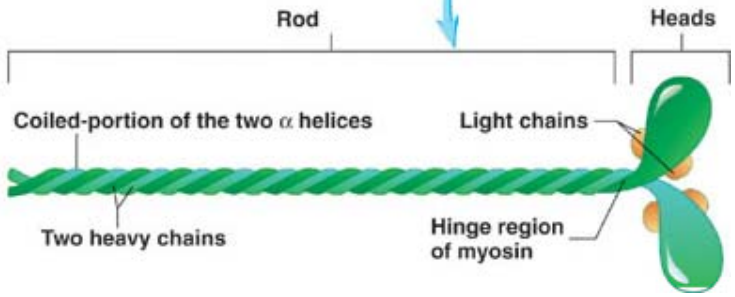
Myomesin

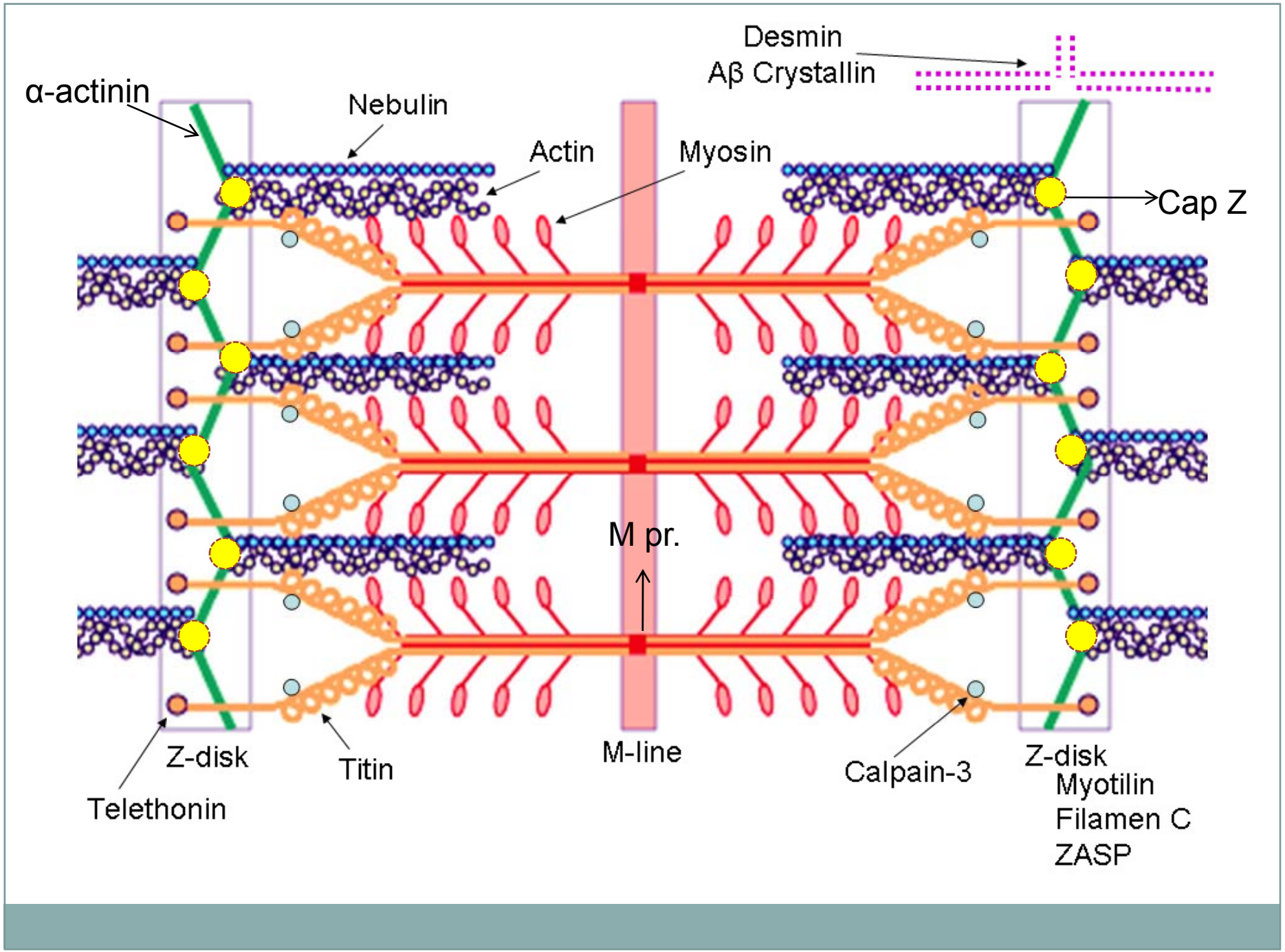
Creatine kinase




- Myosin & Actin more than of 50% of muscle pr.
- Hexagonal structure

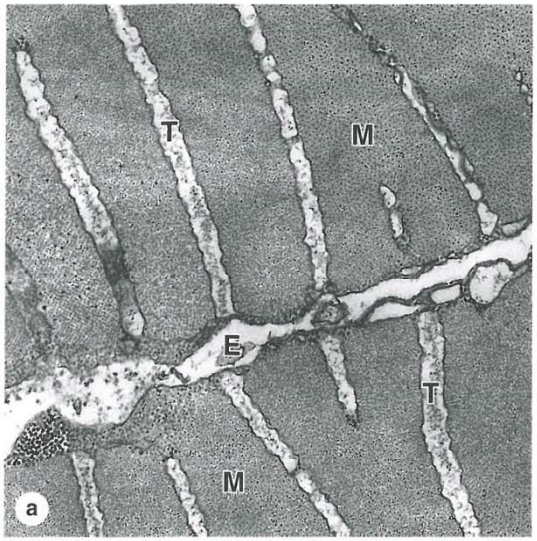


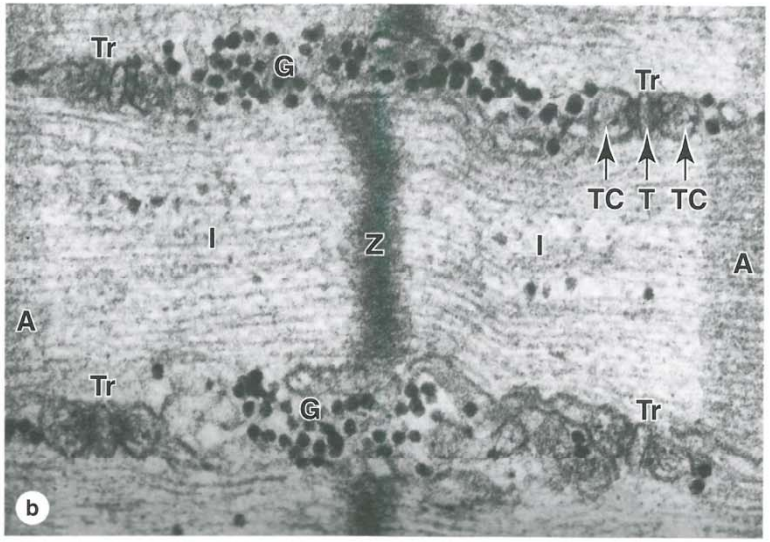




Desmin
Aβ Crystallin
α-actinin
Nebulin
Actin
Myosin
Cap Z
M pr.
Z-disk
Titin
Telethonin
M-line
Calpain-3
Z-disk
Myotilin
Filamen C
ZASP

# Sarcoplasmic reticulum & transverse tubule system

FIGURE **10–10** **Transverse tubule system.**




Transverse tubules are invaginations of the sarcolemma that penetrate deeply into the muscle fiber around all myofibrils.

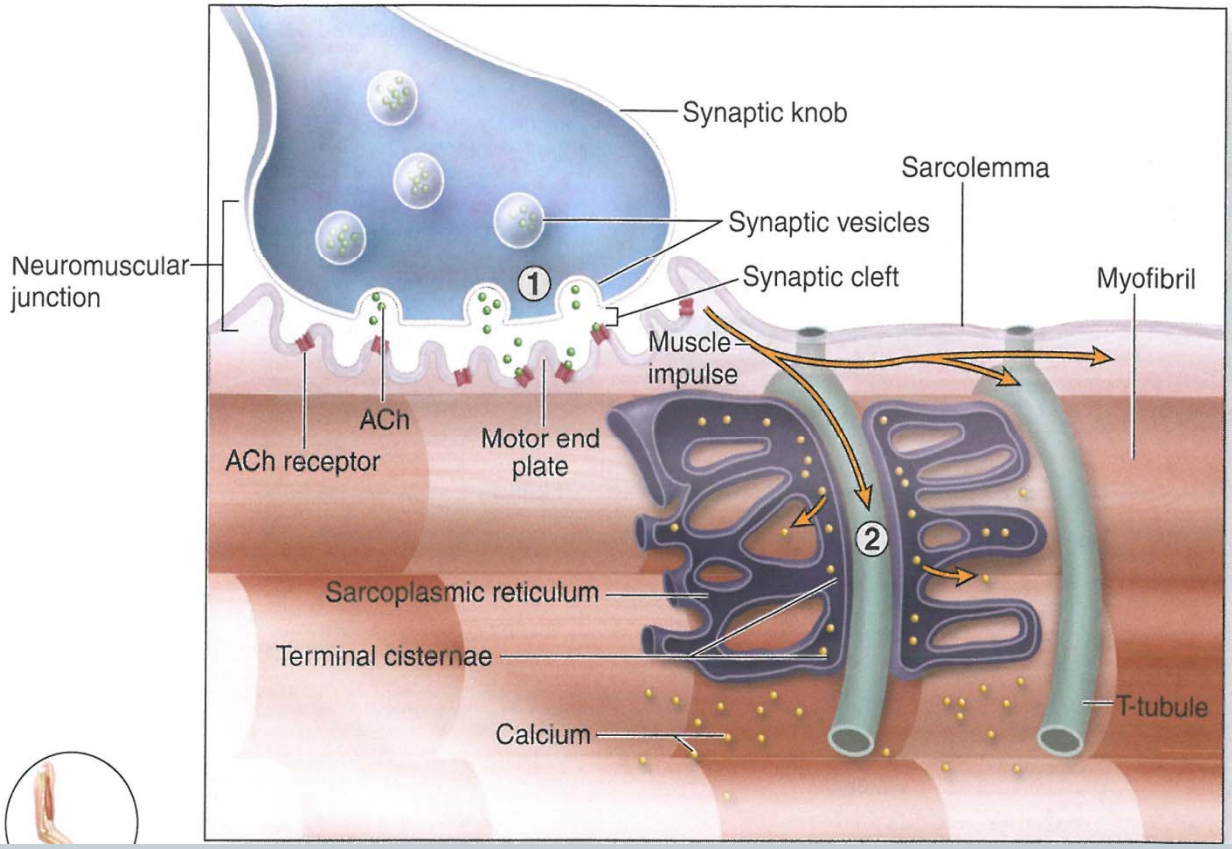
**(a)** TEM cross section of fish muscle shows portions of two fibers and the endomysium (**E**) between them. Several transverse or T tubules (**T**) are shown, perpendicular to the fiber surface, penetrating between myofibrils (**M**). X50,000.

**(b)** Higher-magnification TEM of skeletal muscle in longitudinal section shows four membranous triads (**Tr**) cut transversely near the **A**-band–**I**-band junctions. Each triad consists of a central transverse tubule (**T**) and two adjacent terminal cisterns (**TC**) extending from the sarcoplasmic reticulum. Centrally located is the **Z** disc. Besides elements of the triad, sarcoplasm surrounding the myofibril also contains dense glycogen granules (**G**).

Components of the triad are responsible for the cyclic release of $Ca^{2+}$ from the cisternae and its sequestration again that occurs during muscle contraction and relaxation. The association between SR cisternae and T tubules is shown diagrammatically in Figure 10–11. X90,000.

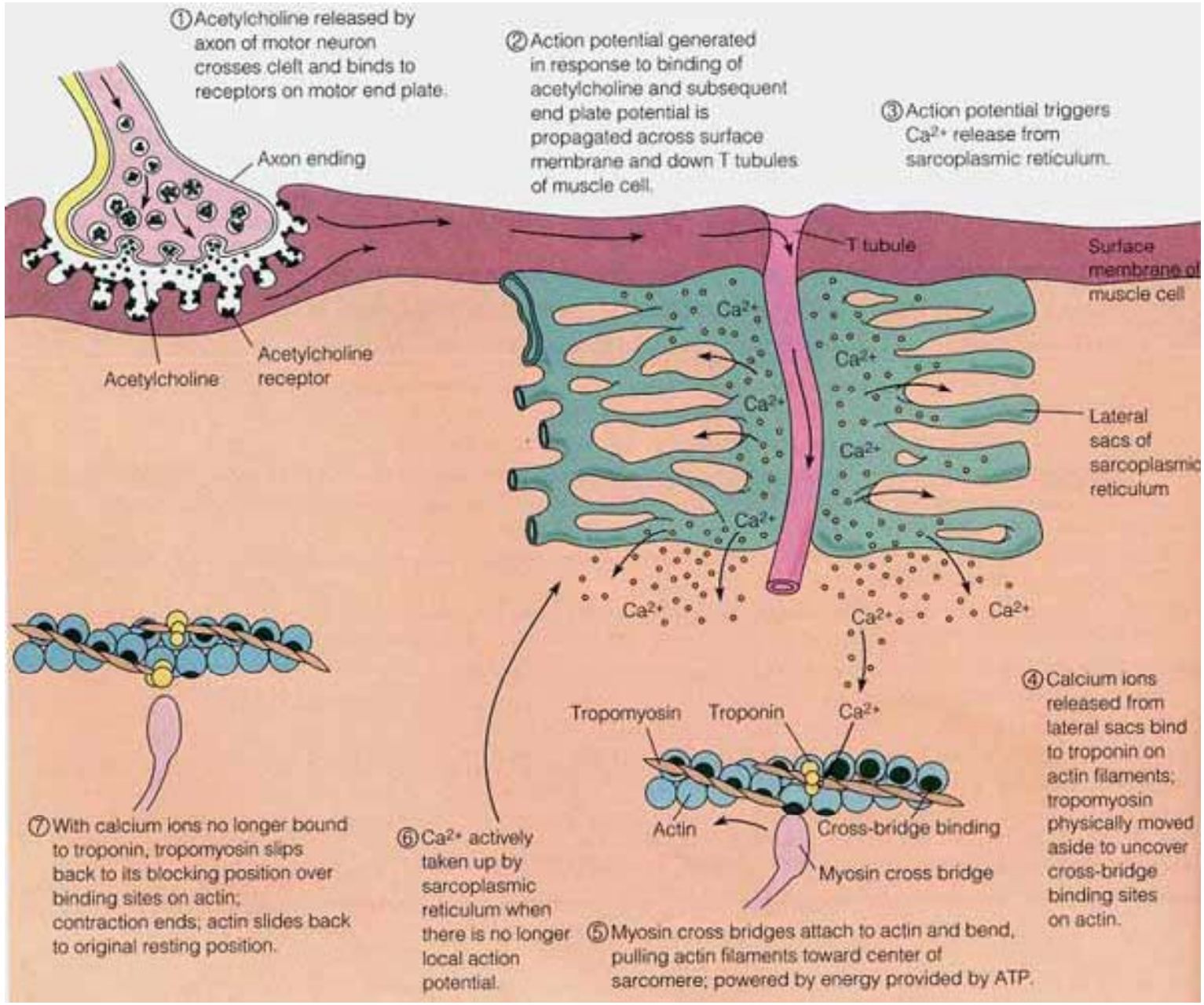
# Sarcoplasmic reticulum & transverse tubule system

FIGURE **10–11** **Events of muscle contraction.**

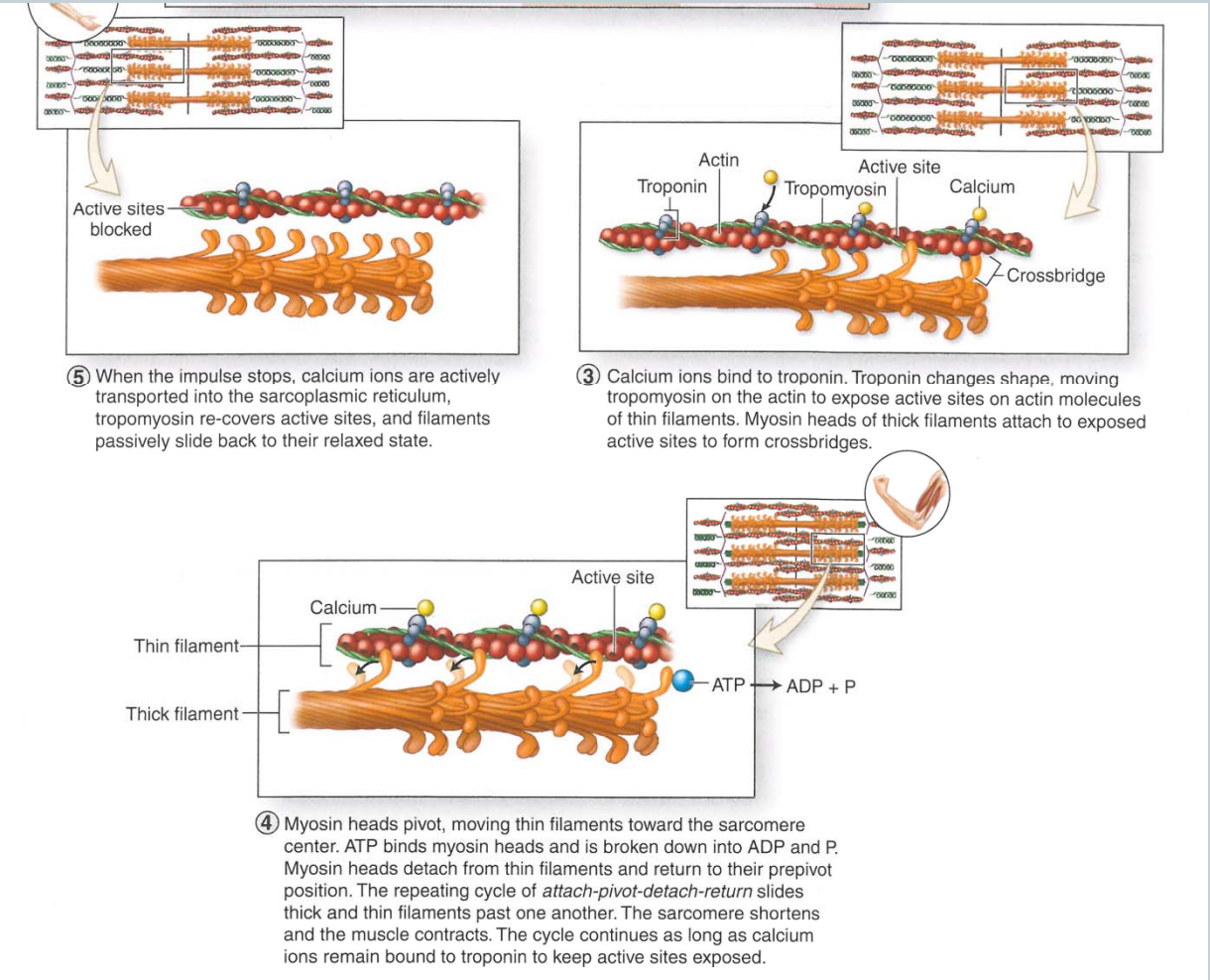



(1) A nerve impulse triggers release of ACh from the synaptic knob into the synaptic cleft. ACh binds to ACh receptors in the motor end plate of the neuromuscular junction, initiating a muscle impulse in the sarcolemma of the muscle fiber.

(2) As the muscle impulse spreads quickly from the sarcolemma along T tubules, calcium ions are released from terminal cisternae into the sarcoplasm.


① Acetylcholine released by axon of motor neuron crosses cleft and binds to receptors on motor end plate.
② Action potential generated in response to binding of acetylcholine and subsequent end plate potential is propagated across surface membrane and down T tubules of muscle cell.
③ Action potential triggers $Ca^{2+}$ release from sarcoplasmic reticulum.
Axon ending
Acetylcholine
Acetylcholine receptor
T tubule
Surface membrane of muscle cell
$Ca^{2+}$
Lateral sacs of sarcoplasmic reticulum
Tropomyosin
Troponin
Actin
Cross-bridge binding
Myosin cross bridge
④ Calcium ions released from lateral sacs bind to troponin on actin filaments; tropomyosin physically moved aside to uncover cross-bridge binding sites on actin.
⑤ Myosin cross bridges attach to actin and bend, pulling actin filaments toward center of sarcomere; powered by energy provided by ATP.
⑥ $Ca^{2+}$ actively taken up by sarcoplasmic reticulum when there is no longer local action potential.
⑦ With calcium ions no longer bound to troponin, tropomyosin slips back to its blocking position over binding sites on actin; contraction ends; actin slides back to original resting position.

# Contraction mechanism

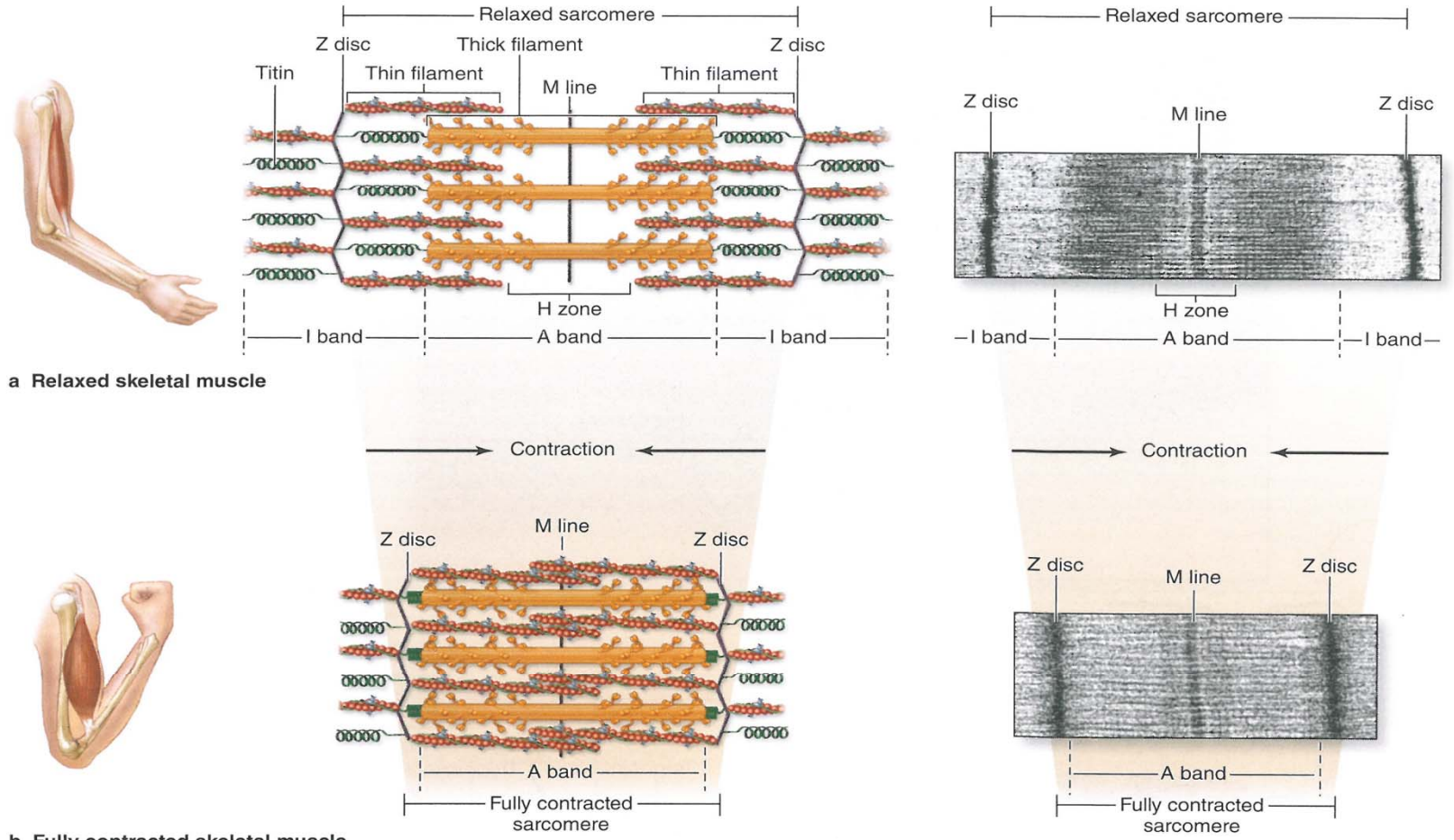



⑤ When the impulse stops, calcium ions are actively transported into the sarcoplasmic reticulum, tropomyosin re-covers active sites, and filaments passively slide back to their relaxed state.

③ Calcium ions bind to troponin. Troponin changes shape, moving tropomyosin on the actin to expose active sites on actin molecules of thin filaments. Myosin heads of thick filaments attach to exposed active sites to form crossbridges.

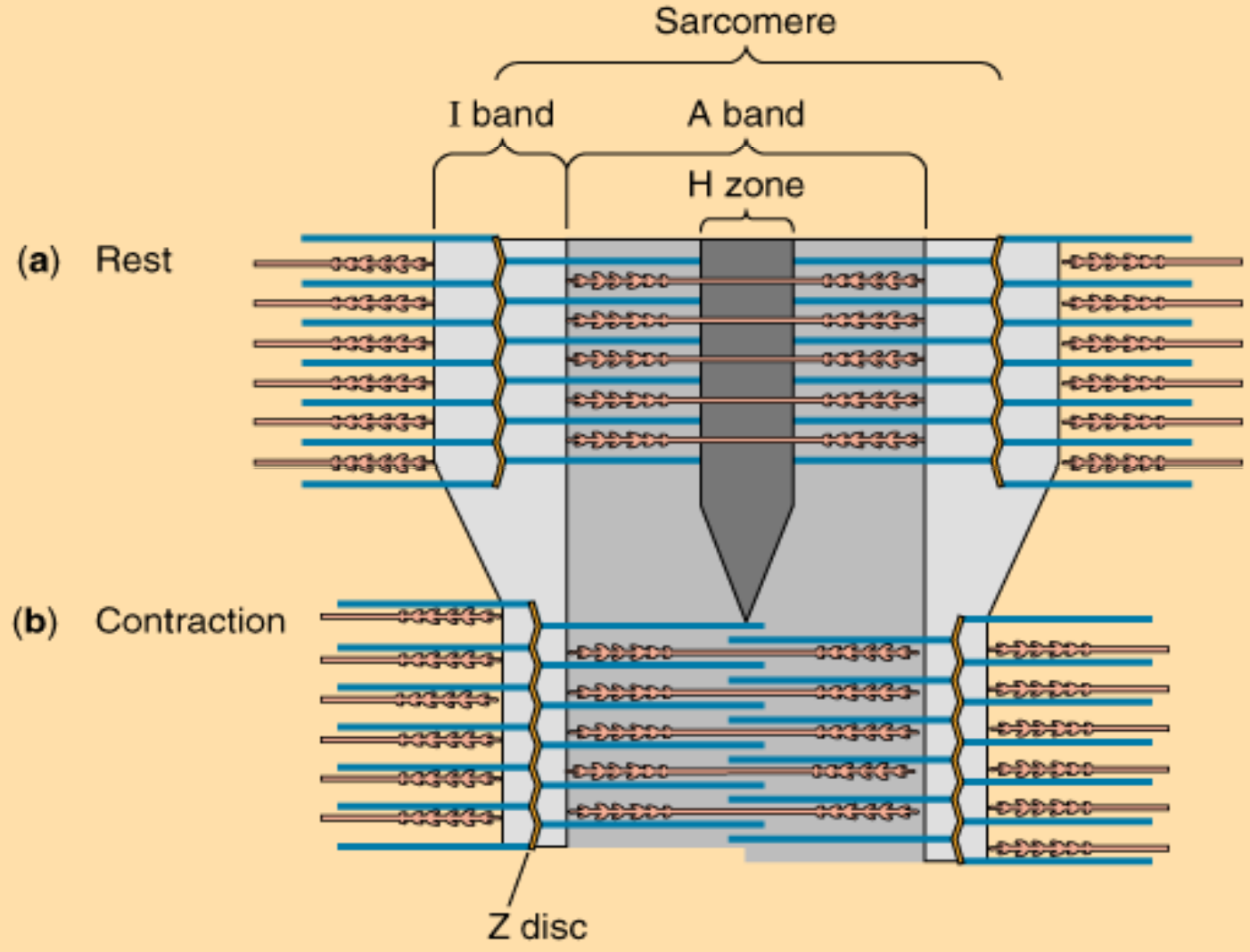
④ Myosin heads pivot, moving thin filaments toward the sarcomere center. ATP binds myosin heads and is broken down into ADP and P. Myosin heads detach from thin filaments and return to their prepivot position. The repeating cycle of *attach-pivot-detach-return* slides thick and thin filaments past one another. The sarcomere shortens and the muscle contracts. The cycle continues as long as calcium ions remain bound to troponin to keep active sites exposed.

## FIGURE 10–12 Sliding filaments and sarcomere shortening in contraction.

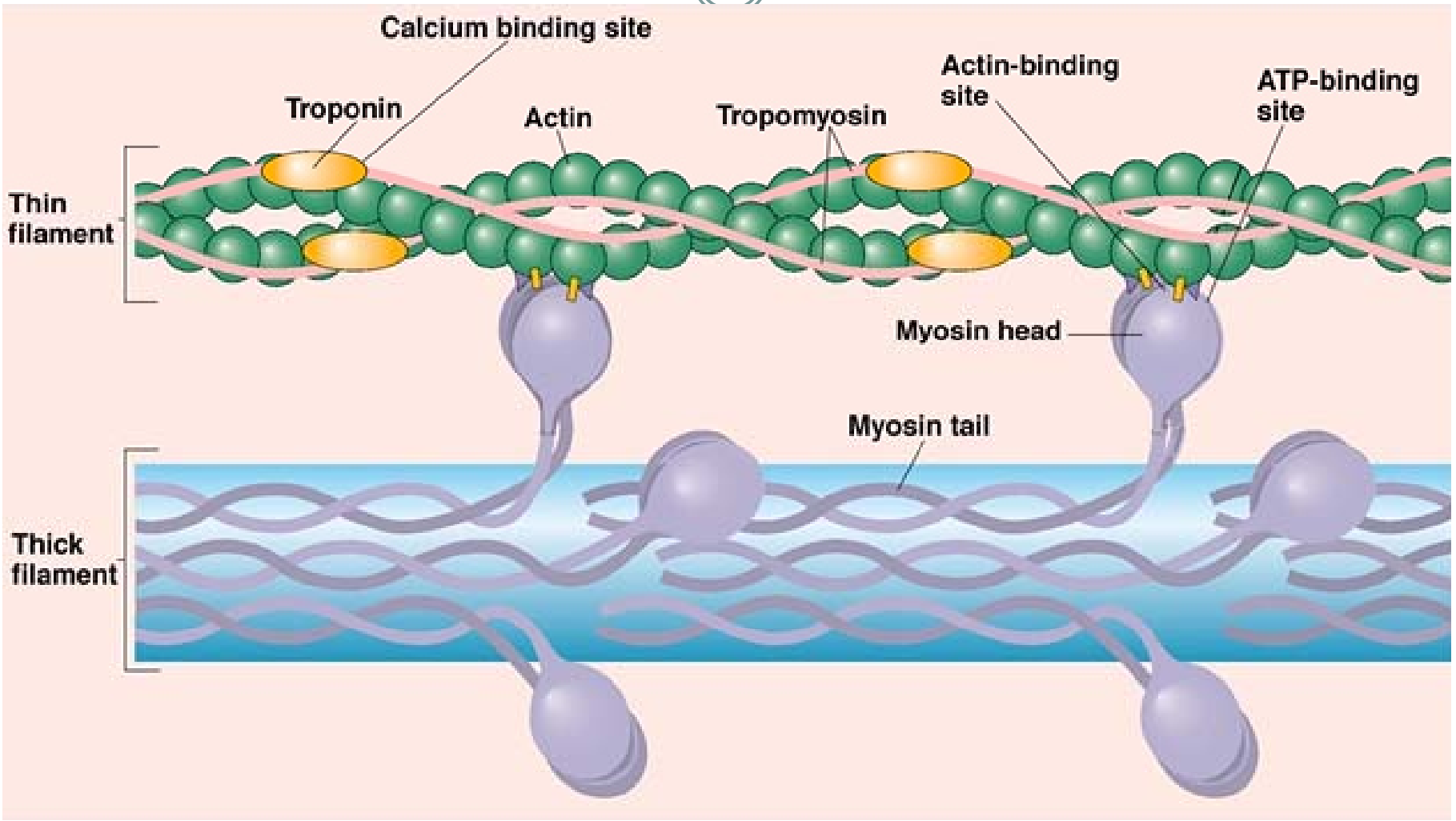



Diagrams and TEM micrographs show sarcomere shortening during skeletal muscle contraction. **(a)** In the relaxed state the sarcomere, I band, and H zone are at their expanded length. The springlike action of titin molecules, which span the I band, helps pull thin and thick filaments past one another in relaxed muscle. **(b)** During muscle contraction, the Z discs at the sarcomere boundaries are drawn closer together as they move toward the ends of thick filaments in the A band. Titin molecules are compressed during contraction.

► **Changes in a Sarcomere During Contraction**



# Cross-Bridge Formation in Muscle Contraction



# Energy for Muscle Contraction

- **ATP is required for muscle contraction**
  - Myosin ATPase breaks down ATP as fiber contracts

- **Sources of ATP**
  - Phosphocreatine (PC)
  - Glycolysis
  - Oxidative phosphorylation

# Innervation

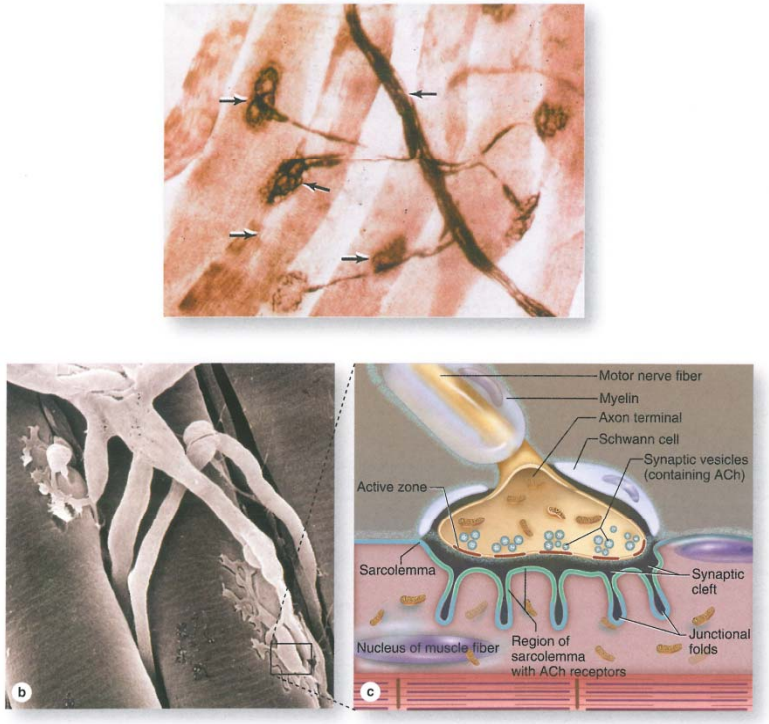
- Myelinated motor nerve
- In perimysium
- Unmyelinated branch to endomysium
- Schwann cell
- Motor end plate (MEP)
- Acetylcholine
- Synaptic cleft
- Junctional folds

- Motor unit

**MEDICAL APPLICATION**

**Myasthenia gravis** is an autoimmune disorder that involves circulating antibodies against proteins of acetylcholine receptors. Antibody binding to the antigenic sites interferes with acetylcholine activation of their receptors, leading to intermittent periods of skeletal muscle weakness. As the body attempts to correct the condition, junctional folds of sarcolemma with affected receptors are internalized, digested by lysosomes, and replaced by newly formed receptors. These receptors, however, are again made unresponsive to acetylcholine by similar antibodies, and the disease follows a progressive course. The extraocular muscles of the eyes are commonly the first affected.

FIGURE **10–13** The neuromuscular junction (NMJ).




Before it terminates in a skeletal muscle, each motor axon bundled in the nerve forms many branches, each of which forms a synapse with a muscle fiber.

**(a)** Silver staining can reveal the nerve bundle (**NB**), the terminal axonal twigs, and the motor end plates (**MEP**, also called neuromuscular junctions or NMJ) on striated muscle fibers (**S**). X1200.

**(b)** An SEM shows the branching ends of a motor axon, each covered by an extension of the last Schwann cell and expanded terminally as an MEP embedded in a groove in the external lamina of the muscle fiber.

**(c)** Diagram of enclosed portion of the SEM indicating key features of a typical MEP: synaptic vesicles of acetylcholine (**ACh**), a synaptic cleft, and a postsynaptic membrane. This membrane, the sarcolemma, is highly folded to increase the number of ACh receptors at the MEP. Receptor binding initiates muscle fiber depolarization, which is carried to the deeper myofibrils by the T tubules.