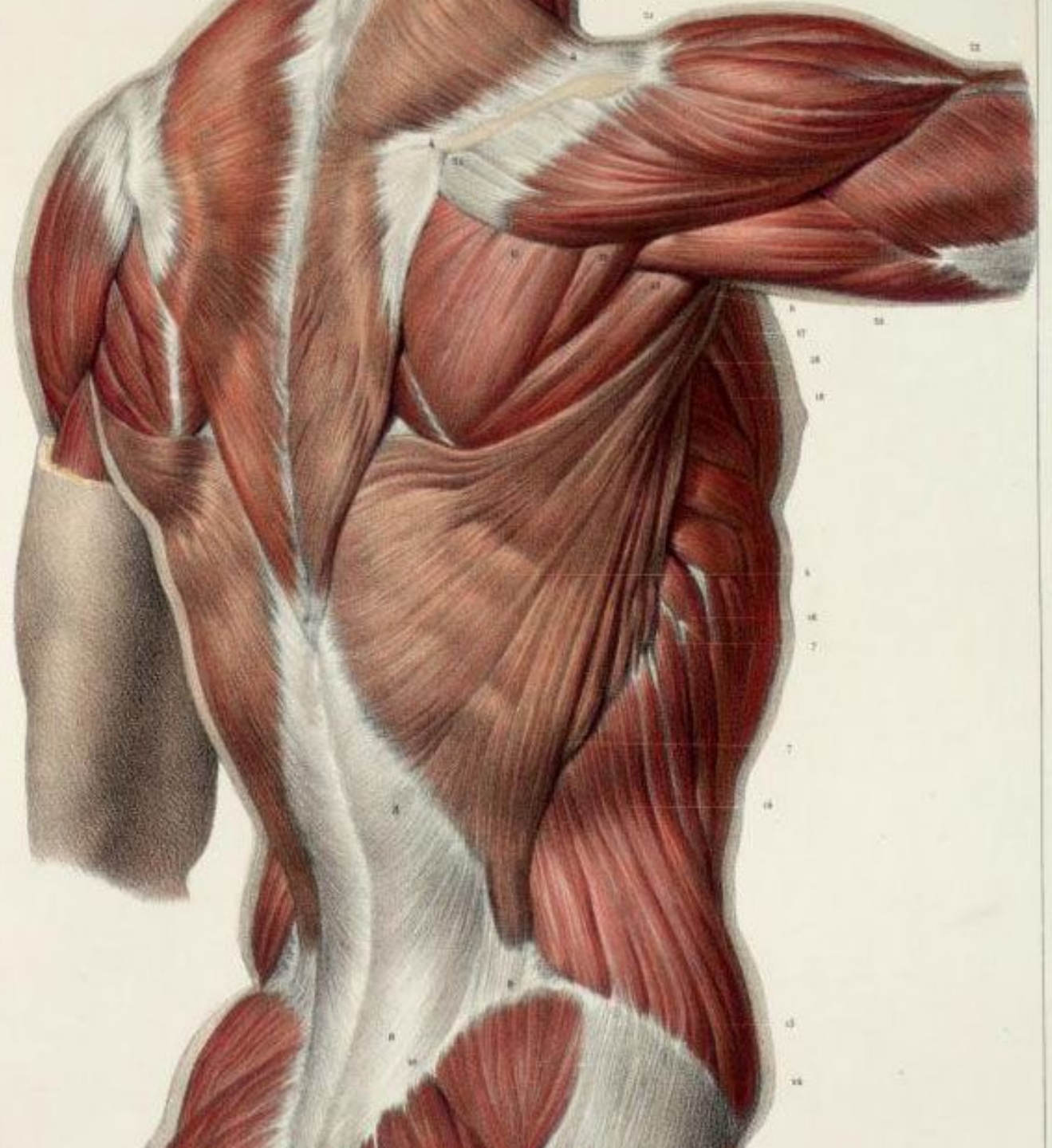


# LAB 5: THE MUSCULAR SYSTEM

---

Protocol slides  
PCB 3702L  
FIU



# LAB 5 PROTOCOL OBJECTIVES

1

Explain the sliding filament theory.

2

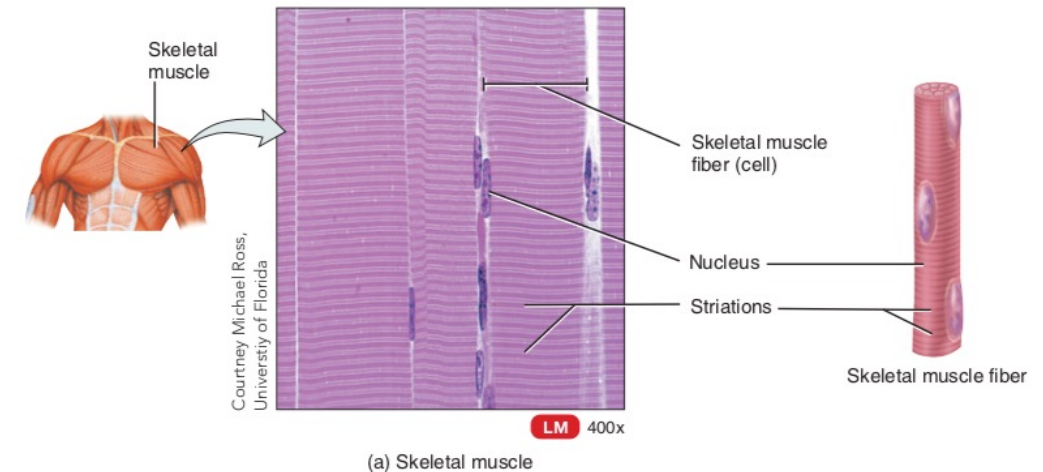
Explain muscle contraction.

3

Describe muscle fatigue.

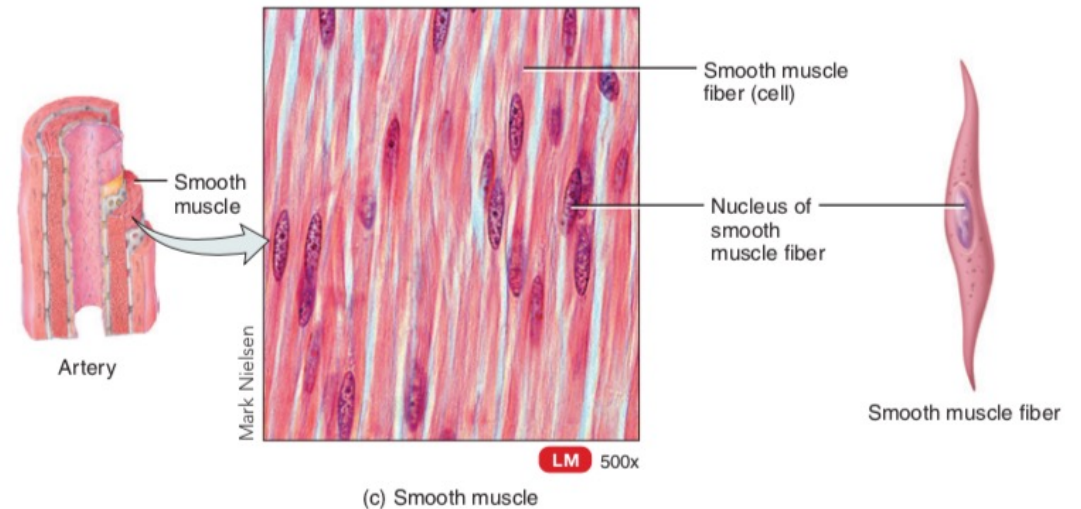
# SKELETAL MUSCLE

- **Striated** - Cross-striations create a striped appearance.
- **Multinucleated** - Multiple nuclei within a single muscle fiber. This contrasts with most cells in the body, which typically have a single nucleus.
- **Voluntary** - Activity is consciously controlled by motor neurons. A part of the somatic nervous system. Allows precise and coordinated movements.
- **Subconscious Control** - Some level of subconscious control is possible. Reflexes and muscle memory contribute to this control. Examples of Subconscious Control:
  - Diaphragm Function - Contracts and relaxes involuntarily to maintain breathing. Ensures continuous respiration without conscious thought.
  - Postural Muscles - Muscles responsible for maintaining posture and body stability. Contract to support your body unconsciously.



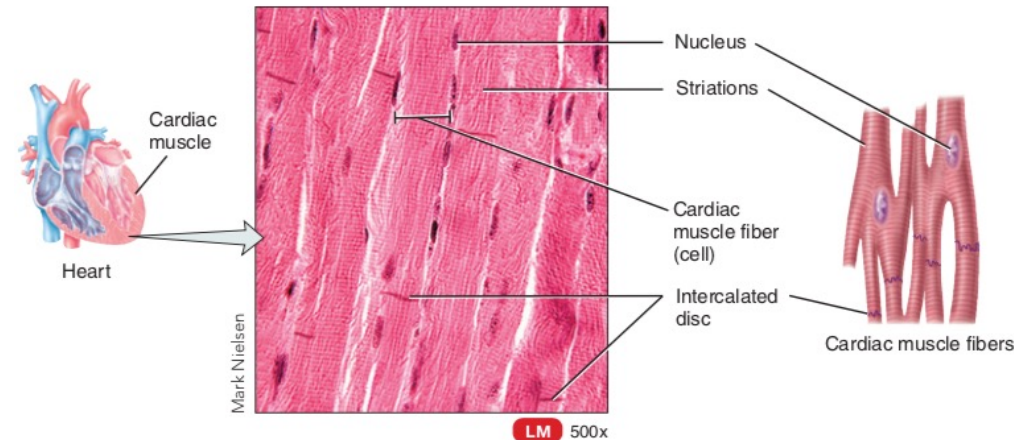
# SMOOTH MUSCLE

- **Lacks striations (nonstriated)** – Absence of cross-striations.
- **Mononucleated** – Contains a single nucleus per cell. Differentiates it from multinucleated skeletal muscle fibers.
- **Involuntary** - Contractions not under conscious control. Contractions occur automatically in response to stimuli.
- **Regulated by the Autonomic Nervous System and Hormones** - Controlled by motor neurons of the autonomic nervous system. Hormones released by endocrine glands also influence its activity.
- **Found in Walls of Hollow Internal Structures** - Located in the walls of various internal organs. Commonly found in blood vessels, airways, stomach, intestines, and uterus.



# CARDIAC MUSCLE

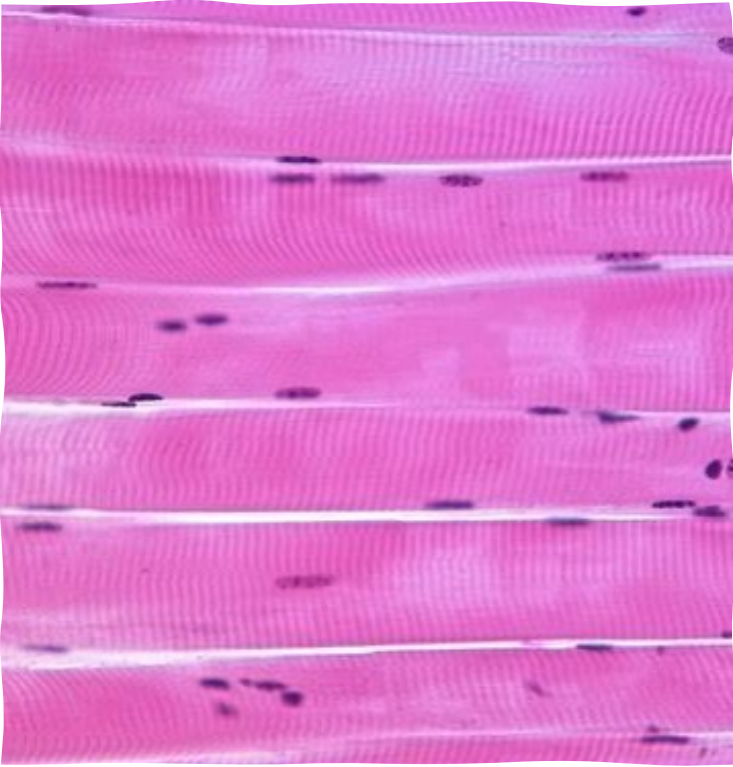
- **Striated** – Exhibits cross-striations similar to skeletal muscle. Contributes to its rhythmic contractile pattern.
- **Mononucleated** – Contains a single nucleus per cell. Unlike multinucleated skeletal muscle fibers.
- **Involuntary** – Contractions occur without conscious control. The heart's pacemaker initiates each contraction.
- **Pacemaker Regulation** – Heart beats due to a pacemaker that initiates contractions. Neurotransmitters and hormones can adjust the heart rate by influencing the pacemaker.
- **Autonomic Nervous System Control** – This can be regulated by the autonomic nervous system. Sympathetic and parasympathetic branches influence heart rate.
- **Intercalated Discs** – Specialized cell junction unique to cardiac muscle. Facilitate rapid conduction of electrical impulses.
- **Unique to the Heart** – Cardiac muscle is exclusively found in the heart. Responsible for maintaining a continuous heartbeat.



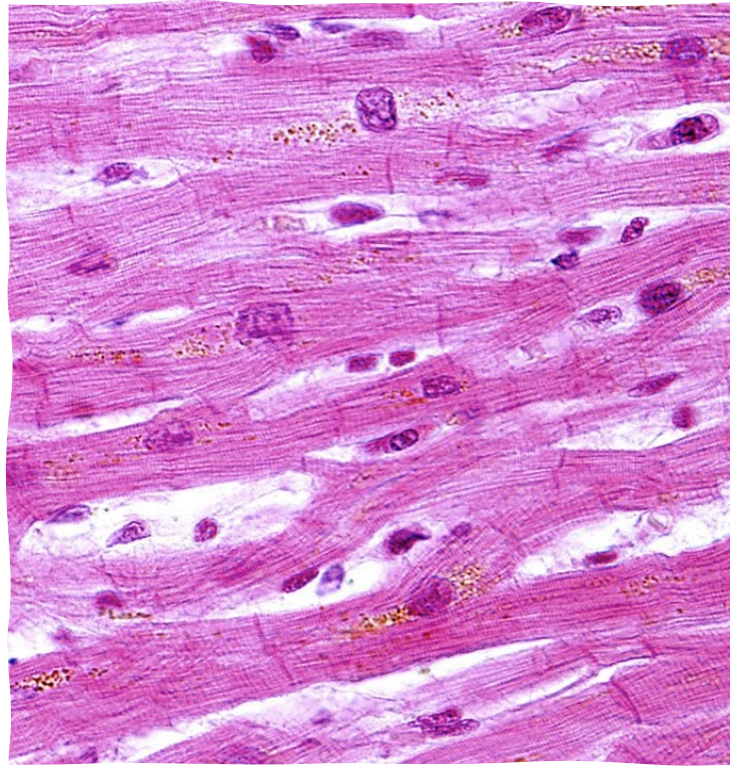


# IDENTIFY THE MUSCLE TYPE FOR EACH IMAGE BELOW:

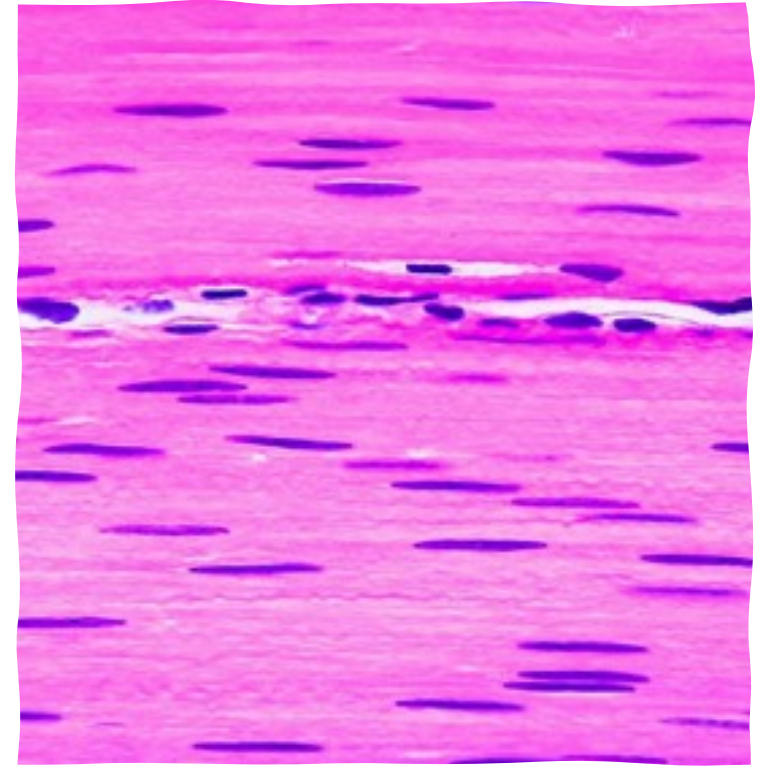
A. Skeletal Muscle



B. Cardiac Muscle



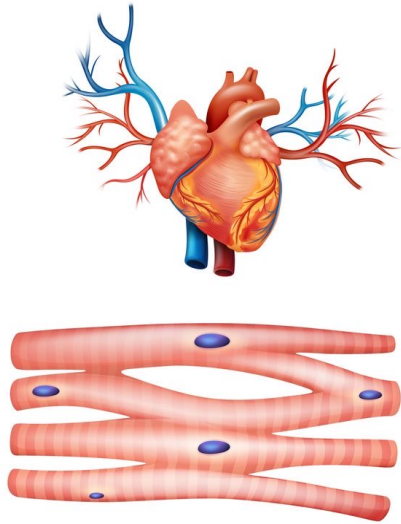
C. Smooth Muscle



Look for striations/intercalated discs/branching/etc.

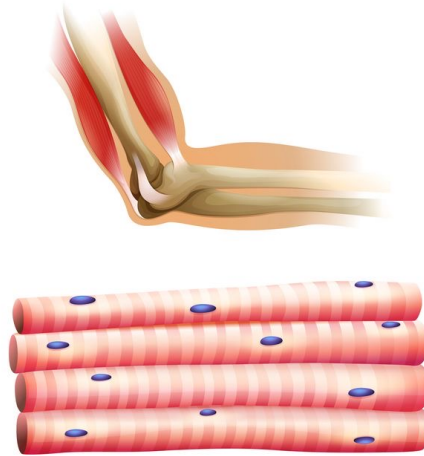
# MUSCLE TYPES OVERVIEW

## Cardiac Muscle



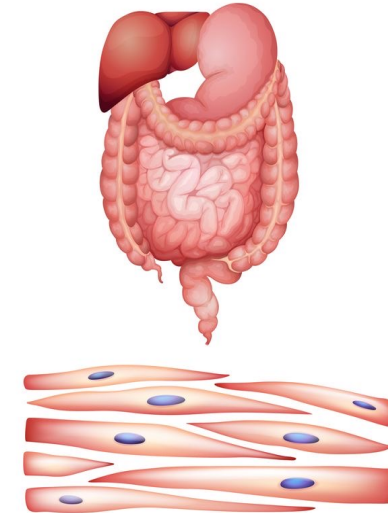
- Short, branched cells
- Usually, one nucleus
- Striations
- Intercalated discs
- Deep

## Skeletal Muscle



- Long, cylindrical cells
- Multiple nuclei
- Striations
- Superficial

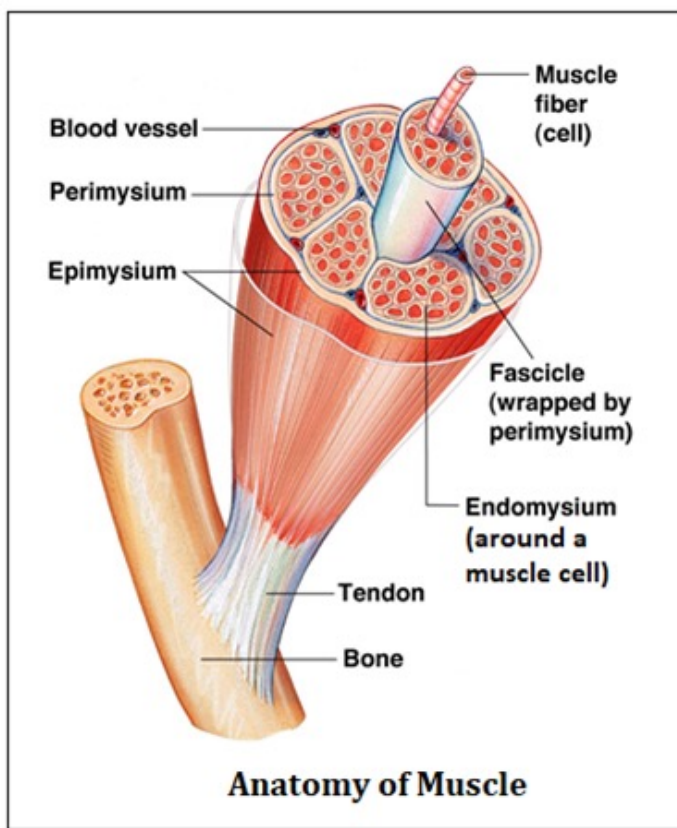
## Smooth Muscle



- Short, spindle-shaped cells
- One central nucleus
- No striations
- Deep

*\*All three muscle types require  $\text{Ca}^{2+}$  for contraction*





# SKELETAL MUSCLE ANATOMY

**Tendons** connect muscle to bone while **ligaments** connect bone to bone

**Epimysium:** Connective tissue around a muscle

A muscle is composed of a bundle of muscle fascicles

**Perimysium:** Connective tissue around a muscle fascicle

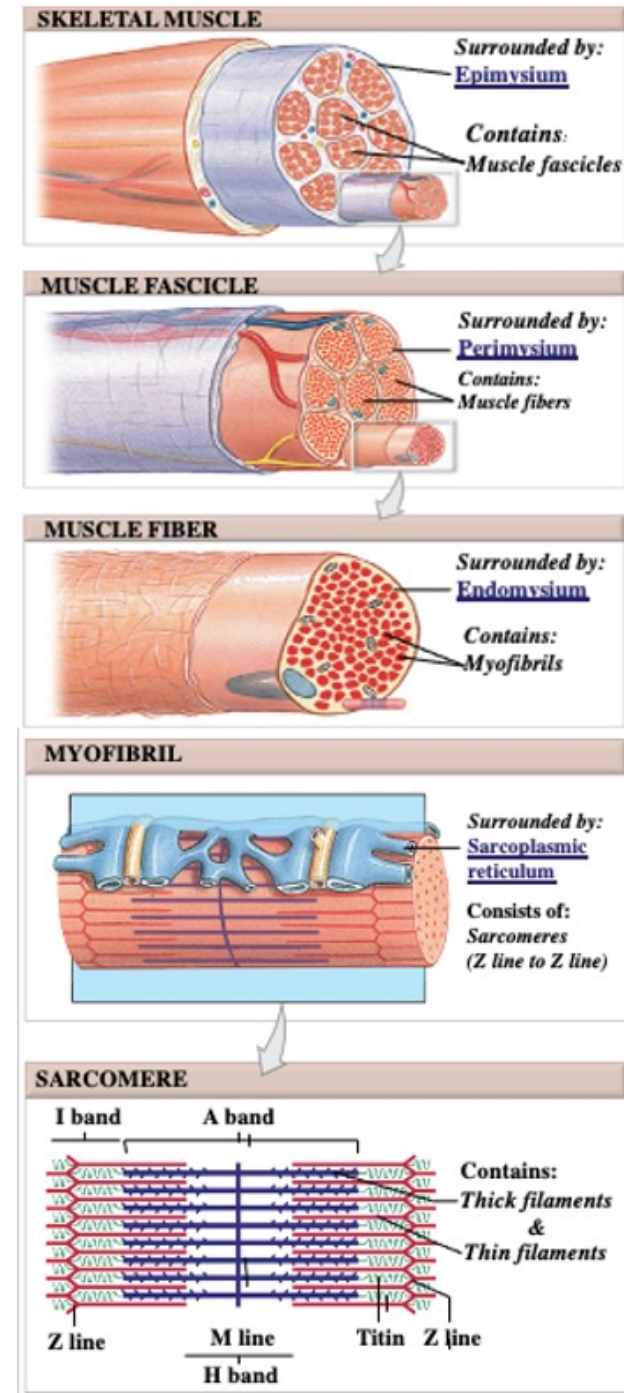
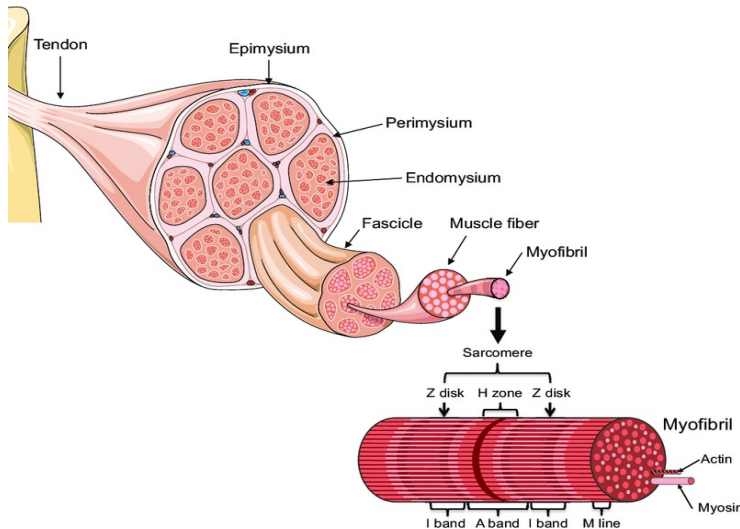
- A fascicle is composed of a bundle of muscle fibers

**Endomysium:** Connective tissue around a muscle fiber

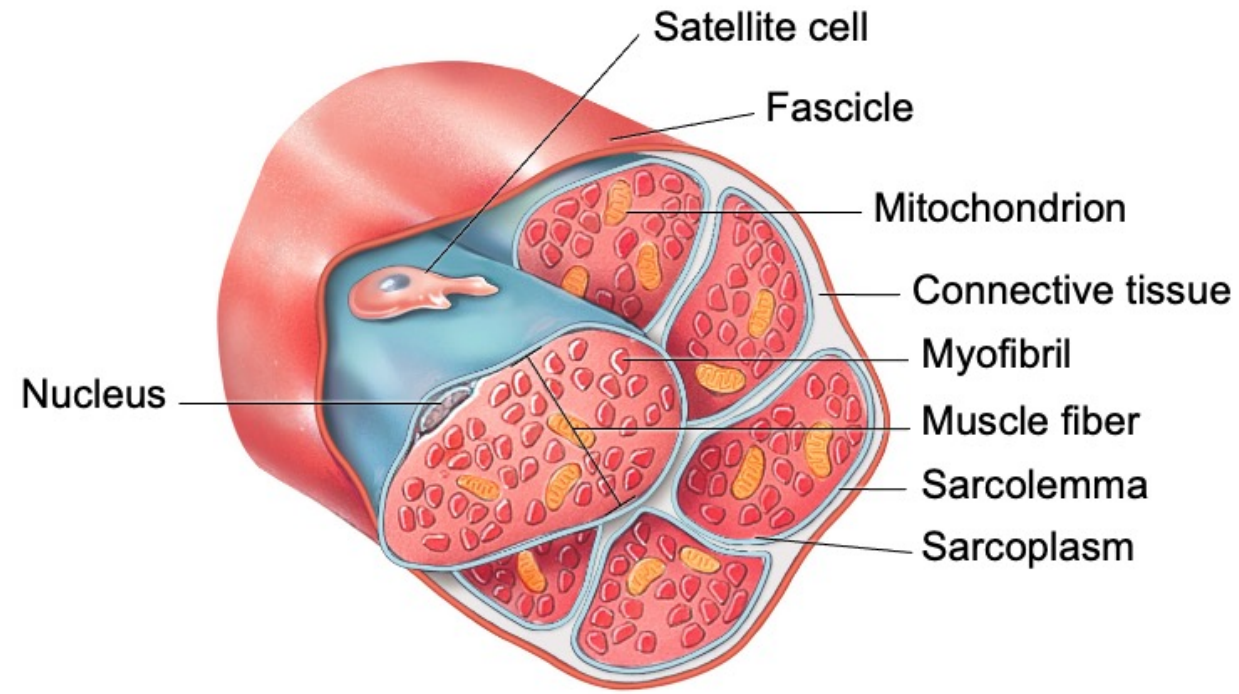
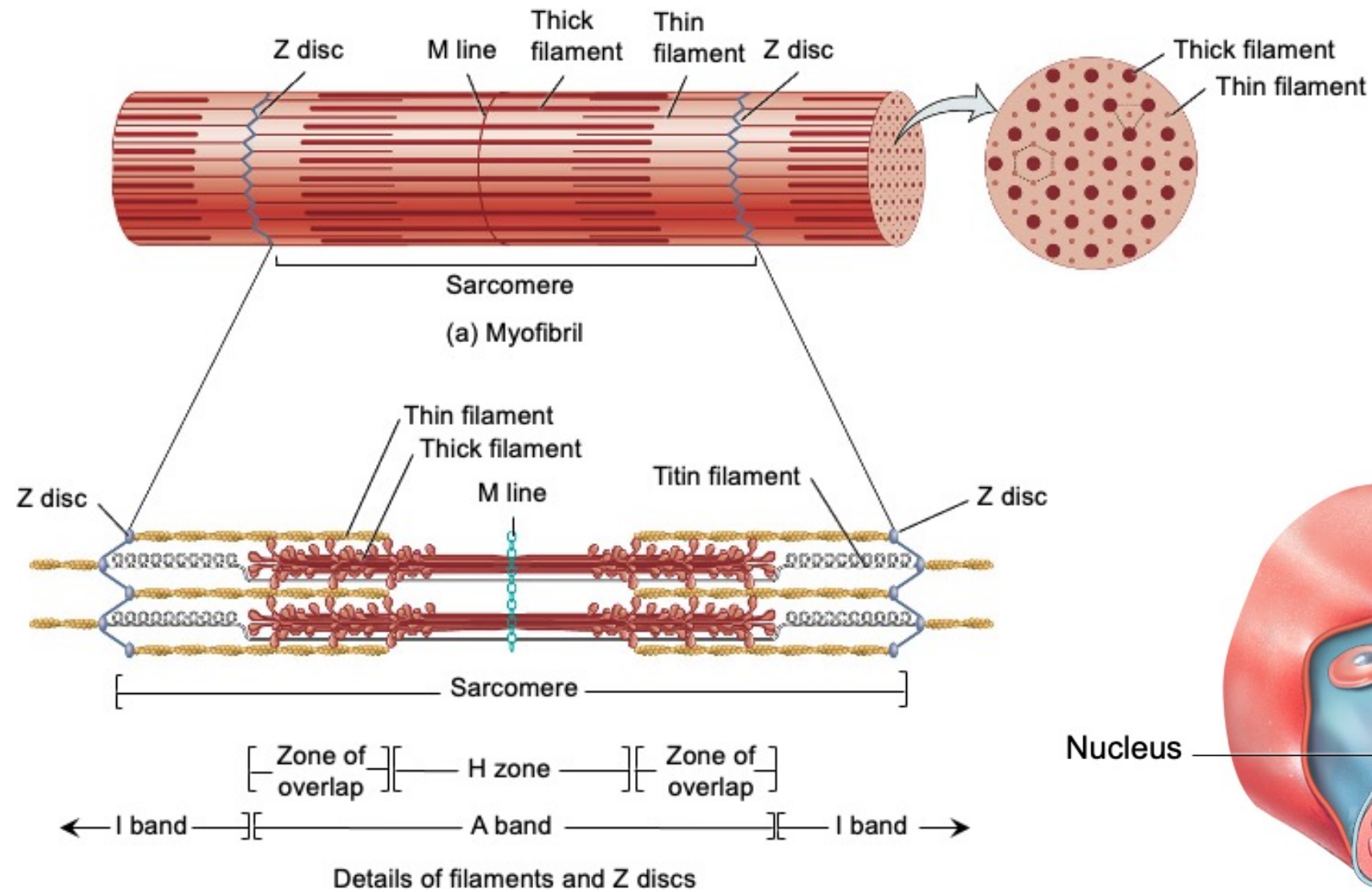
A muscle fiber is composed of a bundle of myofibrils

Organization from largest to smallest: **Muscle > Fascicle > Muscle Fiber > Myofibril**

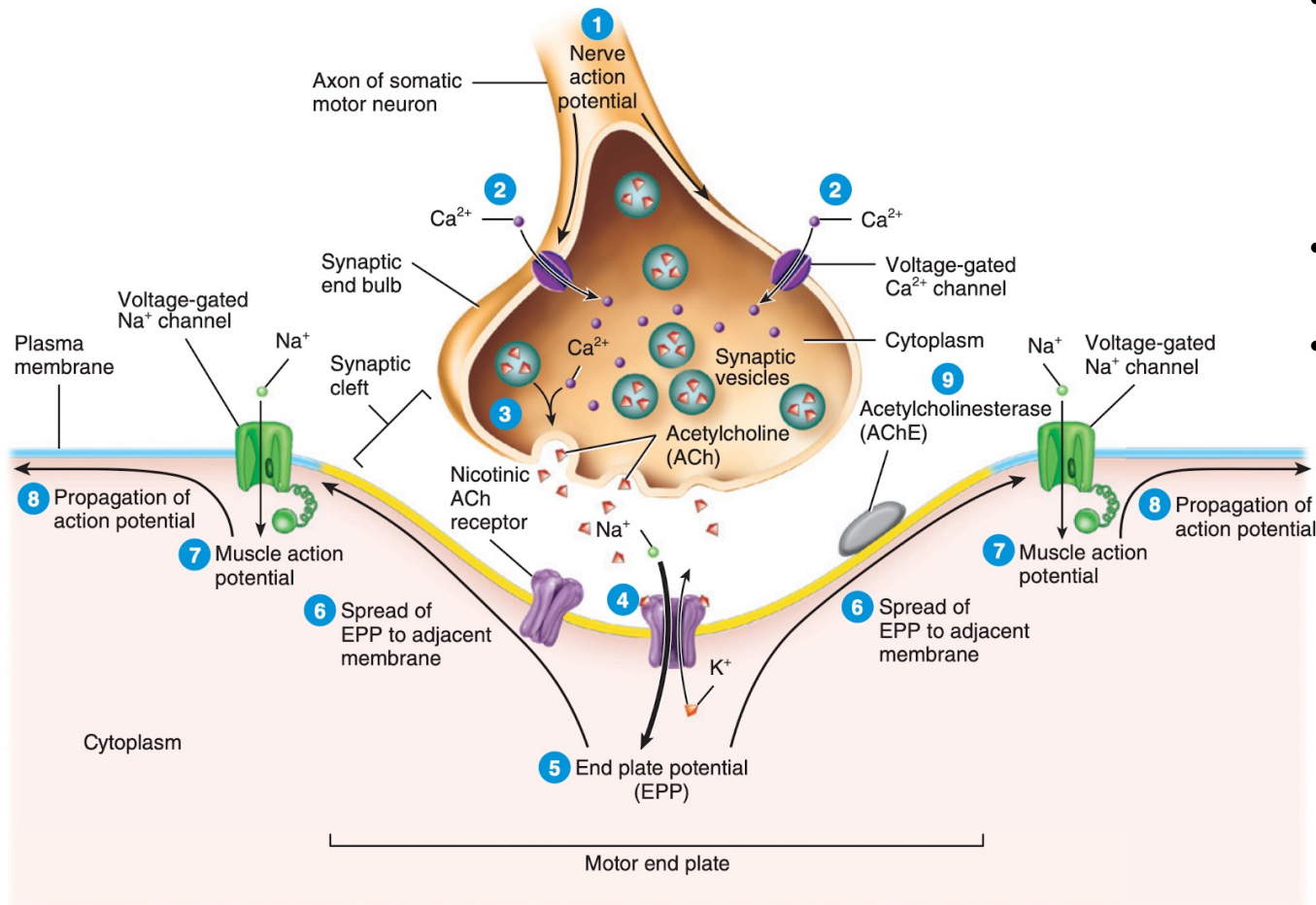
**Be able to identify all structures listed on this slide if provided with a picture!**







# THE NEUROMUSCULAR JUNCTION



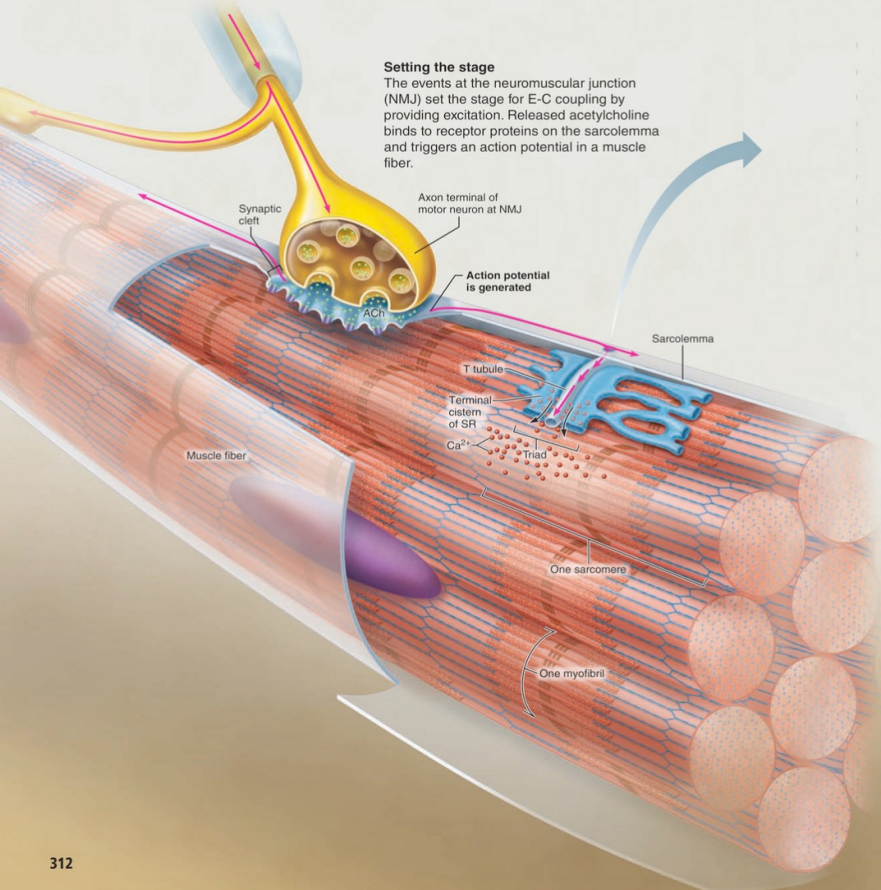
- **Neuromuscular Junction (NMJ):** Chemical synapse between a somatic motor neuron and a skeletal muscle fiber
  - Consists of the synaptic end bulb of a somatic motor neuron, a synaptic cleft (the gap between), and a motor end plate (the part of sarcolemma that has ACh receptors)
- **Neurotransmitter involved with skeletal muscle contraction:**
  - Acetylcholine (ACh) which has an excitatory effect
- **Events at the NMJ leading to muscle contraction:**
  1. A nerve impulse arriving at the axon terminal triggers the opening of voltage-gated calcium channels. Extracellular calcium flows in, binds to synaptic vesicles containing the neurotransmitter acetylcholine, and induces their exocytosis.
  2. Acetylcholine diffuses across the synaptic cleft and binds to its ACh receptors on the motor end plate, causing the opening of ligand-gated sodium ion channels.
  3. The rapid influx of sodium into the muscle cell causes a local depolarization called an end-plate potential, which will lead to a muscle action potential.



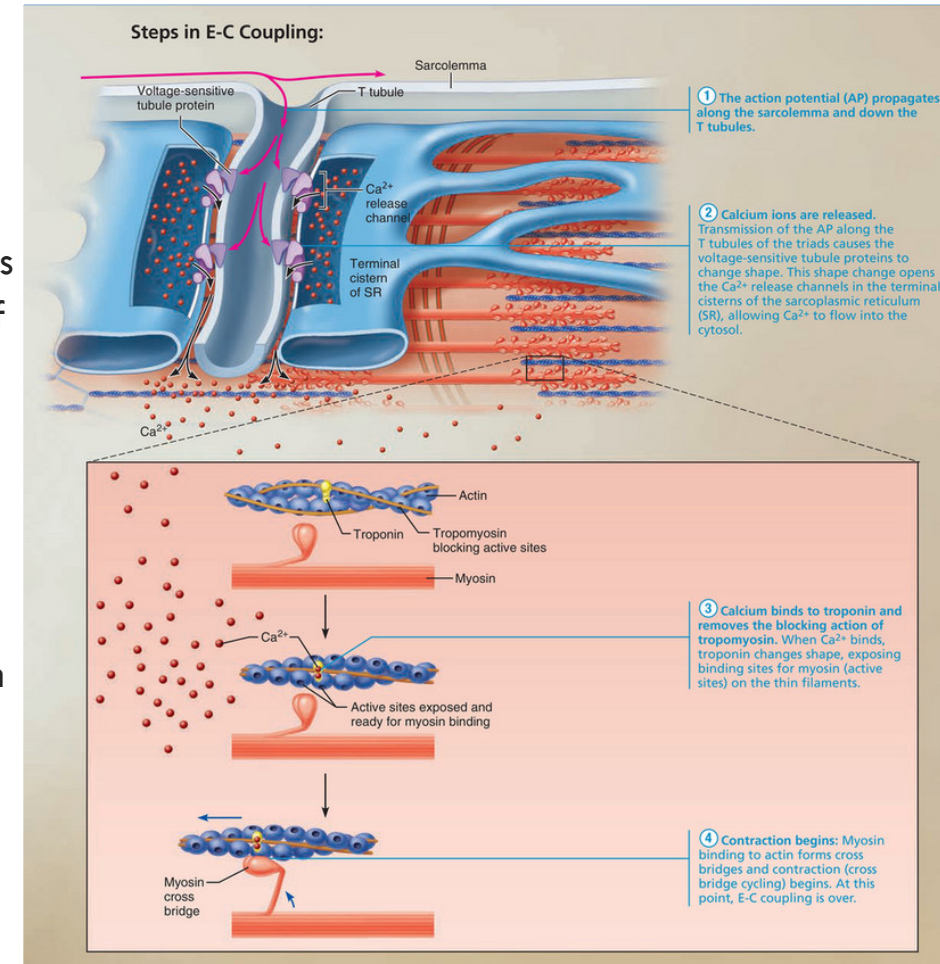
# EXCITATION-CONTRACTION COUPLING

**Focus Figure 9.2** Excitation-contraction (E-C) coupling is the sequence of events by which transmission of an action potential along the sarcolemma leads to the sliding of myofilaments.

Watch full 3-D animations  
MasteringA&P® Study Area A&P Flix



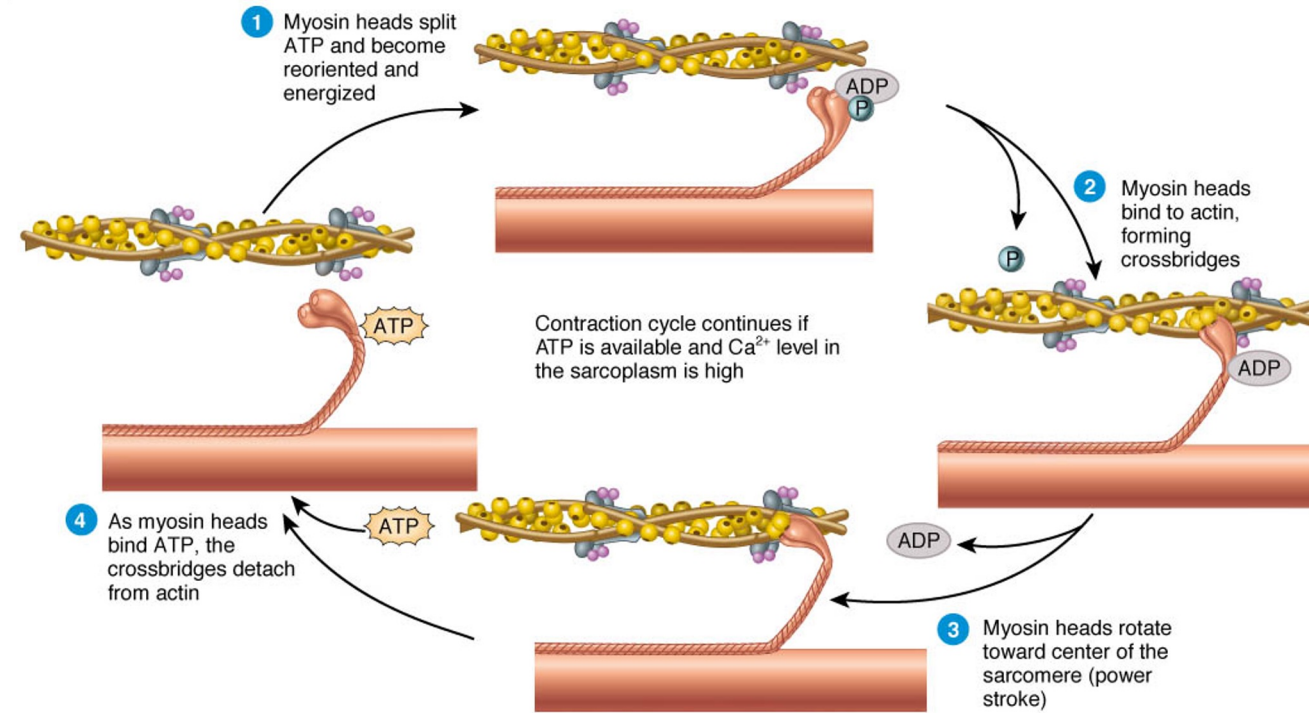
4. The action potential propagates along the sarcolemma of the muscle fiber and down the transverse tubules (t-tubules), which are invaginations of the sarcolemma.
5. This triggers the opening of calcium-release channels on the terminal cisternae of the sarcoplasmic reticulum.
6. Calcium flows out into the sarcoplasm in the vicinity of the thin and thick filaments...





# SLIDING FILAMENT THEORY

1. Calcium ions ( $\text{Ca}^{2+}$ ) in the sarcoplasm bind to Troponin C. This causes changes to Troponin I & T.
2. These changes in Troponin cause a conformational change in Tropomyosin, which exposes the active site on Actin.
3. Myosin will be in a cocked (tilt) position due to the hydrolysis of ATP into ADP and Phosphate ( $[\text{PO}_4]^{3-}$ ).
4. Myosin head binds to actin forming a cross-bridge, Phosphate ion is released simultaneously.
5. **Muscle contraction:** During a power stroke (contraction) Myosin heads rotate towards the center of the sarcomere which causes it to pull on actin filaments.
  - ADP molecule is released because its stored energy has been used up during the power stroke.
6. **Muscle relaxation:** After a power stroke, a new ATP molecule binds to the Myosin head which causes it to detach from Actin (cross-bridge is broken).
  - This allows for the muscle to relax temporarily before another cycle takes place.
7. The cycle begins again when the ATP molecule in Myosin is hydrolyzed to ADP and Phosphate. This repeated power stroke is called the “ratchet mechanism”
8. When contraction stops, Calcium is pumped back into the Sarcoplasmic Reticulum via Calcium pumps located on the Terminal Cisternae.



Calcium is taken back into the SR at the end of muscle contraction. Is ATP required during this process?

- **Yes, ATP is required to pump  $\text{Ca}^{2+}$  back into the sarcoplasmic reticulum.**

The process of muscular contraction can last for as long as there is adequate ATP and  $\text{Ca}^{+}$  stores.

# ACETYLCHOLINE

## 1. What role does Acetylcholine have in skeletal muscle contraction?

- Acetylcholine (ACh) functions as a ligand and binds to acetylcholine receptors (ACh-Rs). This causes the opening of ligand-gated sodium channels, which allows for the influx of sodium from the synaptic cleft. This leads to depolarization and end-plate potential

## 2. What would happen if a drug/poison blocked ACh receptors?

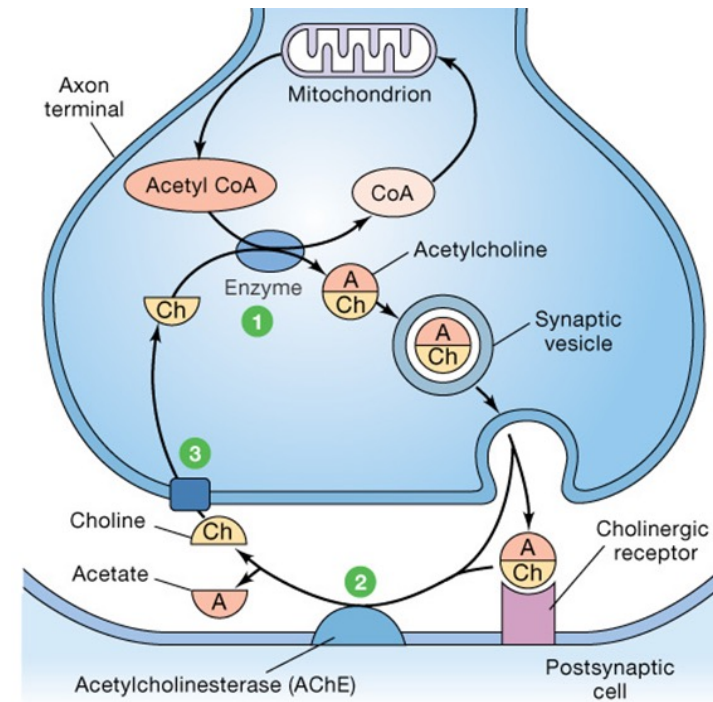
- No muscle contraction as acetylcholine would be unable to bind to its receptors, preventing sodium from entering the post-synaptic cell. No sodium entering means no depolarization can occur. Can prevent movement and depending on severity lead to death

## 3. What would happen if calcium channels on the terminal cisternae were blocked?

- No muscle contraction since calcium would be unable to bind troponin

## 4. What causes its binding effects to be brief?

- The enzyme acetylcholinesterase (AChE) catalyzes the breakdown of acetylcholine (ACh) in the body



1 Acetylcholine (ACh) is made from choline and acetyl CoA.

2 In the synaptic cleft ACh is rapidly broken down by the enzyme acetylcholinesterase.

3 Choline is transported back into the axon terminal and is used to make more ACh.

An anatomical illustration of a human shoulder joint, showing the humerus, scapula, and clavicle bones. The surrounding muscles, including the deltoid and rotator cuff muscles, are depicted in a realistic, reddish-pink color with visible fiber structure. Blue and red lines represent blood vessels, and green lines represent nerves. The word "PROTOCOL" is written in white, uppercase letters across the center of the image.

PROTOCOL

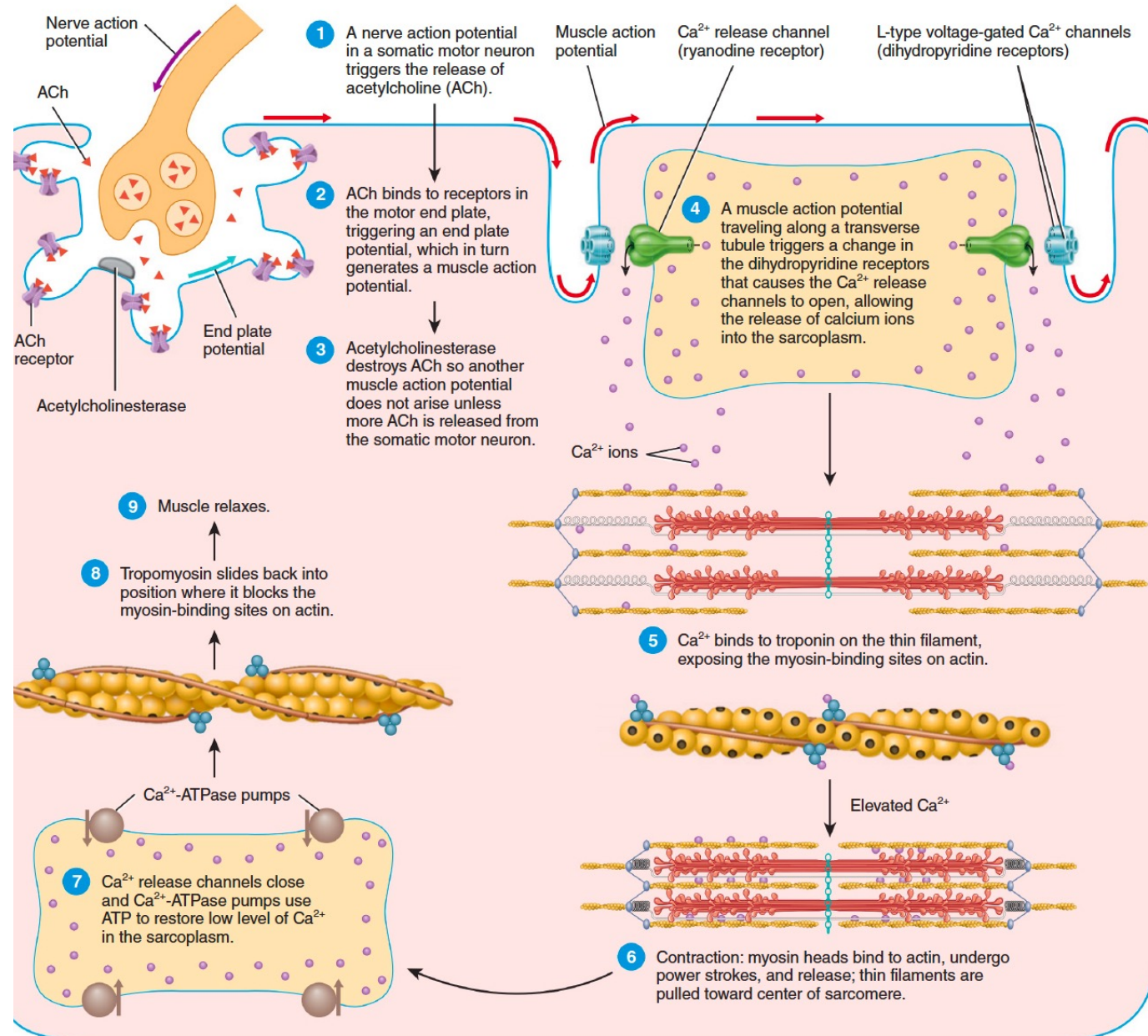


# ACTIVITY 1: SUMMARY

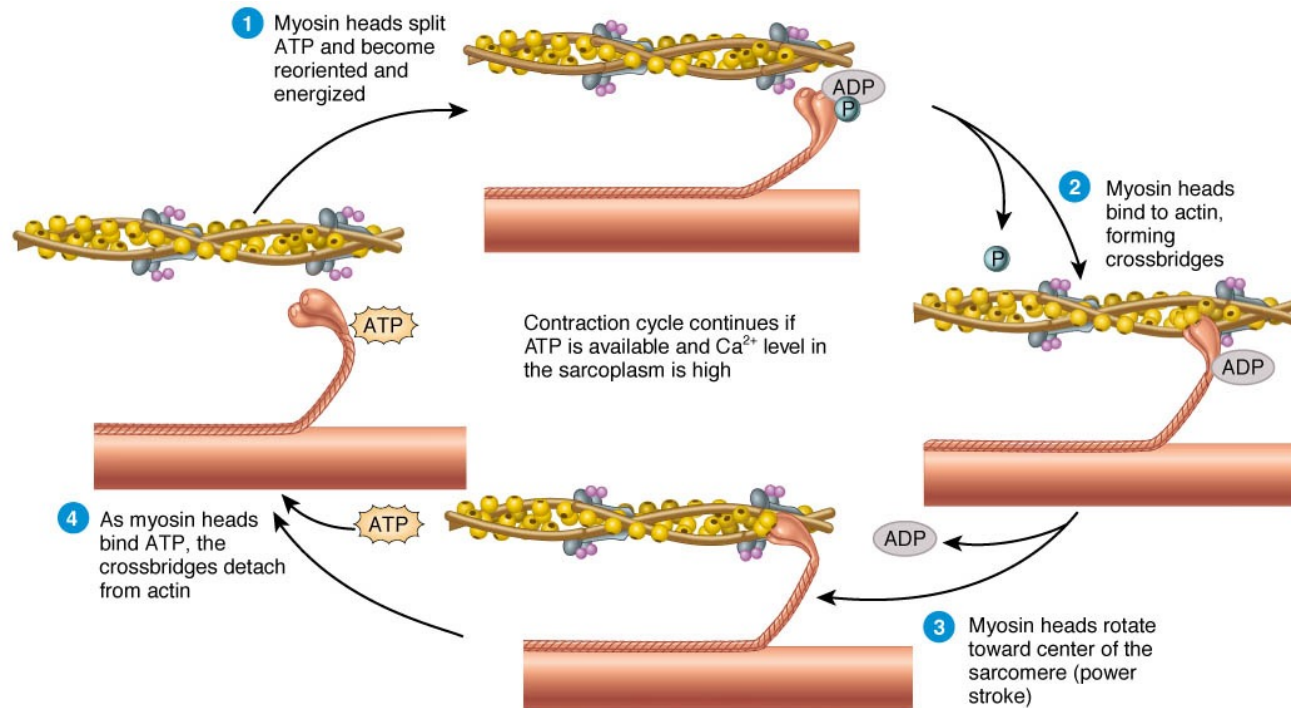
Calcium ions	Troponin	Tropomyosin
Thin	Myosin head	Thick
ATP	contraction	relaxation
Neuromuscular junction	Sarcoplasmic reticulum	Motor end plate

1. When a nerve impulse arrives at the Neuromuscular junction, calcium ions are released from the Sarcoplasmic reticulum.
2. Calcium ions attach to troponin causing a change in conformation (shape)
3. Tropomyosin moves away, exposing myosin binding sites on the actin thin (thin/thick) filaments.
4. Myosin heads on thick (thin/thick) filaments bind with actin binding sites on thin filaments, forming a cross bridge, causing muscle contraction.
5. A (n) ATP molecule binds to the myosin head. This causes the myosin head to detach, causing muscle relaxation.

# ACTIVITY 1: SUMMARY



# SLIDING FILAMENT THEORY - PROTOCOL



## 1. Which ion stimulates contraction?

- Calcium ( $\text{Ca}^{2+}$ )

## 2. Where does this ion bind to?

- Troponin

## 3. Where is it released from?

- Sarcoplasmic Reticulum

## 4. Which filament moves, and which does not move?

- Actin (thin) filaments move
- Myosin (thick) filaments do not move

**Myosin has two binding sites! One is for Actin, the other is for ATP (which will cause ATP hydrolysis).**



# CONTRACTION OF GLYCERINATED MUSCLE WITH ATP

- For contraction to occur, muscles need both ATP and salts
- Unlike living muscle, glycerinated muscle does not require the presence of  $\text{Ca}^{2+}$  to contract. The **glycerination process** disrupts a regulatory mechanism known as the troponin/tropomyosin complex, and with it the need for  $\text{Ca}^{2+}$ . ATP, however, is still needed to induce contraction.
- Three solutions were used during this experiment:
  - A.  $\text{KCl} + \text{MgCl}_2$  in Distilled water → Only ions
  - B. 0.2% ATP + Distilled water (DI water) → Only ATP
  - C. 0.2% ATP +  $\text{KCl} + \text{MgCl}_2$  in Distilled water → ATP & ions

# RESULTS

- **Prominent contraction:**

- 0.2% ATP + KCl + MgCl<sub>2</sub> in Distilled water
- This solution contains ATP and salts (ions) so you will have the most contraction here

- **Slight contraction:**

- 0.2% ATP + Distilled water
- This solution DOES NOT contain ions, but it contains ATP so that causes a slight contraction when the ATP is hydrolyzed

- **No contraction:**

- KCl + MgCl<sub>2</sub> in Distilled water
- This solution contains salts (ions) but it DOES NOT contain ATP and without ATP the muscle cannot contract

**You ALWAYS need ATP for a muscle contraction to take place. If ATP is NOT present, there is no energy to do work.**

# MUSCLE FATIGUE

## What is muscle fatigue?

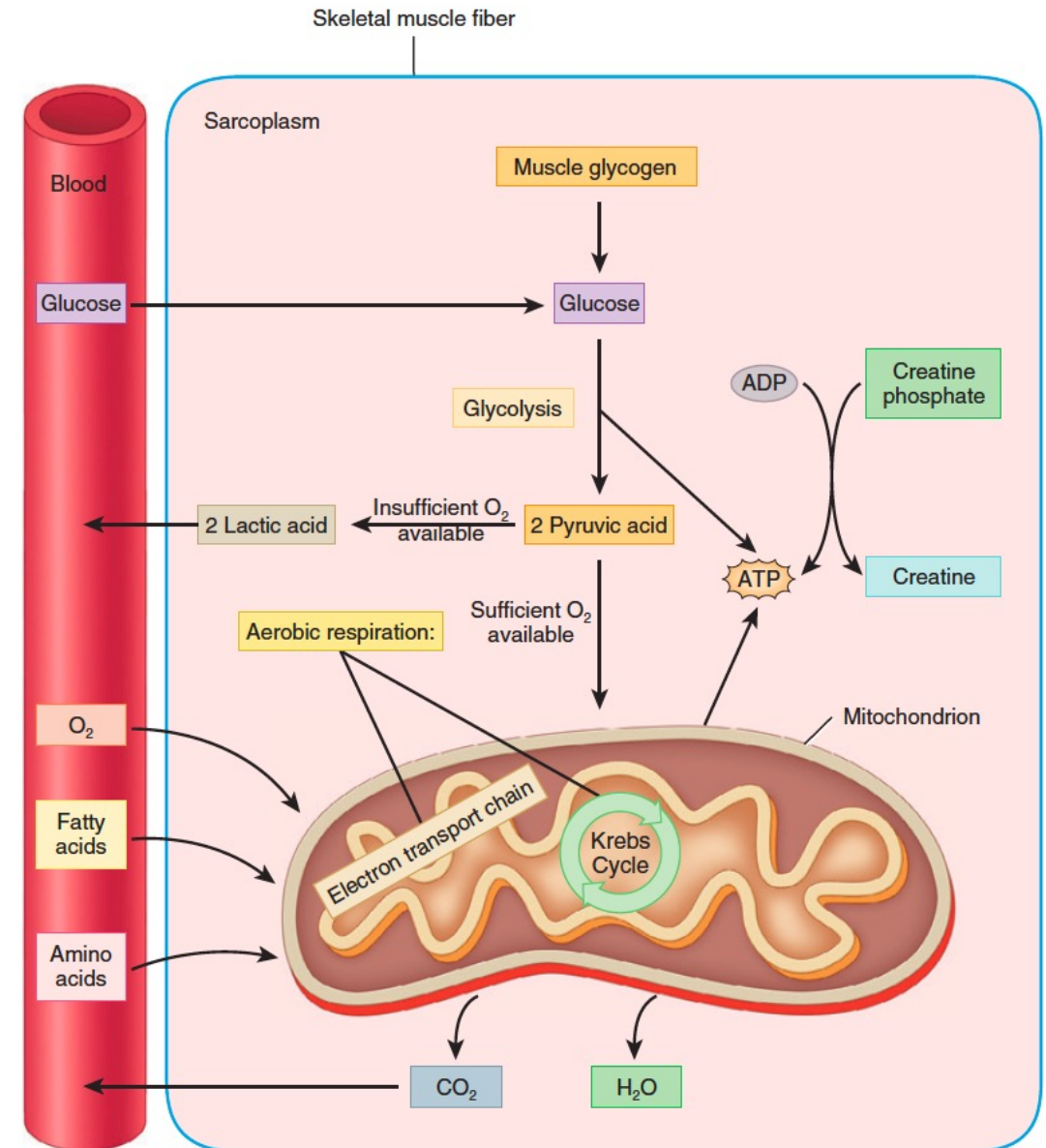
- Muscle fatigue is the decline in ability of muscles to generate force (contractions) because of a lack of energy. This lack in energy is due to the lack of oxygen, which normally fuels the aerobic process of ATP production.

Skeletal muscles generate energy from the process known **cellular respiration**. This process uses glucose, fatty acids, amino acids as reactants to produce ATP.

- When oxygen is not readily available, our muscle cells proceed with the **anaerobic pathway**, converting glucose to pyruvate, which is then converted to lactic acid via **lactic acid fermentation**. This process is faster but generates less ATP
- When oxygen is readily available, pyruvate enters the mitochondria for the **aerobic pathway**: the Krebs cycle and oxidative phosphorylation. This process is slower but generates much more ATP.

## Why is it important to rest in between sets or workouts?

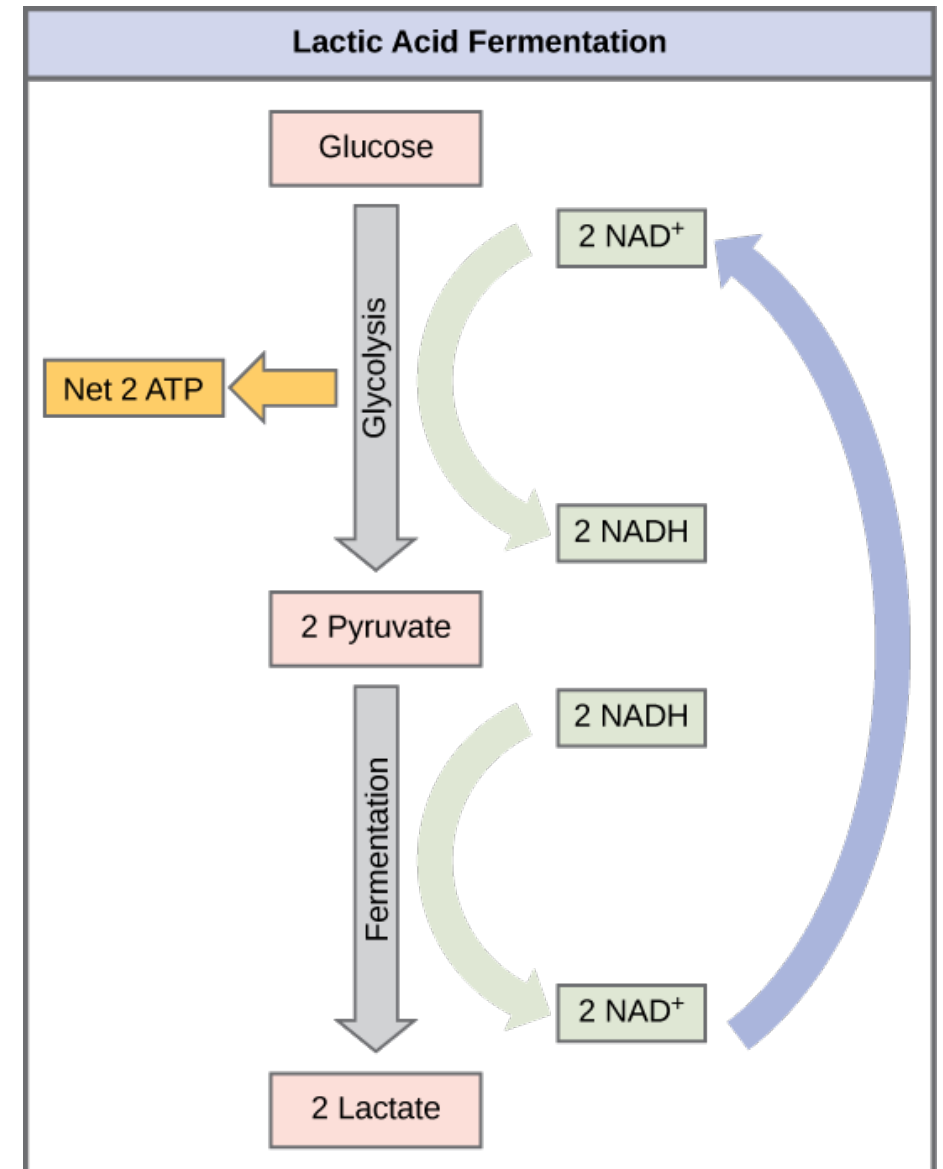
- Resting is important to reduce muscle fatigue and have more endurance during your other sets of exercises. During rest periods, the body switches to aerobic respiration which is a more efficient way to produce ATP, replenishes the oxygen debt incurred during exercise, and clears the accumulated lactic acid.





# MUSCLE FATIGUE

- At what point did you feel the most burning sensations?  
At the beginning, middle, or end?
  - Towards the end
- Lack of which gas, causes the formation of lactic acid?
  - Oxygen
- Why is the body required to create lactate/lactic acid during intense workouts?
  - Not enough oxygen available to break down glucose for energy
- What organ is responsible for getting rid of excess lactate?
  - Lactate exits the cells and is transported to the liver, where it is oxidized back to pyruvate and ultimately converted to glucose via the cori cycle

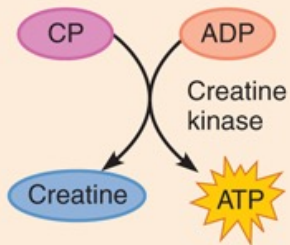


# ENERGY FOR SKELETAL MUSCLE CONTRACTION

## Direct Phosphorylation

Coupled reaction of creatine phosphate (CP) and ADP

Energy source: CP

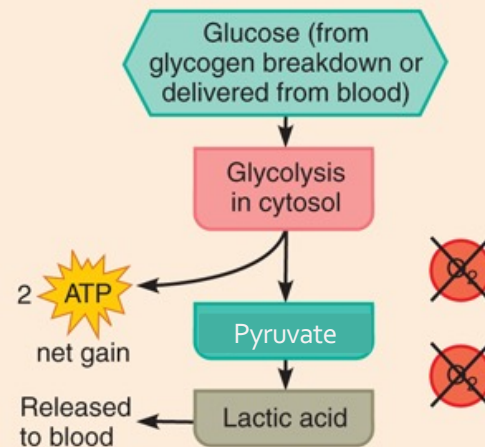


**Oxygen use:** None  
**Products:** 1 ATP per CP, creatine  
**Duration of energy provided:** 15 seconds

## Anaerobic Pathway

Glycolysis and lactic acid formation

Energy source: glucose

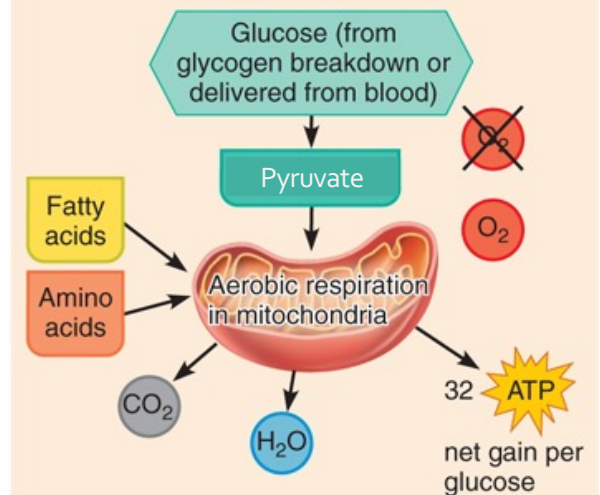


**Oxygen use:** None  
**Products:** 2 ATP per glucose, lactic acid  
**Duration of energy provided:** 30–40 seconds, or slightly more

## Aerobic Pathway

Aerobic cellular respiration

Energy source: glucose; pyruvic acid; free fatty acids from adipose tissue; amino acids from protein catabolism



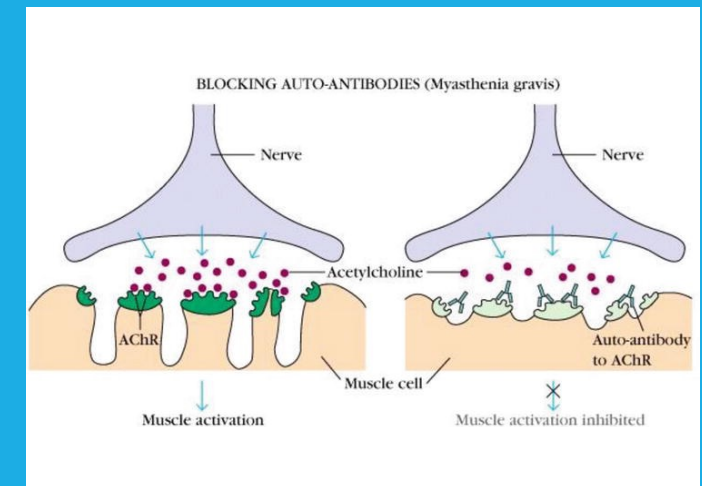
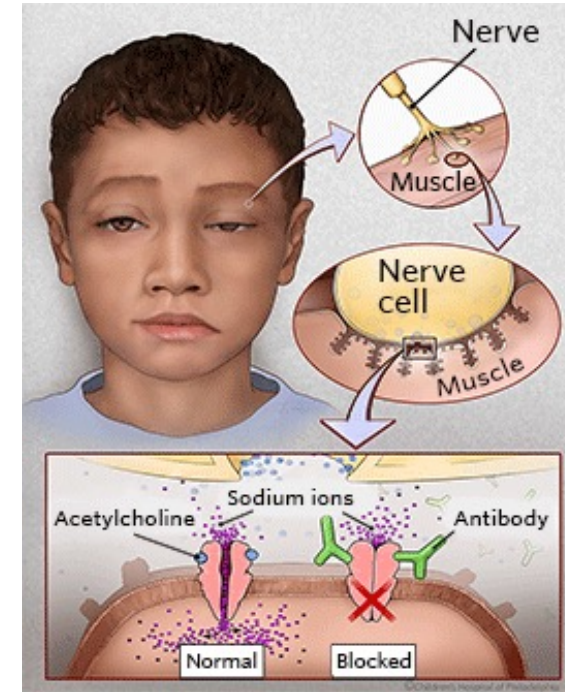
**Oxygen use:** Required  
**Products:** 32 ATP per glucose, CO<sub>2</sub>, H<sub>2</sub>O  
**Duration of energy provided:** Hours

# CLINICAL APPLICATION: MYASTHENIA GRAVIS

- An **autoimmune disease** involving the neuromuscular junction (NMJ), characterized by skeletal muscle weakness.
  - The immune system produces antibodies that bind and block acetylcholine (ACh) receptors. This leads to a decrease in the number of functional receptors at the motor end plate of skeletal muscle. As more and more receptors are damaged and lost, this causes the muscles to get weaker and fatigue quicker.
  - The absence of functional acetylcholine receptors results in the inability of muscle contraction, leading to *acute flaccid paralysis* since the muscles are in a constant state of relaxation. This condition also prevents sodium from entering the post-synaptic muscle cell since ACh cannot bind to its receptors.

## Treatment options include:

- Anticholinesterase drugs, which inhibit the enzyme acetylcholinesterase (AChE). This allows more acetylcholine to be present, which can bind to the receptors that are still functional.
- Immunosuppressants.





# CLINICAL APPLICATION: BOTOX - BOTULINUM TOXIN

- It prevents the release of the neurotransmitter acetylcholine from the axon terminal at the neuromuscular junction, causing flaccid paralysis.
- Although this is the **same toxin** that causes the disease **botulism** — a life-threatening form of food poisoning — its effects vary according to the amount and type of exposure.



# CLINICAL APPLICATION: RIGOR MORTIS

- Latin: rigor "**stiffness**", and mortis "of **death**"
- Characterized as the stiffening of the joints & muscles after death
- After death, the cell membrane becomes leaky, which allows calcium to leak out from the sarcoplasmic reticulum, entering the sarcoplasm
  - Allows for the cross-bridge to form. Myosin heads bind to actin
- After breathing stops, ATP synthesis ceases because there is no oxygen available
  - Cross-bridge cannot be broken down without ATP
- Muscles cannot relax so it remains in a rigid state (spastic paralysis)

# CLINICAL APPLICATION: TETANUS

- Tetanus is an infection caused by the bacteria known as *Clostridium tetani*, which releases tetanus toxin.
- The toxin causes the activation of neurons that stimulate muscles to contract, leading to prolonged and painful muscle spasms, a condition known as tetanus. Without treatment, it can be fatal.
- **Mechanism:** The toxin prevents the release of inhibitory neurotransmitters (Glycine & GABA), thus preventing muscle relaxation (resulting in spastic paralysis) due to continuous stimulation by the excitatory neurotransmitter (ACh).
- Tetanus is commonly referred to as “lockjaw” because it can cause the jaw muscles to lock, making it hard to open the mouth or swallow.

