Lesson 3

- Muscle Stimulation

- stimulation & contraction
- nerve stimulus & action potential
- transmission of impulse to muscle
- sliding filament theory

Stimulation and Contraction of Single Skeletal Muscle Cells

- Excitability (also called responsiveness or irritability)—ability to receive and respond to a stimulus
- Contractility—ability to shorten when an adequate stimulus is received
- Extensibility—ability of muscle cells to be stretched
- Elasticity—ability to recoil and resume resting length after stretching

The Nerve Stimulus and Action Potential

- Skeletal muscles must be stimulated by a **motor neuron** (nerve cell) to contract
- Motor unit— one motor neuron and all the **skeletal muscle** cells stimulated by that neuron

The Nerve Stimulus and Action Potential

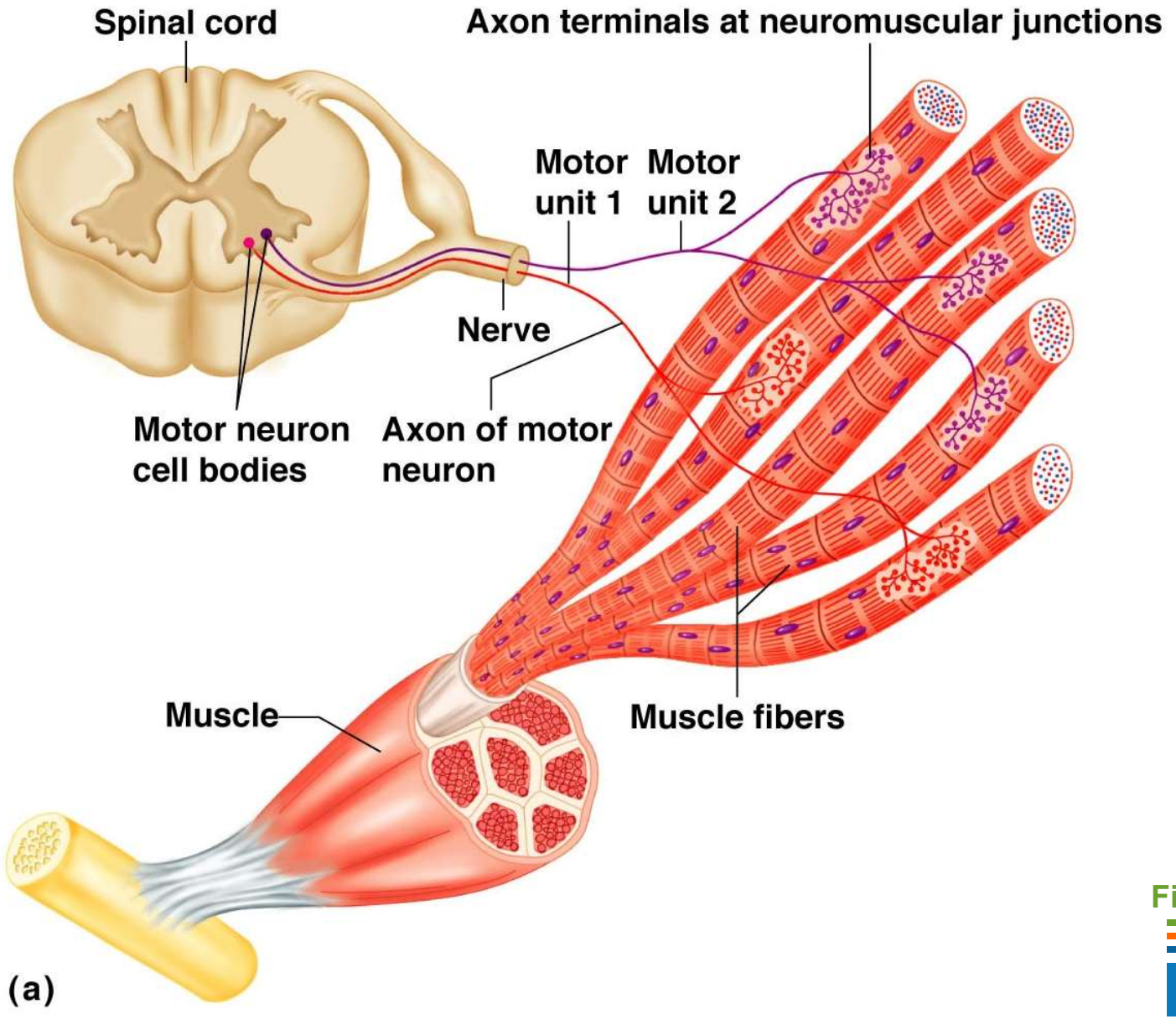


Figure 6.4a

The Nerve Stimulus and Action Potential

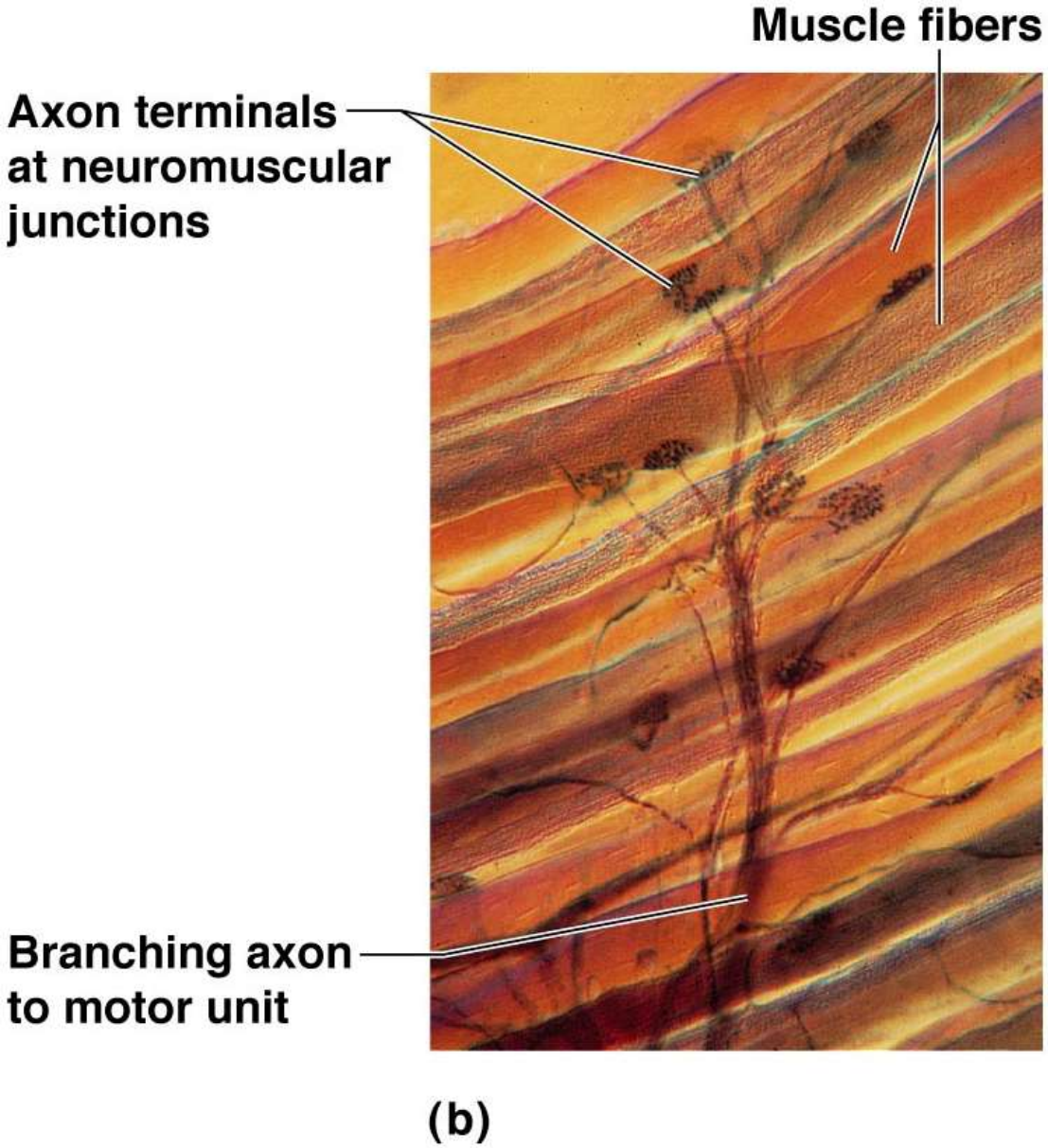


Figure 6.4b

The Nerve Stimulus and Action Potential

- Neuromuscular junction
  - Association site of axon terminal of the motor neuron and muscle
- Synaptic cleft
  - Gap between nerve and muscle
  - Nerve and muscle do not make contact
  - Area between nerve and muscle is filled with interstitial fluid

The Nerve Stimulus and Action Potential

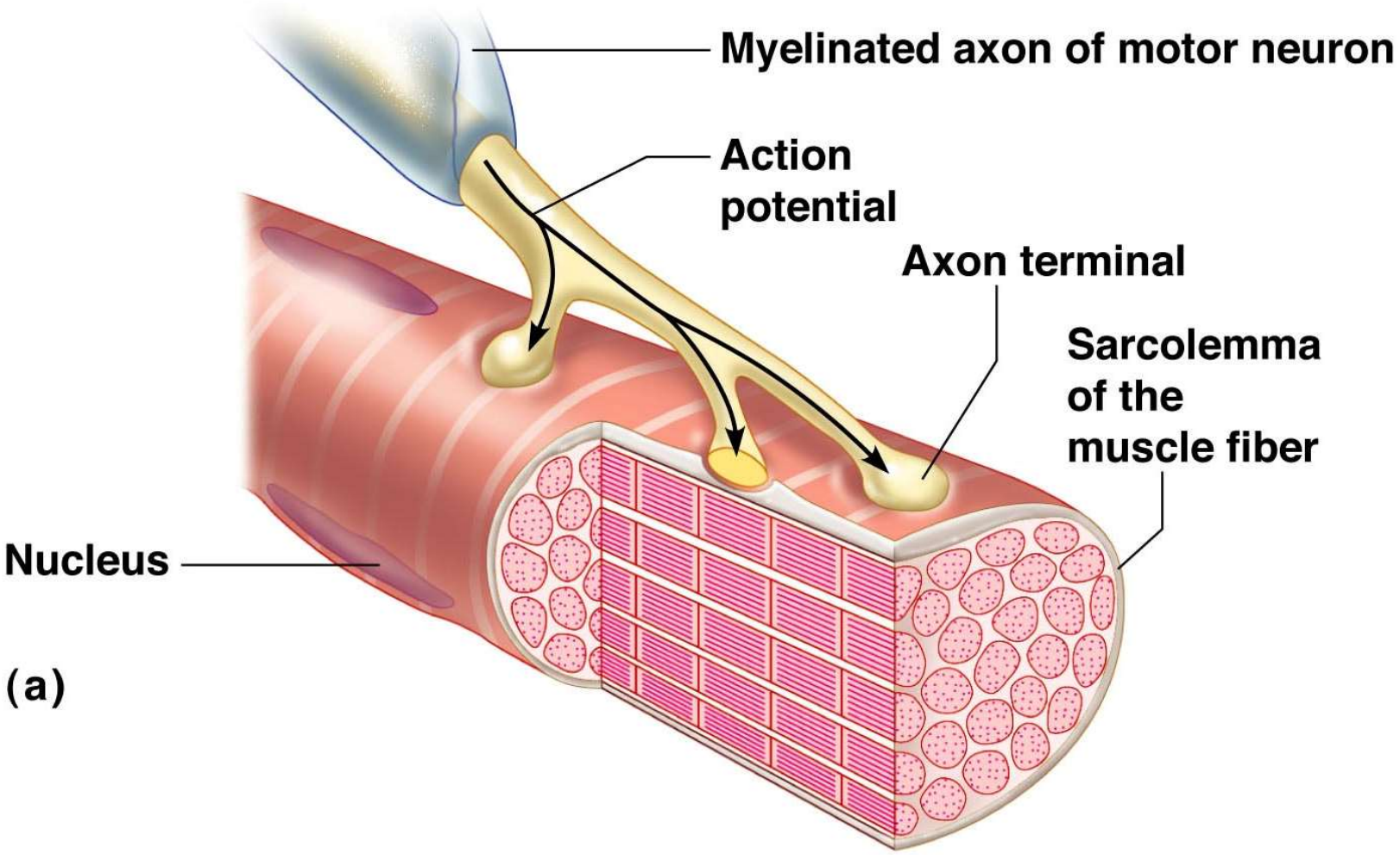
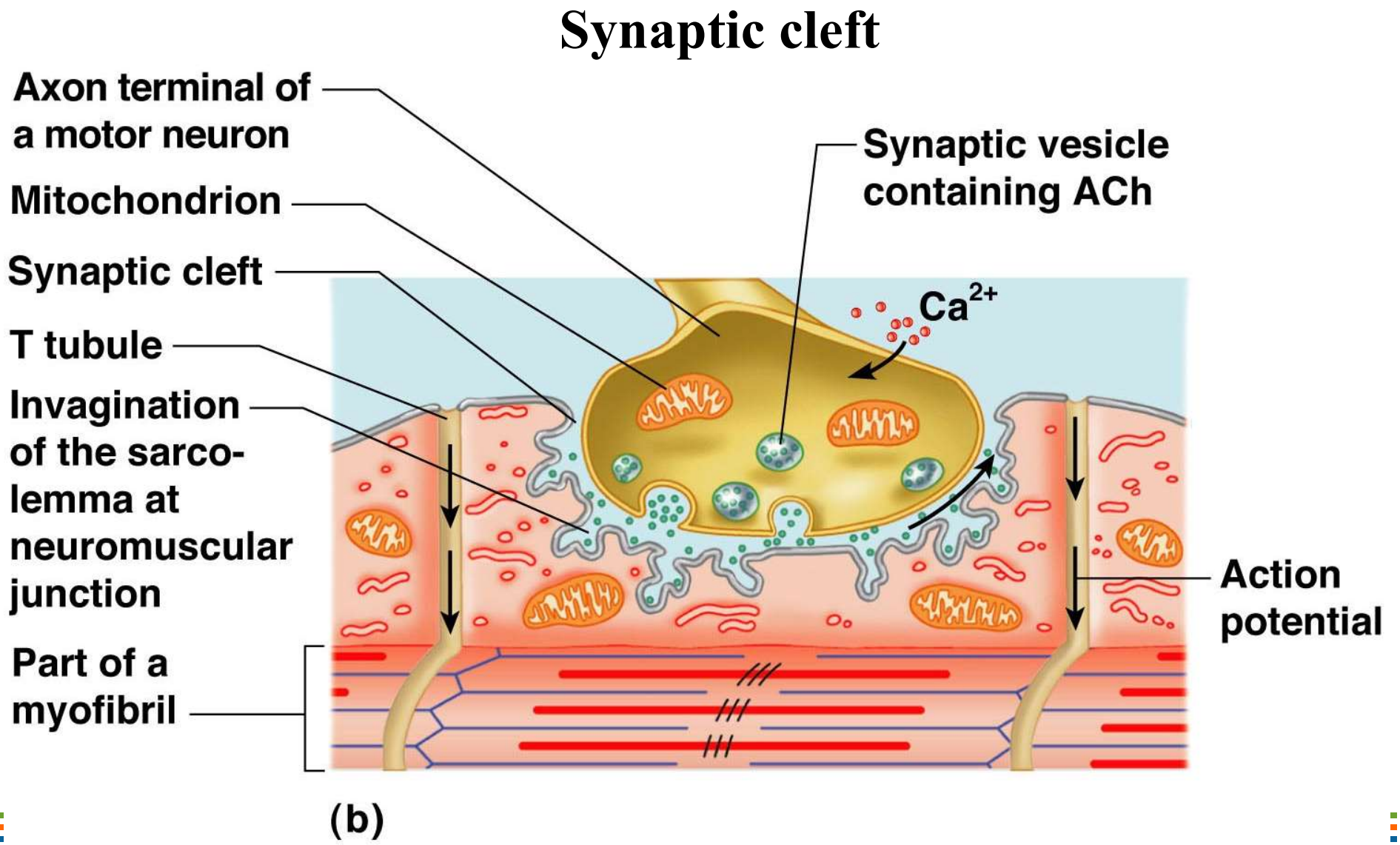


Figure 6.5a

The Nerve Stimulus and Action Potential

Figure 6.5b



Transmission of Nerve Impulse to Muscle

- Neurotransmitter—chemical released by nerve upon arrival of nerve impulse
  - Carries the impulse across the synaptic cleft
  - The neurotransmitter for skeletal muscle is acetylcholine (ACh)
- Acetylcholine attaches to receptors on the sarcolemma of the muscle cells
- Sarcolemma becomes permeable to sodium (Na+)

Transmission of Nerve Impulse to Muscle

- Sodium rushes into the cell generating an **action potential**
- Once started, muscle contraction cannot be stopped

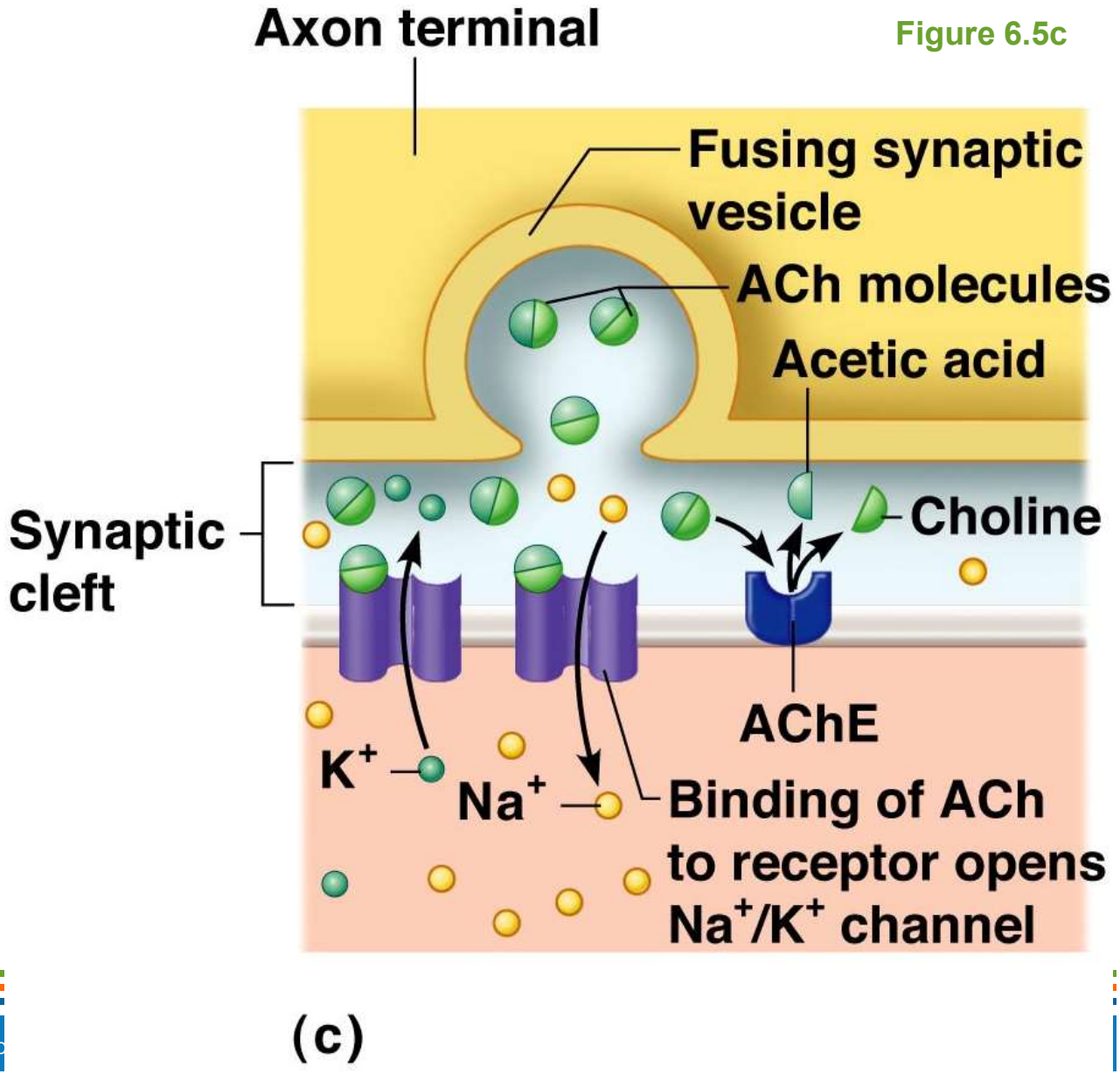


Figure 6.5c

Transmission of Nerve Impulse to Muscle

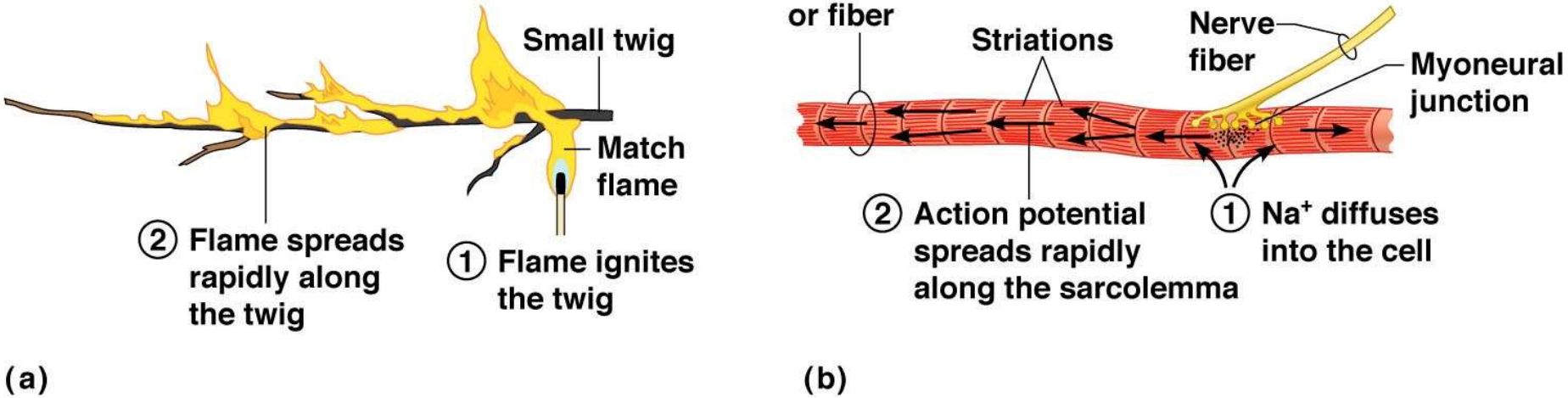


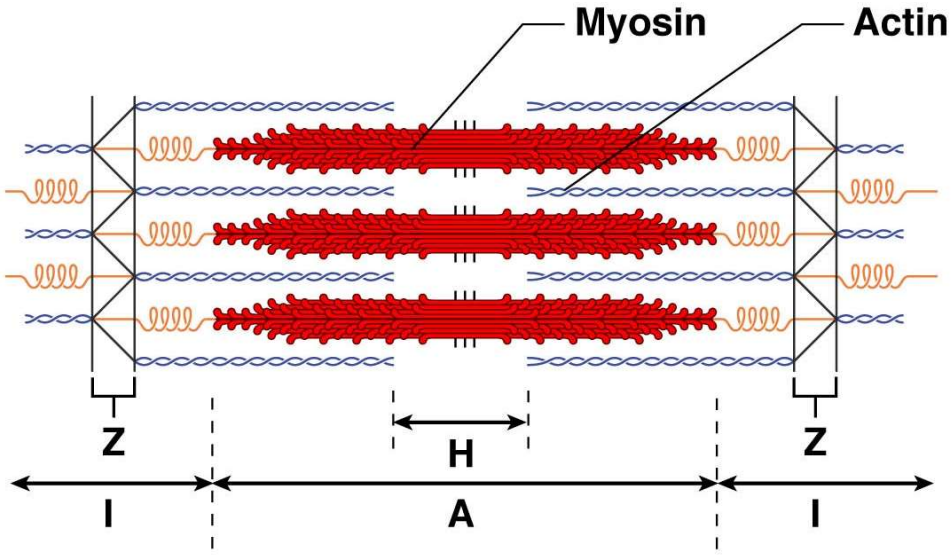
Figure 6.6

The Sliding Filament Theory of Muscle Contraction

- Activation by nerve causes myosin heads (cross bridges) to attach to binding sites on the thin filament; requires energy in form of ATP
- Myosin heads then pull thin filaments toward the center of the sarcomere
- This continued action causes a sliding of the actin past the myosin
- The result is that the muscle is shortened (contracted)

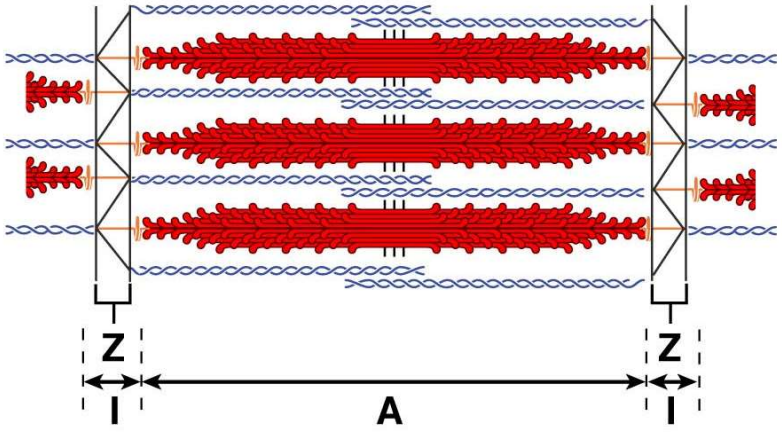
The Sliding Filament Theory of Muscle Contraction

Song - Muscule anatomy & contraction (itsy bitsy spider)



(a)

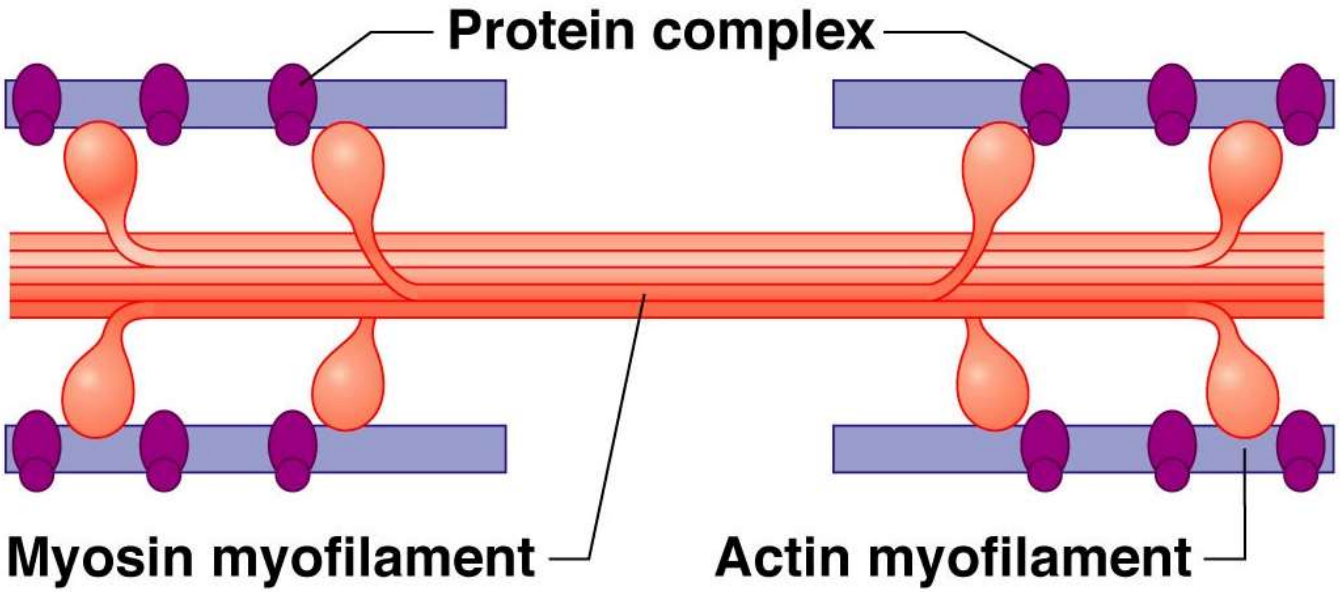
Video: Muscle Contraction Animation



(b)

Figure 6.7a–b

The Sliding Filament Theory

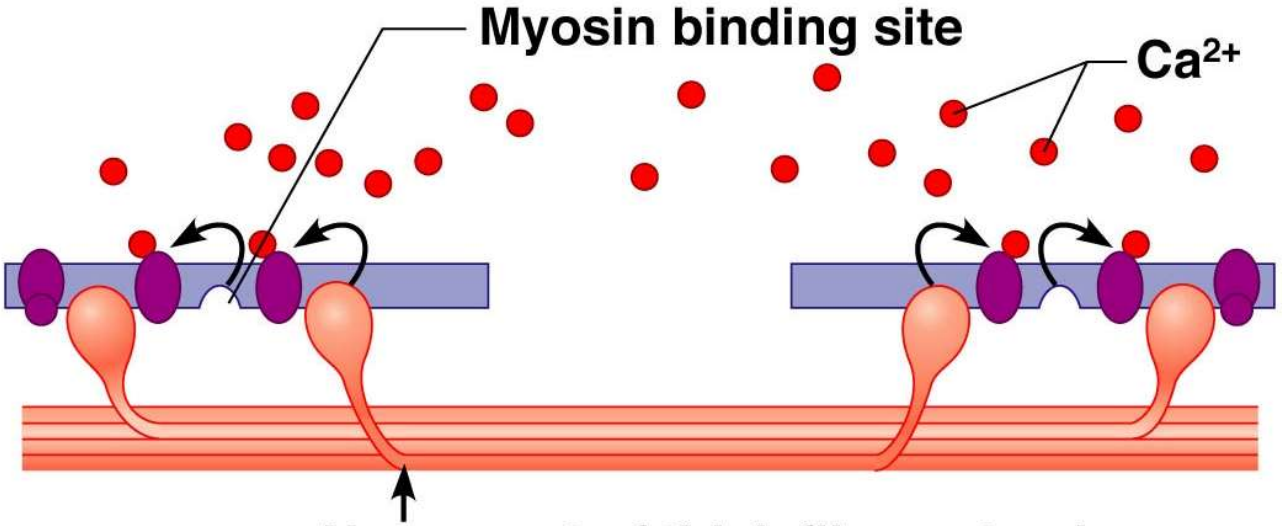


(a)

In a relaxed muscle cell, the regulatory proteins forming part of the actin myofilaments prevent myosin binding (see a). When an action potential sweeps along its sarcolemma and a muscle cell is excited, calcium ions (Ca^{2+}) are released from intracellular storage areas (the sacs of the sarcoplasmic reticulum).

Figure 6.8a

The Sliding Filament Theory



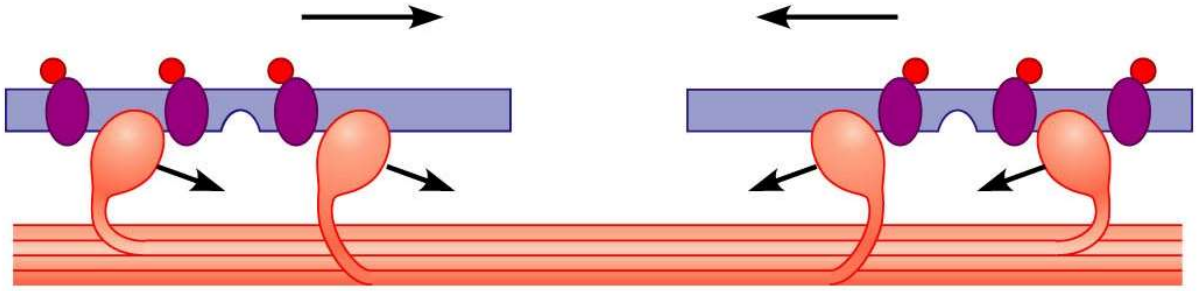
(b)

The flood of calcium acts as the final trigger for contraction, because as calcium binds to the regulatory proteins on the actin filaments, they change both their shape and their position on the thin filaments. This action exposes myosin binding sites on the actin, to which the myosin heads can attach (see b), and the myosin heads immediately begin seeking out binding sites.

Figure 6.8b

The Sliding Filament Theory

Figure 6.8c



(c)

The free myosin heads are "cocked," much like a set mousetrap. The physical attachment of myosin to actin "springs the trap," causing the myosin heads to snap (pivot) toward the center of the sarcomere. Because actin and myosin are still firmly bound to each other when this happens, the thin filaments are slightly pulled toward the center of the sarcomere (see c). ATP provides the energy needed to release and recock each myosin head so that it is ready to take another "step" and attach to a binding site farther along the thin filament. When the action potential ends and calcium ions are reabsorbed into the SR storage areas, the regulatory proteins resume their original shape and position, and again block myosin binding to the thin filaments. Since myosin now has nothing to attach to, the muscle cell relaxes and settles back to its original length.