17 Skeletal Muscle System

OBJECTIVES

After studying the chapter, you should be able to:

- Describe the functions of skeletal muscle tissue.
- Identify the characteristics of muscle tissue that make movement possible.
- Describe the macroscopic and microscopic organization of skeletal muscle tissue.
- Relate the molecular structure of myofilaments to the sliding filament theory of muscle contraction.
- Identify the regions of a sarcomere, and explain the changes that occur in these regions during contraction.
- Discuss the importance of specialized organelles, specifically, the sarcoplasmic reticulum, the T tubules, and the myofibrils.
- Explain the events involved in excitation-contraction coupling.
- Describe the sequence of events in the generation of force within the contractile elements.
- Differentiate muscle fiber types based on their contractile and metabolic properties.
- Discuss the ramifications of fiber type distribution on the likelihood of success in a given athletic event.

Introduction

Muscle contractions provide the basis for all human movement. Movement also involves interactions among different body systems. For instance, the muscle cells (fibers) produce and utilize ATP to provide the energy for contraction and force production. The digestive, respiratory, endocrine, and cardiovascular systems must be operating effectively to provide muscle cells with the oxygen and nutrients needed to produce the energy. For the purposes of this chapter, it is assumed that these other body systems are functioning properly.

Overview of Muscle Tissue

Muscle tissue produces force through the interaction of its basic contractile elements—the myofilaments—which are composed primarily of protein. The three types of muscle tissue (skeletal, smooth, and cardiac) have different general functions. The force of contraction may be used for movement such as locomotion (skeletal muscle), the movement of materials through hollow tubes such as the digestive tract or blood vessels (smooth muscle), or the pumping action of the heart (cardiac muscle). Regardless of type, all muscle tissue can produce force because of certain basic characteristics. This chapter focuses on skeletal muscle (**Figure 17.1**).

Because skeletal muscles have various characteristics, they are often referred to by different names. Skeletal muscles are under conscious control and are often called *voluntary muscles*. Skeletal muscles are also sometimes referred to as *striated muscle* because of the repeating pattern of light and dark bands seen in their microscopic structure. Additionally, to differentiate skeletal muscle fibers from intrafusal fibers found in sensory organs of the muscle (proprioceptors; see Chapter 20), physiologists sometimes refer to skeletal muscle fibers as *extrafusal muscle fibers*.

Functions of Skeletal Muscle

Although movement is the primary function of muscle tissue, the muscular system also has other important roles. In addition to locomotion and manipulation, skeletal muscles maintain body posture, assist in the venous return of blood to the heart, and produce heat (thermogenesis). Heat is a by-product of cellular respiration; because muscles use a great deal of energy for movement, they also generate a great deal of heat. Additionally, muscles act as energy transducers by converting biochemical energy from ingested food into mechanical and thermal energy. Skeletal muscles also help protect internal organs. Because muscles make up most of the protein in the body, they constitute a potential but rarely used form of stored energy. The use of protein as an energy substrate is discussed in the metabolism unit.

Irritability The ability of a muscle to receive and respond to stimuli.

Contractility The ability of a muscle to respond to a stimulus by shortening.

Extensibility The ability of a muscle to be stretched or lengthened.

Elasticity The ability of a muscle to return to resting length after being stretched.

FIGURE 17.1 Bodybuilders.
Bodybuilding poses demonstrate muscle hypertrophy and definition.

Characteristics of Muscle Tissue

The unique characteristics of muscle tissue are specifically suited to its primary function: converting an electrical signal into a mechanical event (contraction of muscle fibers). These characteristics include irritability, contractility, extensibility, and elasticity.

Irritability refers to the ability of a muscle to receive and respond to stimuli. The stimulus is usually a chemical message (from a neurotransmitter), and the response is the generation of an electrical current (action potential) along the cell membrane. **Contractility** refers to the ability of a muscle to shorten in response to a stimulus. This shortening produces force. Muscle tissue is the only body tissue that can generate force. **Extensibility** refers to the ability of a muscle to be stretched or lengthened. Stretching occurs when a muscle is manipulated by another force. **Elasticity** refers to the ability of a muscle to return to its resting length after being stretched. Together, these characteristics of muscles allow for human movement.

Macroscopic Structure of Skeletal Muscles

The human body has over 400 skeletal muscles, which account for 40–45% of the adult male body weight and 23–25% of the adult female body weight (Hunter, 2000).

These muscles function together in remarkable ways to provide smooth, integrated movement for a wide variety of activities, many of which require little conscious thought. Muscle action is also the basis of sport and fitness activities. To understand how muscles function in various sports and exercise activity, or in any other activity, it is necessary to look beneath the skin.

Organization and Connective Tissue

Skeletal muscles are organized systematically, as shown in **Figure 17.2**. Some of this organization is apparent to the naked eye, but other aspects are apparent only when muscle fibers are viewed through a microscope.

Skeletal muscles are attached to bones by tendons, which allow the contraction of a muscle to move a bone. Each muscle is bound together by a thick layer (sheath) of connective tissue called *fascia*. Just beneath the fascia is a more delicate layer of connective tissue called *epimysium* that directly covers the muscle.

The interior of the muscle is subdivided into bundles of muscle fibers called *fasciculi* (singular: *fasciculus* or *fascicle*), which are also surrounded by connective tissue. The sheath of connective tissue that separates fasciculi within a skeletal muscle is called *perimysium*. The fasciculi are comprised of many individual muscle fibers (cells), each of which is surrounded by its own sheath of connective tissue called *endomysium*.

The three layers of connective tissue (the epimysium, the perimysium, and the endomysium) provide the framework that holds the muscle together. These layers of connective tissue come together at each end of the muscle to form the tendons that attach the muscle to bone. As a muscle contracts, it pulls on the connective tissue in which it is wrapped, causing the tendon to pull on the bone to which it is attached.

Architectural Organization

Different arrangements of fasciculi within a muscle account for the different shapes of muscles. Muscles can be described as longitudinal, fusiform, radiate, unipennate, bipennate, or circular, as shown in **Figure 17.3**. The shape of a muscle in part determines its range of motion

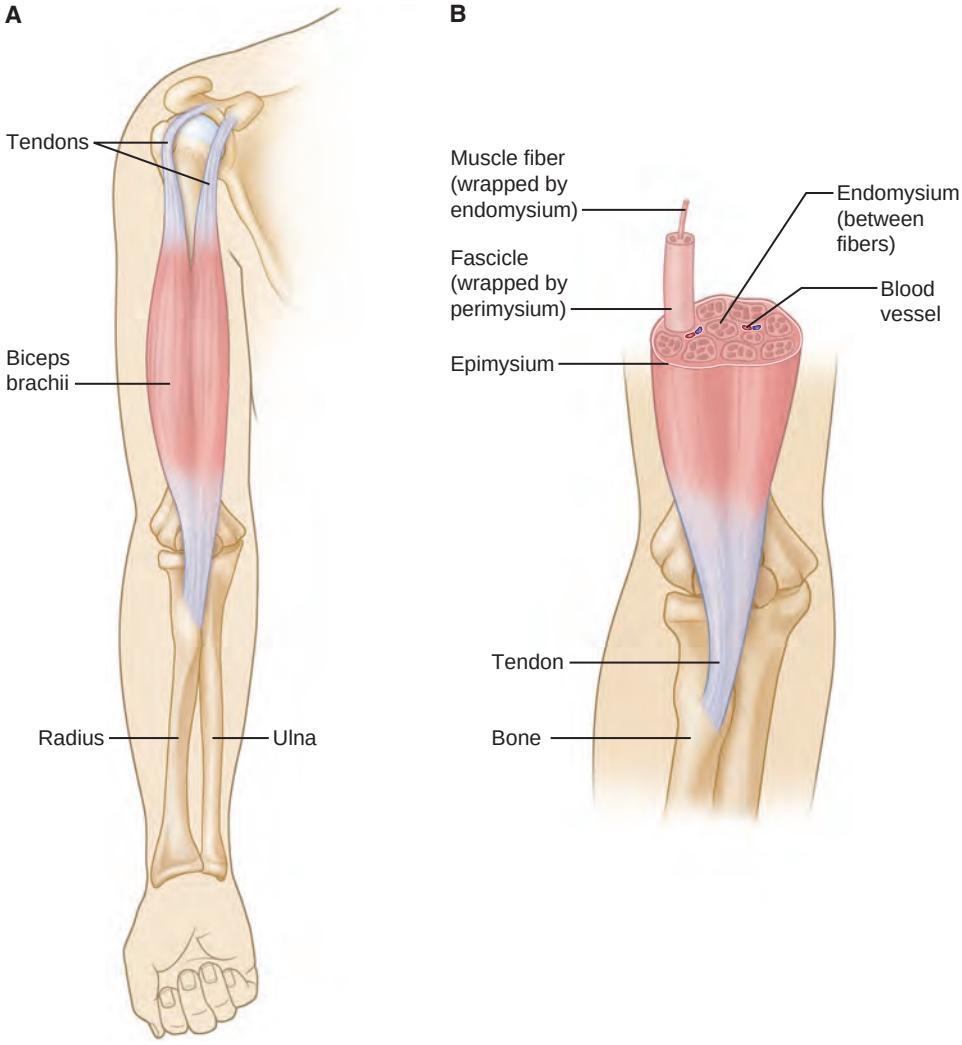


FIGURE 17.2 Organization of Skeletal Tissue.
A. Intact skeletal muscle. Biceps brachii are attached to bones through tendons. **B.** Connective tissue. The entire muscle is surrounded by connective tissue called epimysium. The muscle is organized into bundles called fasciculi, which are surrounded by connective tissue called perimysium. Each fasciculus contains many individual fibers surrounded by connective tissue called endomysium.

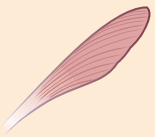
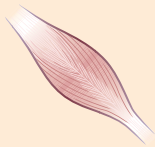
Classification	Example	Diagram
Longitudinal	Sartorius	
Fusiform	Biceps brachii	
Radiate	Gluteus medius	
Unipennate	Tibialis posterior	
Bipennate	Gastrocnemius	
Circular	Orbicular oculi (and sphincters)	

FIGURE 17.3 Arrangement of Fasciculi.

and influences its power production. Longer and more parallel muscle fibers, as are present in longitudinal muscles, allow for greater muscle shortening. Bipennate muscles, in contrast, shorten very little but are more powerful.

Microscopic Structure of a Muscle Fiber

Individual muscle fibers are composed primarily of smaller units called myofibrils, which are in turn made up of myofilaments. Refer to the organization of skeletal muscle shown in **Figure 17.4** as you read the following sections.

Sarcoplasmic Reticulum (SR) The specialized muscle cell organelle that stores and releases calcium.

Transverse Tubules (T Tubules) Organelles that carry the electrical signal from the sarcolemma into the interior of the cell.

Muscle Fibers

Muscle fibers, also called muscle cells, are long, cylinder-shaped cells ranging from 10 to 100 µm in diameter and 1–400 mm in length (Hunter, 2000; Marieb and Hoehn, 2010). The major structures of a muscle fiber and their functions are summarized in **Table 17.1**.

A skeletal muscle fiber contains many nuclei, which are located just below the cell membrane. The *sarcolemma is the polarized plasma membrane* of a muscle cell, whose properties account for the irritability of muscle. The *sarcoplasm* of a muscle cell is similar to the cytoplasm of other cells, but it has specific adaptations to serve the functional needs of muscle cells, namely, increased amounts of glycogen and the oxygen-binding protein myoglobin.

A muscle fiber contains the same organelles found in other cells (including a large number of mitochondria) along with some specialized organelles. Organelles of specific interest are the transverse tubules (T tubules), the sarcoplasmic reticulum (SR), and the myofibrils. Myofibrils are composed primarily of the protein myofilaments and are responsible for the contractile properties of muscles. Skeletal muscle cells also have a highly organized complex cytoskeleton that provides the framework for the organelles and plays an important role in transmitting force from muscle tissue to bone.

Sarcoplasmic Reticulum and Transverse Tubules

Figure 17.4 illustrates the relationship among the myofibrils, the sarcoplasmic reticulum, and the transverse tubules. The **sarcoplasmic reticulum (SR)** is a specialized organelle that stores and releases calcium. It is an interconnecting network of tubules running parallel with and wrapped around the myofibrils. (In **Figure 17.4** the sarcolemma has been partially removed to illustrate the SR and myofibrils.) The major significance of the sarcoplasmic reticulum is its ability to store, release, and take up calcium and thereby control muscle contraction. Calcium is stored in the portion of the sarcoplasmic reticulum called the *lateral sacs* or *cisterns*.

The **transverse tubules (T tubules)** are organelles that carry the electrical signal from the sarcolemma to the interior of the cell. T tubules are continuous with the sarcolemma and protrude into the sarcoplasm of the cell. As their name implies, T tubules run perpendicular (transverse) to the myofibril. Each T tubule runs between two lateral sacs of the sarcoplasmic reticulum, creating what is known as a triad; this ensures that that the spread of an electrical signal (action potential) through the T tubules causes the release of calcium from the lateral sacs of the sarcoplasmic reticulum.

Myofibrils and Myofilaments

Each muscle fiber contains hundreds to thousands of smaller cylindrical units, or rod-like strands, called

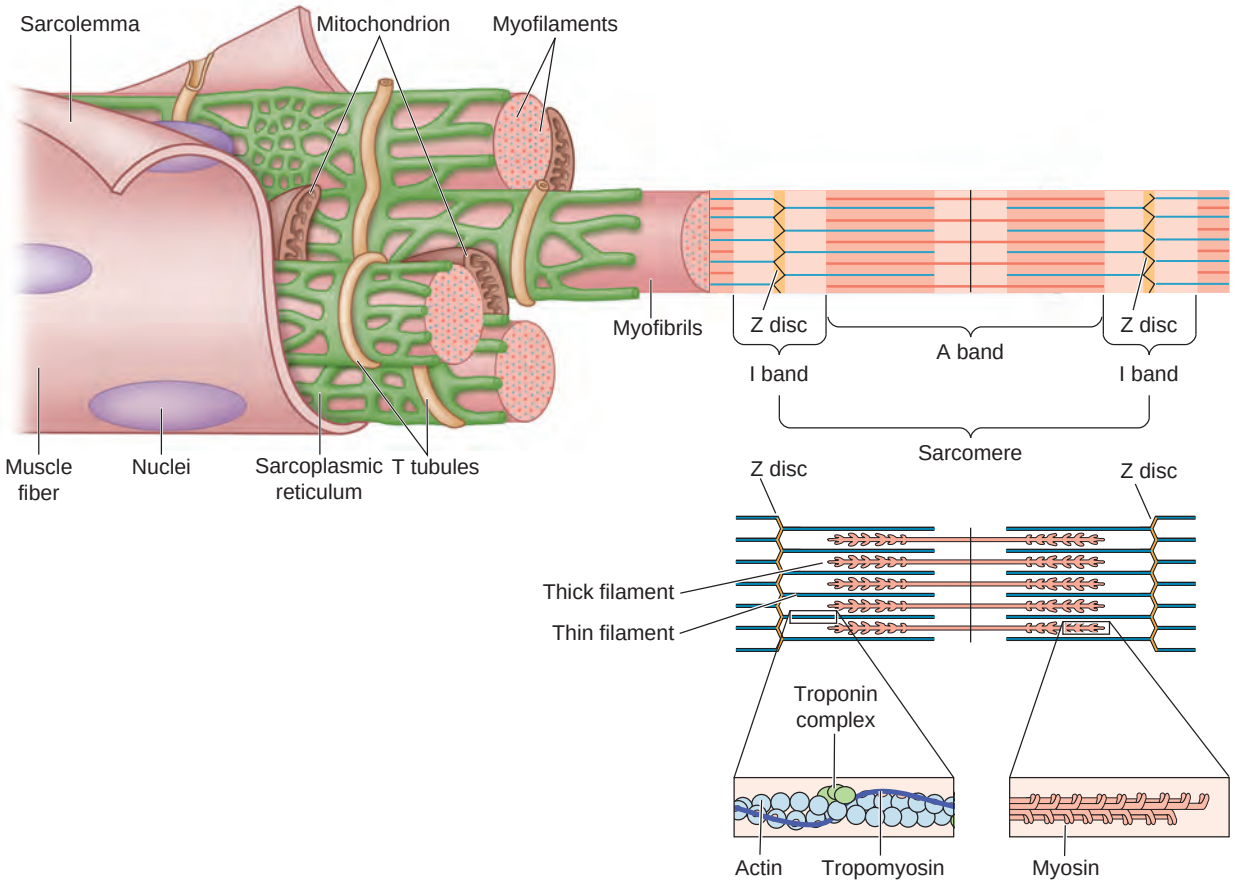


FIGURE 17.4 Organization of a Muscle Fiber.
There is a close anatomical relationship among the organelles, specifically the myofibrils, T tubules, and sarcoplasmic reticulum (SR). The repeating pattern of the myofibrils is due to the arrangement of the myofilaments.

TABLE 17.1 Summary of Major Components of a Skeletal Muscle Cell

Cell Part	Description	Function
Nucleus	Multinucleated	Is the control center for the cell
Sarcolemma	Polarized cell membrane	Is capable of receiving stimuli from the nervous system
Sarcoplasm	Intracellular material	Holds organelles and nutrients; provides the medium for glycolytic enzymatic reactions
Organelles		
Myofibrils	Rod-like structures composed of smaller units called myofilaments; account for 80% of muscle volume	Contain contractile proteins (myofilaments), which are responsible for muscle contraction
T tubules	Series of tubules that run perpendicular (transverse) to the cell and are open to the external part of cell	Spread polarization from the cell membrane into the interior of cell, which triggers the sarcoplasmic reticulum to release calcium
Sarcoplasmic reticulum	Interconnecting network of tubules running parallel with and wrapped around the myofibrils	Stores and releases calcium
Mitochondria	Sausage- or spherical-shaped organelles; numerous in a muscle cell	Are the major site of energy production

myofibrils (**Figure 17.4**). Myofibrils are specialized contractile organelles composed of myofilaments. These myofibrils, sometimes simply called fibrils, typically lie parallel to the long axis of the muscle cell and extend the entire length of the cell. Myofibrils account for approximately 80% of the volume of a muscle fiber.

As shown in **Figure 17.4**, each myofibril is composed of still smaller myofilaments (or filaments) arranged in a repeating pattern along the length of the myofibril. **Myofilaments** are contractile proteins (thick and thin) that are responsible for muscle contraction. Myofilaments account for most of the muscle protein. The repeating pattern of these myofilaments along the length of the myofibril gives skeletal muscle its striated appearance. Each repeating unit is referred to as a sarcomere.

Sarcomeres

A **sarcomere** is the functional unit (contractile unit) of a muscle fiber. As shown in **Figure 17.5**, each sarcomere contains two types of myofilaments. The *thick* filaments are composed primarily of the contractile protein myosin, and the *thin* filaments are composed primarily of the contractile protein actin. Thin filaments also contain the *regulatory proteins*, troponin and tropomyosin. Viewed with an electron microscope, the arrangement of myofilaments has the appearance of alternating bands of light and dark striations. The light bands are called *I bands* and contain only thin filaments. The dark bands are called *A bands* and contain thick and thin filaments, with the thick filaments running the entire length of the A band. The length of the thick filament thus determines the length of the A band.

The letter names of the various regions of the sarcomere derive from the first letter of the German word that describes the appearance of each. The names of the bands relate to the refraction of light through them. The I band is named for the word *isotropic*, which means that this area appears lighter because more light passes through it. The A band is named for its *anisotropic* properties, meaning that it appears darker because not as much light passes through. These properties relate to the types of filament present in the bands.

Each A band is interrupted in the midsection by an *H zone* (from the German *Hellerscheibe*, for "clear disk"), where there is no overlap of thick and thin filaments. Running through the center of the H zone is a dense line called the *M line* (from the German *Mittelscheibe*, for "middle disk"). The I bands are also interrupted at the midline by a darker area called the Z disk (from the German *Zwischenscheibe*, for "between disc"). A sarcomere extends from one Z disk to the successive Z disk. The Z disk serves to anchor the thin filaments to adjacent sarcomeres.

Myofibril Contractile organelles composed of myofilaments.

Myofilaments Contractile (thick and thin) proteins responsible for muscle contraction.

Sarcomere The functional unit (contractile unit) of muscle fibers.

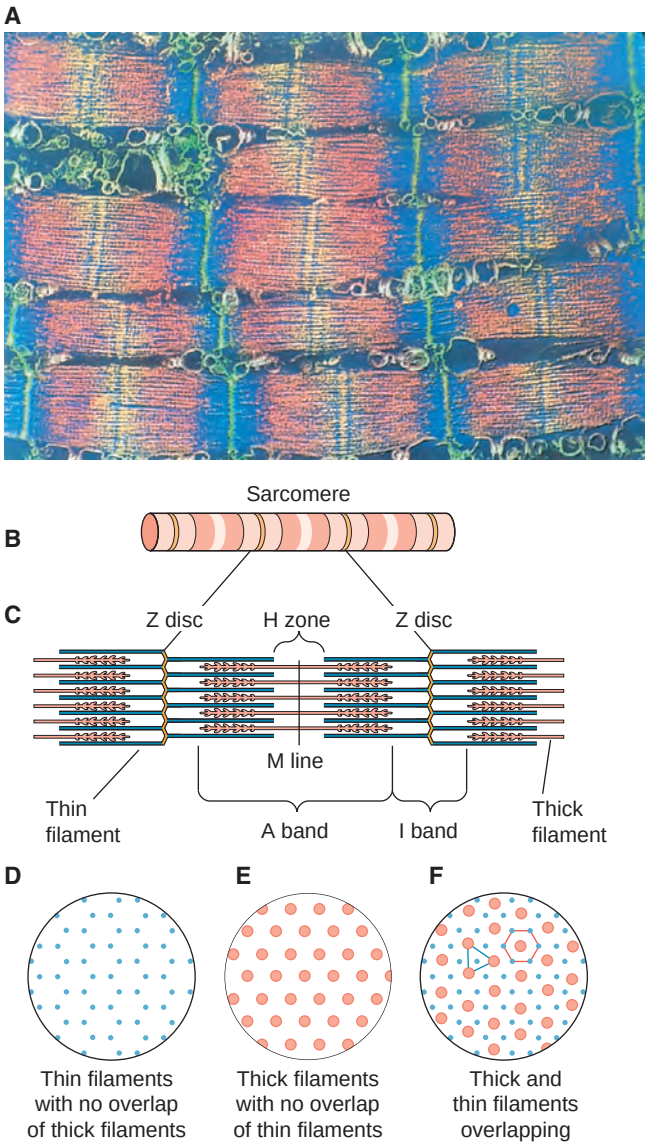


FIGURE 17.5 Arrangement of Myofilaments in a Sarcomere. **A.** Micrograph of sarcomeres. **B.** Model of sarcomeres. **C.** Relationship between thick and thin filaments. **D.** Cross-sectional view of thin filaments. **E.** Cross-sectional view of thick filaments. **F.** Cross-sectional view of thick and thin filaments.

Myofilaments occupy three-dimensional space. The arrangement of the myofilaments at different points in the sarcomere is shown in **Figure 17.5D–F**. **Figure 17.5D** presents a cross section of thin filaments in regions where there is no overlap with thick filaments (i.e., the I band), whereas **Figure 17.5E** presents a cross section of thick filaments in a region where there is no overlap with thin filaments (H zone). Notice that

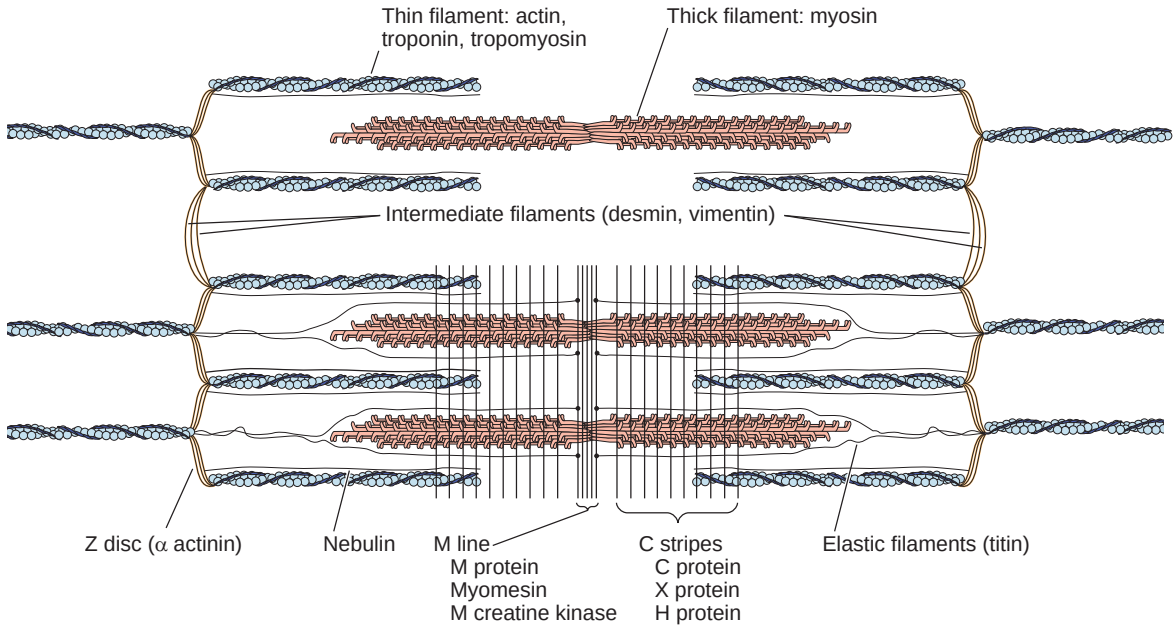


FIGURE 17.6 Representation of Auxiliary Proteins in the Sarcomere.
***Source*:** From Billeter, R. & H. Hoppeler: Muscular basis of strength. In Komi, P. V. (ed.): *Strength and Power in Sport*. Champaign, IL: Human Kinetics. 45 (1992). Copyright 1992 by International Olympic Committee. Reprinted by permission.

in regions where the thick and thin filaments overlap (**Figure 17.5F**), each thick filament is surrounded by six thin filaments, and each thin filament is surrounded by three thick filaments.

A sarcomere consists of contractile, regulatory, and structural proteins. Structural proteins make up much of the cytoskeleton. In recent years, researchers have identified dozens of proteins that contribute to a highly organized and complex sarcomere (Caiozzo and Rourke, 2006). **Figure 17.6** diagrams some of the major auxillary structural proteins in the cytoskeleton of the sarcomere and indicates the relationship between the structural and contractile proteins (Billeter and Hoppler, 1992). Proteins of the M line and the Z disk hold the thick and the thin filaments in place, respectively. Titan, an elastic filament, helps keep the thick filament in the middle of the sarcomere during contraction. Structural proteins also serve as mechanical links between sarcomeres and the extracellular matrix. Collectively, these connection sites are called costameres (Caiozzo and Rourke, 2006).

Molecular Structure of the Myofilaments

The contractile proteins of the myofilaments slide over one another during muscular contraction. Hence, the **sliding filament theory of muscle contraction** explains how muscles contract. Knowing the structure of the myofilaments is essential to understanding how muscles contract.

Thick Filaments

Thick filaments are composed of myosin molecules, primarily the contractile form, myosin heavy chain (MHC). Myosin light chain (MLC) molecules are also present and assist in regulating the rate of contractions (Caiozzo and Rourke, 2006). The term myosin as used in this text refers to the contractile form (MHC) unless otherwise specified. Each molecule of myosin has a rod-like tail and two globular heads (**Figure 17.7A**). A typical thick filament contains approximately 200–300 myosin molecules (Caiozzo and Rourke, 2006). These molecules are oriented so that the tails form the central rod-like structure of the filament (**Figure 17.7B**). The globular myosin heads extend outward and form *cross-bridges* when they interact with thin filaments. The myosin heads have two reactive sites: One allows it to bind with the actin filament, and one binds to ATP. Only when the myosin heads bind strongly to the active sites on actin, forming a cross-bridge, can contraction occur.

The myosin subunits are oriented in opposite directions along the filament, forming a central section that lacks projecting heads (**Figure 17.7C**). The result is a bare zone in the middle of the filament, which is the H zone seen in the middle of the A band (**Figure 17.5C** and **D**).

Thin Filaments

Thin filaments are composed primarily of the contractile protein actin. As illustrated in **Figure 17.8A** and **B**, actin is composed of small globular subunits (G actin) that form long strands called fibrous actin (F actin). A filament of

FOCUS ON APPLICATION: *CLINICALLY RELEVANT*

Increasing Protein Synthesis—Interaction of Training and Nutrition

Many athletes, especially those engaged in resistance training, are interested in increasing protein synthesis. Increased protein synthesis increases the amount of contractile proteins and thus makes the muscles larger and stronger. Protein synthesis is enhanced in several circumstances: (a) following resistance exercise, (b) when amino acid availability is increased, and (c) when blood insulin levels are high. Recent research by Rasmussen et al. (2000) suggests that when these three conditions occur together, their effect on protein synthesis is additive. Participants in this study ingested a drink containing six essential amino acids and 35 g of sucrose following a bout of resistance training. The participants consumed the drink at either 1 or 3 hours after training, and the results were compared to a control group that consumed a flavored placebo drink. The ingestion of sucrose caused an elevation in blood insulin levels.

The combination of essential amino acids, elevated insulin levels, and resistance training stimulated protein synthesis approximately 400% above predrink levels when the drink was consumed 1 or 3 hours after resistance exercise. This increase in protein synthesis is greater than that reported following resistance training alone (~100% increase in protein synthesis), increased amino acid availability alone (~150% increase in protein synthesis), and the combination of resistance training and increased amino acid availability (~200% increase in protein synthesis). Based on these results, fitness professionals may recommend that exercise participants interested in increasing muscle size consider consuming a drink containing essential amino acids and carbohydrate following resistance training workouts. The supplement is equally effective when consumed 1 or 3 hours after a workout.

Source: Rasmussen et al. (2000).

actin is formed by two strands of F actin coiled about each other to form a double-helical structure; this structure, which resembles two strands of pearls wound around each other, may be referred to as a *coiled coil* (**Figure 17.8C**). The actin molecules contain active sites to which myosin heads bind during contraction.

The thin filaments also contain the regulatory proteins called tropomyosin and troponin, which regulate the interaction of actin and myosin. *Tropomyosin* is a long, double-stranded, helical protein wrapped about the long axis of the actin backbone (**Figure 17.8D**). Tropomyosin blocks the active site on actin, thereby inhibiting actin and myosin from binding under resting conditions.

Troponin is a small, globular protein complex composed of three subunits that control the position of the tropomyosin (**Figures 17.8E** and **17.9**). The three units are troponin C (TnC), troponin I (TnI), and troponin T (TnT). TnC contains the calcium-binding sites,

Sliding Filament Theory of Muscle Contraction The theory that explains muscle contraction as the result of myofilaments sliding over each other.

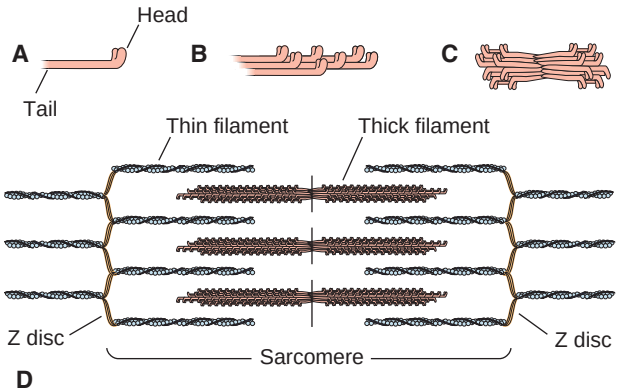


FIGURE 17.7 Molecular Organization of Thick Filaments. **A**. Individual myosin molecules have a rod-like tail and two globular heads. **B**. Individual molecules are arranged so that the tails form a rod-like structure and the globular heads project outward to form cross-bridges. **C**. Myosin subunits are oriented in opposite directions along the filament forming a central bare zone in the middle of the filament (H zone). **D**. Thick filament (myosin) within a single sarcomere showing the myosin heads extending toward the thin filament.

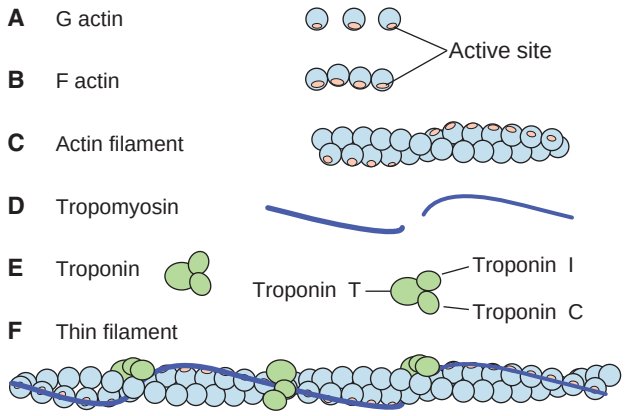


FIGURE 17.8 Molecular Organization of Thin Filaments. **A.** Individual actin subunits (globular, G actin) shown with active site for binding to myosin heads. **B.** Fibrous actin (F actin). **C.** Actin filament with two strands of fibrous actin wound around itself to form a coiled coil. Active sites are exposed. **D.** Tropomyosin is a regulatory protein that covers the binding sites on actin. **E.** Troponin is a regulatory protein that when bound to Ca^{2+} removes tropomyosin from its blocking position on actin. **F.** The thin filament is composed of actin, tropomyosin, and troponin.

TnT binds troponin to tropomyosin, and TnI inhibits the binding of actin and myosin in the resting state (**Figure 17.9B**). When calcium binds to the TnC subunit, the troponin complex undergoes a configurational change. Because troponin is attached to tropomyosin, the change in the shape of troponin causes tropomyosin to be removed from its blocking position, thus exposing the active sites on actin (Marieb and Hoehn, 2010). Once the active sites are exposed, the myosin heads can bind to the actin, forming the cross-bridges (**Figure 17.9C**). Thus, calcium is key to controlling the interaction of the filaments and, thus, muscle contraction.

Contraction of a Muscle Fiber

For a muscle to contract, three major events must happen:

1. An *action potential* must be generated in the motor neuron that innervates the muscle fibers.
2. The motor neuron must release a neurotransmitter that travels across the neuromuscular junction and binds to receptors on the muscle cell membrane (sarcolemma).
3. An action potential in the muscle fiber must lead to the sliding of the myofilaments—thus shortening the muscle cell.

The first two of these necessary events are discussed in detail in Chapter 20. The following section addresses the third event in the sequence: how a muscle fiber produces force when stimulated. The process whereby electrical events in the sarcolemma of the muscle fiber are linked to the movement of the myofilaments is called *excitation-contraction coupling*.

Before detailing the physiological changes within the muscle fiber (and their myofilaments) as a result of electrical stimulation, however, it is useful to consider the major tenets of the sliding filament theory as revealed through microscopic studies.

The Sliding Filament Theory of Muscle Contraction

The sliding filament theory of muscle contraction is commonly used to describe how muscle contraction generates force. A great deal of data have been amassed from X-ray, light microscopic, and electron microscopic studies to support the sliding filament theory of muscle contraction. This theory accounts for force production during concentric (shortening) contractions very well. However, there is some concern about the extent to which the sliding filament theory adequately explains force generation during eccentric (lengthening) contractions. The basic principles of this theory are as follows:

1. The force of contraction is generated by the process that slides the actin filament over the myosin filament.
2. The lengths of the thick and the thin filaments do not change during muscle contraction.
3. The length of the sarcomere decreases as the actin filaments slide over the myosin filaments and pull the Z disks toward the center of the sarcomere.

See animation [Sliding Filament Theory] at http://thePoint.lww.com/Plowman4e.

Changes in the Sarcomere during Contraction

Much of the evidence for the sliding filament theory comes from observed changes in the length of a sarcomere during muscular contraction. **Figure 17.10** diagrams the sarcomere during rest (**Figure 17.10A**) and during shortening with contraction (**Figure 17.10B**). Notice the following changes in the sarcomere:

1. The A band does not change length, but the Z disks do move closer together. The length of the A band is preserved because the thick filament length does not change.
2. The I band shortens and may disappear. The I band shortens because the thin filaments are pulled over the thick filaments toward the center of the sarcomere. Thus, there is little or no area where the thin filaments do not overlap the thick filaments.
3. The H zone shortens and may disappear because the thin filaments are pulled over the thick filaments toward the center of the sarcomere. If the thin filament overlaps the thick filament for the entire length of the thick filament, there is no H zone.

As detailed in the next section, the sarcomere shortens as the result of the attachment of the myosin heads with the active site on actin and the subsequent release of stored energy that swivels the myosin cross-bridges. This step causes the actin to pull the Z disk toward the center of the sarcomere, which in turn causes the sarcomere, and thus the muscle fiber, to shorten.

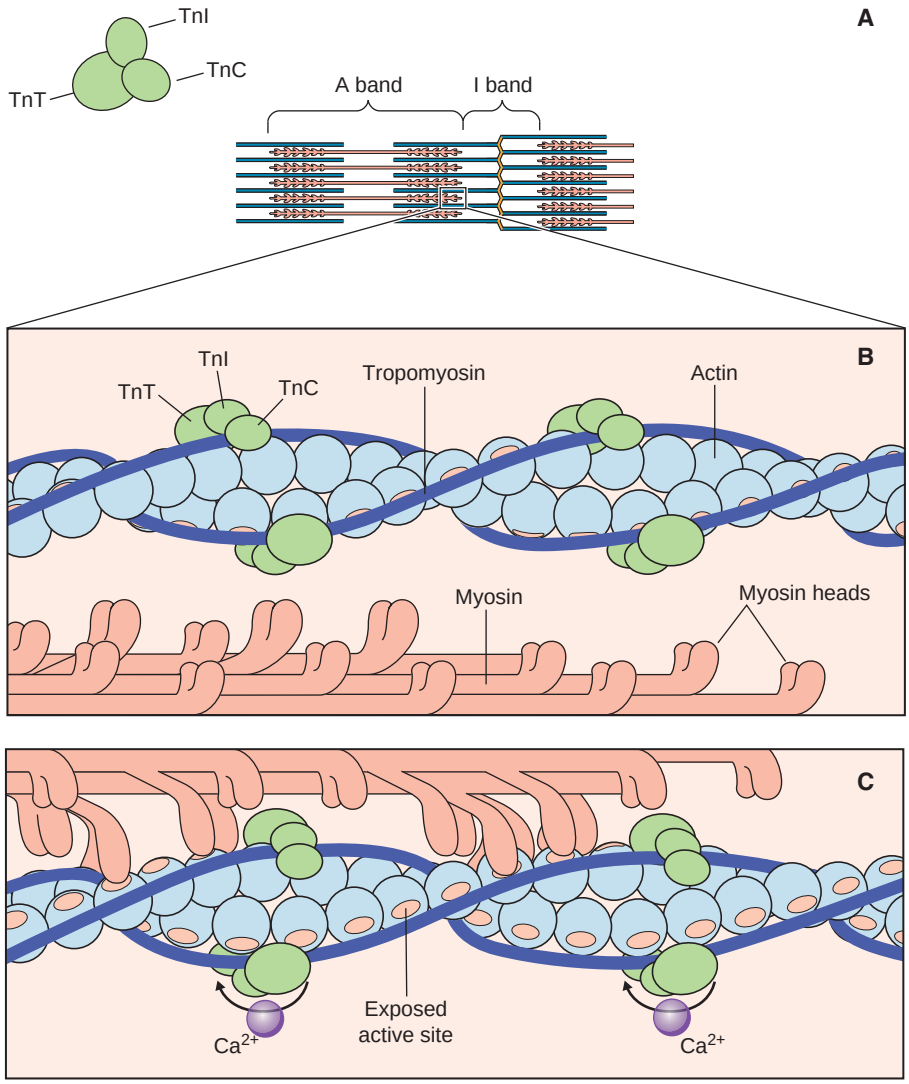


FIGURE 17.9 Regulatory Function of Troponin and Tropomyosin.
A. Troponin is a small globular protein with three subunits. **B.** Resting condition: Tropomyosin blocks the active sites on actin, preventing actin and myosin from binding. **C.** Contraction: When troponin binds with Ca^{2+}, it undergoes a configurational change and pulls tropomyosin from the blocking position on the actin filament, allowing myosin heads to form cross-bridges with actin.

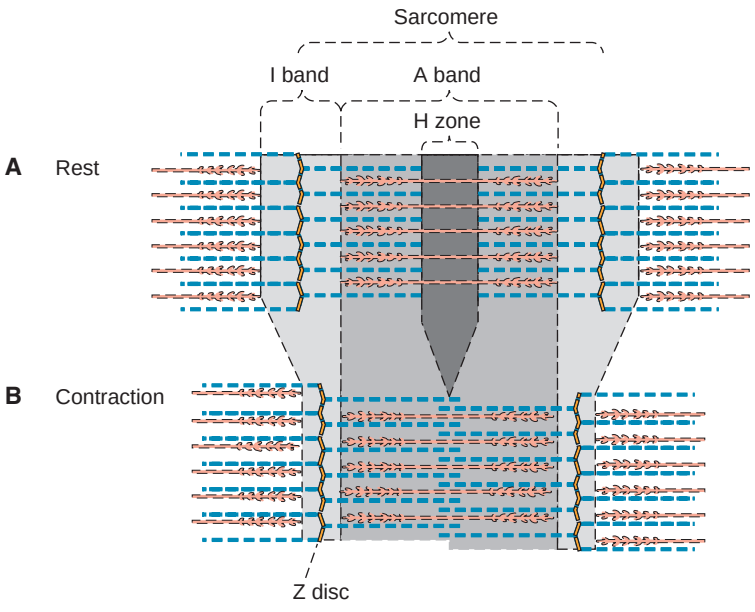


FIGURE 17.10 Changes in a Sarcomere during Contraction.
A. Sarcomere at rest. **B.** During contraction of the sarcomere, the lengths of actin and myosin filaments are unchanged. Sarcomere shortens because actin slides over myosin, pulling Z disks toward the center of the sarcomere. The H zone disappears, the I band shortens, and the A band remains unchanged.

Excitation-Contraction Coupling

Excitation-contraction coupling is the sequence of events by which an action potential (AP; an electrical event) in the sarcolemma of the muscle cell initiates the sliding of the myofilaments, resulting in contraction (a mechanical event). Excitation-contraction coupling occurs in three phases:

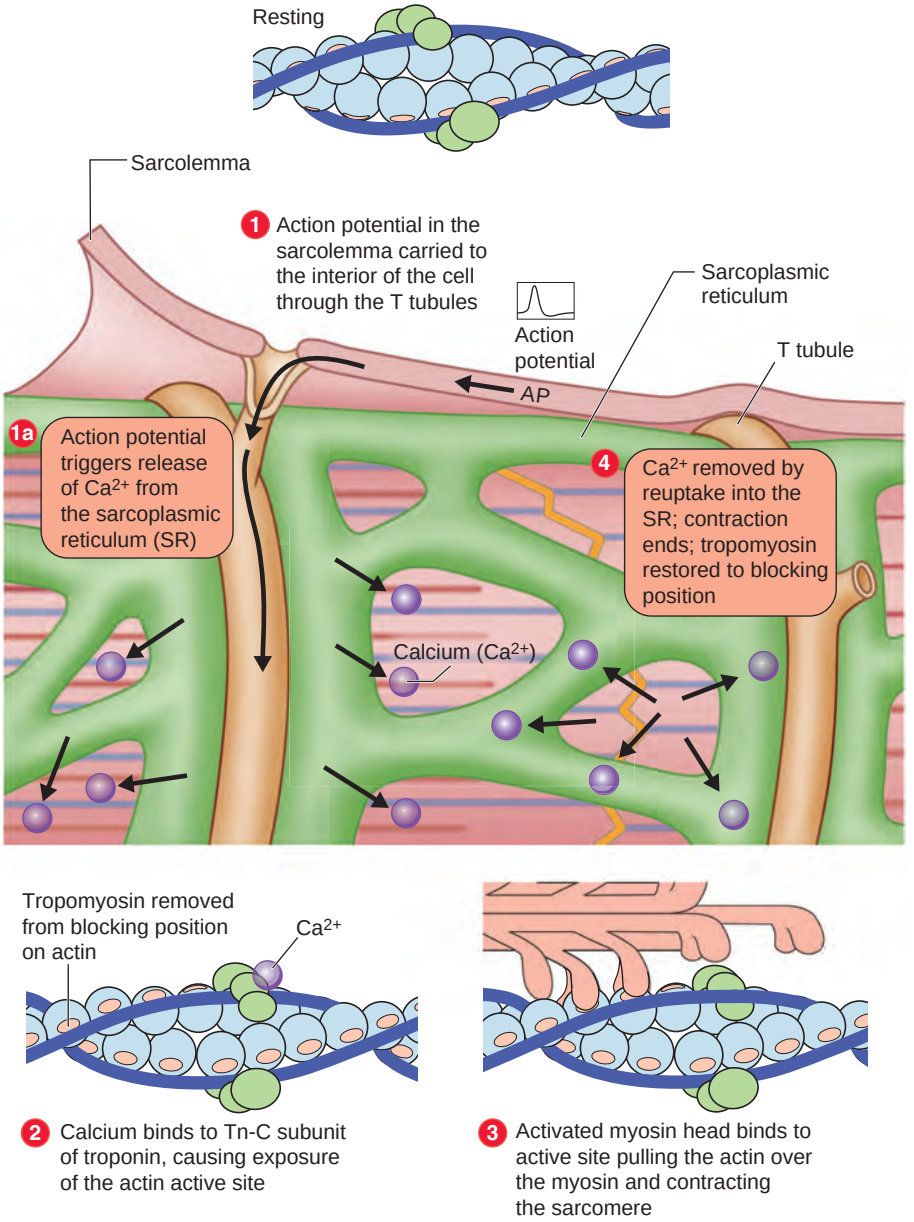
1. The spread of depolarization
2. The binding of calcium to troponin
3. The generation of force (cross-bridge cycling)

Figure 17.11 summarizes what occurs in each phase of excitation-contraction coupling. In the resting state, the regulatory protein tropomyosin is covering the active sites on actin. Excitation-contraction coupling begins with depolarization and the spread of an action potential (AP) along the sarcolemma (labeled 1 in **Figure 17.11**) and continues with the propagation of the action potential into the T tubules. The action potential in the T tubules causes the release of calcium from the adjacent lateral sacs of the sarcoplasmic reticulum (labeled 1a **Figure 17.11**).

The calcium that is released from the SR binds to the troponin molecules (TnC subunit) on the thin filament during the second phase. This causes troponin to undergo a configurational change, thereby removing tropomyosin from its blocking position on the actin filament (labeled 2 in **Figure 17.11**).

The third phase of excitation-contraction coupling is the cross-bridging cycle (labeled 3 in **Figure 17.11** and

FIGURE 17.11 Phases of Excitation-Contraction Coupling.



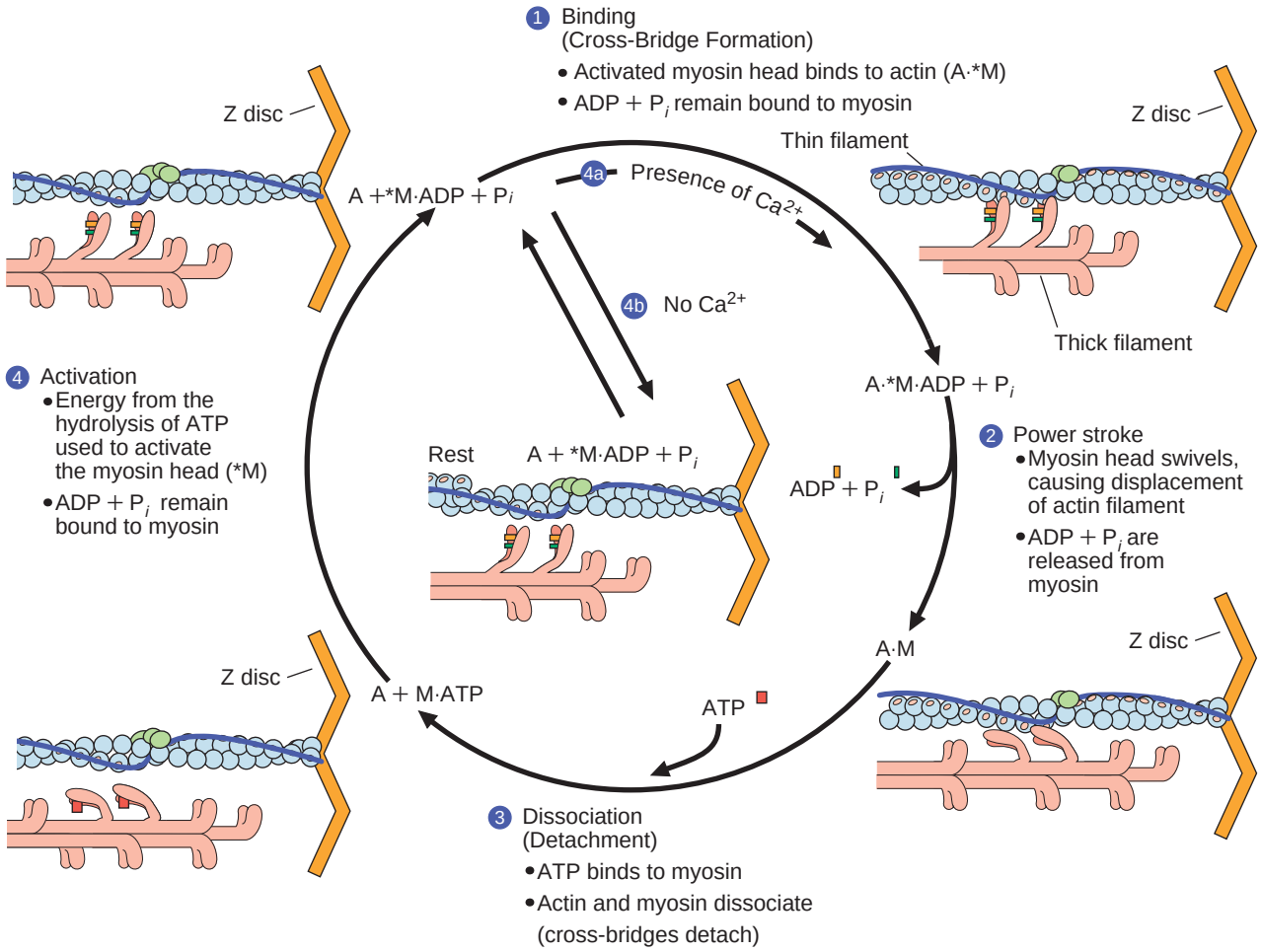


FIGURE 17.12 Force Generation of the Contractile Elements: The Cross-Bridging Cycle.

detailed fully in **Figure 17.12**). The **cross-bridging cycle** involves myosin heads binding to the active sites on actin and a series of cyclic events necessary for the generation of tension within the myosin heads during muscle contraction. The tension within the contractile elements results from the binding of the myosin heads to actin and the subsequent release of stored energy in the myosin heads.

As detailed in **Figure 17.12**, the cross-bridging cycle occurs in four steps (Marieb and Hoehn, 2010; Vander et al., 2001):

1. Binding of myosin heads to actin (cross-bridge formation)
2. Power stroke
3. Dissociation of myosin and actin
4. Activation of myosin heads

Excitation-Contraction Coupling The sequence of events by which an action potential in the sarcolemma initiates the sliding of the myofilaments, resulting in contraction.

Cross-Bridging Cycle The cyclic events necessary for the generation of force or tension within the myosin heads during muscle contraction.

The first step in the *cross-bridge cycle* is the binding of activated myosin heads (*M) with the active sites on actin, forming cross-bridges. In **Figure 17.12**, a centered dot (·) indicates binding, and an asterisk (*) indicates activated myosin heads. Thus, A·*M means that the activated myosin heads are bound to actin (A), whereas A + M indicates that actin and myosin are unbound.

The second step in the cross-bridging cycle is the power stroke. During this step, activated myosin heads swivel from their high-energy, activated position to a low-energy configuration (M with no*). This movement of the myosin cross-bridges results in a slight displacement (sliding) of the thin filament over the thick filament toward the center of the sarcomere. As shown in **Figure 17.12**, during the second step, ADP and P*i* are released from the myosin heads, resulting in myosin bound only to actin (A·M).

The third step involves the binding of ATP to the myosin heads and the subsequent dissociation (detachment) of the myosin cross-bridges from actin, thus producing A + M·ATP.

Note the role of ATP in steps 3 and 4. The *binding of ATP* molecules to the myosin head in step 3 allows the myosin heads to detach from actin. In the fourth step, the *breakdown of ATP* provides the energy to activate the myosin heads (*M). The activation of the myosin head is extremely important because it provides the cross-bridges with the stored energy to move the actin during the power stroke. The breakdown of ATP at this step depends on the presence of myosin ATPase (also known as myofibrillar ATPase), as depicted in the following reaction:

$$M \cdot ATP \xrightarrow{\text{myosin ATPase}} *M \cdot ADP + P_i$$

Notice that the products of ATP hydrolysis, ADP + P_i, remain bound to the myosin heads and that the myosin is now in its high-energy or activated state.

The cross-bridging cycle continues as long as ATP is available and calcium is bound to troponin (TnC), causing the active sites on actin to be exposed. On the other hand, activated myosin will remain in the resting state awaiting the next stimulus if calcium is not available in sufficient concentration to remove tropomyosin from its blocking position on actin (labeled 4b in **Figure 17.12**). Because each cycle of the myosin cross-bridges barely displaces the actin, the myosin heads must bind to the actin and be displaced many times for a single contraction to occur. Thus, myosin makes and breaks its bond with actin hundreds or even thousands of times during a single muscle twitch. For this make-and-break cycle to occur, the myosin heads must detach from actin and then be reactivated. This detaching and reactivating process requires the cycle to be repeated and requires the presence of ATP (step 3).

An analogy helps explain the role of ATP in providing energy to activate the myosin head. Visualize a spring-loaded mousetrap. It takes energy to set the trap, just as it takes the splitting of ATP to set or activate the myosin head. Once set, however, the trap will release energy when it is sprung. In a similar manner, the myosin head possesses stored energy that is released when the myosin heads bind to actin and swivel.

It may be useful to review the cycle of events in **Figure 17.12** several times, paying attention to a different aspect (the symbols, the wording, the diagrams, the role of ATP) in each review. Also, keep in mind that ATP plays several important roles in muscle contraction:

1. ATP breakdown provides the energy to activate and reactivate the myosin cross-bridge prior to binding with actin.
2. ATP binding to the myosin head is necessary to break the cross-bridge linkage between the myosin heads and the actin so that the cycle can repeat.
3. ATP is used for the return of calcium into the sarcoplasmic reticulum and restoration of the resting membrane potential once contraction has ended.

The final phase of muscular contraction is a return to muscular relaxation. Relaxation occurs when the nerve impulse ceases and calcium is pumped back into the sarcoplasmic reticulum by active transport (labeled 4, **Figure 17.12**). In the absence of calcium, tropomyosin returns to its blocking position on actin, and myosin heads are not able to bind to actin. While emphasis is usually placed on muscle contraction, relaxation of a muscle following contraction is just as important.

All-or-None Principle

According to the **all-or-none principle**, when a motor neuron is stimulated, all the muscle fibers in that motor unit contract to their fullest extent or do not contract at all. A **motor unit** is defined as a motor neuron (α_1 or α_2) and the muscle fibers it innervates. The minimal stimulus necessary to initiate that contraction is referred to as the *threshold stimulus*. When the threshold is reached, a muscle fiber contracts to its fullest extent. This principle involves the electrical properties of the stimulated cell membrane and thus applies to a motor unit or a single muscle fiber only, not to the entire muscle. Consider the analogy of a light switch. When enough pressure (threshold) is applied to the switch to flip it on, the light will turn on to its fullest extent. When the switch controls a group of lights (like a motor neuron innervating multiple muscle fibers), all the lights will turn on to their fullest extent. You cannot make the lights brighter by pushing the switch harder, because it is an all-or-none response. The same is true for an individual muscle fiber or a motor unit: Either a threshold stimulus is reached and contraction occurs or a threshold stimulus is not reached and contraction does not occur.

Muscle Fiber Types

Muscle fibers are typically described by two characteristics: their contractile (twitch) properties and their metabolic properties (**Figure 17.13**).

Contractile (Twitch) Properties

Based on differences in *contractile (twitch) properties*, human muscle fibers can be categorized as *slow-twitch (ST)* or *fast-twitch (FT) fibers*. Slow-twitch fibers are sometimes called Type I fibers, and fast-twitch fibers Type II fibers. The difference between ST and FT fibers appears to be absolute—like the difference between black and white. Some FT fibers contract and relax slightly faster than other fast-twitch fibers, but both of them are clearly much faster than ST fibers. Understanding the difference between twitch speeds begins with understanding the integration of muscles and nerves.

Skeletal muscle fibers are innervated by alpha (α) motor neurons, which exist in two categories, α_1 and α_2.

Muscle Fibers			
Twitch properties	Slow	Fast	
Metabolic properties	Oxidative	Oxidative/ glycolytic	Glycolytic
Name based on twitch and metabolic properties	SO	FOG	FG
Other nomenclature	ST, Type I	FTa, FTA, Type IIA	FTx, FTX, Type IIX
Motor Neurons			
Neuron type	α_2	α_1	α_1
Neuron size	Small	Large	Large
Conduction velocity	Slow	Fast	Fast
Recruitment threshold	Low	High	High

FIGURE 17.13 Properties of Motor Units.

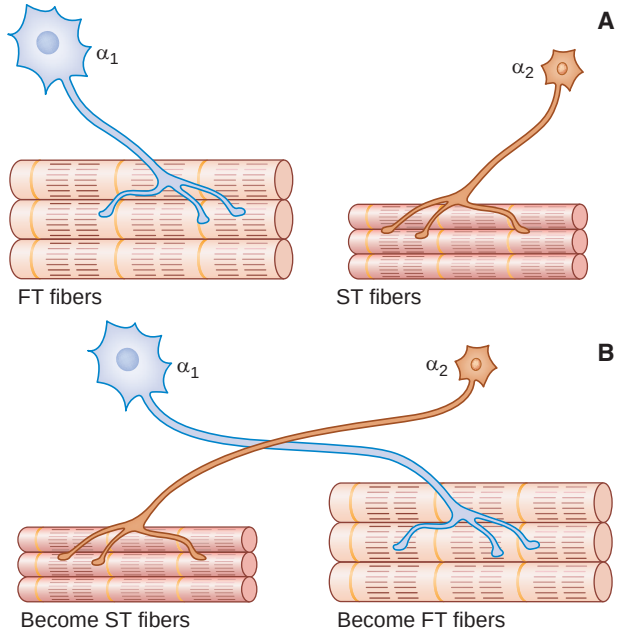


FIGURE 17.14 Results of Cross Innervation.
A. Under normal conditions, α_1 motor neurons innervate FT fibers and α_2 motor neurons innervate ST fibers. **B.** If the neurons supplying the muscles are switched (cross-innervated), the muscle fibers acquire the properties of the new motor neuron.

The α_1 motor neurons innervate FT fibers, and the α_2 motor neurons innervate ST fibers. **Figure 17.14** depicts the results of an experiment that manipulated the innervation of muscle fibers. The α_1 motor neuron was severed from the FT fibers and connected to the ST fibers, and the α_2 motor neuron was cut from the ST fibers and connected to the FT fibers. Importantly, the ST fibers became FT fibers when the α_1 motor neuron replaced the α_2 motor neuron, and vice versa. Therefore, it is logical to conclude that the contractile property of muscle depends on the type of motor neuron that innervates the muscle fibers (Buller et al., 1960; Noth, 1992).

Other elements in the muscle, especially the contractile enzyme myosin ATPase, also contribute to the variation in twitch speed. Indeed, when biopsied muscle fibers are typed, the amount of stain for myosin ATPase is often used to distinguish twitch speed, since the motor neurons are not typically biopsied.

Note that α_2 motor neurons are the smaller of the two nerves and innervate the ST muscle fibers; the α_1 motor neurons are the larger nerves and innervate the FT muscle fibers. The size difference is important because small motor neurons have low excitation thresholds and slow conduction velocities and are thus recruited at low workloads. In contrast, larger motor neurons have a higher excitation threshold and are not recruited until high force output is needed. Thus, motor neurons are recruited according to the *size principle*. Smaller motor units (α_2 motor neurons innervating ST fibers) are recruited during activities that require low force output, such as maintaining posture. As the need for force production increases, such as lifting heavy weights, larger motor units (α_1 motor neurons innervating FT fibers) are recruited.

All-or-None Principle When a motor neuron is stimulated, all of the muscle fibers in that motor unit contract to their fullest extent or do not contract at all.

Motor Unit A motor neuron and the muscle fibers it innervates.

Metabolic Properties

On the basis of differences in *metabolic properties*, human muscle fibers can be described as *glycolytic*, *oxidative*, or a combination of both, *oxidative/glycolytic*. All muscle fibers can produce energy both anaerobically (without oxygen, labeled as glycolytic) and aerobically (with oxygen, labeled as oxidative). These processes and terms are fully explained in the unit on metabolism.

Despite the ability of all muscle fibers to produce energy by both glycolytic and oxidative processes, one or the other type of energy metabolism may predominate or the production may be balanced. Thus, the metabolic properties of muscle fibers are not absolute characteristics as much as a continuum (oxidative to glycolytic). This continuum involves shades of gray, unlike the black-or-white typing of slow or fast twitch fibers. The metabolic properties of a muscle specimen are determined by staining for key enzymes (often phosphofructokinase [PFK] for glycolytic processes and succinate dehydrogenase [SDH] for oxidative processes) (Saltin et al., 1977).

Integrated Nomenclature

Slow-twitch fibers rely primarily on oxidative metabolism to produce energy and are therefore referred to as **slow oxidative (SO) fibers** (Type I fibers). Fast-twitch fibers that can work under both oxidative and glycolytic conditions are called **fast oxidative glycolytic (FOG) fibers**; these fibers are also referred to as Type IIA or Type IIa fibers. Other fast-twitch fibers that perform predominantly under glycolytic conditions are called **fast glycolytic (FG) fibers** and are also known as Type IIX or Type IIx fibers.

Skeletal muscle has been classified in numerous ways based on the composition of the myosin molecules, particularly the isoforms of the myosin heavy chain (MHC) and to a lesser extent on the myosin light chain (MLC) isoform (Caiozzo and Rourke, 2006). This chapter primarily relies on the integrated nomenclature of SO, FOG, and FG for clarity but occasionally the designations Type I, IIA (or IIa), and IIX (or IIx) will be used. **Figure 17.13** and **Table 17.2** summarize the properties of motor units and muscle fibers (Caiozzo and Rourke, 2006; Harris and Dudley, 2000).

A motor unit consists of a motor neuron (α_1 or α_2) and the muscle fibers it innervates. As previously described, the twitch speed of a muscle fiber depends largely on the motor neuron that innervates it. Thus, all muscle fibers within a motor unit will be either FT or ST. In addition, because all muscle fibers in a motor unit are recruited to contract together, they have the same metabolic capabilities. Therefore, a motor unit is composed exclusively of SO, FOG, or FG muscle fibers. That means that references to muscle fiber types also refer to motor unit types.

Table 17.2 further compares the different muscle fiber types in reference to important structural, neural, functional, and metabolic characteristics (Harris and Dudley, 2000; Zierath and Hawley, 2004). The diameters of the individual ST and FT fibers differ. The size of the muscle fiber is related to the size of the motor neuron innervating it, but primarily its size reflects the amount of contractile proteins within the muscle cell. ST fibers are smaller than FT fibers and have smaller motor neurons. The larger size of FT fibers is the result of their having more contractile proteins, which, in turn, enables them to produce greater force. **Figure 17.15** shows functional differences in the three types of muscle fibers, including different force production curves (**A**), different fatigue curves (**B**), and different contractile force and power at various speeds (**C**).

Other structural differences among fiber types are related directly to their predominant metabolic pathway for energy production. The SO fibers, which rely mainly on oxidative pathways for energy production, have a high number of mitochondria, high capillary density, high myoglobin content, and high oxidative enzyme activity. The FG fibers, which rely primarily on glycolytic pathways for energy production, have few mitochondria, low capillary density, low myoglobin content,

TABLE 17.2 Characteristics of Muscle Fibers

	Type I	Type II	
Contractile (Twitch):	ST	FTa	FTx
Metabolic:	SO	FOG	FG
Structural Aspects			
Muscle fiber diameter	Small	Intermediate	Large
Mitochondrial density	High	Intermediate	Low
Capillary density	High	Intermediate	Low
Myoglobin content	High	Intermediate	Low
Functional Aspects			
Twitch (contraction) time	Slow	Fast	Fast
Relaxation time	Slow	Fast	Fast
Force production	Low	Intermediate	High
Fatigability	Low	Intermediate	High
Metabolic Aspects			
Phosphocreatine stores	Low	High	High
Glycogen stores	Low	Intermediate	High
Triglyceride stores	High	Intermediate	Low
Myosin-ATPase activity	Low	Intermediate	High
Glycolytic enzyme activity	Low	Intermediate	High
Oxidative enzyme activity	High	Intermediate	Low

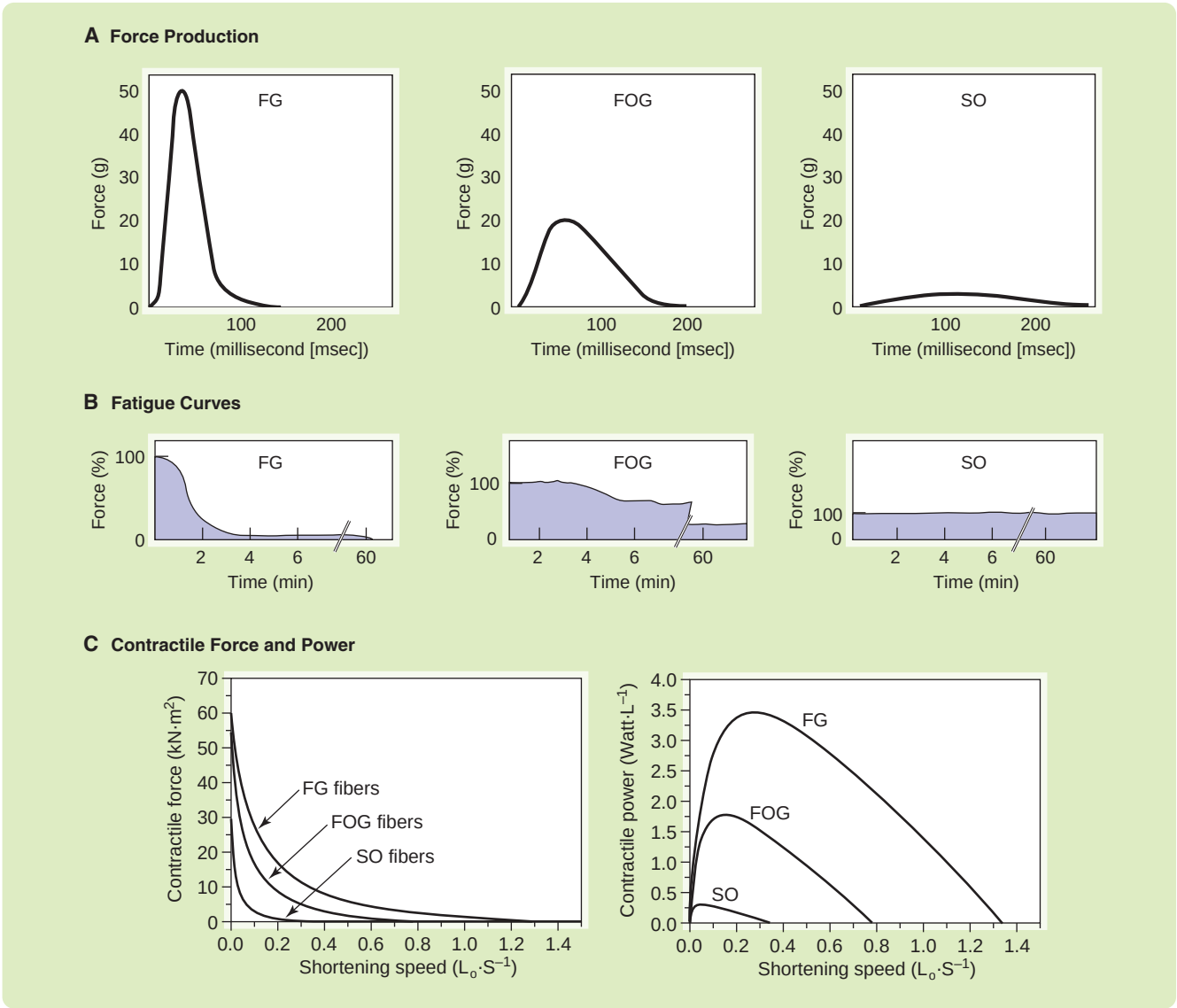


FIGURE 17.15 Fiber Types have Different Properties.
A. Force Production **B**. Fatigue Curves **C**. Contractile Force and Power at Different Contraction Speeds.
***Sources*:** Adapted from Edington, D. W. & V. R. Edgarton: The Biology of Physical Activity. Boston: Houghton Mifflin (1986) and Caiozzo, V. J. & B. Rourke: The muscular system: Structural and functional plasticity. In: *ACSM's Advanced Exercise Physiology*. Philadelphia, PA: Lippincott, Williams & Wilkins (2006).

Slow Oxidative (SO, Type I) Fibers Slow-twitch muscle fibers that rely primarily on oxidative metabolism to produce energy.

Fast Oxidative Glycolytic (FOG, Type IIA or IIa) Fibers Fast-twitch muscle fibers that can work under oxidative and glycolytic conditions.

Fast Glycolytic (FG, Type IIX or IIx) Fibers Fast-twitch muscle fibers that perform primarily under glycolytic conditions.

and high glycolytic enzyme activity. The FOG fibers share characteristics of both SO and FG but also have unique characteristics. Specifically, the FOG fibers have intermediate mitochondrial density, capillary density, myoglobin content, and oxidative enzyme activity and high PC stores, glycogen stores, and glycolytic enzyme activity.

The metabolic differences among muscle fibers both require and reflect differences in energy substrate availability. All muscles store and utilize glycogen; but since glycogen is the only substrate (along with its constituent parts—glucose) that can be used to fuel glycolysis, it makes sense that the FOG and FG fibers would have

higher glycogen stores than SO fibers have. Conversely, since triglycerides can only be broken down and used oxidatively, it would be anticipated that SO fibers would have more triglyceride storage than either FG or FOG fibers. Furthermore, FOG fibers would have an intermediate amount of triglycerides—more than FG but less than SO fibers.

Because SO fibers are so well supplied by the cardiovascular system and have ample fuel supplies (energy substrate), particularly from triglycerides, they are very resistant to fatigue. Because the FOG fibers have a substantial oxidative capability and the FG fibers do not, the FG fibers are the quickest to fatigue. The FOG fibers are somewhat less resistant to fatigue than the SO fibers and somewhat more resistant to fatigue than the FG fibers.

Table 17.2 summarizes key information about fiber types. Take a few minutes now to study this table and check your understanding of how this information is interrelated. Remember that although the fiber types have been labeled at the top of the columns separately for their contractile and metabolic properties, in practice the designations ST, SO, and Type I; Type IIa and FOG; and Type IIx and FG are used interchangeably.

Assessment of Muscle Fiber Type

Muscle fiber type is typically determined by a needle biopsy, an invasive procedure that involves collecting a small sample of skeletal muscle (**Figure 17.16**). Muscle biopsy samples are most commonly obtained from the gastrocnemius, vastus lateralis, or deltoid muscles. First, the skin is thoroughly cleaned and a topical anesthetic is applied to numb the area. A small incision is made through the skin, subcutaneous tissue, and fascia. The biopsy needle is then inserted into the belly of the muscle to extract a small amount of skeletal muscle tissue (~20–40 mg). This sample is then frozen in liquid nitrogen and sliced into very thin cross sections.

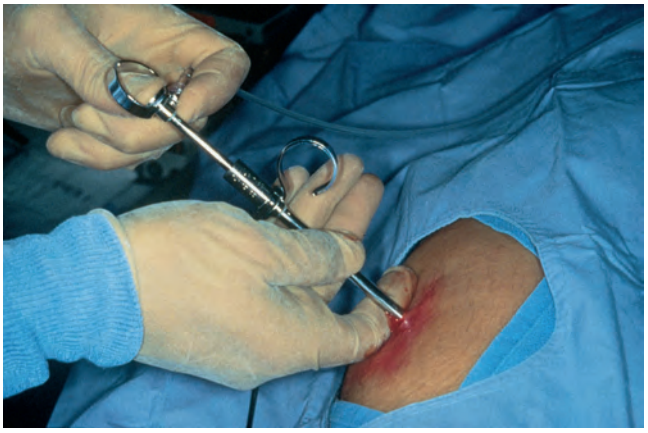


FIGURE 17.16 Muscle Biopsy
A biopsy needle is inserted into a small incision to obtain a sample of skeletal tissue.

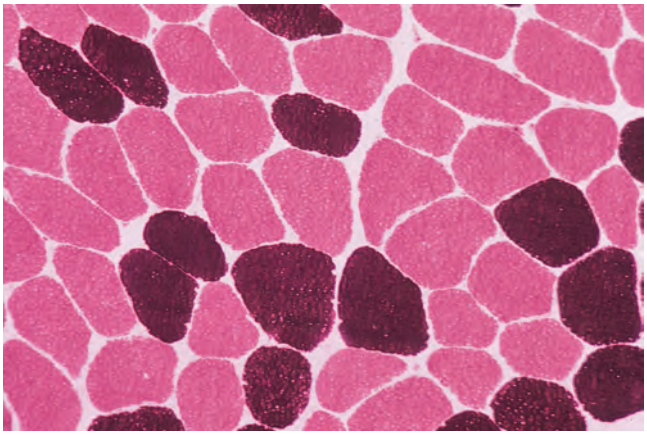


FIGURE 17.17 Mosaic Pattern of FT and ST Muscle Fibers Seen in a Microphotograph of Skeletal Muscle.
The darker stained fibers are the ST fibers, the lighter stained fibers are the FT Fibers.

The cross sections are chemically stained so that the muscle fibers can be differentiated into categories. Muscle samples may be stained for the enzyme myosin ATPase and for glycolytic and oxidative enzymes. When stained muscle fibers are viewed in cross section under a microscope, different muscle fiber types appear in different colors. **Figure 17.17** shows a skeletal muscle sample that has been histochemically stained, revealing ST (dark) and FT (light) fibers. Notice that the fiber types are intermingled, creating a mosaic pattern. Counting fibers of each type allows calculating the percentage of each fiber type (see the Check your Comprehension box). In addition to these percentages, researchers often measure the diameter of the muscle fibers.

Muscle fibers also can be typed noninvasively by nuclear magnetic resonance spectroscopy (NMR), but this laboratory technique has not yet gained widespread acceptance (Baguet et al., 2011; Boicelli et al., 1989). Attempts to use the vertical jump as a field measure of fiber type have met with varying success (see the Focus on Application) (Costill, 1978; Fry et al., 2003).

Knowledge about fiber types is important for at least three reasons:

1. Fiber type differences help explain individual differences in performance and response to training.
2. Fiber type differences help explain what training can and cannot do.
3. The relationship between fiber types and training and performance in elite athletes helps in the design of training programs for others who wish to be successful in specific events even if they do not know their exact fiber type percentages or distribution.

FOCUS ON APPLICATION

The Relationship between Muscle Fiber Characteristics and Physical Performance

Athletes in strength or power sports have higher percentages of fast-twitch (FT) fibers than athletes in endurance events. Additionally, the cross-sectional area of FT fibers is larger in weightlifters and strength athletes than in sedentary individuals or endurance-trained athletes. Based on the structure-function relationship that underlies physiology, a strong relationship would be expected between fiber type percentage and performance variables. Indeed, when a group of researchers investigated the relationship (correlations) between fast oxidative glycolytic (FOG) fibers (both percentage of fibers and area) and performance, they found several statistically significant correlations (see table below).

Correlations between Muscle Fiber Characteristics and Performance Variables

	% FOG Fibers	% Area of FOG Fibers
1 RM snatch	0.94	0.83
Vertical jump	0.83	0.75

The high percentage and large cross-sectional area of fast oxidative fibers in weightlifters is strongly related to performance in both weightlifting and in vertical jump performance. These results support the theoretical relationship between muscle fiber characteristics and actual physical performance. The results also suggest that a simple test for lower-body power, the vertical jump, may be a useful field test to provide a noninvasive indicator of muscle fiber characteristics.

Source: Fry et al. (2003).

CHECK YOUR COMPREHENSION

Fiber typing involves determining the percentage of a sample's fast-twitch or slow-twitch fibers. Count how many total fibers are visible in **Figure 17.17**. Now count how many of those fibers are darkly stained (indicating, in this example, that they are ST fibers). Based on these two numbers, what is the percentage of ST fibers in this muscle sample? What is the percentage of FT fibers in this sample?
Check your answer in Appendix C.

Distribution of Fiber Types

All muscles in humans are composed of a combination of slow-twitch and fast-twitch muscle fibers arranged in a mosaic pattern. This arrangement is thought to reflect the variety of tasks that human muscles must perform. The relative distribution, or percentage, of these fibers, however, may vary greatly from one muscle to another. For example, the soleus muscle may have as much as 85% ST fibers, and the triceps and ocular muscles may have as few as 30% ST fibers. The distribution may also vary considerably among individuals for the same muscle group (Saltin et al., 1977). The following are general characteristics of the distribution of fiber types:

1. Although distribution of fiber type varies within and between individuals, most individuals possess between 45% and 55% ST fibers.
2. The distribution of fiber types is not different for males and females, although males tend to show greater variation than females.
3. After early childhood, the fiber distribution does not change significantly as a function of age.
4. Fiber type distribution is primarily genetically determined.
5. Muscles involved in sustained postural activity have the highest number of slow-twitch muscle fibers.

Fiber Type in Athletes

Few topics in exercise physiology evoke more interest and debate than issues of fiber type in athletes. **Figure 17.18** shows the distribution of fiber types in male and female athletes. Athletes in endurance activities typically have a higher percentage of slow-twitch fibers, while athletes in power activities have a higher percentage of fast-twitch muscle fibers. The distribution of fiber type ranges widely in each group, however, indicating that athletic success is not determined solely by fiber type.

Not only do endurance athletes differ in general fiber type from power or resistance athletes but often these differences relate to specific muscles within these athletic groups. The results of one study of fiber type distribution are reported in **Figure 17.19** (Tesch and Karlsson, 1985). According to this study, the vastus lateralis muscles of the legs possess a greater percentage of ST fibers in endurance athletes primarily using the legs for their activity (such as runners). In contrast, athletes whose sport requires endurance of the upper body possess a greater percentage of ST fibers in the deltoid muscle.

An interesting question arises from such comparisons of fiber type distribution in various athletes: Did training and participation in a given sport influence the fiber type, or did fiber type influence the type of athletic participation? Although some researchers have

FOCUS ON RESEARCH

Does Fiber Type Distribution Affect Maximal Oxygen Uptake?

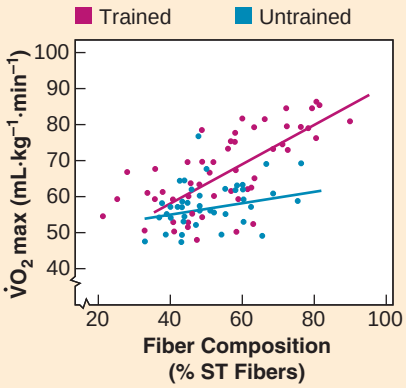
Researchers have long been interested in the distribution of fiber type in athletes. At the time that Bergh et al. undertook this classic study, researchers knew that aerobically trained individuals were characterized by a high percentage of ST fibers (and hence a lower percentage of FT fibers) and that anaerobically trained individuals (e.g., sprinters) were more likely to have a high percentage of FT fibers. Researchers also knew that aerobic training was associated with a high $\dot{V}O_2$max. $\dot{V}O_2$max is the greatest amount of oxygen an individual can take in, transport, and use during strenuous work; it is considered the best measure of an individual's aerobic (or cardiovascular) fitness. Thus, Bergh et al. proposed that there would be a relationship between the percentage of slow-twitch fibers and a person's $\dot{V}O_2$max.

Their results, shown here in the graph, support their hypothesis.

These data lead to two important conclusions:

1. There is a strong linear relationship between $\dot{V}O_2$max and %ST fibers. This makes sense because the ST fibers have the greatest oxidative ability, that is, the ability to use oxygen to produce large amounts of ATP to support long-duration activities.
2. At any given %ST (above ~40%), an athlete has a greater $\dot{V}O_2$max than a nonathlete. This is consistent with what we know about the trainability of muscle fibers. Endurance training increases the oxidative capacity of muscle, thereby allowing the muscle to use more oxygen and thus achieve a higher $\dot{V}O_2$max.

Source: Bergh, U., A. Thorstensson, B. Sjodin, B. Hulten, K. Piehl, & J. Karlsson: Maximal oxygen uptake and muscle fiber types in trained and untrained humans. *Medicine and Science in Sports and Exercise*. 10(3):151–154 (1978).



theorized that changes are possible in the contractile properties of muscle, most available evidence indicates that the distribution of ST and FT fibers (the types involving contractile properties) is genetically determined and cannot be altered in humans by exercise training (Kraemer, 2000; Saltin et al., 1977; Williams, 1994). Evidence does show, however, that training can alter the metabolic properties of the cell (enzyme concentration, substrate storage, and so on). These changes may lead to a conversion of FT fiber subdivisions. Indeed, with endurance training, the oxidative potential of FOG and FG fibers can exceed that of SO fibers of sedentary individuals (Saltin et al., 1977).

In summary, the distribution of fiber types varies considerably within the muscle groups of an individual and among individuals. The basic distribution of fiber type appears to be genetically determined. It is generally thought that exercise training does not alter the

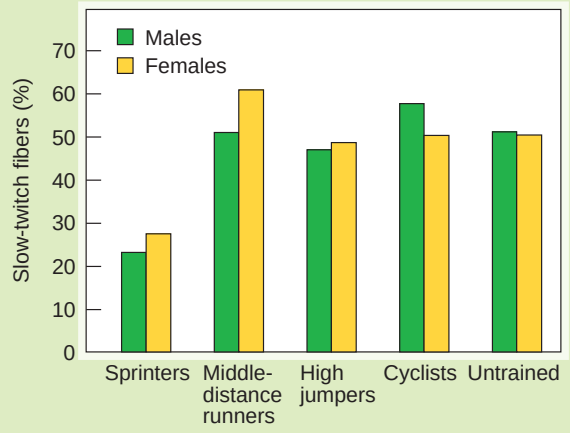


FIGURE 17.18 Fiber Type Distribution among Athletes.
***Source*:** Data from Fox, E. L., R. W. Bowers, & M. L. Foss: *The Physiological Basis for Exercise and Sport*. Dubuque, IA: Brown & Benchmark, 94–135 (1993).

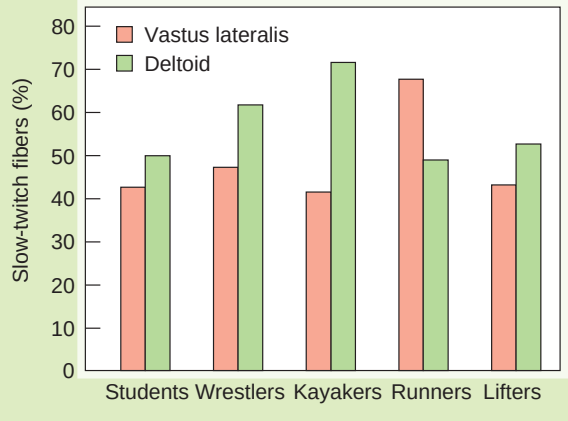


FIGURE 17.19 Fiber Type Distribution of Different Muscle Groups Among Athletes.
***Source*:** Data from Tesch, P. A. & J. Karlsson: Muscle fiber types and size in trained and untrained muscles of elite athletes. *Journal of Applied Physiology*. 59:1716–1720 (1985).

FOCUS ON RESEARCH

Are There Sex Differences in Muscle Fiber Power Production in Older Adults?

It is well known that males are generally stronger than females. The purpose of this study was to determine if differences in power at the single muscle fiber level contribute to the sex difference in whole muscle power production in elderly individuals. Sixteen older adults (mean age = 72 years) participated in the study. A muscle biopsy procedure was performed to obtain muscle fibers.

As expected, the males were stronger in a double-knee press and had greater right knee extension power than females (although some measures of strength and power did not differ between these males and females). However, the slow oxidative (SO) and fast oxidative glycolytic (FOG) fibers of these males and females did not differ significantly in power production.

Thus, it appears that power-generating capacity differs by muscle fiber type (SO<FOG), but not by sex. Rather, it appears that the primary reason that males are stronger than females is that they possess greater muscle mass.

Source: Krivickas, L. S., R. A. Fielding, A. Murray, D. Callahan, A. Johansson, D. J., Dorer, & W. R. Frontera: Sex differences in single muscle fiber power in older adults. *Medicine & Science in Sports & Exercise*. 38(1):57–64 (2006).

contractile properties of muscle fibers. The possibility remains, however, that training adaptations can alter the metabolic capabilities of muscle fibers sufficiently to change the classification of fiber types within the FT fibers (i.e., from FG to FOG or vice versa).

SUMMARY

1. Skeletal muscles provide for locomotion and manipulation, maintain body posture, and play an important role in heat generation.
2. The muscle characteristics that allow production of movement include irritability, contractility, extensibility, and elasticity.
3. A motor neuron along with the muscle fibers it innervates is called a motor unit. Because each muscle fiber in a motor unit is connected to the same neuron, the electrical activity in the motor neuron controls the contractile activity of all the muscle fibers in a given motor unit.
4. Skeletal muscle fibers are bundled together into groups of fibers called fasciculi. A muscle fiber is itself comprised of smaller units called myofibrils, which are made up of myofilaments.
5. The two types of myofilaments are the thick and thin filaments. The repeating pattern of these myofilaments along the length of the myofibril gives skeletal muscle its striated appearance.
6. The repeating unit, or sarcomere, is the functional unit of the muscle.
7. Tropomyosin is a regulatory protein that blocks the active site on actin, thereby inhibiting actin and myosin from binding under resting conditions. The position of tropomyosin is controlled by troponin.
8. Excitation-contraction coupling is the sequence of events by which an action potential in the sarcolemma initiates the contractile process of the myofilaments.
9. Excitation-contraction coupling has three phases: the spread of depolarization, the binding of calcium to troponin, and the generation of force (cross-bridge cycling).
10. Force is generated in the cross-bridging cycle. This cycle consists of the binding of myosin to actin, the power stroke, the dissociation of myosin and actin, and the activation of myosin heads.
11. The spread of depolarization (action potential) is carried into the interior of the muscle fiber by the T tubules. As the electrical signal moves into the cell, it causes the release of calcium, which is stored in the lateral sacs of the sarcoplasmic reticulum.
12. Calcium released from the sarcoplasmic reticulum binds to the troponin molecules, which undergo a configuration change, thereby removing tropomyosin from its blocking position on the actin filament. This allows the myosin cross-bridges (heads) to bind with the actin filaments.

13. The generation of tension within the contractile elements results from the binding of actin and myosin, which causes the release of stored energy in the myosin heads.
14. ATP plays several important roles in muscle contraction. The hydrolysis of ATP provides the energy to activate or reactivate the myosin head before binding with actin. ATP binding is also necessary to break the linkage between the myosin cross-bridge and actin so that the cycle can repeat. ATP is also used to return calcium to the sarcoplasmic reticulum and to restore the resting membrane potential.
15. During relaxation, calcium is pumped back into the sarcoplasmic reticulum (by active transport), and troponin no longer keeps tropomyosin from its blocking position.
16. When a muscle fiber or motor unit is stimulated to contract, it contracts to its fullest extent or does not contract at all. This is known as the all-or-none principle.
17. Human muscle fibers are categorized as two different types, ST and FT, based on their contractile properties. The FT fibers can be further classified as FOG or FG fibers based on their metabolic properties. ST fibers are metabolically oxidative or SO.
18. Athletes involved in endurance activities typically have a high percentage of slow-twitch fibers. Athletes involved in power activities typically have a high percentage of fast-twitch muscle fibers.
19. Training alters the metabolic capabilities of muscle fibers, but not their contractile properties. It is possible that metabolic alterations could be significant enough to change the classification of fibers with the FT fibers (FOG to FG, and vice versa).

REVIEW QUESTIONS

1. List, in order of largest to smallest, the major components of the whole muscle.
2. What causes the striated appearance of skeletal muscle fibers?
3. What are the T tubules and the sarcoplasmic reticulum? What is the function of each?
4. Relate each region of the sarcomere to the presence of thick and thin myofilaments.
5. Diagram a sarcomere at rest and at the end of a contraction, and identify each of the areas.
6. Describe the role of the regulatory proteins in controlling muscle contraction.
7. Describe the sequence of events in excitation-contraction coupling.
8. Identify the role of ATP in the production of force within the contractile unit of muscle.
9. What is the role of calcium in muscle contraction?
10. Describe the all-or-none principle as it relates to the contraction of a single muscle fiber.
11. Diagram the force production, twitch speed, and fatigue curve for the different fiber types.
12. Discuss the possibility of influencing fiber type distribution by exercise training.

For further review and additional study tools, visit the website at http://thePoint.lww.com/Plowman4e.

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