

# What is the Sliding Filament Theory?

## Worksheet

According to the sliding filament theory, myosin heads repeatedly bind actin, pull it toward the sarcomere's center (power stroke), release, and re-bind further along - sliding actin over myosin and shortening the muscle.

## Questions

1. According to the sliding filament theory, muscle contraction occurs because:
  - A) Actin and myosin filaments shorten
  - B) Actin filaments slide past myosin filaments, pulling Z-discs closer together
  - C) Myosin filaments shrink in length
  - D) Sarcomeres are destroyed and rebuilt
2. What triggers the exposure of myosin-binding sites on actin?
  - A) ATP hydrolysis
  - B) Calcium binding to troponin
  - C) Sodium influx into the sarcoplasm
  - D) Myosin head detachment
3. What happens to the myosin head immediately after the power stroke?
  - A) It binds a new ATP molecule and detaches from actin
  - B) It stays permanently bound to actin
  - C) It splits into two separate heads
  - D) It converts directly into actin
4. During muscle contraction, which sarcomere zone narrows?
  - A) A band
  - B) I band
  - C) M line only
  - D) Titin filament length
5. During a bicep curl, calcium ions flood the sarcoplasm. What happens to the troponin-tropomyosin complex, and why does it matter for contraction?
6. A muscle cell completely runs out of ATP (as happens after death, causing rigor mortis). What happens to its cross-bridges?
7. A sarcomere shortens from 2.2 m to 2.0 m during contraction. What happens to the A band versus the I band?
8. Define: What is the sliding filament theory?
9. Define: What role does calcium play in contraction?
10. Define: What powers the cross-bridge cycle?

## Answer Key

1. B) Actin filaments slide past myosin filaments, pulling Z-discs closer together - Filament lengths don't change - thin actin filaments slide over thick myosin filaments, pulling the Z-discs inward and shortening the sarcomere.
2. B) Calcium binding to troponin -  $\text{Ca}^{2+}$  released from the sarcoplasmic reticulum binds troponin, shifting tropomyosin and exposing actin's myosin-binding sites.
3. A) It binds a new ATP molecule and detaches from actin - A fresh ATP molecule binds the myosin head, causing it to release from actin and reset for another cycle.
4. B) I band - The I band (actin-only region) narrows as actin slides deeper into the A band; the A band's own width stays constant.
5.  $\text{Ca}^{2+}$  released from the sarcoplasmic reticulum binds to troponin Troponin changes shape and pulls tropomyosin away from the myosin-binding sites on actin Myosin heads can now attach to actin, beginning the cross-bridge cycle and contraction
6. Without ATP, myosin heads cannot release from actin (ATP binding is required for detachment) All cross-bridges lock in the bound position The muscle becomes stiff and cannot relax - this is rigor mortis
7. The A band (spans the full length of the myosin filament) stays the same width - myosin doesn't shrink The I band (actin only, no myosin overlap) narrows, because actin slides further into the A band The H zone (myosin only, no actin overlap) also narrows or disappears as overlap increases
8. The model explaining that muscle contraction occurs when actin filaments slide over myosin filaments, shortening the sarcomere, without either filament changing length.
9.  $\text{Ca}^{2+}$  binds troponin, shifting tropomyosin off actin's myosin-binding sites so cross-bridges can form.
10. ATP hydrolysis - it cocks the myosin head and, when new ATP binds, detaches it from actin.

### **Bounlu**

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