Lecture (9)

Muscles

※Muscle classified into three types: ***skeletal muscle***, ***cardiac muscle***, and ***smooth muscle***.

※The three muscle tissues have some properties in common; they all exhibit ***excitability*** as their plasma membranes can change their electrical states (from polarized to depolarized) and send action potential along the membrane. While the nervous system can influence the excitability of cardiac and smooth muscle to some degree,.

※ Skeletal muscle completely depends on signaling from the nervous system to work properly while both cardiac muscle and smooth muscle can respond to other stimuli, such as hormones and local stimuli.

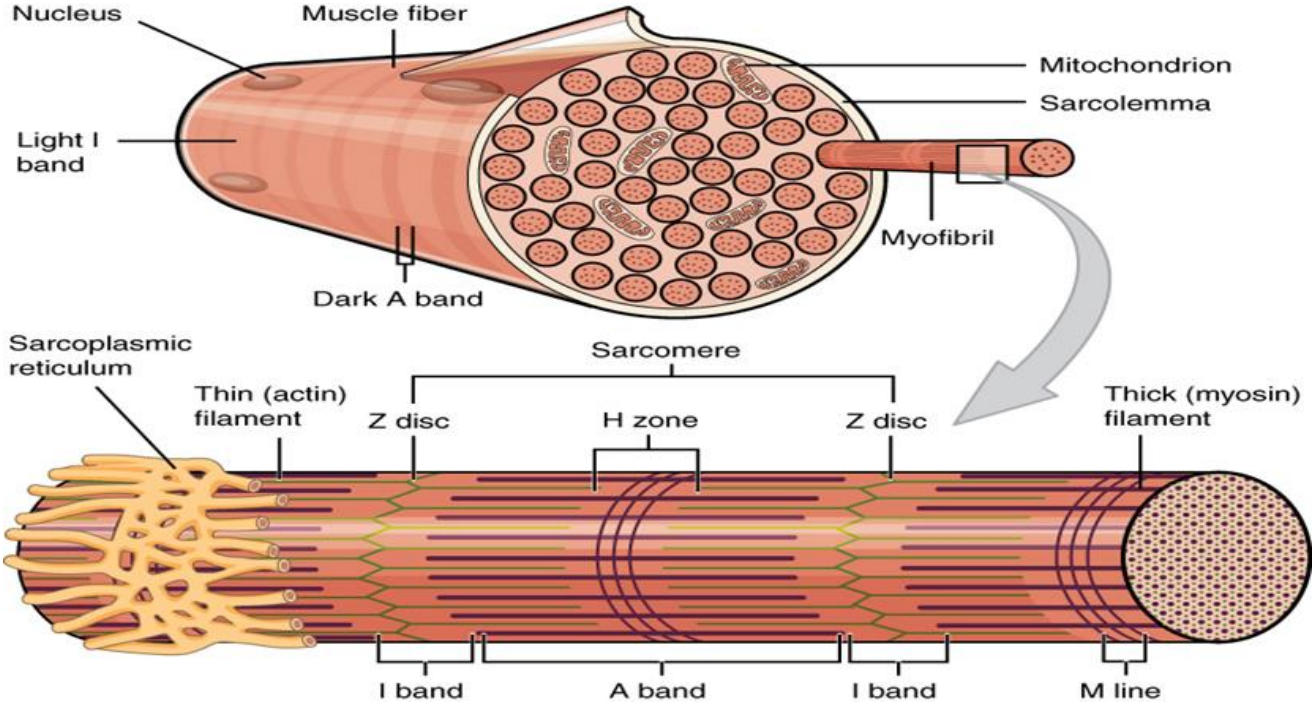
※**The muscles all** begin the actual process of **contracting** (shortening). A muscle can return to its original length when relaxed due to a quality of muscle tissue called ***elasticity*** due to elastic fibers. Muscle tissue also has the quality to stretch or extend (***extensibility)***. ***Contractility*** allows muscle tissue to pull on its attachment points and shorten with force. Differences among the three muscle types include the microscopic organization of their contractile proteins—actin and myosin. The actin and myosin proteins are arranged very regularly in the cytoplasm of individual muscle cells (referred to as fibers) in both **skeletal muscle** and **cardiac muscle**, which creates a pattern, or stripes, called **striations**.

Skeletal muscle

Each skeletal muscle is an organ that consists of muscle fibers, blood vessels, nerve fibers, and connective tissue. The myofibrils (skeletal muscle cells), are the contractile organelles in the muscle fibers. Muscle fiber is rooted in the Greek **sarco**, which means "flesh." The **plasma membrane** of muscle fibers is called the **sarcolemma**, the **cytoplasm** is referred to as **sarcoplasm**, and the **specialized smooth endoplasmic reticulum, which stores, releases, and retrieves calcium ions (Ca++)** is called the **sarcoplasmic reticulum (SR).** And the myofibrils are composed of **sarcomeres**, **the functional contractile unit of a skeletal muscle fiber**. The **sarcomere** is a highly organized arrangement of the contractile myofilaments, ***thin filament*** formed of ***actin*** proteins along with other support and regulatory proteins likes ***troponin*** and ***tropomyosin*** and ***thick filament*** formed of ***myosin*** proteins. The special

arrangement of the actin and myosin myofilaments in the sarcomere gives skeletal muscle fibers the striated appearance.

※Skeletal muscle formed of muscle fibers►myofibriles►myofilaments.

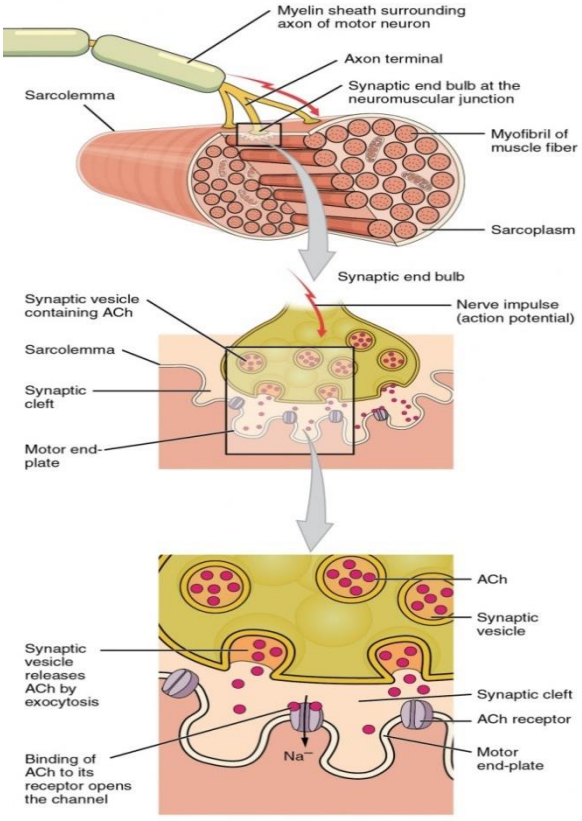


Molecular characteristics of the contractile filaments:

The motor end plate (Neuromuscular junction)

When **several skeletal muscle fibers** individually **innervated** by **branches of the same motor neuron** they formed with each other what's called **Motor unit**. Nerve fiber innervated skeletal muscle is Aα which considered the fastest between all nerve fibers, contains neurotransmitter vesicles and mitochondria provides energy for vesicular exocytosis. **Excitation signals from the neuron** are the **only way** to functionally stimulate the **fiber to contract.** There is no cytoplasmic continuity between the nerve and muscle but a small space between them called the **synaptic cleft**.

The motor end plate (MEP) or neuromuscular junction is the area on the side of the muscle fiber directly opposite to the nerve terminal. Signaling begins when a neuronal **action potential** travels along the axon of a motor neuron towards axon.



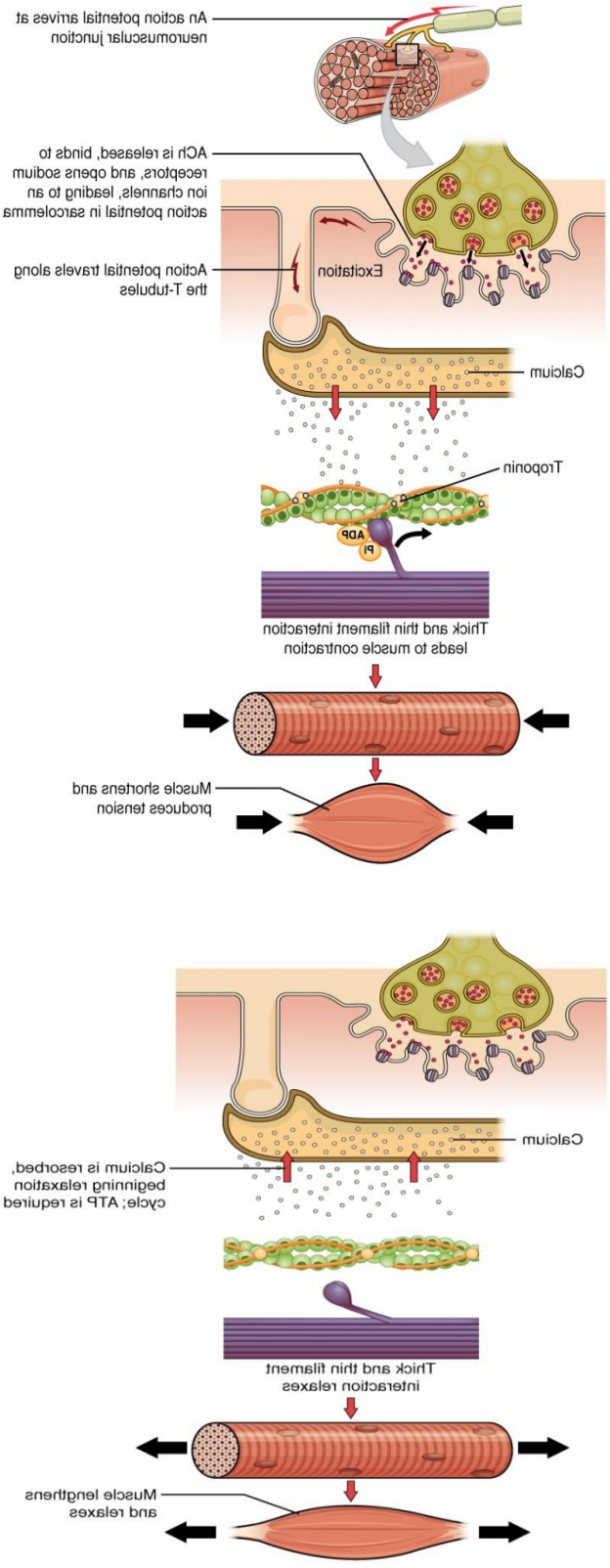
When the action potential reaches the axon terminal, vesicles at the neurons end releasing their content of **neurotransmitter** called **acetylcholine (ACh)** into the synaptic cleft. **ACh crosses synaptic cleft**, **binds to ACh receptors** in the motor end-plate of the sarcolemma causing a channel in the ACh receptor **opens (ligand gated channel)** and positively charged ions **(Na^+)** can pass through into the muscle fiber, causing it to **depolarize** (motor end plate potential) causing **muscle fiber contraction**. In the motor end plate, **acetylcholinesterase** is an enzyme **hydrolyzes ACh** which helps to **end stimulation** and prevent prolongation of muscle contraction.

The muscles concerned with fine movements (hand and ocular muscles) have 3-6 muscle fiber/motor units to allow gradation of contraction (higher central control). The muscles concerned with gross movements (large back muscles) have 120-160 muscle fiber/motor unit to allow maintained strong postural contractions.

The End Plate Potential (EPP):

Partial depolarization at the MEP caused by acetylcholine is proportional to the amount of acetylcholine released similar to the local excitatory state (LES) of the neuron.

The sequence of events resulted in the individual muscle fiber contraction begins with a signal—the neurotransmitter, **ACh**—from the motor neuron innervating that fiber. The local **membrane of the fiber** will **depolarize** as positively charged sodium ions **(Na^+) enter**, triggering an **action potential** that **spreads to the rest of the membrane** will depolarize, including the **T-tubules** where voltage-sensitive proteins that are **linked to proteins in the membrane of the sarcoplasmic reticulum (SR)** activating them and **triggers the release of calcium ions (Ca^{++})** from the sarcoplasmic reticulum. **Ca^{++} then initiates contraction**, which is sustained by ATP. As long as Ca^{++} ions remain in the sarcoplasm to bind to troponin, which keeps the actin-binding sites "unshielded," and as long as ATP is available to drive the cross-bridge cycling and the pulling of actin strands by myosin, the muscle fiber will continue to shorten to an anatomical limit. **The cross-bridge forms between actin and the myosin** heads triggering **contraction**. Muscle contraction usually stops when signaling from the motor neuron ends, which repolarizes the sarcolemma and T-tubules,



and closes the voltage-gated calcium channels in the SR. **Ca^{++} ions** are then **pumped back** into the **SR**, which causes the tropomyosin to reshield (or re-cover) the binding sites on the actin strands. A muscle also can **stop contracting** when it runs out of ATP and becomes fatigued.

※**Skeletal muscle potential** longer than that **of the nerve**, and in the **velocity of conduction being slower than the nerve**.

※Notice that the **absolute refractory period** in skeletal muscle is **very short** and it ends completely before the mechanical response begins i.e **the muscle fiber can responds to another stimulus when reaching the muscle during its contraction phase**.

※Shortly after contraction (Within 30 milliseconds after the action potential ends), **calcium is pumped back into the sarcoplasmic reticulum** by an **active pump (Ca^2-Mg^2 pump)** → ↓ Ca^{+2} concentration in the sarcoplasm → release of Ca^{+2} from troponin C → troponin I binds strongly with actin and tropomyosin covers the active sites of actin → stopping the interaction between actin and myosin → relaxation.