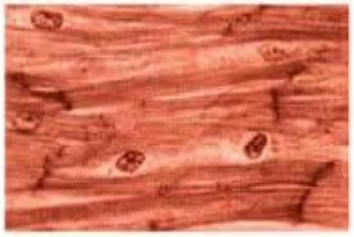
PHYSIOLOGY OF MUSCLE CONTRACTION

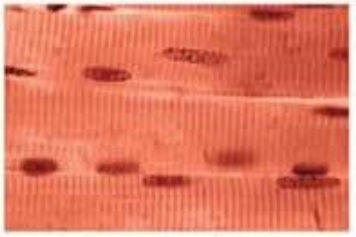
Prof. Sultan Ayoub Meo
MBBS, M.Phil, Ph.D (Pak), M Med Ed (Dundee), FRCP (London), FRCP (Dublin), FRCP (Glasgow), FRCP (Edinburgh)
Professor and Consultant, Department of Physiology,
College of Medicine, King Saud University, Riyadh, KSA

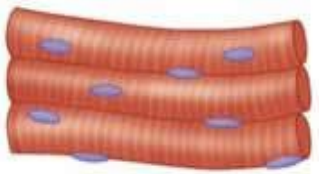
TYPES OF MUSCLES

Smooth muscle
- has narrow, tapered rod-shaped cells.
- has nonstriated, uninucleated fibers.
- occurs in walls of internal organs and blood vessels.
- is involuntary.



Cardiac muscle
- has striated, tubular, branched, uninucleated fibers.
- occurs in walls of heart.
- is involuntary.

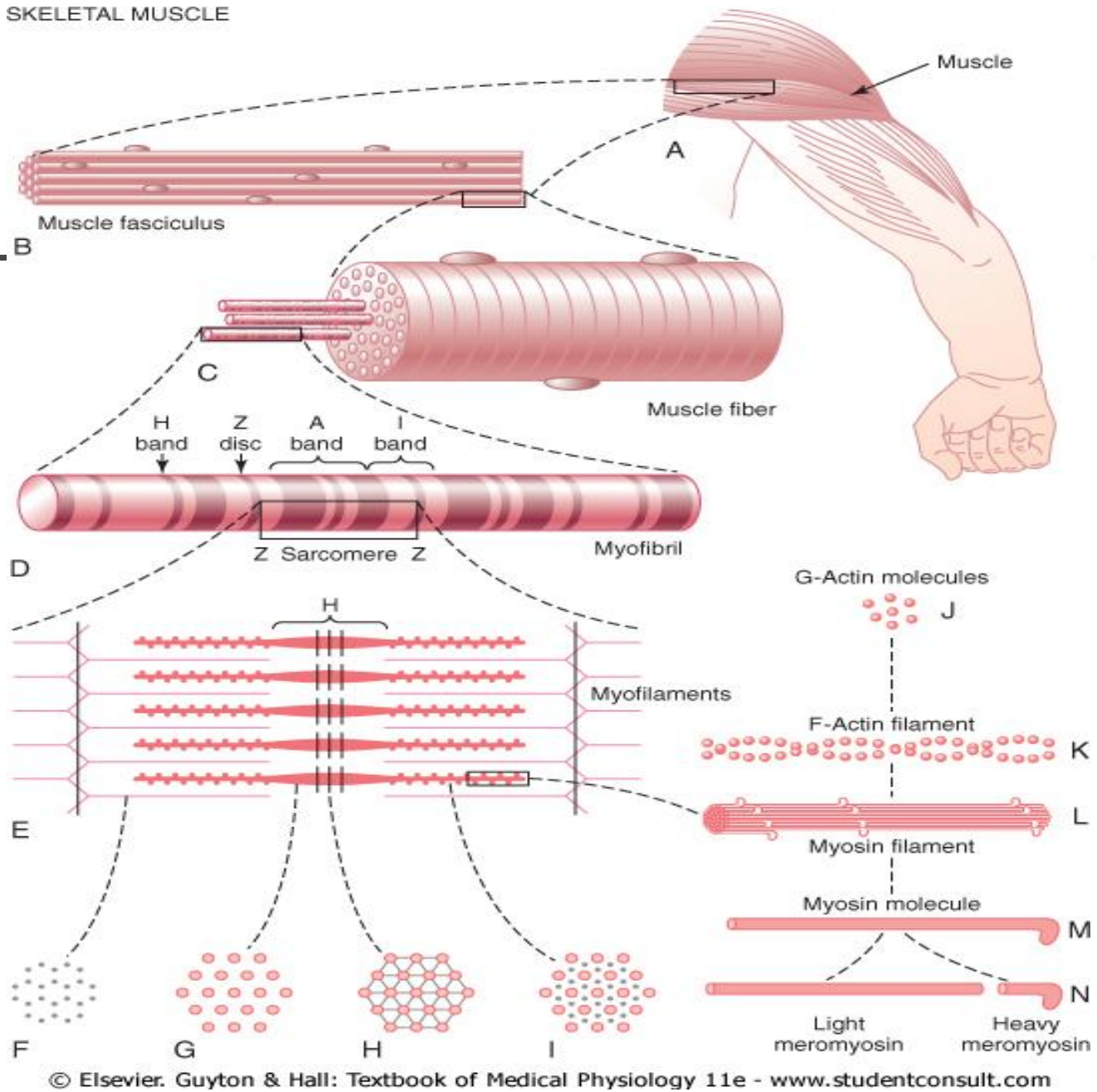




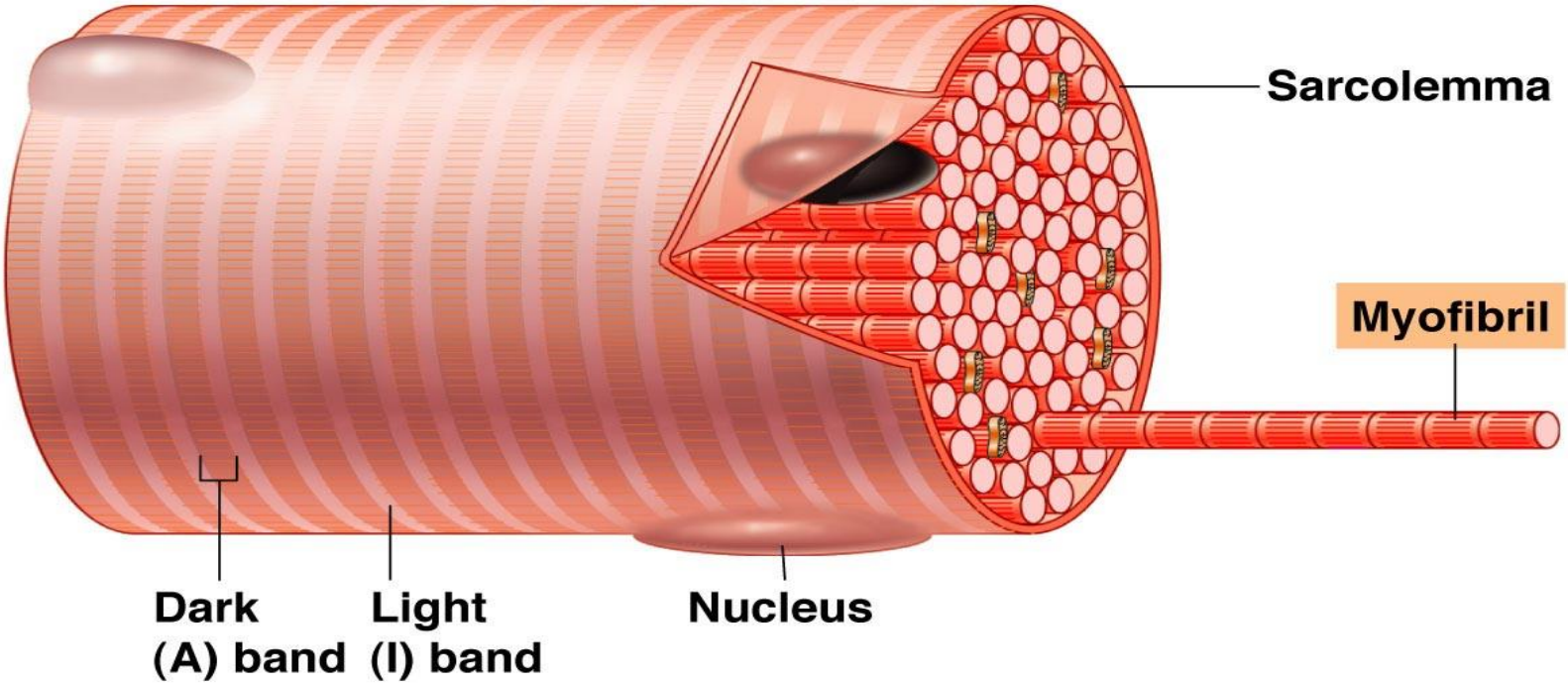
Skeletal muscle
- has striated, tubular, multinucleated fibers.
- is usually attached to skeleton.
- is voluntary.

TYPES OF MUSCLE CONTRACTION

- **Isometric contraction:** Contraction occurs without an appreciable decrease in the length of the whole muscle.
- **Isotonic contraction:** Contraction against a constant load, with approximation of the ends of the muscle.
- **Twitch contraction:** This is a rapid, jerky response to a single threshold or greater stimulus.
- **Treppe contraction:** This a type of contraction in which skeletal muscle contracts more forcefully in response to the same strength of stimulus after it has contracted several times.
- **Tetanus:** This occurs when muscle is stimulated at progressively greater frequencies. At a certain higher frequency, contraction is forced together and cannot be differentiated from one another. This state is known as tetanization.

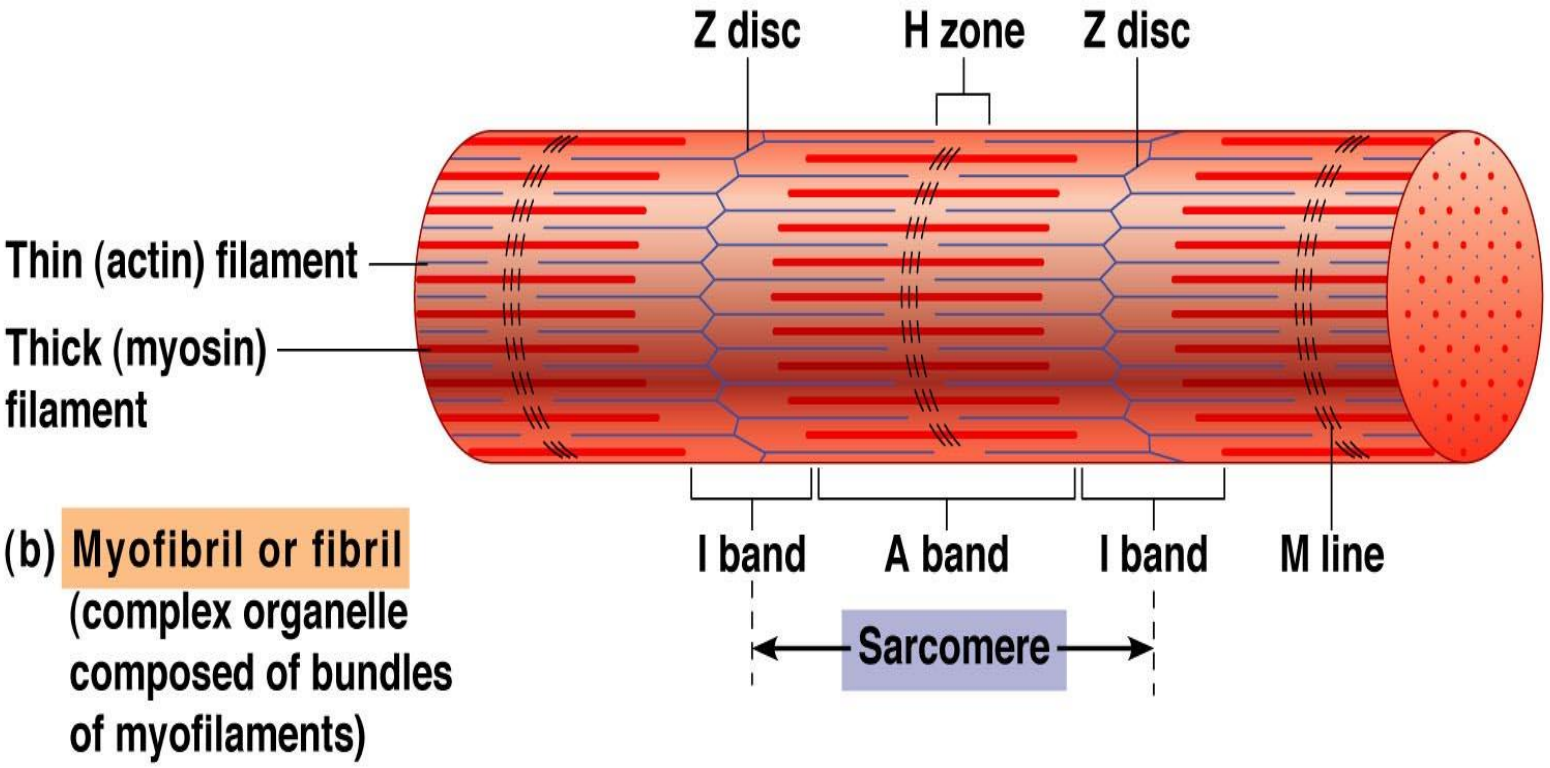

SKELETAL MUSCLE
Muscle
A
Muscle fasciculus
B
C
Muscle fiber
H band
Z disc
A band
I band
Z Sarcomere Z
Myofibril
D
G-Actin molecules
J
H
Myofilaments
F-Actin filament
K
L
Myosin filament
Myosin molecule
M
N
E
Light meromyosin
Heavy meromyosin
F
G
H
I
© Elsevier. Guyton & Hall: Textbook of Medical Physiology 11e - www.studentconsult.com

SKELETAL MUSCLE

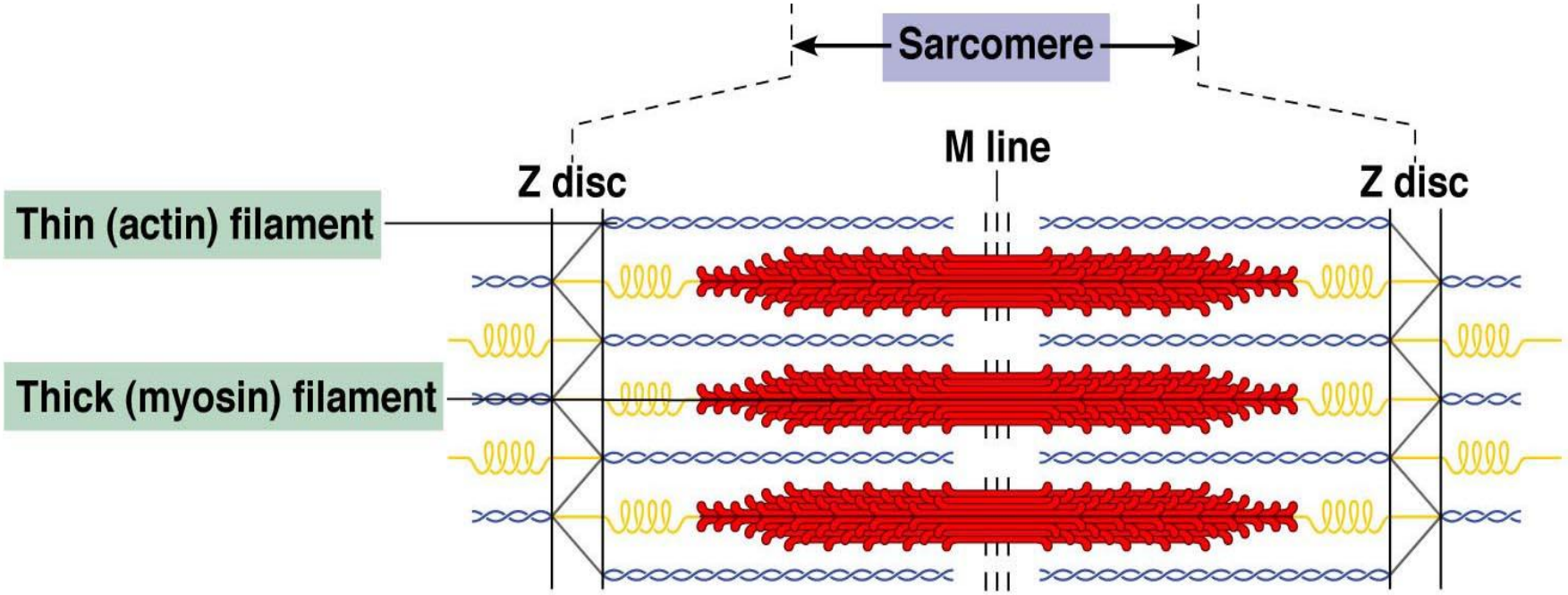


(a) Segment of a muscle fiber (cell)

SKELETAL MUSCLE

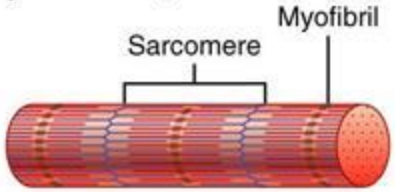
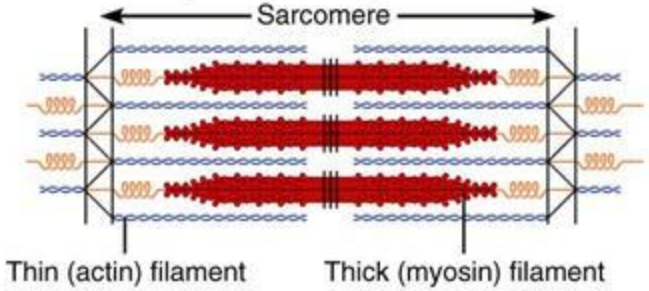
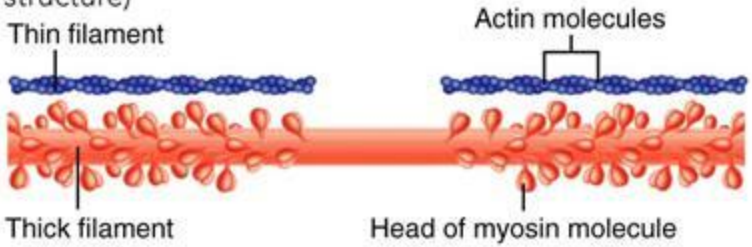


SKELETAL MUSCLE

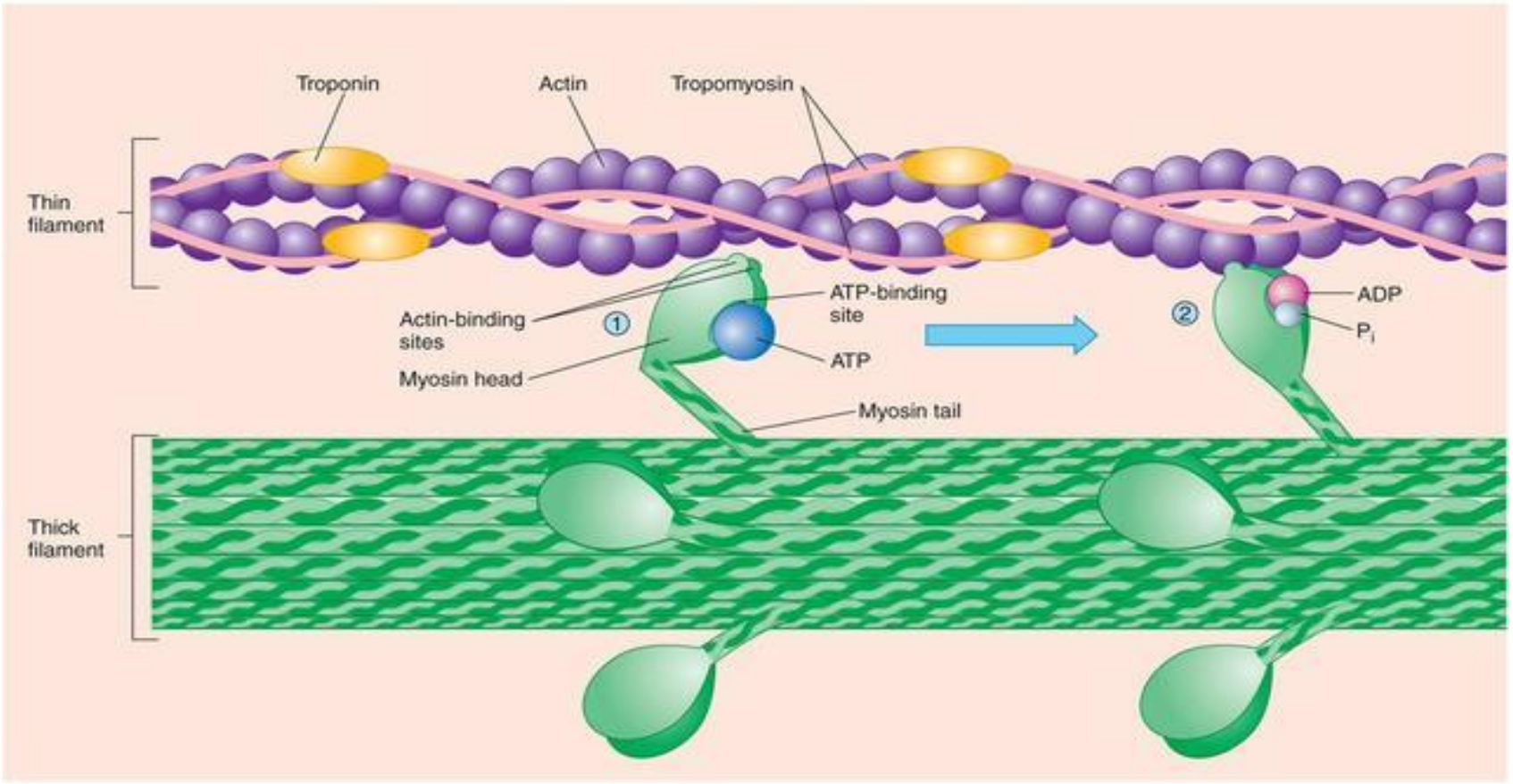


(c) Sarcomere (segment of a myofibril)

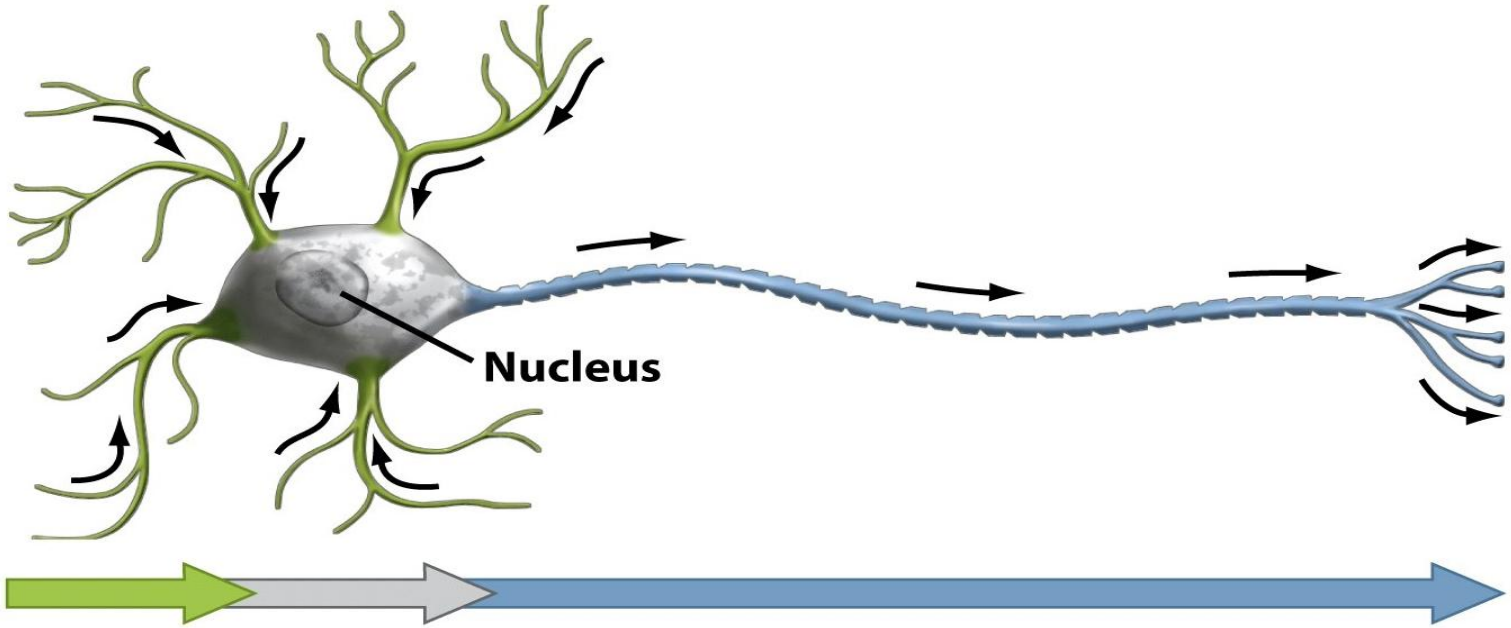
TABLE 9.1 Structure and Organizational Levels of Skeletal Muscle

STRUCTURE AND ORGANIZATIONAL LEVEL	DESCRIPTION	CONNECTIVE TISSUE WRAPPINGS
Myofibril or fibril (complex organelle composed of bundles of myofilaments) 	Rodlike contractile element; myofibrils occupy most of the muscle cell volume; composed of sarcomeres arranged end to end; appear banded, and bands of adjacent myofibrils are aligned	
Sarcomere (a segment of a myofibril) 	The contractile unit, composed of myofilaments made up of contractile proteins	
Myofilament or filament (extended macromolecular structure) 	Contractile myofilaments are of two types—thick and thin: the thick filaments contain bundled myosin molecules; the thin filaments contain actin molecules (plus other proteins); the sliding of the thin filaments past the thick filaments produces muscle shortening. Elastic filaments (not shown here) maintain the organization of the A band and provide for elastic recoil when muscle contraction ends	

SKELETAL MUSCLE



NEURON

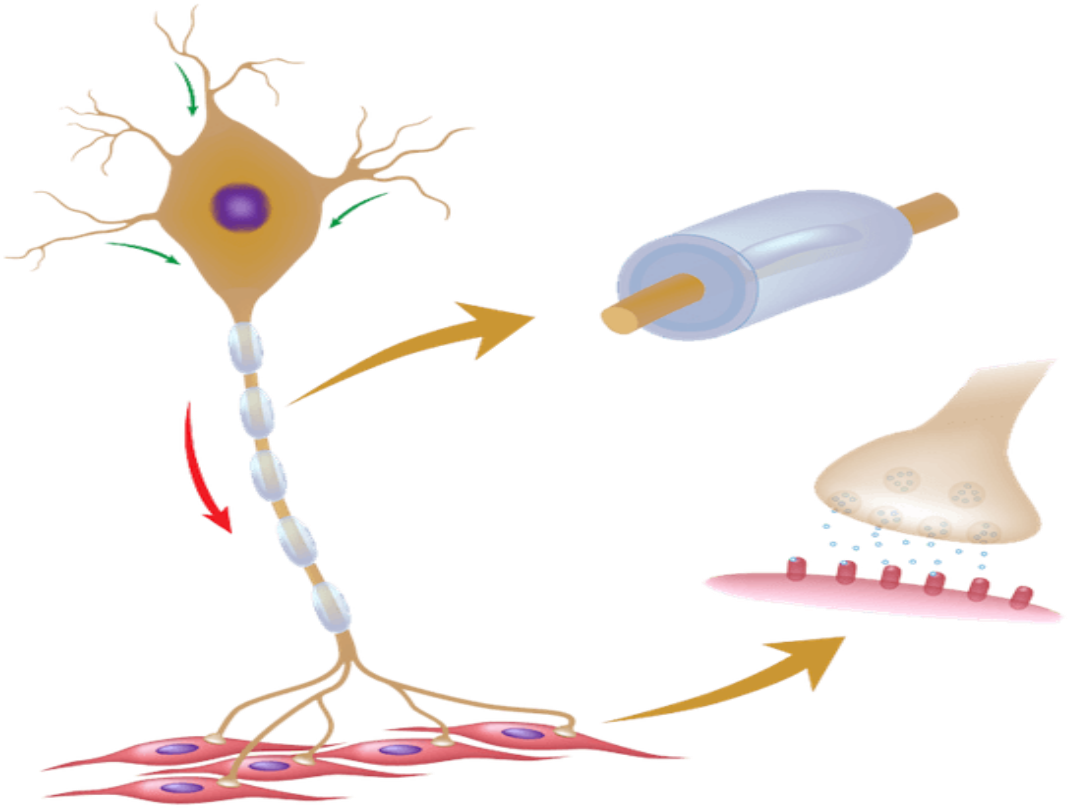


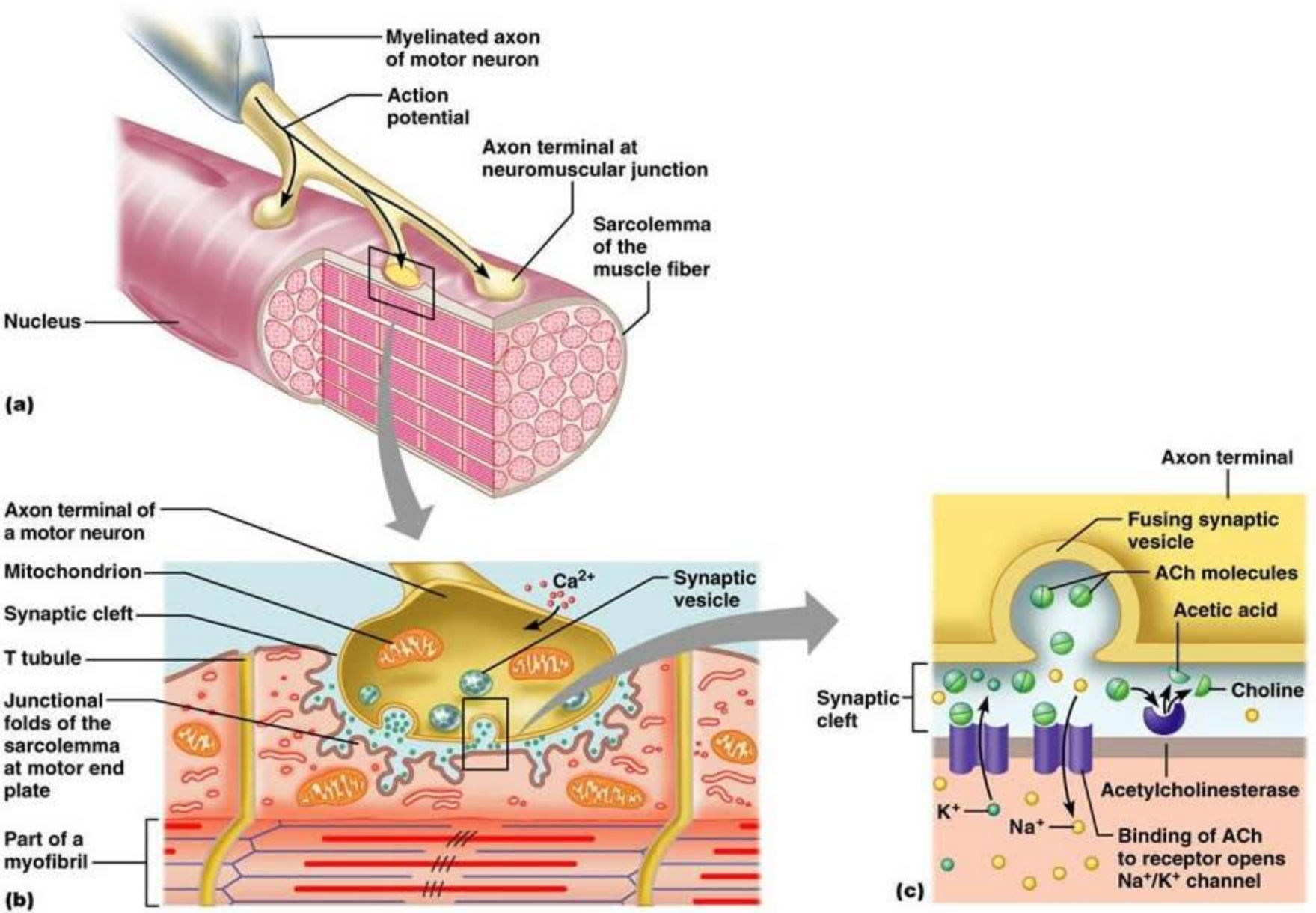
Dendrites
Collect electrical signals

Cell body
Integrates incoming signals and generates outgoing signal to axon

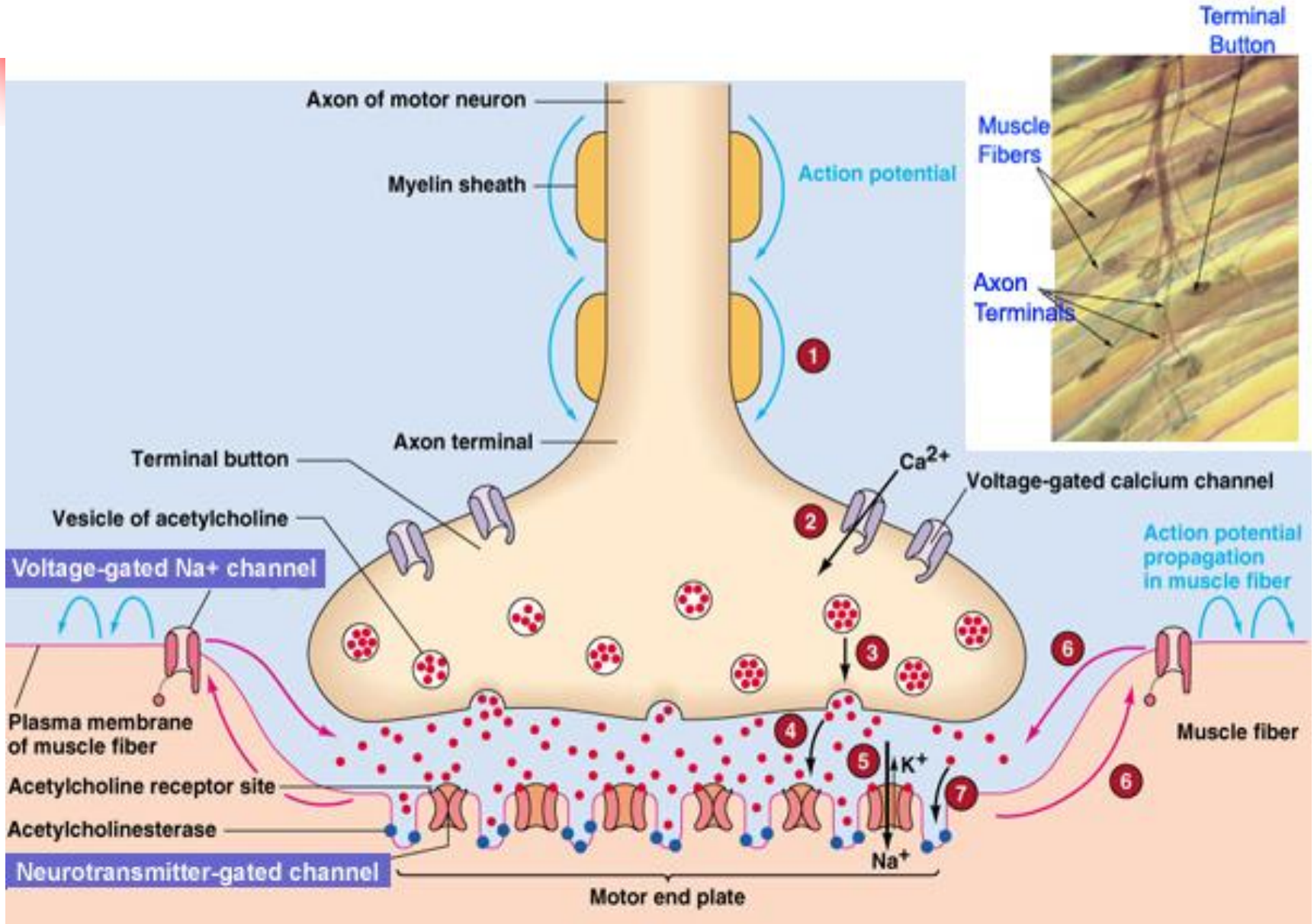
Axon
Passes electrical signals to dendrites of another cell or to an effector cell

NEURON

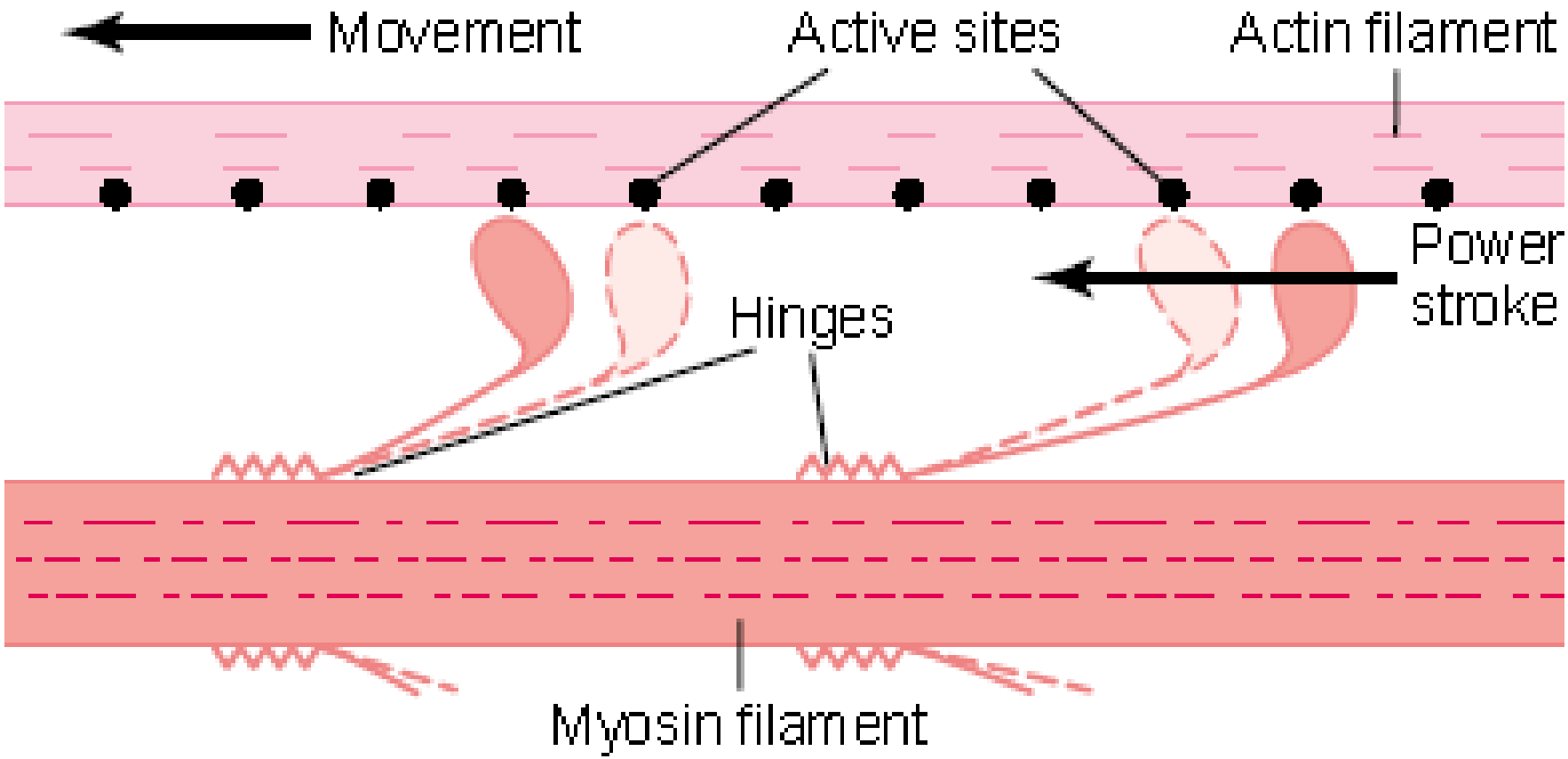



Myelinated axon of motor neuron
Action potential
Axon terminal at neuromuscular junction
Sarcolemma of the muscle fiber
Nucleus
(a)
Axon terminal of a motor neuron
Mitochondrion
Synaptic cleft
T tubule
Junctional folds of the sarcolemma at motor end plate
Part of a myofibril
(b)
Ca2+
Synaptic vesicle
Axon terminal
Fusing synaptic vesicle
ACh molecules
Acetic acid
Synaptic cleft
Choline
Acetylcholinesterase
K+
Na+
Binding of ACh to receptor opens Na+/K+ channel
(c)

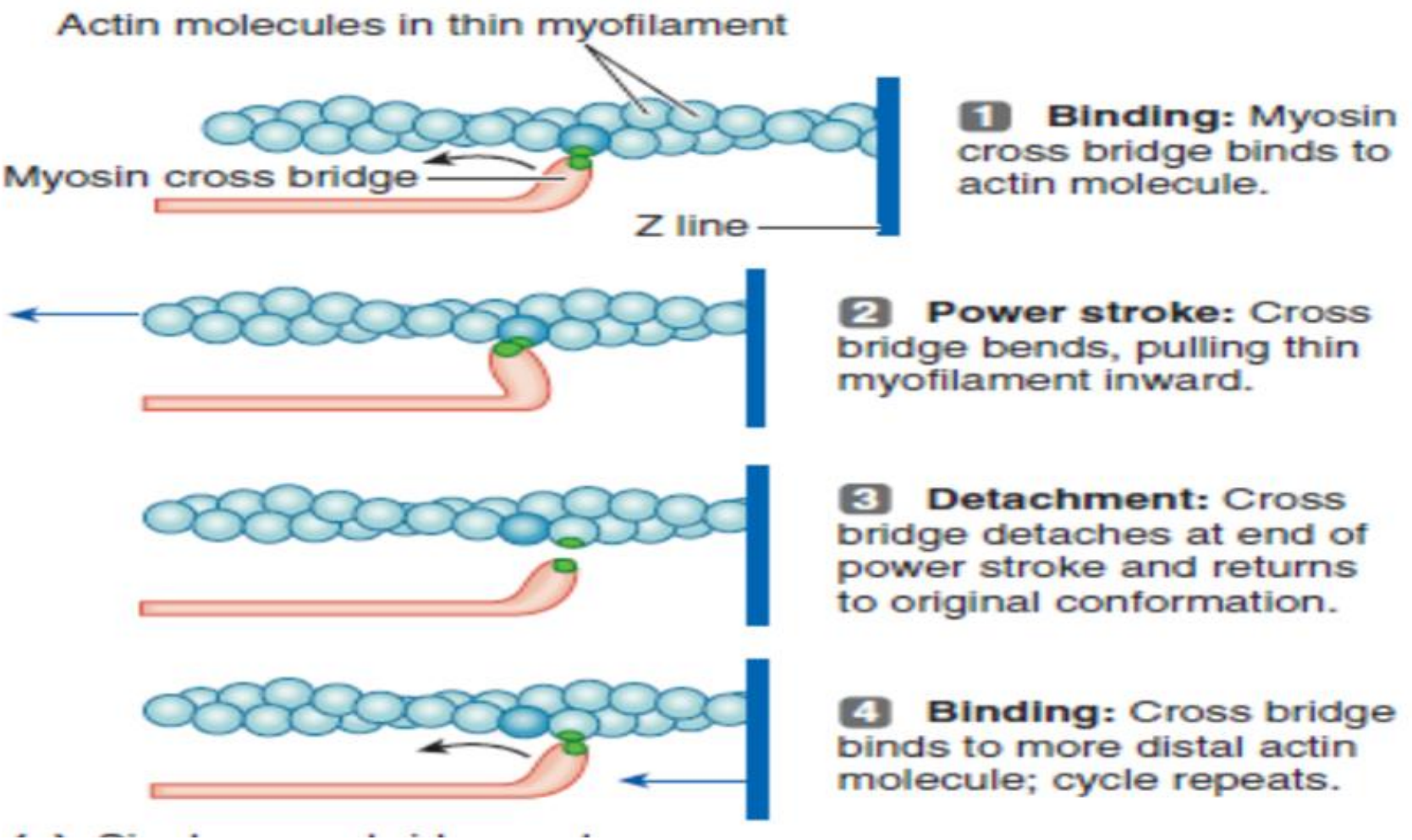
The Neuromuscular Junction



Power Stroke



Power Stroke



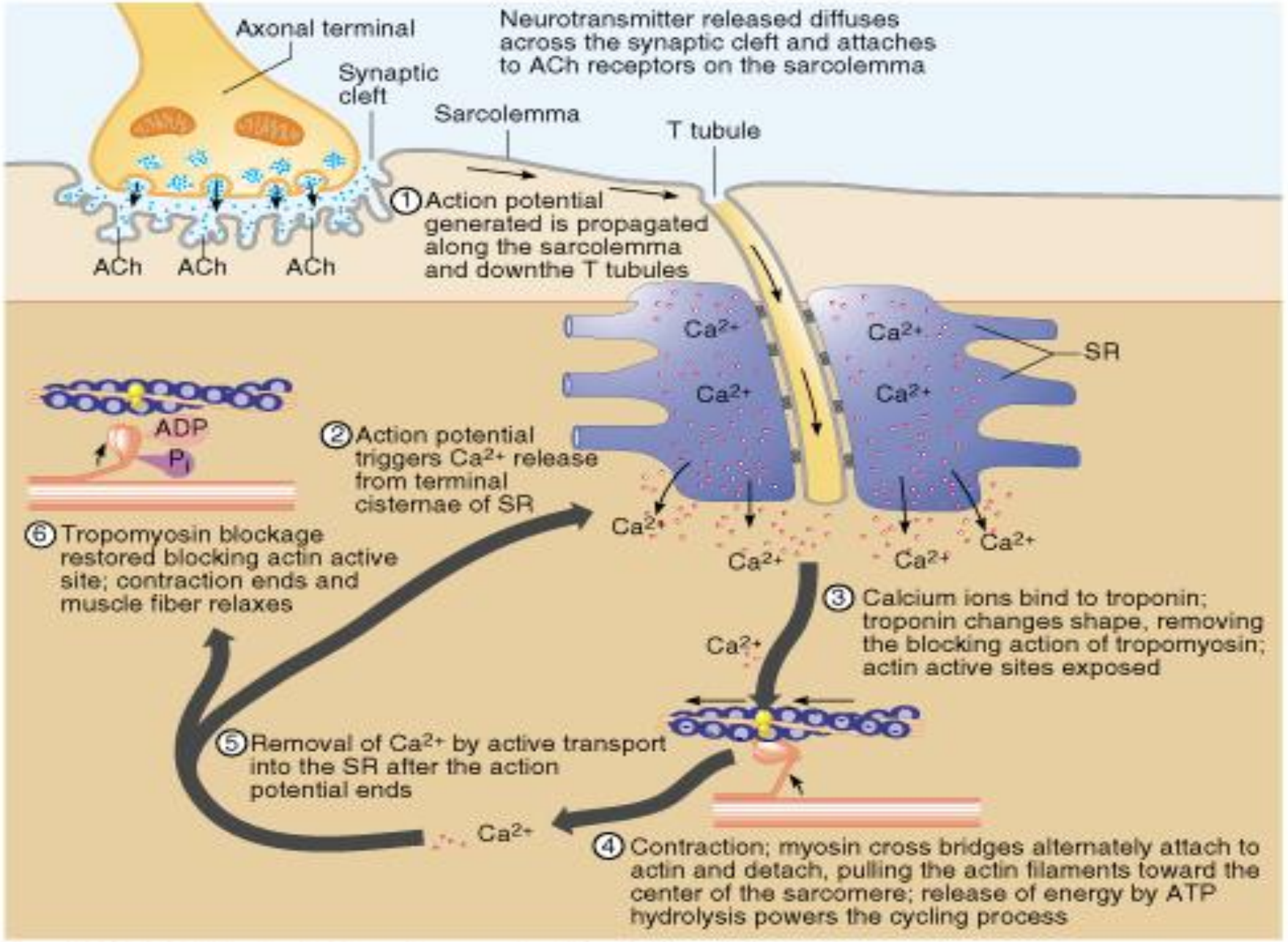
SLIDING FILAMENT THEORY

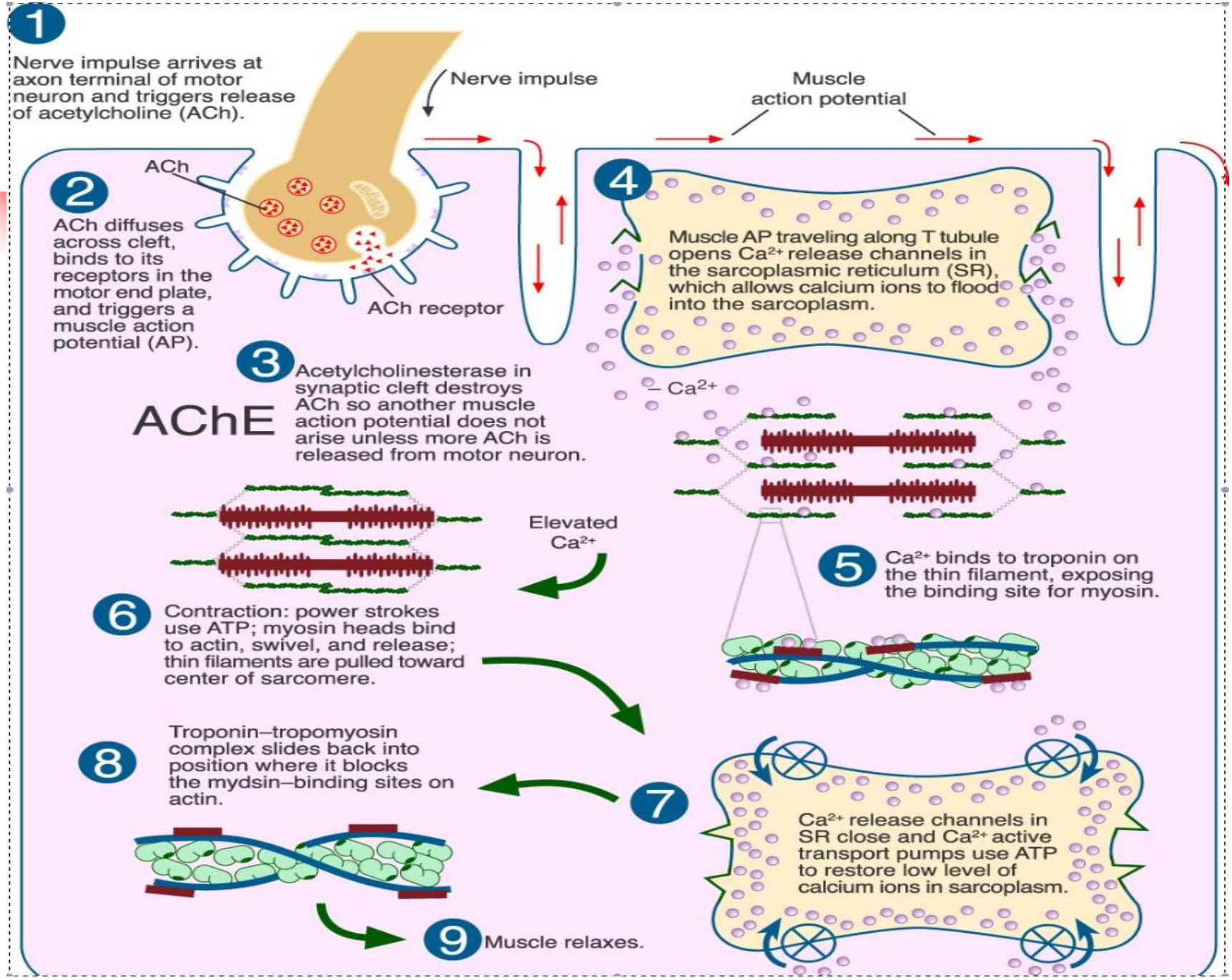
When a muscle cell contracts, the thin filaments slide past the thick filaments, and the sarcomere shortens. This process comprised of several steps is called the Sliding Filament Theory. It is also called the ***Walk Along Theory*** or the ***Ratchet Theory***.

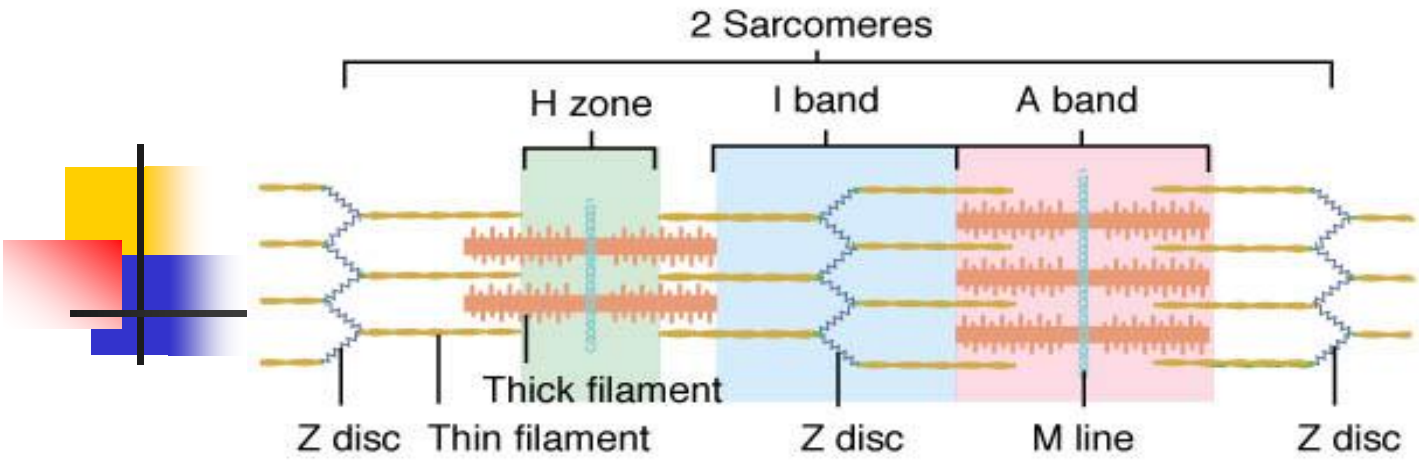
SLIDING FILAMENT THEORY

Table 12.2 | Summary of the Sliding Filament Theory of Contraction

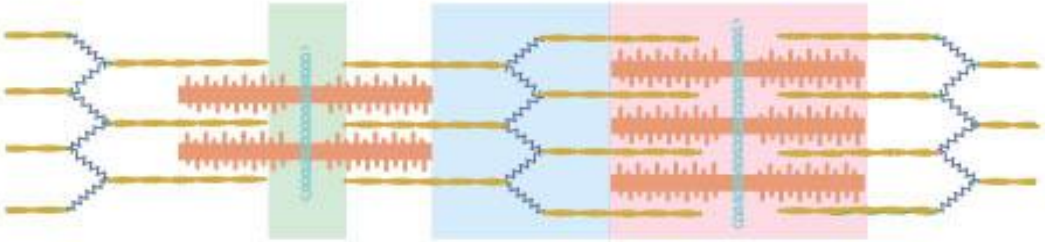
1. A myofiber, together with all its myofibrils, shortens by movement of the insertion toward the origin of the muscle.
2. Shortening of the myofibrils is caused by shortening of the sarcomeres—the distance between Z lines (or discs) is reduced.
3. Shortening of the sarcomeres is accomplished by sliding of the myofilaments—the length of each filament remains the same during contraction.
4. Sliding of the filaments is produced by asynchronous power strokes of myosin cross bridges, which pull the thin filaments (actin) over the thick filaments (myosin).
5. The A bands remain the same length during contraction, but are pulled toward the origin of the muscle.
6. Adjacent A bands are pulled closer together as the I bands between them shorten.
7. The H bands shorten during contraction as the thin filaments on the sides of the sarcomeres are pulled toward the middle.



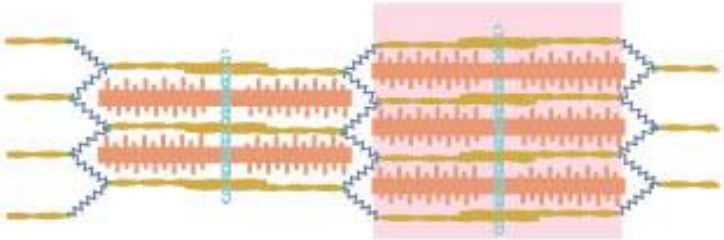

1
Nerve impulse arrives at axon terminal of motor neuron and triggers release of acetylcholine (ACh).
Nerve impulse
Muscle action potential
ACh
2
ACh diffuses across cleft, binds to its receptors in the motor end plate, and triggers a muscle action potential (AP).
ACh receptor
4
Muscle AP traveling along T tubule opens Ca2+ release channels in the sarcoplasmic reticulum (SR), which allows calcium ions to flood into the sarcoplasm.
Ca2+
3
AChE
Acetylcholinesterase in synaptic cleft destroys ACh so another muscle action potential does not arise unless more ACh is released from motor neuron.
Elevated Ca2+
5
Ca2+ binds to troponin on the thin filament, exposing the binding site for myosin.
6
Contraction: power strokes use ATP; myosin heads bind to actin, swivel, and release; thin filaments are pulled toward center of sarcomere.
8
Troponin–tropomyosin complex slides back into position where it blocks the mydsin–binding sites on actin.
7
Ca2+ release channels in SR close and Ca2+ active transport pumps use ATP to restore low level of calcium ions in sarcoplasm.
9
Muscle relaxes.



(a) Relaxed muscle



(b) Partially contracted muscle



(c) Maximally contracted muscle

TABLE 9.3 Comparison of Skeletal, Cardiac, and Smooth Muscle

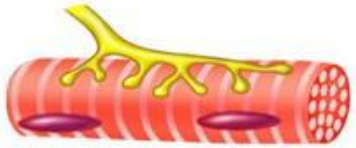
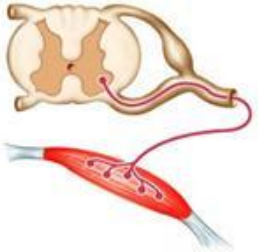
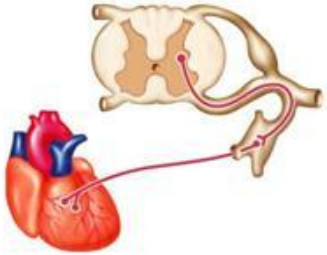
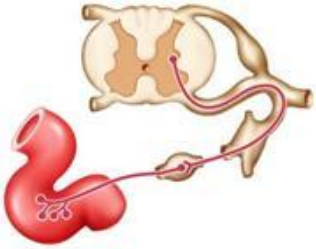
CHARACTERISTIC	SKELETAL	CARDIAC	SMOOTH
Presence of gap junctions	No	Yes; at intercalated discs	Yes; in single-unit muscle
Cells exhibit individual neuromuscular junctions	Yes	No	Not in single-unit muscle; yes in multiunit muscle
			
Regulation of contraction	Voluntary via axon terminals of the somatic nervous system	Involuntary; intrinsic system regulation; also autonomic nervous system controls; hormones; stretch	Involuntary; autonomic nerves, hormones, local chemicals; stretch
			
Source of Ca^{2+} for calcium pulse	Sarcoplasmic reticulum (SR)	SR and from extracellular fluid	SR and from extracellular fluid

Table 9.3.3

TABLE 9.3 Comparison of Skeletal, Cardiac, and Smooth Muscle

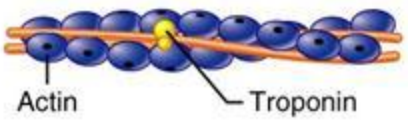
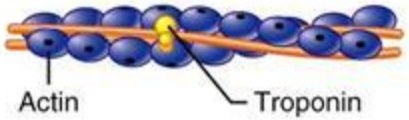
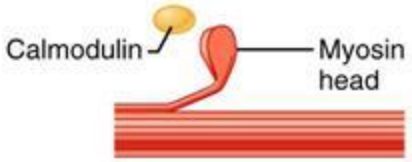
CHARACTERISTIC	SKELETAL	CARDIAC	SMOOTH
Site of calcium regulation	Troponin on actin-containing thin filaments	Troponin on actin-containing thin filaments	Calmodulin in the sarcoplasm
			
Presence of pacemaker(s)	No	Yes	Yes (in single-unit muscle only)
Effect of nervous system stimulation	Excitation	Excitation or inhibition	Excitation or inhibition
Speed of contraction	Slow to fast	Slow	Very slow
			
Rhythmic contraction	No	Yes	Yes in single-unit muscle
Response to stretch	Contractile strength increases with degree of stretch (to a point)	Contractile strength increases with degree of stretch	Stress-relaxation response
Respiration	Aerobic and anaerobic	Aerobic	Mainly aerobic

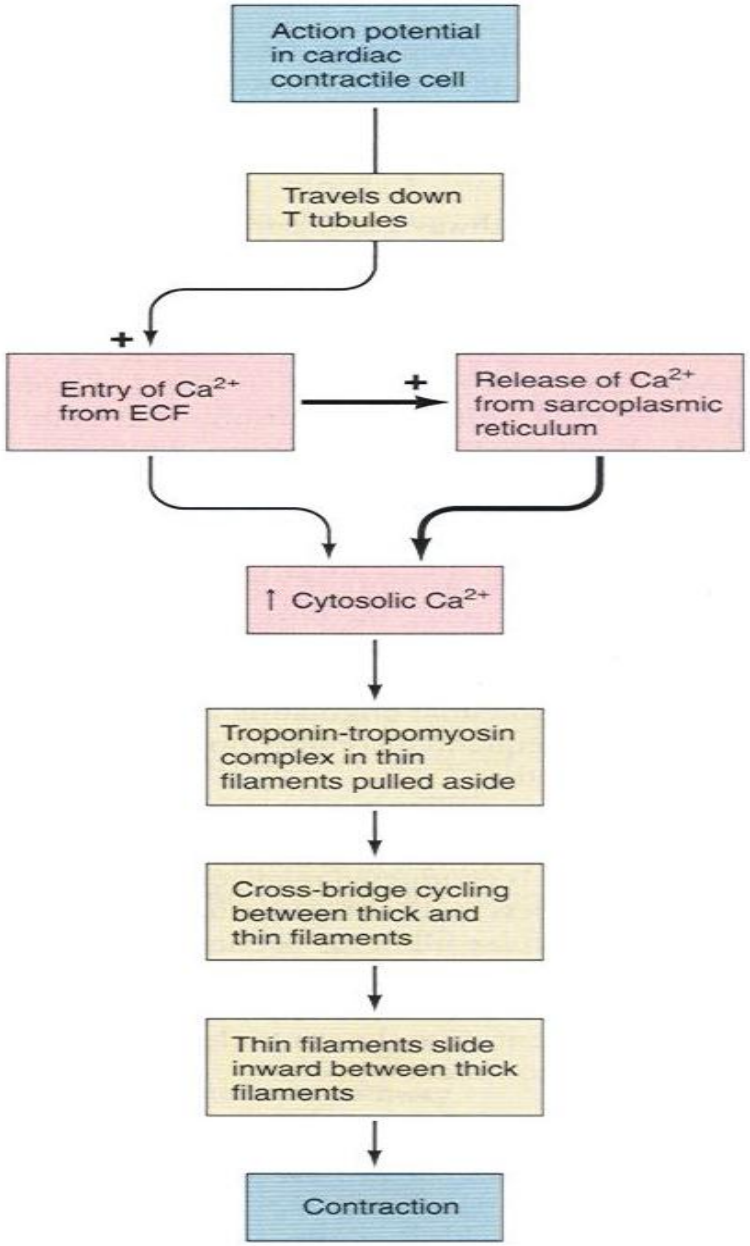
Table 3–2. Sequence of events in contraction and relaxation of skeletal muscle.

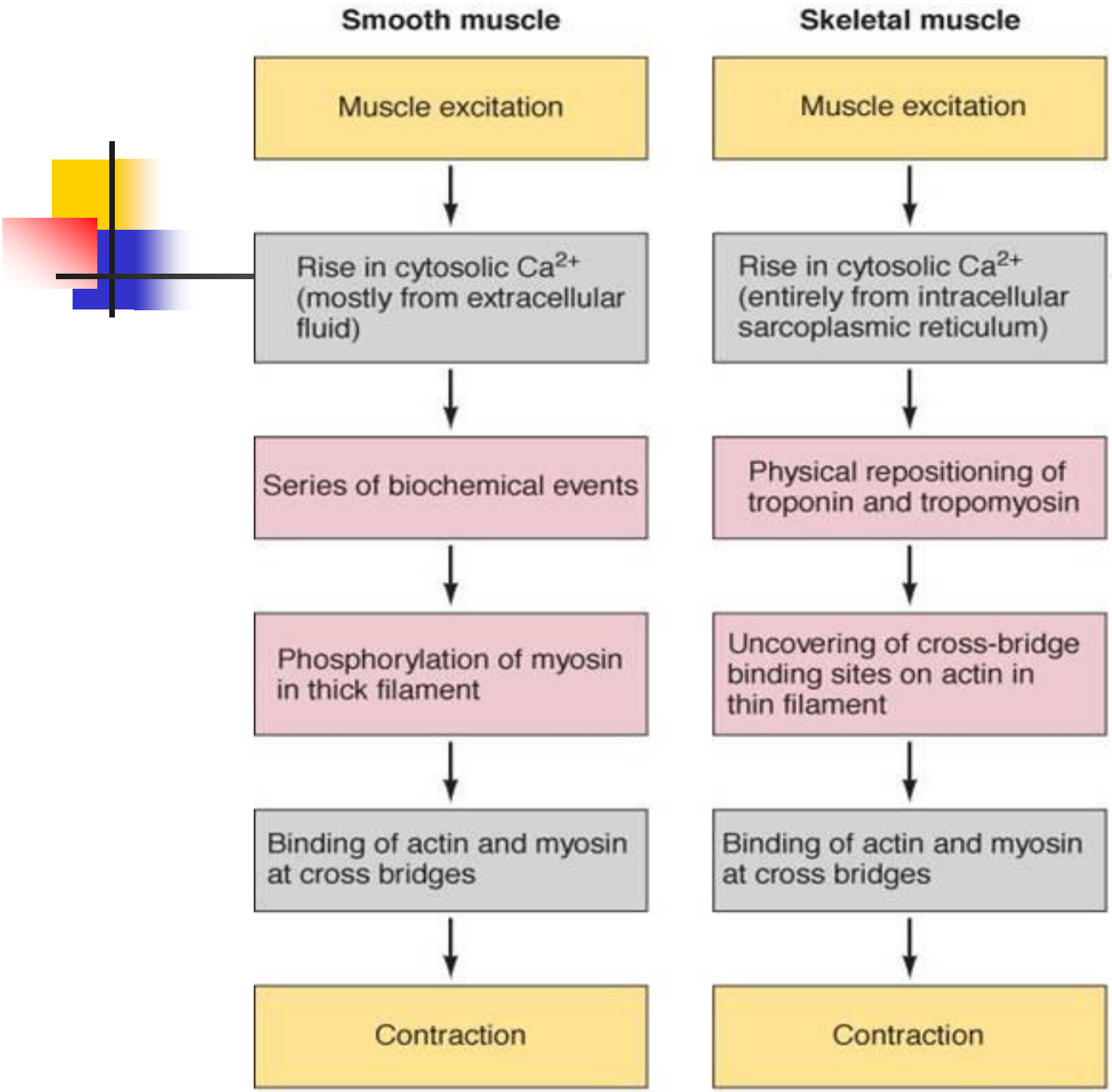
Steps in contraction[1]

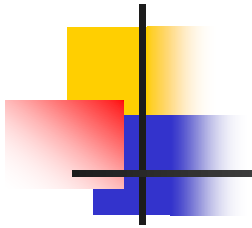
(1) Discharge of motor neuron.
(2) Release of transmitter (acetylcholine) at motor end-plate.
(3) Binding of acetylcholine to nicotinic acetylcholine receptors.
(4) Increased Na^+ and K^+ conductance in end-plate membrane.
(5) Generation of end-plate potential.
(6) Generation of action potential in muscle fibers.
(7) Inward spread of depolarization along T tubules.
(8) Release of Ca^{2+} from terminal cisterns of sarcoplasmic reticulum and diffusion to thick and thin filaments.
(9) Binding of Ca^{2+} to troponin C, uncovering myosin-binding sites on actin.
(10) Formation of cross-linkages between actin and myosin and sliding of thin on thick filaments, producing shortening.

Steps in relaxation

(1) Ca^{2+} pumped back into sarcoplasmic reticulum.
(2) Release of Ca^{2+} from troponin.
(3) Cessation of interaction between actin and myosin.


Action potential in cardiac contractile cell
Travels down T tubules
+
Entry of Ca^{2+} from ECF
+
Release of Ca^{2+} from sarcoplasmic reticulum
↑ Cytosolic Ca^{2+}
Troponin-tropomyosin complex in thin filaments pulled aside
Cross-bridge cycling between thick and thin filaments
Thin filaments slide inward between thick filaments
Contraction





THANK YOU