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10.1 OVERVIEW OF MUSCLE TISSUE

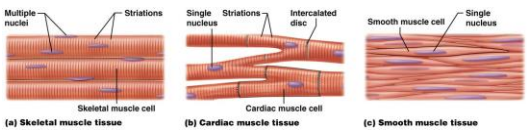
TYPES OF MUSCLE TISSUE

- The three types of cells in muscle tissue are **skeletal**, **cardiac**, and **smooth muscle** (**Figure 10.1**)
- Generating a force called **muscle tension** is a basic function *common* to <u>each</u> muscle tissue type

TYPES OF MUSCLE TISSUE

- Other **functions** of muscle tissue are to:
  - create *movement*
  - maintain *posture*
  - *stabilize* joints
  - generate *heat*
  - regulate the *flow* of materials through hollow organs

TYPES OF MUSCLE TISSUE



Both skeletal and cardiac muscle cells have **striations**, giving both cell types a *striped* appearance.

Figure 10.1 Three types of muscle tissue.

STRIATED MUSCLE TISSUE

- Skeletal muscle tissue is made up of long muscle cells arranged **parallel** to one another; some are <u>quite</u> long, extending nearly the <u>entire</u> *length* of the muscle
- Skeletal muscle cells are known as **fibers** due to their *length* and *appearance*; they are **multinucleated** cells whose contractions are **voluntary** (controlled by *conscious* thought)

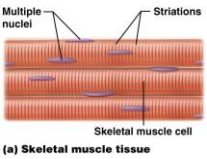


Figure 10.1a Three types of muscle tissue.

STRIATED MUSCLE TISSUE

- Most are found attached by **connective tissue** to the **skeleton**, where their contraction can produce *movement* of a body part

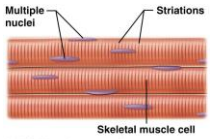


Figure 10.1a Three types of muscle tissue.

STRIATED MUSCLE TISSUE

- Cardiac muscle cells are found only in the **heart**
  - Each cell is short and highly **branched**, and has *one* to *two* **nuclei**
  - **Intercalated discs** join *adjacent* cells; they contain **gap junctions** and **desmosomes** (modified **tight junctions**) that both *unite* the cells and permit them to *coordinate contraction*
  - Contraction is **involuntary**, or not controlled by *conscious* thought

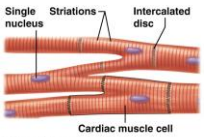


Figure 10.1b Three types of muscle tissue.

SMOOTH MUSCLE TISSUE

- Smooth muscle cells do not have **striations**, unlike skeletal and cardiac muscle tissue
- Smooth muscle cells are *long* and *flat* with "**spindle-shaped**" pointed ends and a single **centrally** located nucleus

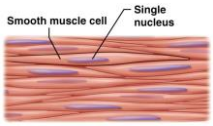


Figure 10.1c Three types of muscle tissue.

SMOOTH MUSCLE TISSUE

- Smooth muscle cells are found lining most **hollow organs** in the **eye**, **skin**, and some **glandular ducts**; their contractions are **involuntary**
- Many smooth muscle cells are linked to one another by **gap junctions**, allowing for **synchronized contraction**

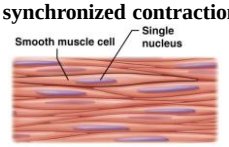


Figure 10.1c Three types of muscle tissue.

PROPERTIES OF MUSCLE CELLS

1. **Contractility** is the ability to *contract* where proteins in the cell draw closer together; this does not necessarily involve *shortening* of the cell
2. **Excitability** is the ability of a cell to respond to a **stimulus** (chemical, mechanical stretch, or local electrical signals)
3. **Conductivity** is the ability of a cell to conduct **electrical changes** across the entire **plasma membrane**

PROPERTIES OF MUSCLE CELLS

4. **Extensibility** is the ability of a cell that allows it to be **stretched** without being **ruptured** (up to 3 times their resting length without damage)
5. **Elasticity** is the ability of a cell that allows it to return to its **original length** after it has been **stretched**

Structure of Muscle Cells

- **Myocytes**, or muscle cells, are described using specialized terminology
  - The **sarcoplasm** is the myocyte's *cytoplasm*
  - The **sarcolemma** is the myocyte's *plasma membrane*
  - The **sarcoplasmic reticulum** (**SR**) is modified *endoplasmic reticulum* that:
    - Forms a *weblike network* surrounding the **myofibrils**
    - Varies in *structure* in the three types of muscle tissue (discussed later)

Structure of Muscle Cells

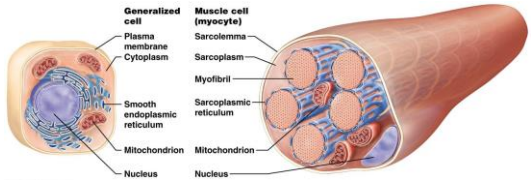


Figure 10.2 A generalized cell (left) compared with a generic muscle cell (right).

Structure of Muscle Cells

Myofibrils are unique structures found in each of the three muscle cell types:

- Cylindrical **organelles**, found in great numbers (100s to 1000s) in the myocyte, make up ***50–80***% of the **cell volume**
- Measure about *one micrometer* in **diameter** (about 1/100 the thickness of a human hair)

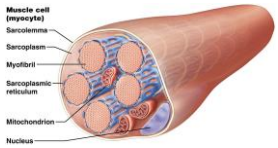


Figure 10.2 A generalized cell (left) compared with a generic muscle cell (right).

Structure of Muscle Cells

Myofibrils (continued):

- Made up of bundles of *specialized proteins* that allow for contraction
- Other *organelles* (such as mitochondria) are packed between the myofibrils
- Smooth muscle cells' myofibril arrangement is different than cardiac and skeletal muscle cells

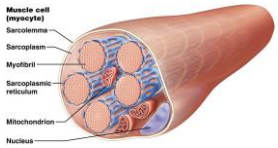


Figure 10.2 A generalized cell (left) compared with a generic muscle cell (right).

10.2 Structure and Function of the Skeletal Muscle Fiber

Structure of the Skeletal Muscle Fiber

Skeletal muscle tissue consists of many **fibers** and their surrounding **endomysium** (*extracellular matrix*)

- Skeletal muscle fibers are *thin cylinders* but can be quite long and thick
  - Fibers can reach *lengths* up to *30 centimeters* (the RBC below is only *7.5 micrometers* in diameter)
  - Fibers can reach a *thickness* up to *100 micrometers*

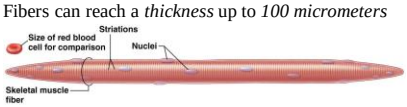


Figure 10.3 Size and shape of a skeletal muscle fiber.

Structure of the Skeletal Muscle Fiber

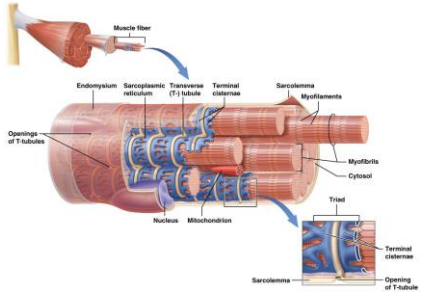


Figure 10.4 Structure of a skeletal muscle fiber.

Structure of the Skeletal Muscle Fiber

Skeletal muscle fibers are formed by the *fusion* of many embryonic **myoblasts** giving each fiber multiple nuclei

- The **myofibrils**, the most abundant organelle, are made up of mostly *contractile proteins*
- The **sarcoplasmic reticulum** (**SR**) surrounds the myofibrils and stores and releases **calcium ions**

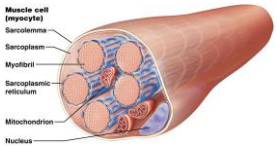
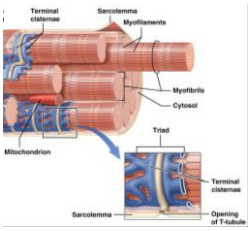


Figure 10.2 A generalized cell (left) compared with a generic muscle cell (right).

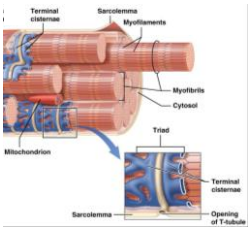
Structure of the Skeletal Muscle Fiber

- **Transverse tubules** (**T-tubules**) are deep inward extensions of sarcolemma that surround each myofibril
- T-tubules form a *tunnel-like* network within the muscle fiber, continuous with the **exterior** of the cell, and are therefore filled with **extracellular fluid**



Structure of the Skeletal Muscle Fiber

- **Terminal cisternae** are enlarged sections of SR found flanking each **T-tubule**
- Two terminal cisternae and their corresponding T-tubule form a **triad**



Structure of the Myofibril

Each myofibril is made of hundreds to thousands of **myofilaments**, which consist of one or more of the following types of proteins:

- **Contractile proteins** that generate *tension*
- **Regulatory proteins** that dictate when a fiber may *contract*
- **Structural proteins** that maintain proper myofilament *alignment* and fiber *stability*

Structure of the Myofibril

There are three types of **myofilaments** (**Figure 10.5**):

- **Thick filaments** are composed of bundles of the contractile protein **myosin**
- **Thin filaments** are composed of the proteins **actin**, **tropomyosin**, and **troponin**
- **Elastic filaments** are composed of a single massive, spring-like structural protein called **titin** that stabilizes the myofibril structure and resists excessive stretching force

Structure of the Myofibril

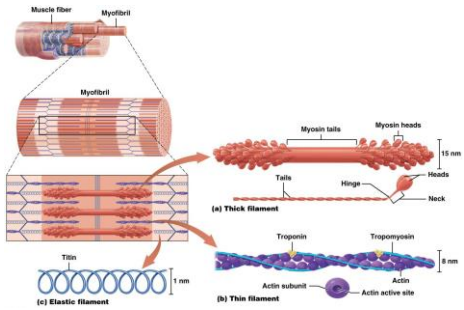


Figure 10.5 Structure of myofilaments.

Structure of the Myofibril

Thick filaments are composed of the contractile protein **myosin**

- Each myosin has **globular heads** at each end linked by *intertwining* **tails**
- Myosin heads are connected to the tails by a *hinge-like* **neck**
- Each myosin head has an **active site** that *binds* with **actin**

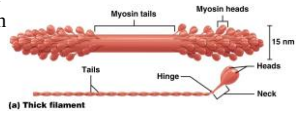


Figure 10.5a Structure of myofilaments.

Structure of the Myofibril

Thin filaments are composed of **actin**, **tropomyosin**, and **troponin**:

- **Multiple actin** subunits string together like beads on a necklace to form the *two intertwining strands* in the functional **thin filament**; each bead-shaped actin has an **active site** that binds with the **myosin heads** of the **thick filament**

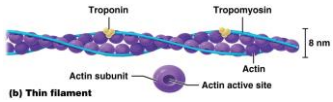


Figure 10.5b Structure of myofilaments.

Structure of the Myofibril

Thin filaments are composed of **actin**, **tropomyosin**, and **troponin** (continued):

- **Tropomyosin** is a long, *rope-like* **regulatory protein** that twists around **actin**, covering up its **active sites**
- **Troponin** is a small *globular* **regulatory protein** that holds **tropomyosin** in place and assists with turning contractions *on* and *off*

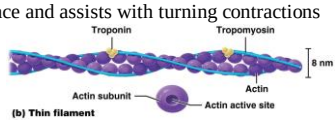
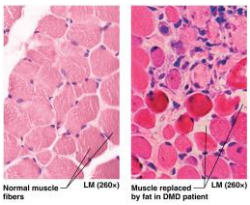


Figure 10.5b Structure of myofilaments.

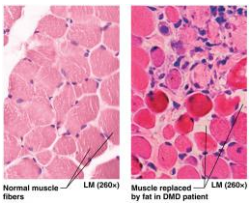
Duchenne Muscular Dystrophy (DMD)

- **DMD** is a degenerative muscular disease occurring almost exclusively in *boys*
- Caused by a defective gene for the protein **dystrophin**, coded on **X chromosome**
  - **Dystrophin** is a structural protein found in **striated muscle fibers** that anchors the sarcolemma to the surrounding connective tissue and to the myofibrils



Duchenne Muscular Dystrophy (DMD)

- In the absence of normal **dystrophin**, the sarcolemma breaks down and the muscle fiber is destroyed and replaced with **fatty** and **fibrous connective tissue**
- **Symptoms** (arising between 2 and 12 years of age) include weakness of the proximal limb muscles and a waddling gait; generally wheelchair-bound by age 12 and dead from **respiratory** or **cardiac failure** by age 20



Putting It All Together: The Big Picture of Skeletal Muscle Structure

- Multiple muscle fibers (surrounded by extracellular matrix called the **endomysium**) form a **fascicle**
- Each **fascicle** is surrounded by a layer of connective tissue called the **perimysium**
- **Bundles** of **fascicles** make up a skeletal **muscle**, which is surrounded by the **epimysium**, a connective tissue layer

Putting It All Together: The Big Picture of Skeletal Muscle Structure

- The **perimysium** and **epimysium** come together at the end of the muscle to form a **tendon** that *binds* the muscle to its attaching structure (usually bone)
- Skeletal muscles are enclosed by a layer of thick connective tissue called **fascia**, which *anchors* them to the surrounding tissues and holds *groups* of muscles together

Putting It All Together: The Big Picture of Skeletal Muscle Structure

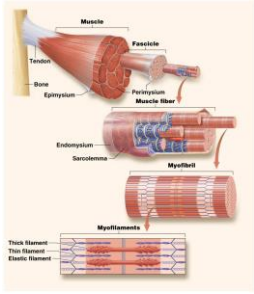


Figure 10.6 The Big Picture of Skeletal Muscle Structure.

Putting It All Together: The Big Picture of Skeletal Muscle Structure

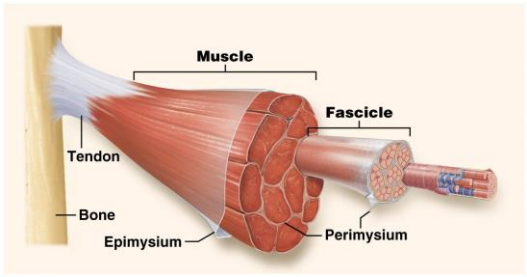


Figure 10.6 The Big Picture of Skeletal Muscle Structure.

Myofilament Arrangement and the Sarcomere

Striations appear microscopically as alternating:

- **Light bands**, where only **thin filaments** are found
- **Dark bands**, where both **thin** and **thick** filaments are found

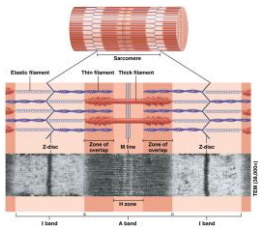


Figure 10.7 Structure and bands of the sarcomere.

Myofilament Arrangement and the Sarcomere

Dark and light areas of striations include the following *specific regions* (**Figure 10.8**):

- The **I band** ("i" in light, mnemonic) is composed only of **thin filaments**
- The **Z disc** is found in the *middle* of the **I band** and is composed of *structural proteins* that:
  - *Anchor* the thin filaments in place and to one another
  - Serve as *attachment points* for elastic filaments
  - Attach myofibrils to one another across the *entire diameter* of the muscle fiber

Myofilament Arrangement and the Sarcomere

Dark and light areas of striations include the following *specific regions* (continued):

- The **A band** ("a" in dark, mnemonic) contains the **zone of overlap**, the region where we find both **thick** and **thin filaments** and where **tension** is generated during contraction
- In the middle of the A band where only **thick filaments** exist is the **H zone** (HA, mnemonic, H is in the A band)
- The **M line** (M is in the *middle*, mnemonic) is a dark line in the middle of the A band made up of structural proteins that hold the **thick filaments** in place and serve as an *anchoring point* for elastic filaments

Myofilament Arrangement and the Sarcomere

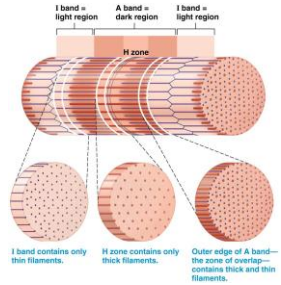
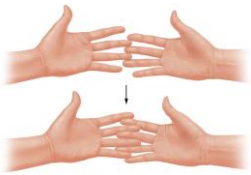


Figure 10.8 Three-dimensional structure of the sarcomere.

The Sliding-Filament Mechanism of Contraction

Your *hands* represent a single, large **sarcomere**, and where your fingers overlap represents the **zone of overlap** of the **thick** and **thin filaments**:

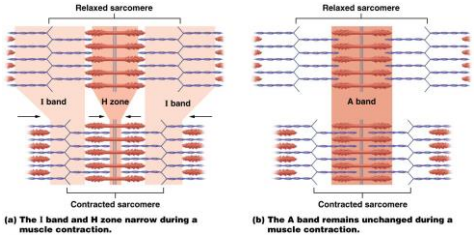


- The inner edges of your *palms* are the **z-discs**
- Now move your fingers slowly *together*
- As you can see, your "**sarcomere**" (the width of your two hands) grows progressively shorter (but your fingers do not *change in length*)

The Sliding-Filament Mechanism of Contraction

- The **sarcomere** extends from one **Z-disc** to the next; it is the **functional unit** where contraction occurs
- The **sliding filament mechanism** explains how tension is generated during muscle contraction
  - During a contraction, both the **I band** and the **H zone** narrow while the **A band** remains unchanged
  - **Myosin heads** attach to **actin** to pull the **thin filaments** toward the **M line**, which brings Z-discs *closer together*, shortening the sarcomere
  - **Sarcomeres** are arranged *end to end* within each **myofibril** and when *simultaneously contracted*, shorten the *whole* **muscle fiber**

The Sliding-Filament Mechanism of Contraction



Remember – none of the **filaments** themselves actually **shorten**; the thin filaments simply **move** toward the **M line**

Figure 10.9 The sliding filament mechanism; changes in the bands of the sarcomere.

10.3 Skeletal Muscle Fibers as Electrically Excitable Cells

Membrane Potentials in our Cells

Membrane potentials are due to an unequal distribution of ions near the plasma membrane resulting in a **polarized** resting state

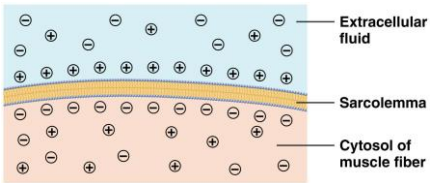
- A thin layer of *negatively* charged ions exists in the cytosol on the inside of the cell while a thin layer of *positively* charged ions exists on the outside of the cell (see the following image)
- This separation of charges creates an **electrical gradient** (**Core Principle**) that represents a source of **potential energy**

Membrane Potentials in our Cells

Membrane potentials are due to an unequal distribution of ions near the plasma membrane resulting in a **polarized** resting state (continued):

- When the **barrier** separating the ions is removed, they follow their gradients, creating a *flow of electrical charges*, and the **potential energy** becomes **kinetic energy**
- This is why the **electrical gradient** can be referred to as an **electrical potential**

Membrane Potentials in our Cells



- Away from sarcolemma, positive and negative ions are present in equal numbers
- This means that the cytosol and extracellular fluid are always electrically **neutral**

Membrane Potentials in our Cells

- The **membrane potential** of a cell is the **electrical potential** (*charge* difference) that exists across the plasma membrane
- A difference in charge (**potential**) between two points is called a **voltage**
- The potential across the sarcolemma is quite small, and is therefore measured in a unit called a **millivolt** (**mV**), or 1/1000 of a **volt**
- The resting **membrane potential** is the **electrical potential** across the sarcolemma of a resting **muscle fiber** and it measures –85 mV, meaning the cytosol is 85 mV more negative than the extracellular fluid

The Na^+/K^+ ATPase Pump, and Na^+ and K^+ Concentration Gradients

- Recall that the **phospholipid bilayer** of any plasma membrane is impermeable to charged particles (ions)
- **Resting membrane potentials** change only when the *barrier* to ion movement is removed from the plasma membrane
- Sodium and potassium ions can then move through the sarcolemma using **protein channels** and **carriers**
- They will only move by **diffusion** if a **gradient** exists between *two regions* (in other words, *across* the membrane)

The Na^+/K^+ ATPase Pump, and Na^+ and K^+ Concentration Gradients

- This concentration gradient is maintained by the **Na^+/K^+ pump**
  - The pump moves three Na^+ ions *out* and two K^+ ions *into* the cell, per ATP hydrolyzed
  - **ATP hydrolyis** is necessary because this pump moves the ions against their concentration gradients
  - This creates a high concentration of Na^+ in the extracellular fluid while the concentration in the cytosol remains lower
  - This creates a high concentration of K^+ in the cytosol while the concentration in the extracellular fluid remains lower

THE Na^+/K^+ ATPASE PUMP, AND Na^+ AND K^+ CONCENTRATION GRADIENTS

- The sarcolemma of a skeletal muscle fiber has *millions* of these pumps, which work together constantly to maintain steep concentration gradients of sodium and potassium

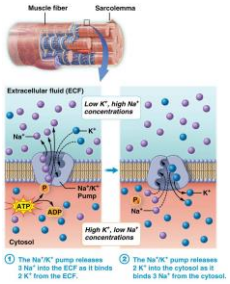


Figure 10.10 Ion gradients maintained by the Na^+/K^+ pump.

THE Na^+/K^+ ATPASE PUMP, AND Na^+ AND K^+ CONCENTRATION GRADIENTS

- These gradients are critical because they provide much of the *driving force* for the passive diffusion of sodium and potassium ions through membrane channels

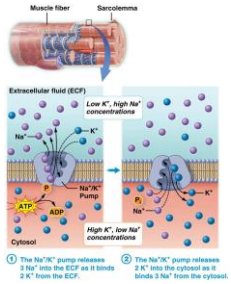


Figure 10.10 Ion gradients maintained by the Na^+/K^+ pump.

ACTION POTENTIALS

- **Action potentials** are brief changes in the membrane potential of a cell from a resting negative value to a positive value, then back to its resting negative value
- These changes can be used to electrically *communicate* with and sometimes *stimulate* a response from other cells

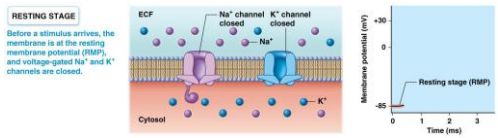
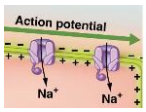


Figure 10.11a Stages of an action potential.

ACTION POTENTIALS

- **Gated channels** contain "**gates**" that are normally *closed* and *open* only in response to some sort of *stimulus*
- Action potentials are generated by opening two types of gated ion channels in the plasma membrane:
  - **Ligand-gated channels** (**chemically gated**) open in response to the presence of a chemical or **ligand**
  - **Voltage-gated channels** open and close in response to changes in the membrane **potential** of the plasma membrane



ACTION POTENTIALS

An **action potential** occurs in two stages (**Figure 10.11**):

1. **Depolarization** begins when **voltage-gated Na^+ channels** open, allowing Na^+ to flow *inward*
   - The *entry* of these positively charged sodium ions makes the membrane potential become LESS *negative* / LESS *polarized* (hence the term **depolarization**)
   - The membrane potential quickly reaches 0 mV and peaks at approximately +30 mV

ACTION POTENTIALS

An **action potential** occurs in two stages (continued):

2. **Repolarization** begins after Na^+ channels have closed and voltage-gated K^+ channels have opened, allowing K^+ to diffuse out of the cell
   - The *loss* of positively charged potassium ions makes the membrane potential MORE *negative*, returning the sarcolemma to the resting potential
   - K^+ channels *close* once the cell returns to its resting membrane potential

Action Potentials

- Recall that a property of muscle fibers is **conductivity**, which means that electrical changes across the sarcolemma are not isolated events
- Action potentials don't stay in one place; rather, they are **conducted**, or **propagated**, throughout the entire sarcolemma like *ripples on the surface of a pond*
- This process is very *fast* and results in depolarization of the entire sarcolemma, including the T-tubules
- The arrival of the action potential at the T-tubules is what initiates a **muscle contraction** (discussed next)

Action Potentials

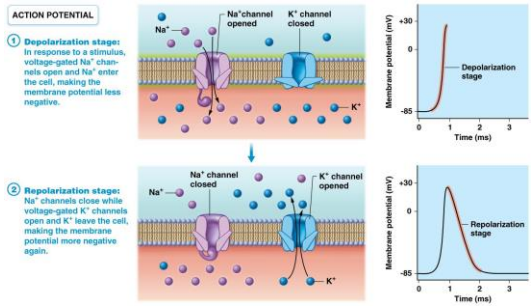


Figure 10.11 Stages of an action potential.

10.4 The Process of Skeletal Muscle Contraction and Relaxation

The Neuromuscular Junction (NMJ)

- All skeletal muscles are **innervated**, which means they are connected to a **neuron**
- A single neuron, called a **motor neuron**, communicates with many muscle fibers; each connection is referred to as a **synapse**
- The **NMJ** is the **synapse** where a single **motor neuron** communicates with many **muscle fibers**
- The **function** of the NMJ is to transmit a signal, called a **nerve impulse** (an **action potential**), from the neuron to the sarcolemma of the muscle fiber

The Neuromuscular Junction (NMJ)

There are three components of the **NMJ** (**Figure 10.12**):

- The **axon terminal** of the neuron contains **synaptic vesicles** filled with the neurotransmitter **acetylcholine** (**ACh**); **neurotransmitters** are chemicals that trigger changes in a **target tissue** when released, allowing for *cell to cell communication* (**Core Principle**)
- The **synaptic cleft** is the **space** between **axon terminal** and muscle fiber, filled with **collagen fibers** and a gel that *anchors* the neuron in place

The Neuromuscular Junction (NMJ)

There are three components of the **NMJ** (**Figure 10.12**) (continued):

- The **motor end plate** is a specialized region of the muscle fiber plasma membrane whose *folded* surface has many **ligand-gated Na^+ channels**; ACh is the ligand that *opens* these gates, allowing Na^+ to diffuse into the muscle cell

The Neuromuscular Junction (NMJ)

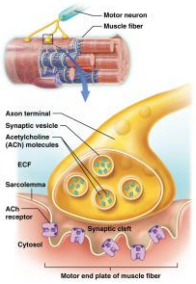


Figure 10.12 Structures of the neuromuscular junction.

Skeletal Muscle Contraction

Muscle contraction can be broken down into **three phases**:

- The **excitation phase** begins when an action potential signals the release of acetylcholine from the axon terminal into the synaptic cleft
- **Excitation-contraction coupling** is the link between the *stimulus* and the *contraction*
- The **contraction phase** begins when Ca^{++} ions bind troponin, which pulls tropomyosin away from actin's active site; the **crossbridge cycle** then begins

Skeletal Muscle Contraction

The **excitation phase** (**Figure 10.13**)

- An action potential from the *brain* or *spinal cord* arrives at the synaptic terminus of a motor neuron, signaling the release of acetylcholine from the axon terminal into the synaptic cleft
- Acetylcholine diffuses across the synaptic cleft where it can *bind* to ligand-gated channels found in the motor end plate of the muscle fiber sarcolemma
- Ligand-gated channels *open* when they bind acetylcholine which allows Na^+ ions to *enter* the muscle fiber generating an **end-plate potential**

Skeletal Muscle Contraction

The **excitation phase** (continued):

- An **end-plate potential** (which is simply a **local depolarization** in the area of the motor end plate) occurs as a result of sodium ion influx
- *Multiple* end-plate potentials must typically be generated in order to stimulate an **action potential** in the *surrounding membrane* that will trigger a functional **muscle contraction**
- Motor neurons continue to fire action potentials as acetylcholine is rapidly *degraded* by the enzyme **acetylcholinesterase** present in the synaptic cleft

Skeletal Muscle Contraction

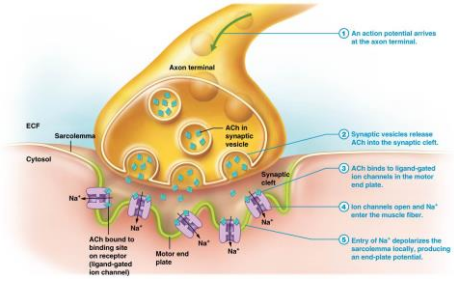
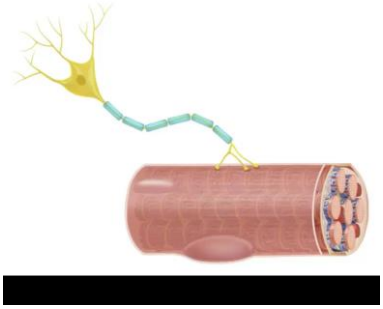


Figure 10.13 Excitation phase: events at the neuromuscular junction.

Big Picture Animation: Excitation



REVIEW

The end plate potential is generated by the influx of _____ into the motor end plate.

a. calcium
b. sodium
c. potassium
d. chloride

REVIEW

The end plate potential is generated by the influx of _____ into the motor end plate.

a. calcium
b. sodium
c. potassium
d. chloride

REVIEW

Acetylcholine is released from the synaptic terminus in response to

a. A ligand binding to a receptor on the synaptic terminus
b. Sodium flowing into the synaptic terminus
c. Potassium entering the synaptic terminus
d. An action potential arriving at the synaptic terminus

REVIEW

Acetylcholine is released from the synaptic terminus in response to

a. A ligand binding to a receptor on the synaptic terminus
b. Sodium flowing into the synaptic terminus
c. Potassium entering the synaptic terminus
d. An action potential arriving at the synaptic terminus

REVIEW

The term "synaptic cleft" refers to

a. A fold on the motor end plate
b. A vesicle in the synaptic terminus
c. The gap between the neuron and the muscle fiber
d. The space between adjacent muscle fibers

REVIEW

The term "synaptic cleft" refers to

a. A fold on the motor end plate
b. A vesicle in the synaptic terminus
c. The gap between the neuron and the muscle fiber
d. The space between adjacent muscle fibers

REVIEW

The sodium channels of the motor end plate are

a. Ligand-gated channels
b. Voltage-gated channels
c. Na^+/K^+ pumps
d. Mechanically gated channels

REVIEW

The sodium channels of the motor end plate are

a. Ligand-gated channels
b. Voltage-gated channels
c. Na^+/K^+ pumps
d. Mechanically gated channels

REVIEW

The end plate potential is

a. An action potential
b. A local repolarization
c. A local depolarization
d. A local hyperpolarization

REVIEW

The end plate potential is

a. An action potential
b. A local repolarization
c. A local depolarizaton
d. A local hyperpolarization

SKELETAL MUSCLE CONTRACTION

Excitation-contraction coupling is the link between the events at the NMJ and the contraction (**Figure 10.14**):

1. An **end-plate potential** leads to the opening of *VOLTAGE-gated* Na^+ channels in the sarcolemma *surrounding* the motor end plate, which triggers an **action potential**
2. Action potentials **propagate**; depolarization of *one* area of the membrane triggers the next few voltage-gated sodium ion channels to open, and the process continues like a *chain reaction* down the muscle fiber to the **triads**
3. The action potential signals the terminal cisternae to open **voltage-gated Ca^{++} channels**, releasing Ca^{++} into the cytosol

SKELETAL MUSCLE CONTRACTION

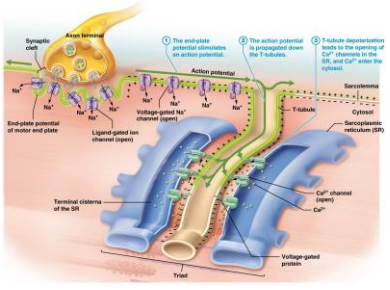
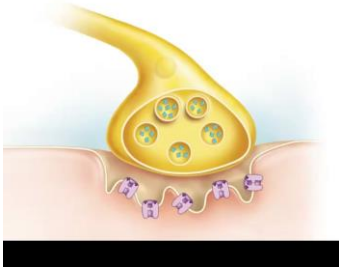


Figure 10.14 Excitation-contraction coupling: events at the sarcolemma and sarcoplasmic reticulum.

Big Picture Animation: Excitation-Contraction Coupling



Review

The channels that open in the sarcolemma surrounding the motor endplate and generate an action potential are

a. Ligand-gated channels
b. Voltage-gated channels
c. Na^+/K^+ pumps
d. Mechanically gated channels

Review

The channels that open in the sarcolemma surrounding the motor endplate and generate an action potential are

a. Ligand-gated channels
b. Voltage-gated channels
c. Na^+/K^+ pumps
d. Mechanically gated channels

Review

The term "propagate" when referring to an action potential means

a. Stimulate
b. Inhibit
c. Magnify
d. Spread

Review

The term "propagate" when referring to an action potential means

a. Stimulate
b. Inhibit
c. Magnify
d. Spread

Review

In order to trigger a muscle contraction, an action potential must reach the

a. Sarcomeres
b. Mitochondria
c. Triads
d. Nuclei

REVIEW

In order to trigger a muscle contraction, an action potential must reach the

a. Sarcomeres
b. Mitochondria
c. Triads
d. Nuclei

REVIEW

A triad consists of

a. Two terminal cisternae and a T-tubule
b. An M line and two zones of overlap
c. Two T-tubules and a terminal cisterna
d. A zone of overlap and two M lines

REVIEW

A triad consists of

a. Two terminal cisternae and a T-tubule
b. An M line and two zones of overlap
c. Two T-tubules and a terminal cisterna
d. A zone of overlap and two M lines

REVIEW

_____ is released from the SR in response to arrival of an action potential

a. Na^+
b. K^+
c. P_i
d. Ca^{++}

REVIEW

_____ is released from the SR in response to arrival of an action potential

a. Na^+
b. K^+
c. P_i
d. Ca^{++}

SKELETAL MUSCLE CONTRACTION

In preparation for muscle contraction (**Figure 10.15**):

1. **Calcium ions** released from the **terminal cisternae** bind to **troponin**
   - Troponin has three subunits
   - One subunit binds calcium ions, one binds actin, and the other binds tropomyosin
2. Tropomyosin moves, and the **active sites** of actin are exposed

Skeletal Muscle Contraction

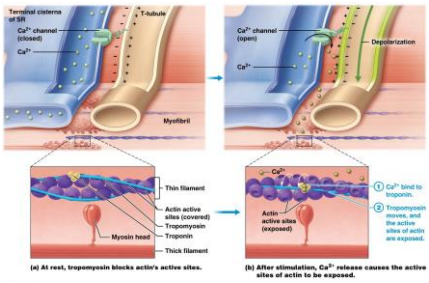
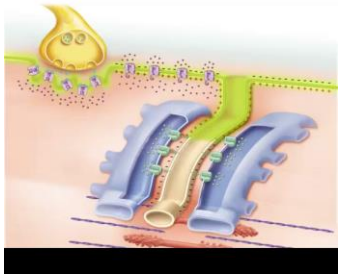


Figure 10.15 Preparation for contraction: regulatory events at the myofibril.

Big Picture Animation: Preparation for Contraction



Review

Tropomyosin

a. Covers actin active sites
b. Binds calcium ions
c. Is a small, globular protein
d. Has three subunits

Review

Tropomyosin

a. Covers actin active sites
b. Binds calcium ions
c. Is a small, globular protein
d. Has three subunits

Review

Troponin has three subunits. Which of the following does NOT bind to one of these subunits?

a. Actin
b. Myosin
c. Calcium
d. Tropomyosin

Review

Troponin has three subunits. Which of the following does NOT bind to one of these subunits?

a. Actin
b. Myosin
c. Calcium
d. Tropomyosin

REVIEW

Choose the correct sequence of events that occur in preparation for contraction

a. Action potential arrives at triad, calcium is released from the terminal cisternae, calcium binds to troponin, tropomyosin exposes the actin active sites
b. Tropomyosin exposes the actin active sites, calcium binds to troponin, action potential arrives at triad, calcium is released from the terminal cisternae
c. Calcium is released from the terminal cisternae, calcium binds to troponin, action potential arrives at triad, tropomyosin exposes the actin active sites
d. Calcium binds to troponin, action potential arrives at triad, calcium is released from the terminal cisternae, tropomyosin exposes the actin active sites

REVIEW

Choose the correct sequence of events that occur in preparation for contraction

a. Action potential arrives at triad, calcium is released from the terminal cisternae, calcium binds to troponin, tropomyosin exposes the actin active sites
b. Tropomyosin exposes the actin active sites, calcium binds to troponin, action potential arrives at triad, calcium is released from the terminal cisternae
c. Calcium is released from the terminal cisternae, calcium binds to troponin, action potential arrives at triad, tropomyosin exposes the actin active sites
d. Calcium binds to troponin, action potential arrives at triad, calcium is released from the terminal cisternae, tropomyosin exposes the actin active sites

SKELETAL MUSCLE CONTRACTION

The **contraction phase** begins when actin's active site is exposed, initiating the **crossbridge cycle** (**Figure 10.16a**):

1. The myosin head becomes **cocked** once an ATP is bound and its energy is gathered by **hydrolysis**

$$ATP \rightarrow ADP + P_i$$

2. Once cocked into its *high energy position* (ready to work), with ADP and P_i remaining attached to the myosin head, the head is able to bind to the active site of **actin**; note that the crossbridge is at a *90° angle* relative to the thick filament

SKELETAL MUSCLE CONTRACTION

The **contraction phase** (continued):

3. A **power stroke** occurs when ADP + P_i are released from the myosin head; myosin pulls the actin toward the M line as it pivots to its *relaxed* (low energy) *position*; the crossbridge is now at about a 45° angle relative to the thick filament
4. Myosin can bind to another ATP which breaks the link with the actin active site; detachment of the myosin head does not allow the thin filaments to *slide backward*, because at any given time some of the myosin heads will still be *attached to actin*

SKELETAL MUSCLE CONTRACTION

The crossbridge cycle may be repeated as long as the stimulus to contract *continues* and ATP is *available*

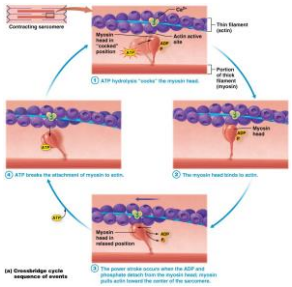
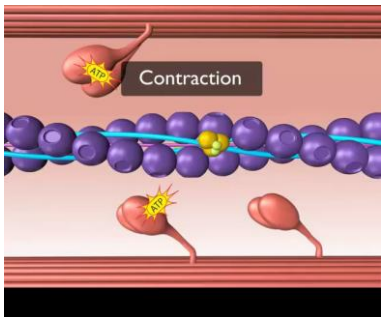


Figure 10.16a Contraction phase: the crossbridge cycle of the sliding filament mechanism.

BIG PICTURE ANIMATION: CONTRACTION



REVIEW

Hydrolysis of ATP is responsible for

a. Release of the myosin heads from the actin active sites
b. Recocking of the myosin heads
c. The power stroke
d. The movement of tropomyosin, exposing the actin active sites

REVIEW

Hydrolysis of ATP is responsible for

a. Release of the myosin heads from the actin active sites
b. Recocking of the myosin heads
c. The power stroke
d. The movement of tropomyosin, exposing the actin active sites

REVIEW

The binding of ATP to myosin is responsible for

a. Release of the myosin heads from the actin active sites
b. Recocking of the myosin heads
c. The power stroke
d. The movement of tropomyosin, exposing the actin active sites

REVIEW

The binding of ATP to myosin is responsible for

a. Release of the myosin heads from the actin active sites
b. Recocking of the myosin heads
c. The power stroke
d. The movement of tropomyosin, exposing the actin active sites

REVIEW

The release of ADP and P_i from myosin occurs during

a. Release of the myosin heads from the actin active sites
b. Recocking of the myosin heads
c. The power stroke
d. The movement of tropomyosin, exposing the actin active sites

REVIEW

The release of ADP and P_i from myosin occurs during

a. Release of the myosin heads from the actin active sites
b. Recocking of the myosin heads
c. The power stroke
d. The movement of tropomyosin, exposing the actin active sites

REVIEW

The myosin heads return to their low-energy (relaxed) state during

a. Release of the myosin heads from the actin active sites
b. Recocking of the myosin heads
c. The power stroke
d. The movement of tropomyosin, exposing the actin active sites

REVIEW

The myosin heads return to their low-energy (relaxed) state during

a. Release of the myosin heads from the actin active sites
b. Recocking of the myosin heads
c. The power stroke
d. The movement of tropomyosin, exposing the actin active sites

REVIEW

The power stroke

a. Pulls the thick filaments toward the Z lines
b. Positions the myosin heads in their high-energy position
c. Shortens the length of the thin filaments
d. Pulls the thin filaments toward the M lines

REVIEW

The power stroke

a. Pulls the thick filaments toward the Z lines
b. Positions the myosin heads in their high-energy position
c. Shortens the length of the thin filaments
d. Pulls the thin filaments toward the M lines

SKELETAL MUSCLE CONTRACTION

The **contraction cycle** is repeated (**Figure 10.16b**):

- The myosin head is *recocked*, it binds to the first actin molecule, and the power stroke repeats
- Mysosin then binds to the second actin, and so on, over and over
- For an average contraction, this process will repeat about *20–40 times* for each myosin head in each sarcomere of the muscle fiber

SKELETAL MUSCLE CONTRACTION

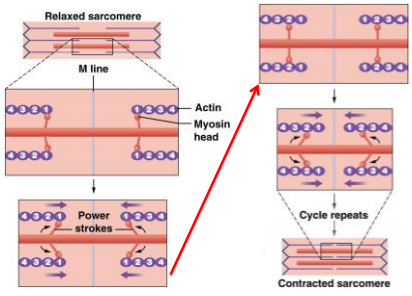


Figure 10.16b Contraction phase: the crossbridge cycle of the sliding filament mechanism.

Botulism and Botox

- The bacterium ***Clostridium botulinum*** produces the most lethal known biological poison—as little as one gram of crystalline toxin is enough to kill about **one million adults**
- Exposure to the **botuminum toxin** through contaminated food causes the disease **botulism**:
  - The toxin binds to motor neurons of the NMJ and **blocks** the release of acetylcholine from synaptic vesicles
  - This *paralyzes* the affected muscle, and without proper treatment, death from **respiratory failure** will follow
- The toxin can be used to treat painful muscle spasm and migraine headaches when injected in minute quantities; also used cosmetically to relax facial muscles (as **Botox**)

Skeletal Muscle Relaxation

Muscle Relaxation has two components:

- Motor neuron action potentials stop signaling for the release of acetylcholine from axon terminals
- Calcium ions are actively pumped back into the SR terminal cisternae

Skeletal Muscle Relaxation

The specific sequence of relaxation events is as follows (**Figure 10.17**):

1. **Acetylcholinesterase** degrades the remaining ACh, ligand-gated sodium channels *close*, the end plate potential *ends*, and the final *repolarization* begins
2. The sarcolemma returns to its *resting membrane potential* as a result of K^+ efflux through voltage-gated K^+ channels, and calcium ion channels in the SR close as the T-tubules repolarize

Skeletal Muscle Relaxation

The specific sequence of relaxation events is as follows (continued):

3. Calcium ions are pumped back into the SR, returning the calcium ion concentration in the cytosol to its *resting level*
4. In the absence of calcium, troponin and tropomyosin block the active sites of actin, and the muscle *relaxes*; myofilaments slide back into their *original positions*, with support from titin and other structural proteins

Skeletal Muscle Relaxation

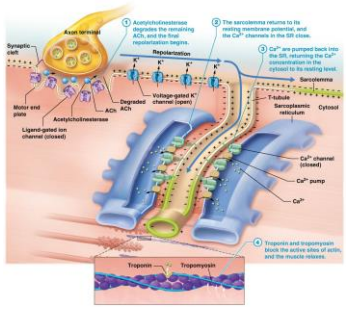
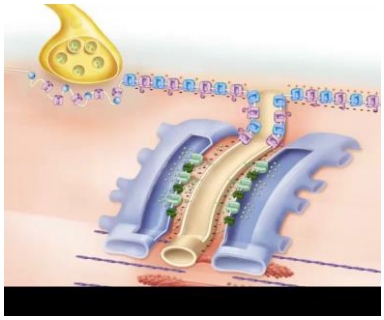


Figure 10.17 Relaxation phase: the process of muscle relaxation.

Big Picture Animation: Relaxation



REVIEW

During muscle fiber relaxation, calcium channels in the SR close because

a. The resting membrane potential is restored
b. Calcium levels in the SR are depleted
c. Calcium is released from the SR
d. Calcium is pumped into the extracellular fluid

REVIEW

During muscle fiber relaxation, calcium channels in the SR close because

a. The resting membrane potential is restored
b. Calcium levels in the SR are depleted
c. Calcium is released from the SR
d. Calcium is pumped into the extracellular fluid

REVIEW

During muscle fiber relaxation

a. Calcium levels in the sarcoplasm rise
b. Calcium is pumped back into the SR
c. Calcium is released from the SR
d. Calcium is pumped into the extracellular fluid

REVIEW

During muscle fiber relaxation

a. Calcium levels in the sarcoplasm rise
b. Calcium is pumped back into the SR
c. Calcium is released from the SR
d. Calcium is pumped into the extracellular fluid

REVIEW

Acetylcholinesterase in the synaptic cleft degrades acetylcholine, allowing

a. Depolarization of the motor end plate
b. Calcium levels in the sarcoplasm to rise
c. Tropomyosin to expose actin active sites
d. Ligand-gated sodium channels to close

REVIEW

Acetylcholinesterase in the synaptic cleft degrades acetylcholine, allowing

a. Depolarization of the motor end plate
b. Calcium levels in the sarcoplasm to rise
c. Tropomyosin to expose actin active sites
d. Ligand-gated sodium channels to close

REVIEW

Sarcolemma repolarization during relaxation

a. Means that the interior of the cell becomes less negative

b. Restores the resting membrane potential

c. Only occurs at the motor end plate of the fiber

d. Is caused by closure of calcium channels

REVIEW

Sarcolemma repolarization during relaxation

a. Means that the interior of the cell becomes less negative

b. Restores the resting membrane potential

c. Only occurs at the motor end plate of the fiber

d. Is caused by closure of calcium channels

REVIEW

Which aspect of muscle relaxation requires ATP?

a. Motor end plate repolarization

b. Blockage of actin active sites by tropomyosin

c. Sarcomeres returning to their original length

d. Pumping calcium ions back into the SR

REVIEW

Which aspect of muscle relaxation requires ATP?

a. Motor end plate repolarization

b. Blockage of actin active sites by tropomyosin

c. Sarcomeres returning to their original length

d. Pumping calcium ions back into the SR

RIGOR MORTIS

- The progressive stiffening (*contraction*) of skeletal muscles begins about 3–4 hours after death, as the pumps that drive calcium ions back *into the SR* no longer have ATP to *fuel their activity*
- As a result, Ca^{++} ions remain in the cytosol, where they bind to troponin and initiate muscular contraction all over the body
- The muscle fibers are unable to *relax* without ATP, so the myosin heads cannot detach from actin
- The muscles remain contracted until the proteins of the myofilaments begin to *degenerate*, about 48–72 hours after death

PUTTING IT ALL TOGETHER: THE BIG PICTURE OF SKELETAL MUSCLE CONTRACTION AND RELAXATION

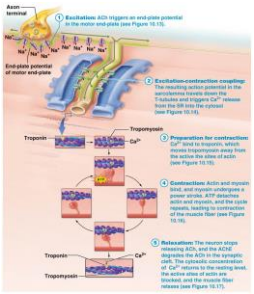


Figure 10.18 The Big Picture of Skeletal Muscle Contraction and Relaxation.

Module 10.5 Energy Sources for Skeletal Muscle

Sources of Energy for Muscle Contraction

- In skeletal muscle, ATP is *required* to
  - *Power* the Na^+/K^+ pumps that maintain the ion gradients involved in action potentials
  - *Release* the myosin heads from the actin active sites and *recock* the heads in preparation for another power stroke
  - *Pump* calcium back into the SR during relaxation

Sources of Energy for Muscle Contraction

- The required ATP is generated by:
  - Immediate cytosolic reactions (**Figure 10.19a**)
  - **Glycolytic catabolism** in the *cytosol* (**Figure 10.19b**)
  - **Oxidative catabolism** in the *mitochondria* (**Figure 10.19b**)
- *All three processes* may occur simultaneously in muscle fibers during contractions, but they are used in *different proportions*, depending on the resources and needs of the cells

Immediate Sources of Energy for Muscle Contraction

- The main immediate energy is stored as ATP in the muscle fiber and is *rapidly consumed* during muscle contraction
- **Creatine phosphate** concentration in the cytosol is 5–6 times *higher* than ATP; it can immediately regenerate enough ATP for about *10 seconds of maximum muscle activity*

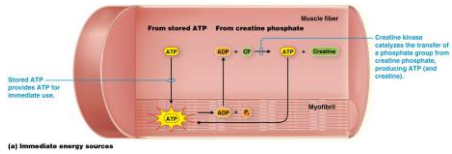


Figure 10.19a Sources of energy for muscle fibers.

Creatine Supplementation

- Research has demonstrated that supplementation with **creatine** does mildly *improve performance* for activities that require *short bursts* of muscle activity
- The effects on **endurance-type activities** are *minimal to nonexistent*

Creatine Supplementation

- Creatine may actually be *detrimental* in some cases:
  - Causes **weight gain** from **water retention**
  - **Massive doses** may cause **kidney damage**
- Skeletal muscles have a *maximal storage capacity* for creatine; therefore, huge doses are a waste of money because the excess is simply excreted in the urine

GLYCOLYTIC ENERGY SOURCES

- **Glycolysis** is a series of reactions that occurs in all cells' cytosol to break glucose down into **pyruvate**; it provides energy for muscle contraction once immediate sources of energy are depleted
- Glycolysis uses **glucose** found in the blood and stored in muscle (or liver) cells as **glycogen**; it can replenish ATP for *30–40 seconds* of sustained contraction

GLYCOLYTIC ENERGY SOURCES

- **Glycolysis**, or **anaerobic catabolism**, does not require **oxygen** directly, but the amount of oxygen present leads to the following two possible scenarios:
  - If oxygen is abundant, pyruvate formed by glucose catabolism enters the mitochondria for **oxidative catabolism**, which will then occur *simultaneously* with glycolysis as long as glucose is available
  - If oxygen is not abundant, the pyruvate is converted into two molecules of **lactic acid** which can later either be converted back into glucose by the liver (about 20%) or taken up in the mitochondria for oxidative catabolism

GLYCOLYTIC ENERGY SOURCES

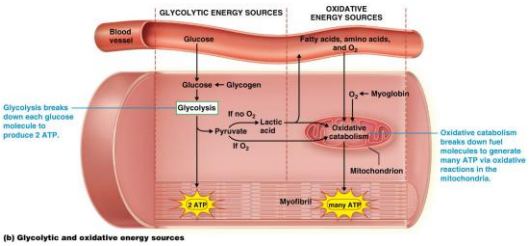


Figure 10.19b Sources of energy for muscle fibers.

OXIDATIVE ENERGY SOURCES

- **Oxidative catabolism**, or **aerobic catabolism**, requires **oxygen** directly; it allows for *longer lasting* muscle contractions because these reactions produce many more ATP than glycolysis
  - The amount of ATP produced depends on the *type of fuel* used by the fiber
  - Muscle fibers prefer to use glucose, but as it becomes unavailable, they will catabolize **fatty acids** and **amino acids**

OXIDATIVE ENERGY SOURCES

- Oxidative catabolism is the *predominant* energy source after one minute of contraction and provides nearly 100% of the necessary ATP after several minutes; it can provide ATP for hours, as long as oxygen and fuels are available

OXIDATIVE ENERGY SOURCES

- **Electrons** are removed from fuel molecules, and the energy derived from this catabolism is used to synthesize ATP in the mitochondria, where the electrons are transferred to oxygen as the *final step* in aerobic catabolism
- Oxygen is supplied by the blood, diffusing into the fiber from the extracellular fluid
- Within the fiber, it is bound to the oxygen-carrier **myoglobin**, (which is similar to **hemoglobin**, the oxygen carrier found in the blood), which *releases* the oxygen as needed

OXIDATIVE ENERGY SOURCES

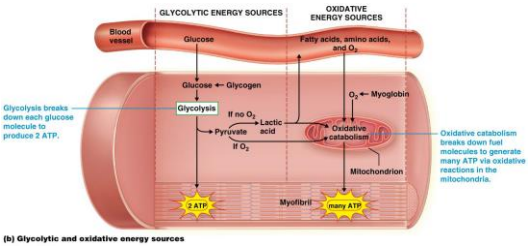


Figure 10.19b Sources of energy for muscle fibers.

10.6 MUSCLE TENSION AT THE FIBER LEVEL

TWITCH CONTRACTION

- A **muscle twitch** is the smallest muscle contraction; occurs in the laboratory, not in whole muscles of the body (**Figure 10.20**)
- The three phases of a twitch on a **myogram** include the following:
  - The **latent period** is the time it takes the action potential to propagate across the sarcolemma
  - The **contraction period** begins as repeated crossbridge cycles generate tension
  - The **relaxation period** begins as calcium ion levels are reduced in the cytosol by SR pumps and tension diminishes

TWITCH CONTRACTION

- The **refractory period** begins at the onset of the latent period and ends at the beginning of the contraction period
- During this time (about 5 ms) the muscle fiber is *unable to respond* to further stimuli
- Cardiac muscle and smooth muscle have refractory periods as *long as their contractions*, so the cells must *fully relax* before they can contract a *second time*

TWITCH CONTRACTION

The tension produced during a twitch varies considerably with *several factors*:

- *Timing* and *frequency* of stimulation
- *Length* of the fiber at rest
- *Type* of muscle fiber (discussed in next sections)

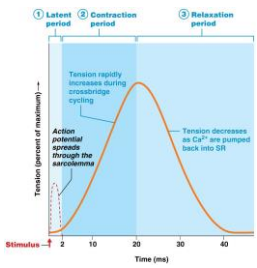


Figure 10.20 Myogram of a twitch contraction.

TENSION PRODUCTION AND THE TIMING AND FREQUENCY OF STIMULATION

- The increase in tension caused by repeated stimulation of the muscle fiber by a motor neuron is known as **wave summation**
- Repeated stimulation results in *progressively greater* tension production because:
  - The pumps in the SR membranes have *inadequate time* to pump all of the released **calcium** ions back into the SR before the fiber is *restimulated*
  - Therefore, the concentration of calcium ions in the cytosol increases with *each stimulation*

Tension Production and the Timing and Frequency of Stimulation

- The tension produced depends on the **frequency** of *motor neuron stimulation*, and results in two possible myogram patterns:
  - **Unfused tetanus** results when fibers are stimulated about *50 times per second* and the fiber *partially relaxes* between stimuli; tension *pulsates* (with individual twitches remaining visible) and increases to about 80% of the maximum (**Figure 10.21a**)

Tension Production and the Timing and Frequency of Stimulation

- The tension produced depends on the **frequency** of *motor neuron stimulation*, and results in two possible myogram patterns (continued):
  - **Fused** (**complete**) **tetanus** occurs when the fiber is stimulated at a rate of *80–100 stimuli per second* and the fiber does not *relax* between stimuli; tension stays constant at nearly 100% of the maximum (**Figure 10.21b**)
    - The increased availability of calcium allows more crossbridges to form, contributing to the increase in tension
    - Note that fused tetanus is possible only because of the *extremely short refractory period* of the skeletal muscle fiber

Tension Production and the Timing and Frequency of Stimulation

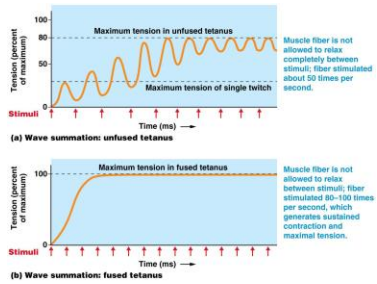


Figure 10.21 Wave summation: unfused and fused tetanus.

The Length-Tension Relationship

- The **length-tension relationship** states that the **optimal length** of a sarcomere is about 100–120% of the *natural length* of the sarcomere (**Figure 10.22**)
- At this optimal length a sarcomere can generate the greatest tension because the number of crossbridges that can form is maximal
  - The length of the sarcomere must be *short enough* to allow for a generous zone of overlap between thin and thick filaments
  - But the length of the sarcomere must be *long enough* for the thick filaments to pull the thin filaments toward the M line without running into the Z-discs

The Length-Tension Relationship

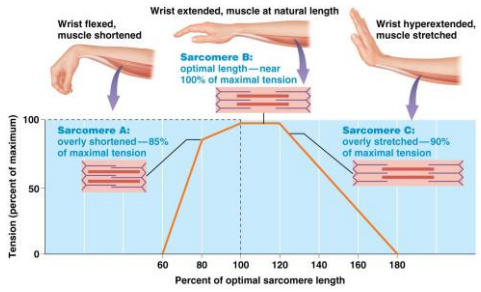


Figure 10.22 The length-tension relationship.

Concept Boost: Understanding How Events at the Myofilaments Produce Tension of a Whole Muscle

- Remember that myofibrils are connected to the sarcolemma of the muscle fiber, so any tension in the myofibrils is transmitted to the muscle fiber as a whole. The muscle fiber then relays that tension to the collagen fibers in the endomysium, which causes contraction of the fascicle as a whole. As the fascicles contract, the tension is in turn conducted from the surrounding perimysium to the epimysium and the tendons of the entire muscle. The muscle then contracts, pulling on the attached bones and causing movement.

Concept Boost: Understanding How Events at the Myofilaments Produce Tension of a Whole Muscle

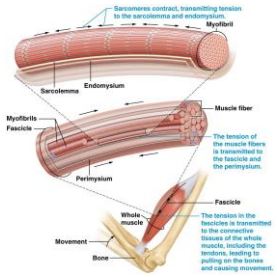


Figure 10.23 How myofilament sliding leads to whole muscle contraction.

Classes of Skeletal Muscle Fibers

- There are two main classes of skeletal muscle fibers:
  - **Type I/slow** and **Type II/fast**
  - Classified based on **myosin ATPase** activity (determines how *fast* or how *slowly* a power stroke can occur), and on the predominant energy source (oxidative versus glycolytic catabolism)
- Most muscles contain combinations of both fiber types
- The *proportion* of fibers, either **Type I/slow** or **Type II/fast**, is dependent on the *function* of the muscle itself (**Structure-Function Core Principle**)

Classes of Skeletal Muscle Fibers

- **Type I fibers** are *small diameter, slow-twitch fibers* that contract slowly to produce less force for a longer period of time
  - Slow-twitch fibers have low myosin ATPase activity
  - Slow fibers rely on oxidative catabolism and have large numbers of mitochondria, a well-developed blood **supply**, and myoglobin molecules; this gives them a characteristic "*dark muscle*" red color
  - Slow fibers predominate in *postural muscles* that must sustain contractions for long durations

Classes of Skeletal Muscle Fibers

- **Type II fibers** are *large diameter, fast twitch fibers* that fatigue quickly
  - Fast fibers have high myosin ATPase activity and rely mainly on glycolytic catabolism for the production of ATP
  - Fast fibers have fewer mitochondria and lower levels of myoglobin and less extensive blood supply, giving them their characteristic "*white muscle*" appearance

Classes of Skeletal Muscle Fibers

- **Type II fibers** (continued):
  - There are three subtypes that are categorized based on their *energy production method*
    - **IIa** (**fast oxidative-glycolytic** or **FOG**)
    - **IIx** (**fast oxidative** or **FO**)
    - **IIb** (**fast glycolytic** or **FG**) – produce extremely fast, powerful twitches
  - Examples of muscles with large numbers of type II fibers include *eye muscles*

Classes of Skeletal Muscle Fibers

- Most muscles contain all fiber classes (**Figure 10.24**), each of which is stimulated under *different conditions*
  - A baseball player sitting in the dugout uses primarily *type I fibers* in the back and abdomen to *remain sitting upright*
  - When the player gets up and *jogs to the plate* to bat, primarily *type IIa fibers* in the legs are used
  - When the player *hits the ball*, the bat is swung using *type IIx and IIb fibers* in the arms

Classes of Skeletal Muscle Fibers

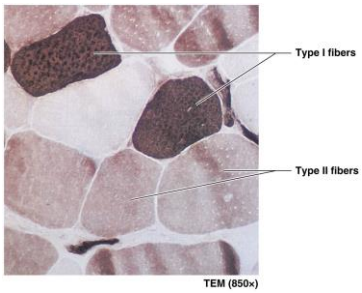


Figure 10.24 Comparison of Type I and type II muscle fibers.

Module 10.7 Muscle Tension at the Organ Level

Motor Units

A single motor neuron and all the muscle fibers that it innervates define a **motor unit** (**Figure 10.25**)

- Motor units are considered **slow**, composed of type I fibers only or **fast**, composed of type II fibers only
- When the motor neuron fires an action potential, all of the muscle fibers within its motor unit respond and produce about the same amount of tension; this applies to only the motor unit, not to the *entire muscle*

Motor Units

- The *number of fibers* in a motor unit varies depending on the motor unit's *function*
  - Muscles requiring fine *motor control* have small motor units (as few as 10 muscle fibers per motor unit, as in the larynx and fingers)
  - Those requiring less *control* (and generation of more *power*) have large motor units (as many as 2000–3000 fibers per motor unit, as in the postural muscles of the back, or the large muscles of the legs)

Motor Units

- Initiation of a contraction activates a *small number* of motor units
- As greater force is required more motor units must be stimulated, a process known as **recruitment**
  - Slow motor units are typically activated first
  - Fast motor units will follow as *additional tension* is needed

Motor Units

- **Muscle tone** is a baseline level of *involuntary* activation of motor units by the brain and spinal cord
  - Muscle tone is vital for the maintenance of erect posture, stabilization of joints, heat production, and preserving a level of preparedness for movement
  - The nervous system *alternates* which motor units it activates, so that some can *rest* while others *contract*

Motor Units

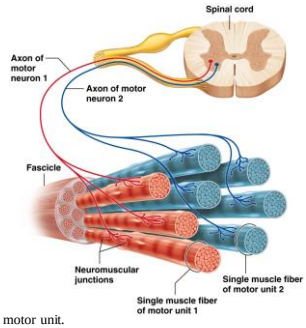


Figure 10.25 The motor unit.

Types of Muscle Contractions

- **Isotonic contractions** (tension generated by the muscle is constant, but muscle length *changes*):
  - **Isotonic concentric contractions** maintain constant **tension** while the muscle *shortens*; the force generated by the muscle is greater than the external force
  - **Isotonic eccentric contractions** maintain constant tension but the muscle *lengthens*, as the external force applied is greater than the force generated by the muscle
- **Isometric contractions** is where the muscle length remains *unchanged* because the external force applied equals that generated by the muscle

Types of Muscle Contractions

- A muscle is able to *lengthen* while it is contracting because the elastic filaments in its myofibrils allow it to *stretch* considerably

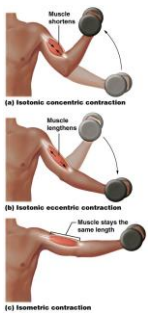


Figure 10.26 The three types of muscle contraction.

Delayed-Onset Muscle Soreness

- The phenomenon of *muscle soreness* following exercise was thought for many years to be due to the accumulation of *lactic acid* produced during glycolysis
- Current research suggests instead that it is more likely due to *minor structural damage*, in particular, that caused by *isotonic eccentric muscle contractions*
- The most effective treatment for DOMS is more *exercise*; unfortunately, once the exercise ceases, the pain returns until the muscle is sufficiently conditioned through **training**
- Other treatment modalities such as massage, topical therapies, acupuncture, and oral medications have shown *little benefit*

Module 10.8 Skeletal Muscle Performance

Changes Caused By Physical Training

- The principle of **myoplasticity** describes the changes in *muscle structure* as a result of changes in *function* related to physical training (**Structure-Function Core Principle**)
  - The majority of mature skeletal muscle fiber nuclei are **amitotic**, meaning that they generally do not undergo *mitosis*
  - **Satellite cells** (a small population of unspecialized cells) do retain mitotic ability, can help *repair* injured skeletal muscle

CHANGES CAUSED BY PHYSICAL TRAINING

- The principle of **myoplasticity** describes the changes in *muscle structure* as a result of changes in *function* related to physical training (**Structure-Function Core Principle**) (continued):
  - Therefore, changes in response to training are *within* the muscle fibers and do not involve changes in the *number* of muscle fibers
  - The precise type of change that occurs depends on the type of *training*—**endurance** or **resistance training**

CHANGES CAUSED BY PHYSICAL TRAINING

- **Endurance training** is defined as training with a large increase in the *frequency* of motor unit activation and a moderate increase in *force production*—in other words, *more repetitions with lighter weight*
- It leads to the following primarily *biochemical* changes most dramatically in type I fibers, but even in type II (**Figure 10.27a**):
  - Increased *oxidative enzymes*, and *mitochondria* (and associated proteins)
  - Increased *fatigue* resistance
  - More efficient use of fatty acids and *non-glucose fuels* for ATP production
  - Increases in the *blood vessel network* supplying the muscle

CHANGES CAUSED BY PHYSICAL TRAINING

- **Resistance**, or **strength**, **training** involves a moderate increase in the *frequency* of motor unit activation and a large increase in *force production*—in other words, *fewer repetitions with heavier weight*
  - It causes primarily *anatomical changes*; both the *number* of myofibrils and the *diameter* of the muscle fibers increase, a change called **hypertrophy** (**Figure 10.27b**)

CHANGES CAUSED BY PHYSICAL TRAINING

- **Resistance**, or **strength**, **training** involves a moderate increase in the *frequency* of motor unit activation and a large increase in *force production*—in other words, *fewer repetitions with heavier weight* (continued):
  - With hypertrophy comes a decreased proportion of mitochondrial proteins and blood supply to the muscle, because of *fiber enlargement*, and not because mitochondria or vessels are actually *lost*
  - This can decrease *endurance*, so a balanced program combining both types of training is recommended for most people

CHANGES CAUSED BY PHYSICAL TRAINING

- *Disuse* leads to anatomical and biochemical changes including a decrease in the number of myofibrils and size of the fiber and a decrease in oxidative enzymes, which is termed **atrophy**
  - The result is a decline in both *strength* and *endurance*
  - In other words, the adage "*use it or lose it*" applies to skeletal muscle
  - Atrophy is a particular problem for the *bedridden*, or those that have lost the *use of their limbs*

CHANGES CAUSED BY PHYSICAL TRAINING

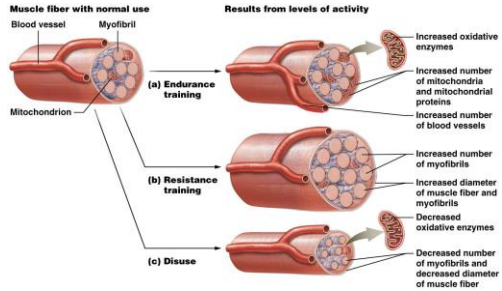


Figure 10.27 Adaptive changes of muscle fibers due to training and disuse.

Muscular Fatigue

- **Fatigue** is the inability to maintain a given level of *intensity* during activity
- Fatigue is caused by *multiple factors*:
  - The depletion of *key metabolites* (creatine phosphate, glycogen, and glucose) involved in ATP production
  - Decreased availability of *oxygen* to muscle fibers (increased demand during exercise coupled with depleted myoglobin-bound oxygen and inadequate oxygen intake in the lungs)
  - The accumulation of certain chemicals in the fiber such as Ca^{++}, ADP, and phosphate
  - Environmental conditions, particularly *extreme heat*; sweating in response to heat may also cause *electrolyte disturbances*

Excess Postexercise Oxygen Consumption and the Recovery Period

- It takes time after exercising to return to the *pre-exercise* state; this is termed the **recovery period**
- The increased *rate of breathing* that occurs during this period supplies the necessary oxygen, and is called the **excess postexercise oxygen consumption** (**EPOC**)
- EPOC is the mechanism that allows the body to *recover* from *exercise-induced homeostatic imbalances* (changes in body temperature, imbalances of intracellular and extracellular ion concentrations, and blood pH imbalances)

Module 10.9 Smooth and Cardiac Muscle

Smooth Muscle

- **Smooth muscle** has the following **functions**:
  - *Propels materials* through hollow organs, a process called **peristalsis** (**Figure 10.28a**)
  - Forms **sphincters** (in the digestive and urinary systems) that control the *passage of materials* by opening and closing
  - *Regulates flow rates* through hollow organs (such as blood vessels, the respiratory tract, and the gastrointestinal tract), by changing the diameter of the tubing

Smooth Muscle

Smooth muscle cells contain myosin and actin filaments arranged differently than in skeletal and cardiac muscle; there are no *sarcomeres*, and therefore no *striations*

- Actin filaments are arranged *obliquely* in the sarcoplasm and are *anchored* to proteins called **dense bodies**
  - Some dense bodies are found in the sarcoplasm, where they are bound to scaffold-like **intermediate filaments** that *connect* the dense bodies to *each other*

Smooth Muscle

- Actin filaments are arranged *obliquely* in the sarcoplasm and are *anchored* to proteins called **dense bodies** (continued):
  - Other dense bodies link a smooth muscle cell to *surrounding* smooth muscle cells which allows for *tension transmission* from cell to cell

SMOOTH MUSCLE

- Several thin filaments radiate from each dense body to surround a *single* thick filament; the *ratio of thin to thick filaments* is therefore higher than in skeletal muscle
- In smooth muscle cell contractile proteins:
  - Both thick and thin filaments are *longer* and the thin filament lacks troponin

SMOOTH MUSCLE

- In smooth muscle cell contractile proteins (continued):
  - Myosin heads are found along the entire length of the thick filament, with *opposite-facing heads* (heads on either side of the filament hinge in opposite directions)
- Smooth muscle cells *lack* motor end plates, the SR is much *less* extensive, and there are *no* T-tubules

SMOOTH MUSCLE

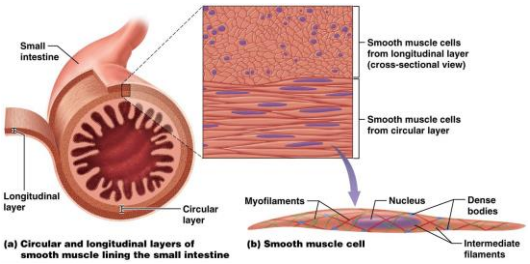


Figure 10.28 Structure of smooth muscle tissue and cells.

SMOOTH MUSCLE

Smooth Muscle Contraction and Relaxation (**Figure 10.29**)

- Contraction of smooth muscle involves a different cascade of events:
  1. Influx of extracellular calcium ions that bind to a protein in the sarcoplasm called **calmodulin**
  2. This complex activates **myosin light chain kinase** (**MLCK**)
  3. This enzyme in turn activates myosin ATPase
  4. The crossbridge cycle then begins

SMOOTH MUSCLE

Smooth Muscle Contraction and Relaxation (continued):

- Repeated contraction cycles pull actin along myosin, causing the cell to *change shape* from thin and flat to fat and globular
- Smooth muscles can contract up to *80% of their resting length*, whereas skeletal muscle can only contract a maximum of 30–40% of their resting length
- Only about 1/100 the amount of ATP is required when compared with skeletal fiber contractions

SMOOTH MUSCLE

Smooth Muscle Contraction and Relaxation (continued):

- Relaxation occurs when Ca^{++} is removed from the cytosol, MLCK is deactivated, and the myosin ATPase is deactivated
- The **latch state** is an alternative to relaxation where the cell remains contracted in an *energy-efficient mode* (important in sphincters that must *stay contracted* to remain closed)

Smooth Muscle

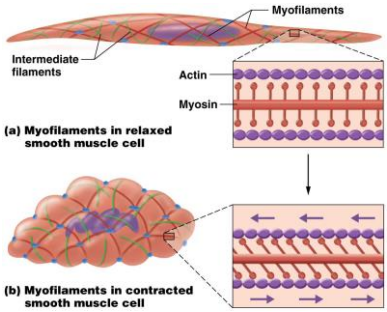


Figure 10.29 Contraction of smooth muscle cells.

Smooth Muscle

Types of Smooth Muscle:

- **Single unit smooth muscle** is
  - The *predominant type* in the body
  - Found in nearly all hollow organ walls where they are *linked electrically* by **gap junctions**
  - Action potentials spread rapidly through the cells via the gap junctions, causing the cells to contract in a coordinated wave as a *single unit*
  - Single unit smooth muscle cells respond to *multiple stimuli* including mechanical, hormonal, neural, and local pacemaker cell stimuli

Smooth Muscle

Types of Smooth Muscle (continued):

- **Multi-unit smooth muscle**:
  - Less common than single-unit smooth muscle
  - Found in the uterus, eye, and skin (arrector pili)
  - Made up of individual cells (not joined by gap junctions) that contract *independently* to allow for precision
  - The amount of tension produced by this type of smooth muscle varies with the *number of cells activated* (as in skeletal muscle)
  - Responds primarily to *nerve stimulation*

Cardiac Muscle

- Cardiac muscle cells are structurally similar to skeletal fibers with some *major differences*:
  - Shorter, branched cells with one or two nuclei and abundant myoglobin
  - Mitochondria account for 30% of the cytoplasmic volume
  - **Intercalated discs** link cells together both *electrically* by gap junctions and *physically* by **desmosomes**, permitting the heart to contract as a *coordinated unit*

Cardiac Muscle

- Unlike skeletal muscle fibers, cardiac fibers do not require stimulation from the nervous system to generate action potentials; their electrical activity is coordinated by **pacemaker cells**
- Cardiac pacemaker cells are found in specific regions of the heart and are **autorhythmic** (like single unit smooth muscle), meaning that they *spontaneously* generate action potentials; these cells coordinate electrical activity and trigger the *contraction* of surrounding cells