<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Skeletal</th>
<th>Cardiac</th>
<th>Smooth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body location</td>
<td>Attached to bones or, for some facial muscles, to skin</td>
<td>Walls of the heart</td>
<td>Mostly in walls of hollow visceral organs (other than the heart)</td>
</tr>
<tr>
<td>Cell shape and appearance</td>
<td>Single, very long, cylindrical, multinucleate cells with very obvious striations</td>
<td>Branching chains of cells; uninucleate, striations; intercalated discs</td>
<td>Single, fusiform, uninucleate; no striations</td>
</tr>
</tbody>
</table>

Table 6.1 Comparison of Skeletal, Cardiac, and Smooth Muscles (1 of 3)
Figure 6.1 Connective tissue wrappings of skeletal muscle.

Word Roots:
epi- upon/over
endo-within
fasci-bundle
mys-muscle
peri-around

Terms for labeling:
Endomysium
Epimysium
Fascicle
Muscle fiber
Perimysium

1. muscle fiber
2. endomysium
3. fascicle
4. perimysium
5. epimysium

Tendon
Bone
Figure 6.3 Anatomy of a skeletal muscle fiber (cell).

(a) Segment of a muscle fiber (cell)

(b) Myofibril or fibril (complex organelle composed of bundles of myofilaments)

(c) Sarcomere (segment of a myofibril)
Figure 6.3a Anatomy of a skeletal muscle fiber (cell).

(a) Segment of a muscle fiber (cell)

- Sarcolemma
- Myofibril
- Dark (A) band
- Light (I) band
- Nucleus
Figure 6.3b Anatomy of a skeletal muscle fiber (cell).

(b) **Myofibril or fibril** (complex organelle composed of bundles of myofilaments)
Figure 6.3c Anatomy of a skeletal muscle fiber (cell).

(c) **Sarcomere** (segment of a myofibril)
Figure 6.4 Motor units.
Figure 6.4a Motor units.

Spinal cord

Motor neuron cell bodies

Muscle

Axon terminals at neuromuscular junctions

Nerve

Motor unit 1

Axon of motor neuron

Motor unit 2

Muscle fibers

(a)
Figure 6.4b Motor units.

Axon terminals at neuromuscular junctions

Muscle fibers

Branching axon to motor unit (b)
Figure 6.5 Events at the neuromuscular junction.

1. Action potential reaches axon terminal of motor neuron.
2. Calcium (Ca\(^{2+}\)) channels open, and Ca\(^{2+}\) enters the axon terminal.
3. Ca\(^{2+}\) entry causes some synaptic vesicles to release their contents (acetylcholine, a neurotransmitter) by exocytosis.
4. Acetylcholine diffuses across the synaptic cleft and binds to receptors in the sarcolemma.
5. ACh binds and channels open that allow simultaneous passage of Na\(^{+}\) into the muscle fiber and K\(^{+}\) out of the muscle fiber. More Na\(^{+}\) ions enter than K\(^{+}\) ions leave, producing a local change in the electrical conditions of the membrane (depolarization). This eventually leads to an action potential.
6. The enzyme acetylcholinesterase breaks down ACh in the synaptic cleft, ending the process.
Figure 6.6 Comparing the action potential to a flame consuming a dry twig.

(a) Flame ignites the twig. Flame spreads rapidly along the twig.

(b) Na⁺ diffuses into the cell. Action potential spreads rapidly along the sarcolemma.
Figure 6.6a Comparing the action potential to a flame consuming a dry twig.

1. Flame ignites the twig.
2. Flame spreads rapidly along the twig.
Figure 6.6b Comparing the action potential to a flame consuming a dry twig.

1. Na\(^+\) diffuses into the cell.
2. Action potential spreads rapidly along the sarcolemma.
Figure 6.7 Diagrammatic views of a sarcomere.

(a) Relaxed sarcomere

(b) Fully contracted sarcomere
Figure 6.7a Diagrammatic views of a sarcomere.

(a) Relaxed sarcomere
Figure 6.7b Diagrammatic views of a sarcomere.

(b) Fully contracted sarcomere
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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