15.4: Muscle Contraction

Arm Wrestling

It’s obvious that a sport like arm wrestling depends on muscle contractions. Arm wrestlers must contract muscles in their hands and arms, and keep them contracted in order to resist the opposing force exerted by their opponent. The wrestler whose muscles can contract with greater force wins the match.

Figure \\
\(\PageIndex{1}\): (CC BY-NC 2.0; Hector Alejandro via Wikicommons).

What Is a Muscle Contraction?

A muscle contraction is an increase in the tension or a decrease in the length of a muscle. Muscle tension is the force exerted by the muscle on a bone or other object. A muscle contraction is isometric if muscle tension changes, but muscle length remains the same. An example of isometric muscle contraction is holding a book in the same position. A muscle contraction is isotonic if muscle length changes, but muscle tension remains the same. An example of isotonic muscle contraction is raising a book by bending the arm at the elbow. The termination of a muscle contraction of either type occurs when the muscle relaxes and returns to its non-contracted tension or length.
How a Skeletal Muscle Contraction Begins

Excluding reflexes, all skeletal muscle contractions occur as a result of conscious effort originating in the brain. The brain sends electrochemical signals through the somatic nervous system to motor neurons that innervate muscle fibers (to review how the brain and neurons function, see the chapter Nervous System). A single motor neuron with multiple axon terminals is able to innervate multiple muscle fibers, thereby causing all of them to contract at the same time. The connection between a motor neuron axon terminal and a muscle fiber occurs at a site called a neuromuscular junction. This is a chemical synapse where a motor neuron transmits a signal to a muscle fiber to initiate a muscle contraction. The process by which a signal is transmitted at a neuromuscular junction is illustrated in Figure \(\PageIndex{2}\).

The sequence of events begins when an action potential is initiated in the cell body of a motor neuron, and the action potential is propagated along the neuron’s axon to the neuromuscular junction. Once the action potential reaches the end of the axon terminal, it causes the release of the neurotransmitter acetylcholine (ACh) from synaptic vesicles in the axon terminal. The ACh molecules diffuse across the synaptic cleft and bind to receptors on the muscle fiber, thereby initiating a muscle contraction. Muscle contraction is initiated with the depolarization of the sarcolemma caused by the entrance of the sodium ions through the sodium channels associated with the ACh receptors.

![Diagram of a neuromuscular junction](https://bio.libretexts.org/Bookshelves/Human_Biology/Book%3A_Human_Biology_(Wakim_and_Grewal)/15%3A_Muscular_S...)

Figure \(\PageIndex{2}\): This diagram represents the sequence of events that occurs when a motor neuron stimulates a muscle fiber to contract. (CC BY-NC 4.0; OpenStax; via wikimedia.org)

Things happen very quickly in the world of excitable membranes (just think about how quickly you can snap your fingers...
as soon as you decide to do it). Immediately following depolarization of the membrane, it repolarizes, re-establishing the negative membrane potential. Meanwhile, the ACh in the synaptic cleft is degraded by the enzyme acetylcholinesterase (AChE) so that the ACh cannot rebind to a receptor and reopen its channel, which would cause unwanted extended muscle excitation and contraction.

Propagation of an action potential along the sarcolemma is the excitation portion of excitation-contraction coupling. Excitation triggers the release of calcium ions ($\text{Ca}^{++}$) from its storage in the cell’s sarcoplasmic reticulum (SR). For the action potential to reach the membrane of the SR, there are periodic invaginations in the sarcolemma, called **T-tubules** (“T” stands for “transverse”). The arrangement of a T-tubule with the membranes of SR on either side is called a **triad** (See figure below). The triad surrounds the cylindrical structure called a **myofibril**, which contains actin and myosin. The T-tubules carry the action potential into the interior of the cell, which triggers the opening of calcium channels in the membrane of the adjacent SR, causing $\text{Ca}^{++}$ to diffuse out of the SR and into the sarcoplasm. It is the arrival of $\text{Ca}^{++}$ in the sarcoplasm that initiates contraction of the muscle fiber by its contractile units, or sarcomeres.

Figure $\PageIndex{3}$: Narrow T-tubules permit the conduction of electrical impulses. The SR functions to regulate intracellular levels of calcium. Two terminal cisternae (where enlarged SR connects to the T-tubule) and one T-tubule comprise a triad—a “threesome” of membranes, with those of SR on two sides and the T-tubule sandwiched between them. (Blausen.com staff (2014) [CC BY 3.0 ]; via wikimedia.org)

**Excitation-contraction coupling**

Although the term **excitation-contraction coupling** confuses or scares some students, it comes down to this: for a skeletal muscle fiber to contract, its membrane must first be “excited”—in other words, it must be stimulated to fire an action potential. The muscle fiber action potential, which sweeps along the sarcolemma as a wave, is “coupled” to the actual contraction through the release of calcium ions ($\text{Ca}^{++}$) from the SR. Once released, the $\text{Ca}^{++}$ interacts with the shielding proteins, troponin and tropomyosin complex, forcing them to move aside so that the actin-binding sites are available for attachment by myosin heads. The myosin then pulls the actin filaments toward the center, shortening the muscle fiber.
In skeletal muscle, this sequence begins with signals from the somatic motor division of the nervous system. In other words, the "excitation" step in skeletal muscles is always triggered by signaling from the nervous system.

### Sliding Filament Theory of Muscle Contraction

Once the muscle fiber is stimulated by the motor neuron, actin and myosin protein filaments within the skeletal muscle fiber slide past each other to produce a contraction. The **sliding filament theory** is the most widely accepted explanation for how this occurs. According to this theory, muscle contraction is a cycle of molecular events in which thick myosin filaments repeatedly attach to and pull on thin actin filaments, so the filaments slide over one another. The actin filaments are attached to Z discs, each of which marks the end of a sarcomere. The sliding of the filaments pulls the Z discs of a sarcomere closer together, thus shortening the sarcomere. As this occurs, the muscle contracts.

![Sliding Filament Theory Diagram](https://bio.libretexts.org/Bookshelves/Human_Biology/Book%3A_Human_Biology_(Wakim_and_Grewal)/15%3A_Muscular_S...)  
Figure (PageIndex(5)): The top diagram shows a relaxed sarcomere, and the bottom diagram shows a contracted sarcomere. (CC BY-by 4.0; [OpenStax](https://openstax.org/); via wikimedia.org)

### Crossbridge Cycling

**Crossbridge cycling** is a sequence of molecular events that underlies the sliding filament theory. There are many projections from the thick myosin filaments, each of which consists of two myosin heads (you can see the projections and heads in the figures above and below). Each myosin head has binding sites for ATP (or the products of ATP...
hydrolysis: ADP and P_i) and for actin. The thin actin filaments also have binding sites for the myosin heads. A cross-bridge forms when a myosin head binds with an actin filament.

The process of cross-bridge cycling is shown in the figure below. A cross-bridge cycle begins when the myosin head binds to an actin filament. ADP and P_i are also bound to the myosin head at this stage. Next, a power stroke moves the actin filament inward toward the center of the sarcomere, thereby shortening the sarcomere. At the end of the power stroke, ADP and P_i are released from the myosin head, leaving the myosin head attached just to the thin filament until another ATP binds to the myosin head. When ATP binds to the myosin head, it causes the myosin head to detach from the actin filament. ATP is once again split into ADP and P_i and the energy released is used to move the myosin head into a "cocked" position. Once in this position, the myosin head can bind to the actin filament again, and another cross-bridge cycle begins.

Figure (\(\PageIndex{6}\)): Crossbridge cycling. (CC BY-SA 4.0; OpenStax; via wikimedia.org)

**Energy for Muscle Contraction**

According to the sliding filament theory, ATP is needed to provide the energy for a muscle contraction. Where does this ATP come from? Actually, there are multiple potential sources, as illustrated in Figure (\(\PageIndex{7}\)).
1. As you can see from the top panel of Figure 7, some ATP is already available in a resting muscle. As a muscle contraction starts, this ATP is used up in just a few seconds. More ATP is generated from creatine phosphate, but this ATP is used up rapidly as well. It’s gone in another 15 seconds or so.

2. Glucose from the blood and glycogen stored in muscle can then be used to make more ATP. Glycogen breaks down to form glucose, and each glucose molecule produces two molecules of ATP and two molecules of pyruvate. Pyruvate (as pyruvic acid) can be used in aerobic respiration if oxygen is available (diagram c). Alternatively, pyruvate can be used in anaerobic respiration, if oxygen is not available. The latter produces lactic acid, which may contribute to muscle fatigue. Anaerobic respiration typically occurs only during strenuous exercise when so much ATP is needed that sufficient oxygen cannot be delivered to the muscle to keep up.

3. Resting or moderately active muscles can get most of the ATP they need for contractions by aerobic respiration. This process takes place in the mitochondria of muscle cells. In the process, glucose and oxygen react to produce carbon dioxide, water, and many molecules of ATP.

Figure 7. Muscles require many ATP molecules to power muscle contractions. The ATP can come from the three sources illustrated in diagrams a-c. (CC BY-SA $.0; OpenStax; via wikimedia.org)

Feature: Human Biology in the News

Interesting and hopeful basic research on muscle contraction is often in the news because muscle contractions are involved in so many different body processes and disorders, including heart failure and stroke.

- **Heart failure** is a chronic condition in which cardiac muscle cells cannot contract forcefully enough to keep body cells adequately supplied with oxygen. In 2016, researchers at the University of Texas Southwestern Medical Center identified a potential new target for the development of drugs to increase the strength of cardiac muscle contractions in patients with heart failure. The UT researchers found a previously unidentified protein involved in muscle contraction. The protein, which is very small, turns off the “brake” on the heart so it pumps blood more vigorously. At the molecular level, the protein affects the calcium-ion pump that controls muscle contraction. The
scientists also found the same protein in slow-twitch skeletal muscle fibers. Interestingly, the protein is encoded by a stretch of mRNA that had been dismissed by scientists as non-coding RNA, commonly referred to as “junk” RNA. According to one of the researchers, “We dipped into the RNA ‘junk’ pile and came up with a hidden treasure.” This result is likely to lead to searches for additional treasures that might be hiding in the RNA junk pile.

- A stroke occurs when a blood clot lodges in an artery in the brain and cuts off blood flow to part of the brain. Damage from the clot would be reduced if the smooth muscles lining brain arteries relaxed following a stroke because the arteries would dilate and allow greater blood flow to the brain. In a recent study undertaken at the Yale University School of Medicine, researchers determined that the muscles lining blood vessels in the brain actually contract after a stroke. This constricts the vessels, reduces blood flow to the brain, and appears to contribute to permanent brain damage. The hopeful takeaway of this finding is that it suggests a new target for stroke therapy.

### Summary

- A muscle contraction is an increase in the tension or a decrease in the length of a muscle. A muscle contraction is isometric if muscle tension changes, but muscle length remains the same. It is isotonic if muscle length changes, but muscle tension remains the same.

- A skeletal muscle contraction begins with electrochemical stimulation of a muscle fiber by a motor neuron. This occurs at a chemical synapse called a neuromuscular junction. The neurotransmitter acetylcholine diffuses across the synaptic cleft and binds to receptors on the muscle fiber. This initiates a muscle contraction.

- Once stimulated, Calcium is released from the sarcoplasmic reticulum. Calcium unshields actin and myosin cross-bridge sites. These protein filaments within the skeletal muscle fiber slide past each other to produce a contraction. The sliding filament theory is the most widely accepted explanation for how this occurs. According to this theory, thick myosin filaments repeatedly attach to and pull on thin actin filaments, thus shortening sarcomeres.

- Crossbridge cycling is a cycle of molecular events that underlies the sliding filament theory. Using energy in ATP, myosin heads repeatedly bind with and pull on actin filaments. This moves the actin filaments toward the center of a sarcomere, shortening the sarcomere and causing a muscle contraction.

- The ATP needed for a muscle contraction comes first from ATP already available in the cell, and more is generated from creatine phosphate. These sources are quickly used up. Glucose and glycogen can be broken down to form ATP and pyruvate. Pyruvate can then be used to produce ATP in aerobic respiration if oxygen is available, or it can be used in anaerobic respiration if oxygen is not available.

### Review

1. What is skeletal muscle contraction?

2. Distinguish between isometric and isotonic contractions of skeletal muscle.

3. How does a motor neuron stimulate a skeletal muscle contraction?

4. What is the sliding filament theory?

5. Describe cross-bridge cycling.

6. Where does the ATP needed for a muscle contraction come from?

7. Explain why an action potential in a single motor neuron can cause multiple muscle fibers to contract.
8. The name of the synapse between a motor neuron and a muscle fiber is the _______________ _________.

9. If the acetylcholine receptors on muscle fibers were blocked by a drug, what do you think this would do to muscle contraction? Explain your answer.

10. **True or False:** According to the sliding filament theory, actin filaments actively attach to and pull on myosin filaments.

11. **True or False:** When a motor neuron produces an action potential, the sarcomeres in the muscle fiber that it innervates become shorter as a result.

12. Explain how cross-bridge cycling and sliding filament theory are related to each other.

13. When does anaerobic respiration typically occur in human muscle cells?

14. Which process produces more ATP: aerobic respiration or anaerobic respiration?

15. If there were no ATP available in a muscle, how would this affect cross-bridge cycling? What would this do to muscle contraction?

**Explore More**

https://bio.libretexts.org/link?16812#Explore_More

Watch this animation to further explore how a muscle contraction occurs:
Check out this video to learn about how a paralyzed man can use his brain waves to play a video game:

https://bio.libretexts.org/Bookshelves/Human_Biology/Book%3A_Human_Biology_(Wakim_and_Grewal)/15%3A_Muscular_S...
The T-tubules carry the action potential into the interior of the cell, which triggers the opening of calcium channels in the membrane of the adjacent SR, causing Ca$^{2+}$ to diffuse out of the SR and into the sarcoplasm. It is the arrival of Ca$^{2+}$ in the sarcoplasm that initiates contraction of the muscle fiber by its contractile units, or sarcomeres.