B L A C K F O X

C O A C H I N G & N U T R I T I O N

B O R N F O R P R O G R E S S

SKELETAL MUSCLE STRUCTURE AND FUNCTION

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SKELETAL MUSCLE STRUCTURE AND FUNCTION

Muscles must first be activated by the nervous system. The muscle activation will then have an effect on a joint to move. A joint can either move (i.e., rotate around its axis) or remain static, depending on how much force the muscle produces.

Each muscle is covered by a thin layer of connective tissue, the epimysium, similar to plastic wrapping around a steak. The epimysium protects the muscle from friction against other muscles or bones. This wrapping does more than provide protection. It's connected to a layer of deep fascia. This means it's like plastic wrapping around a steak that's connected to all the other steaks on the shelf. Moving one of them inevitably affects the others.

Skeletal muscle is made up of bundles of muscle fibers. Each bundle is a fascicle, which is covered by a layer of connective tissue called perimysium. Within the fascicle is a collection of muscle fibers, and each muscle fiber is made up of smaller myofibrils.

Each myofibril contains sarcomeres, which are the functional units that can make the muscle fiber shorten. Sarcomeres are lined up in series within the myofibril to form a rodlike structure. If the myofibril were a yardstick, the sarcomeres would be the inch markers. The borders of the sarcomeres are then formed by a Z line, which we'll cover shortly. Each sarcomere can shorten only a minuscule distance, but the combined effect of all sarcomeres shortening at the same time causes the entire muscle to shorten significantly.

SKELETAL MUSCLE STRUCTURE AND FUNCTION

The sarcomeres shorten due to the sliding of two muscle proteins, myosin and actin, past one another in a process called the cross-bridge cycle. This can be imagined as an overhead view of eight rowers in a boat, moving through a narrow stream. The movement of the oars is similar to myosin, while the water, which gives the oars something to grab against to create movement, is similar to actin. But in this analogy, the boat (i.e., myosin) wouldn't move through water (i.e., actin). The boat would stay in place as the oars move water past it. This entire process is called the Sliding Filament Theory.

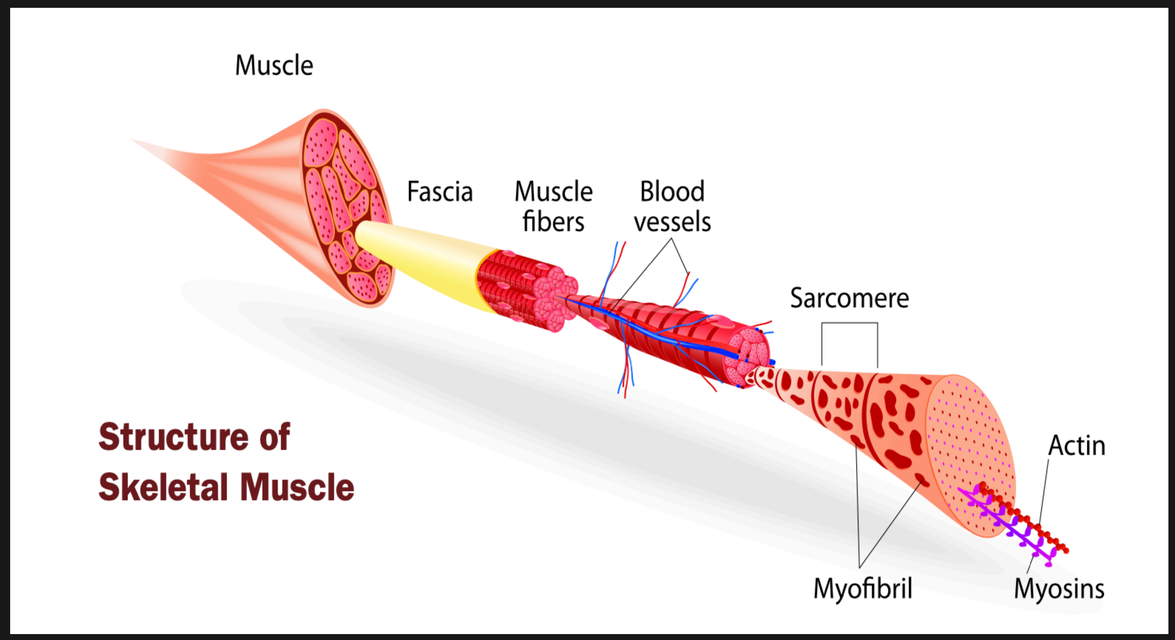
Sliding Filament Theory

- **According to the sliding filament theory of muscle contraction, skeletal muscle shortens because the thick and thin filaments slide past one another. The lengths of the individual thick and thin filaments do not change.**

- **Muscle contraction occurs because myosin heads attach to and "walk" along the actin filaments at both ends of a sarcomere, progressively pulling the thin filaments toward the center of the sarcomere.**

- **The Z lines come closer together. As this occurs simultaneously in sarcomeres throughout the cell, the muscle fiber shortens.**

SCELETAL MUSCLE



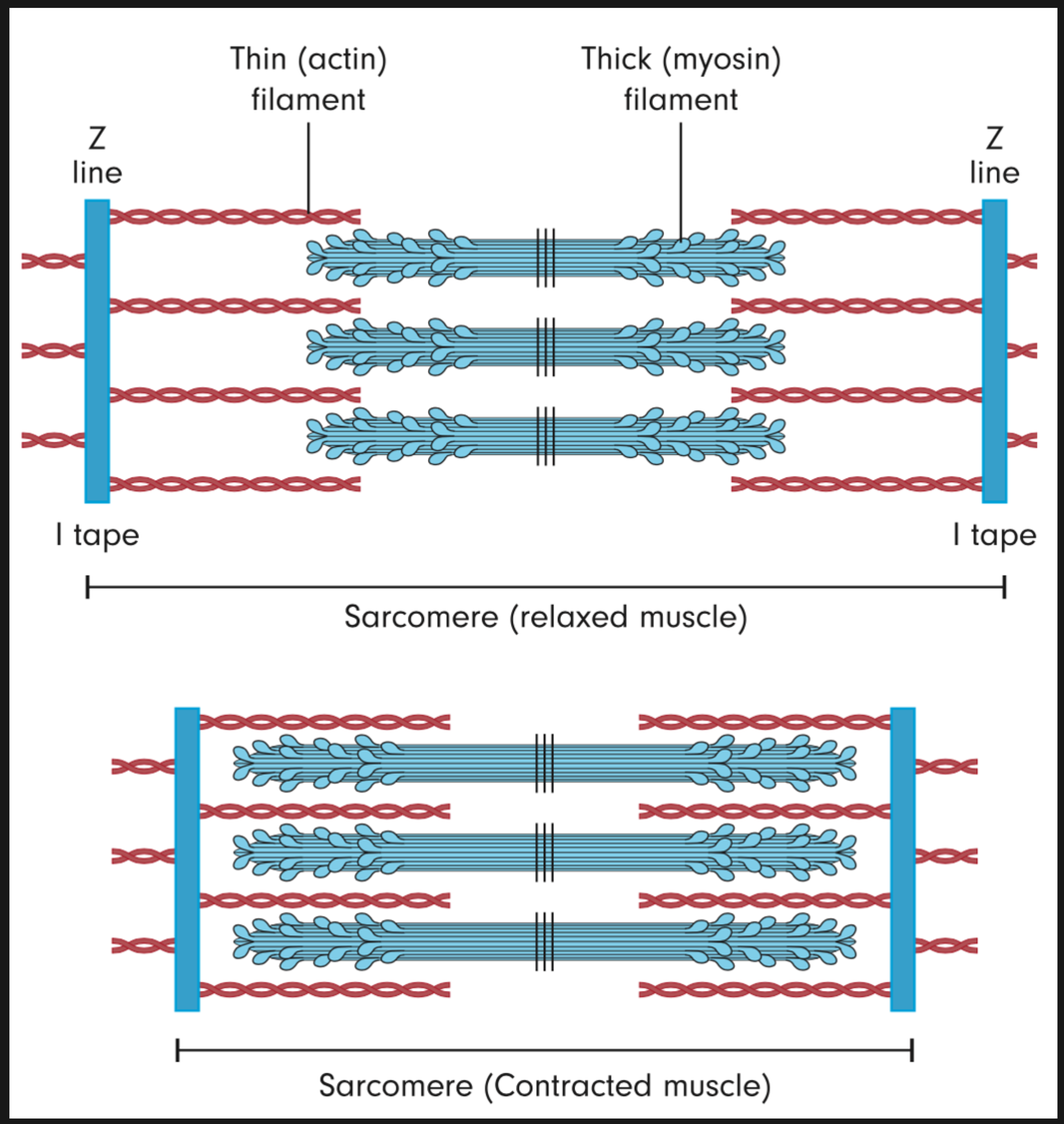
Structural and functional components of muscle. Muscle is made up of fascicles that contain bundles of muscle fibers. Each muscle fiber is a made up of myofibrils that contain sarcomeres, the functional units consisting of myosin and action, that allow it to contract.

MUSCLE CONTRACTIONS

There are two steps before a muscle contracts or relaxes. Using the arm curl exercise as an example, first, a signal is sent from the brain to the biceps. This signal causes the neurotransmitter acetylcholine to release in the biceps' neuromuscular junction—the space between the nerve and muscle. Second, binding of acetylcholine to muscle results in a cascade of events that ends with calcium release within the muscle. This calcium release is fundamental to the cross-bridge cycle, the interaction between actin and myosin. When a muscle is at rest, actin has regulatory proteins wrapped around it, blocking any interaction with myosin. But when calcium levels elevate, it binds to those regulatory proteins, moving them out of the way so myosin can interact with actin. Therefore, calcium is necessary for the cross-bridge cycle.

When a muscle is relaxed, myosin and actin aren't in contact with each other. But as soon as calcium is released in the muscle, myosin's club-shaped heads bind to actin. As the myosin heads move (i.e., cock), they pull actin closer together. Since actin proteins attach to the sarcomeres' Z lines, they cause the sarcomere to shorten, as shown in Figure.

HOW THE SARCOMERE CONTRACTS



MUSCLE CONTRACTIONS

The cross-bridge cycle, like every action in the human body, requires energy. It's provided by adenosine triphosphate (ATP) hydrolysis. ATP hydrolysis is the breakdown of ATP to adenosine diphosphate (ADP) to release energy stored within its phosphate bond.

The following six steps are required for muscle contraction (i.e., shortening of the sarcomeres). This outlined process starts with the muscle in its contracted position, like it is during rigor mortis.

Step 1: ATP binding. In this step the myosin head is bound to actin. Myosin's heads are cocked, as shown in the bottom of Figure 3.3. ATP binds to myosin's head, causing it to release from actin.

Step 2: ATP hydrolysis. The breakdown of ATP to ADP plus one phosphate occurs on the myosin head. This hydrolysis provides the energy to move the myosin head from a cocked to an uncocked position.

Step 3: Weak cross-bridge attachment. At this point, the myosin heads are uncocked. Each head has ADP plus one phosphate attached to it, which causes the head to make contact with actin, forming a weak cross-bridge attachment.

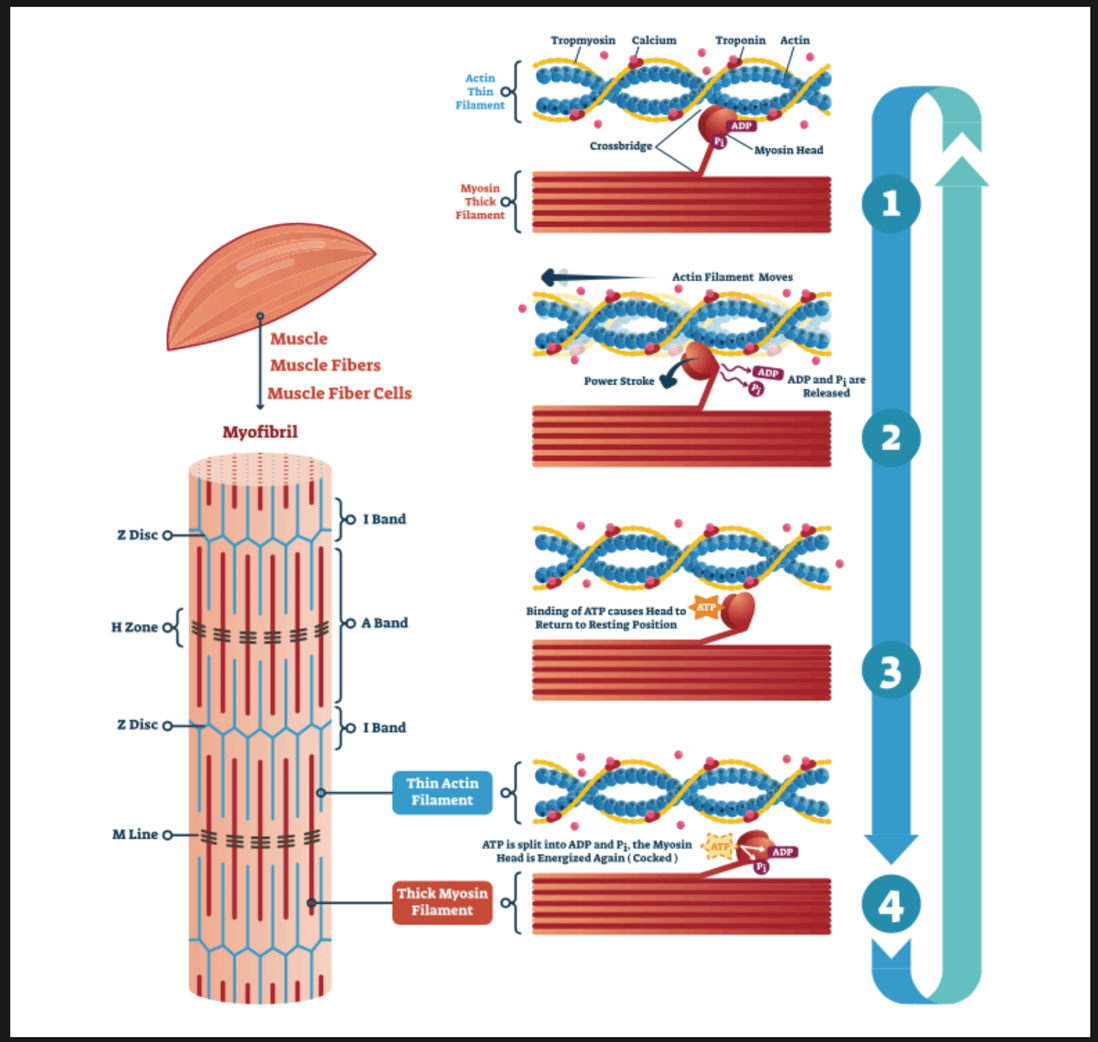
Step 4: Release of phosphate from myosin. Now the phosphate removes from the myosin head, allowing it to form a stronger cross-bridge attachment with actin.

Stepp 5: Power stroke. The myosin necks cock, which then pulls the Z lines closer together. This is how the sarcomere shortens, as shown in the bottom of Figure.

Step 6

: ADP release. Finally, ADP releases from the myosin head. Myosin will remain in a cocked position and bound to actin until another ATP attaches, which then starts the process over at Step 1. Without ATP, the muscle remains rigid in a state of rigor mortis.

ATP MUSCLE CONTRACTION



CALCIUM REGULATION OF MUSCLE ACTIVATION

Muscles will always have ATP available for muscle contractions. But ATP alone isn't enough to run or jump. As a reminder, actin is blocked from interacting with myosin until calcium levels elevate. There are two sources of this calcium:

- **Extracellular space (outside the muscle)**
- **Intracellular space (within the muscle)**

Extracellular calcium enters through special channels; intracellular calcium is released from the muscle's sarcoplasmic reticulum. This calcium elevation, and subsequent muscle contraction, is caused by the neural signal that was sent from your brain to the muscle, a process known as excitation-contraction coupling.

One of the many effects of the fight-or-flight response is a large rapid elevation in the muscle's calcium, which allows it to contract with more force. When the muscle stops receiving a signal from the brain, it causes calcium to reabsorb into the sarcoplasmic reticulum, which allows it to relax. This entire process, regulated by calcium, underpins activation of all muscle types: cardiac, smooth, and skeletal.

Importantly, muscle contractions can't occur in the reverse order. In other words, when a muscle is activated, it can only shorten—or, more specifically, attempt to shorten.

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