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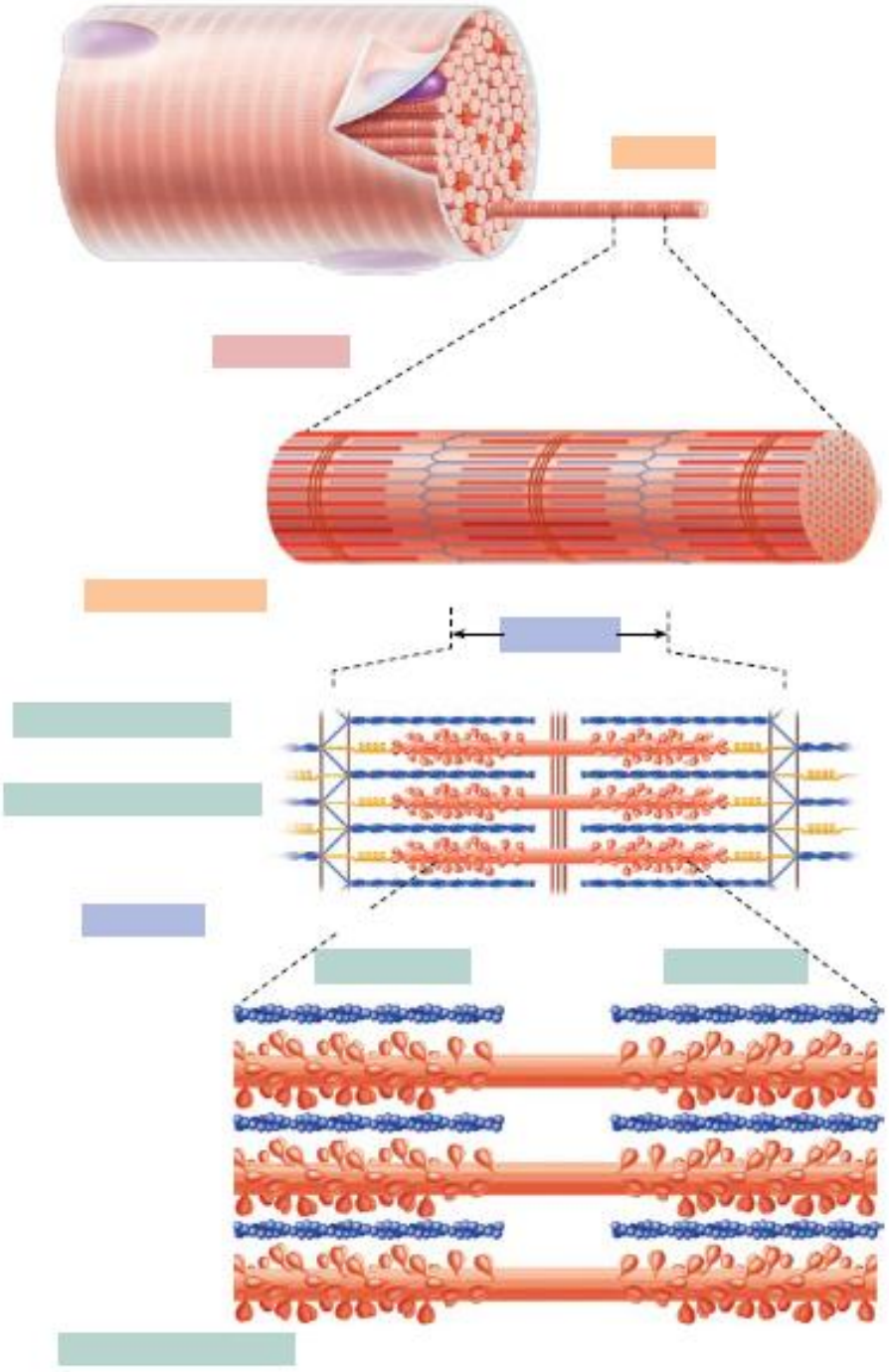

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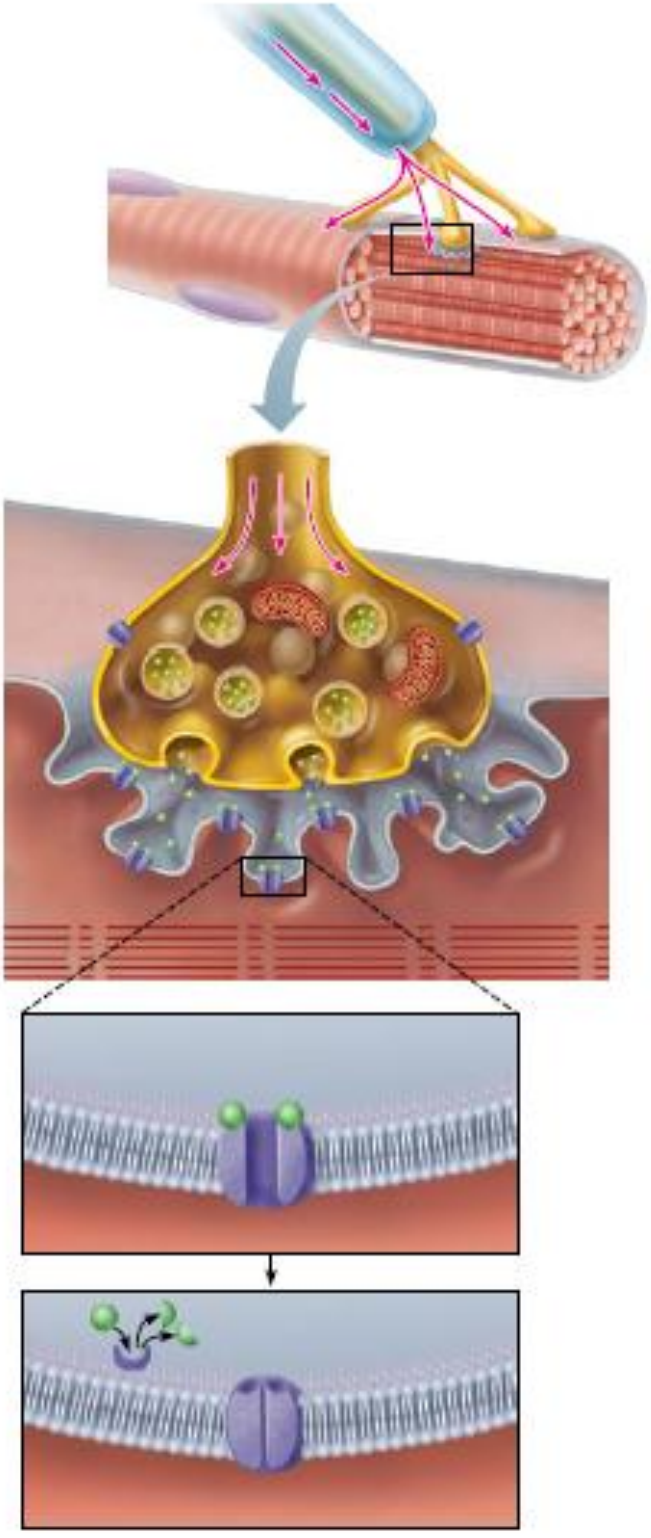

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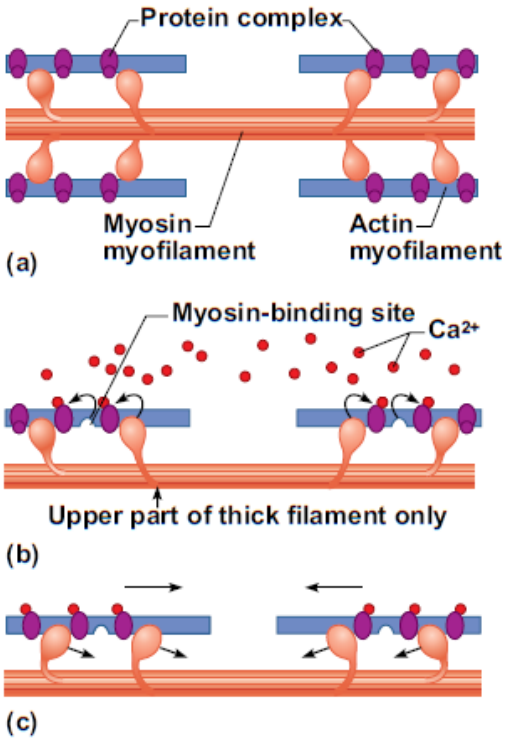
Figure 6.8 Schematic representation of contraction mechanism: The sliding filament theory.



In a relaxed muscle cell, the regulatory proteins forming part of the actin myofilaments prevent myosin binding (see a). When an action potential (AP) 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).

The flood of calcium acts as the final trigger for contraction, because as calcium binds to the regulatory proteins on the actin filaments, the proteins undergo a change in 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.

The free myosin heads are "cocked," much like a set mousetrap. Myosin attachment to actin "springs the trap," causing the myosin heads to snap (pivot) toward the center of the sarcomere. 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 attach to a binding site farther along the thin filament. When the AP ends and calcium ions are returned to SR storage areas, the regulatory proteins resume their original shape and position, and again block myosin binding to the thin filaments. As a result, the muscle cell relaxes and settles back to its original length.

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