

## Muscle Excitation/Contraction (E/C Coupling)

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### INTRODUCTION

Here, we'll learn the coupling of skeletal muscle excitation and contraction.

#### Muscle Cell Environment

##### Sarcolemma

The sarcolemma is the plasma membrane.

##### Myofibril

Myofibrils possess dark/light stripes. They comprise:

- **A Band**, which refers to the length of the thick filaments, "think "A" for d-a-rk.
- **I Band**, which is the region along the thin filaments, between the thick filaments. Think "I" for L-i-ght.

##### Sarcoplasmic reticulum

The sarcoplasmic reticulum is organized into web-like, longitudinal rows. They store calcium and are a key component to coupling muscle cell excitation to myofibril contraction.

##### Terminal cisternae

We show bands of transversely-oriented terminal cisternae (aka lateral sacs) of the sarcoplasmic reticulum. They flank transverse tubules (T-Tubules – tubular invaginations of sarcolemma).

##### Cytosol

Cytosol constitutes the internal milieu of the myofibril.

##### Sarcoplasm

Sarcoplasm fills the internal milieu of the muscle cell.

##### Mitochondria

Mitochondria are the energy powerhouses of the cell – the site of aerobic respiration, which by definition requires oxygen but is capable of generate the most amount of ATP.

##### Synaptic terminal

We also show a synaptic terminal (aka terminal bouton) of a typical motor nerve, which possess molecules of

### MYOFILAMENTS

#### Thin Filaments

Thin filaments notably comprise:

- Actin, which are spherical molecules joined in pairs of double helical strands (like beads on a string),
- Tropomyosin, which are threadlike strands, and

- Troponin protein complexes that bind tropomyosin, actin, and also calcium. We show their calcium-binding sites.

### **Myosin Binding Sites**

Next, indicate the myosin binding sites on actin.

### **ATPase Site**

There is also an ATPase site, an ATP-splitting site.

### **Thick Filaments**

Next, show that thick filaments of myosin, which constitute heavy and light chains form a golfclub shape, and possess a head, neck, and tail.

## **EXCITATION**

Now, let's show the steps of excitation.

### **Ach binds a receptor**

Show that acetylcholine binds a post-synaptic receptor on muscle, which triggers an action potential.

- The action potential proceeds along the T Tubule to a dihydropyridine receptor (it is blocked by dihydropyridine, hence its name).

- Depolarization of the dihydropyridine receptor activates the ryanodine receptor (aka foot proteins, calcium-release channels) within the terminal cisternae, which triggers the release of calcium into the cytosol.

### **Calcium binds troponin**

Show that calcium binds troponin. This causes a shift in tropomyosin, moving it away from its blocking position along actin, which allows myosin to bind actin.

### **Myosin binds actin**

Myosin binds actin and proceeds through thin filament sliding (muscle contraction), which we draw at the end.

### **Contraction cessation**

Show that when the action potentials cease, calcium releases from troponin and returns to the cytoplasm. Tropomyosin shifts and myosin is now unable to bind actin. The contractions cease.

## **HUXLEY SLIDING-FILAMENT MODEL**

Now, let's draw key steps in the Huxley Sliding-Filament Model.

### **Myosin binding**

Draw a myosin head bound to the thin filament.

### **Actin release**

Next, we see that ATP induces release of actin.

### **ATP binds myosin**

Redraw myosin with ATP bound to its head.

### **Actin separates**

Then, draw the actin molecules as separated from (no longer bound to) the myosin.

### **Rigor state**

Thus, we see that ATP is required to move out of the rigor state. If ATP is absent, which occurs after death, rigor will persist, called **rigor mortis**.

### **ATP hydrolysis**

Next, show that ATP is hydrolyzed to ADP and Inorganic phosphate (Pi) prompts the myosin head to rotate on the neck: it is now "cocked" – it's in its high-energy state.

### **"Cocked" state**

Now, show that the "cocked" state causes the thin and thick filaments to again bind via their cross-bridge. Show that ADP and inorganic phosphate (Pi) are still bound to the myosin head.

**Pi release**

Next, show that Pi release initiates the power stroke for the myosin head to release its energy. The thin filament begins its slide.

**Uncocked state**

Redraw the myosin in its uncocked, low energy state. At some point after the power stroke, ADP is released.

- Note that ADP and phosphate may be release simultaneously to initiated the power stroke.