G. Delpho/Peter Arnold, Inc.

Movement in a long-tailed field mouse *(Apodemus sylvaticus)*. Movement of vertebrates occurs as a result of contractions and relaxations of skeletal muscles. When stimulated by the nervous system, actin filaments in the muscles slide over myosin filaments to cause muscle contractions.

41 Muscles, Bones, and Body Movements

Study Plan

41.1 Vertebrate Skeletal Muscle: Structure and Function

The striated appearance of skeletal muscle fibers results from a highly organized internal structure

During muscle contraction, thin filaments on each side of a sarcomere slide over thick filaments

The response of a muscle fiber to action potentials ranges from twitches to tetanus

Muscle fibers differ in their rate of contraction and susceptibility to fatigue

Skeletal muscle control is divided among motor units

Invertebrates move using a variety of striated muscles

41.2 Skeletal Systems

A hydrostatic skeleton consists of muscles and fluid

An exoskeleton is a rigid external body covering

An endoskeleton consists of supportive internal body structures such as bones

Bones of the vertebrate endoskeleton are organs with several functions

41.3 Vertebrate Movement: The Interactions between Muscles and Bones

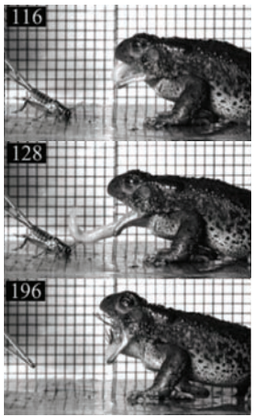
Joints of the vertebrate endoskeleton allow bones to move and rotate

Vertebrates have muscle–bone interactions optimized for specific movements

Why It Matters

A Mexican leaf frog *(Pachymedusa dacnicolor)* sits motionless, its prominent eyes staring into space **(Figure 41.1).** But when the frog detects an approaching cricket, it lunges forward at just the right moment, thrusts out its sticky tongue, and captures the prey. This sequence of events, from the beginning of the movement until the frog's mouth closes, sealing the cricket's fate, requires only 260 milliseconds (ms)—about one quarter of a second. How does the frog move so swiftly, and so surely?

As its prey draws near, neuronal signals travel from the frog's brain to the muscles that extend the frog's hind legs, causing the muscles to contract and propel the frog forward on its forelimbs toward the cricket. Within 50 ms after the jump begins, other signals contract the muscles of the lower jaw, opening the mouth. Then, a muscle on the upper surface of the tongue contracts, which raises the tongue and flips it out of the mouth. As the tongue shoots forward, muscle contractions along the ventral side of the trunk arch the body and direct the head downward toward the prey. Within 80 ms after the lunge begins, the tip of the frog's tongue contacts the cricket. Completion of the lunge folds the tongue—and the cricket—into the frog's mouth, aided by contraction of a muscle



Kiisa Nishikawa/Northern Arizona University

Figure 41.1
A Mexican leaf frog *(Pachymedusa dacnicolor)* capturing a grasshopper.

on the bottom of the tongue. After the mouth closes, further muscle contractions pull the legs forward and fold them under the body.

We know this because Kiisa Nishikawa, Lucie Gray, and James O'Reilly of Northern Arizona University recorded the frog's movements using a high-speed video camera linked to a millisecond timer, with a grid in the background that allowed precise measurement of the distances body parts traveled during the capture. Nishikawa's research group uses the camera's record to study movement in frogs in particular and animals in general.

In Section 36.2 you learned that there are three types of muscle tissue: skeletal, cardiac, and smooth. Skeletal muscle is so named because most muscles of this type are attached by tendons to the skeleton of vertebrates. Cardiac muscle is the contractile muscle of the heart, and smooth muscle is found in the walls of tubes and cavities of the body, including blood vessels and the intestines. In this chapter we describe the structure and function of skeletal muscles, the skeletal systems found in invertebrates and vertebrates, and how muscles bring about movement.

41.1 Vertebrate Skeletal Muscle: Structure and Function

Vertebrate **skeletal muscles** connect to bones of the skeleton. The cells forming skeletal muscles are typically long and cylindrical, and contain many nuclei (shown in Figure 36.6a). Skeletal muscle is controlled by the somatic nervous system.

Most skeletal muscles in humans and other vertebrates are attached at both ends across a joint to bones of the skeleton. (Some, such as those that move the lips, are attached to other muscles or connective tissues under skin.) Depending on its points of attachment, contraction of a single skeletal muscle may extend or bend body parts, or may rotate one body part with respect to another. The human body has more than 600 skeletal muscles, ranging in size from the small muscles that move the eyeballs to the large muscles that move the legs.

Skeletal muscles are attached to bones by cords of connective tissue called *tendons* (see Section 36.2). Tendons vary in length from a few millimeters to some, such as those that connect the muscles of the forearm to the bones of the fingers, that are 20 to 30 cm long.

The Striated Appearance of Skeletal Muscle Fibers Results from a Highly Organized Internal Structure

A skeletal muscle consists of bundles of elongated, cylindrical cells called **muscle fibers**, which are 10 to 100 μm in diameter and run the entire length of the muscle **(Figure 41.2).** Muscle fibers contain many nuclei, reflecting their development by fusion of smaller cells. Some very small muscles, such as some of the muscles of the face, contain only a few hundred muscle fibers; others, such as the larger leg muscles, contain hundreds of thousands. In both cases, the muscle fibers are held in parallel bundles by sheaths of connective tissue that surround them in the muscle and merge with the tendons that connect muscles to bones or other structures. Muscle fibers are richly supplied with nutrients and oxygen by an extensive network of blood vessels that penetrates the muscle tissue.

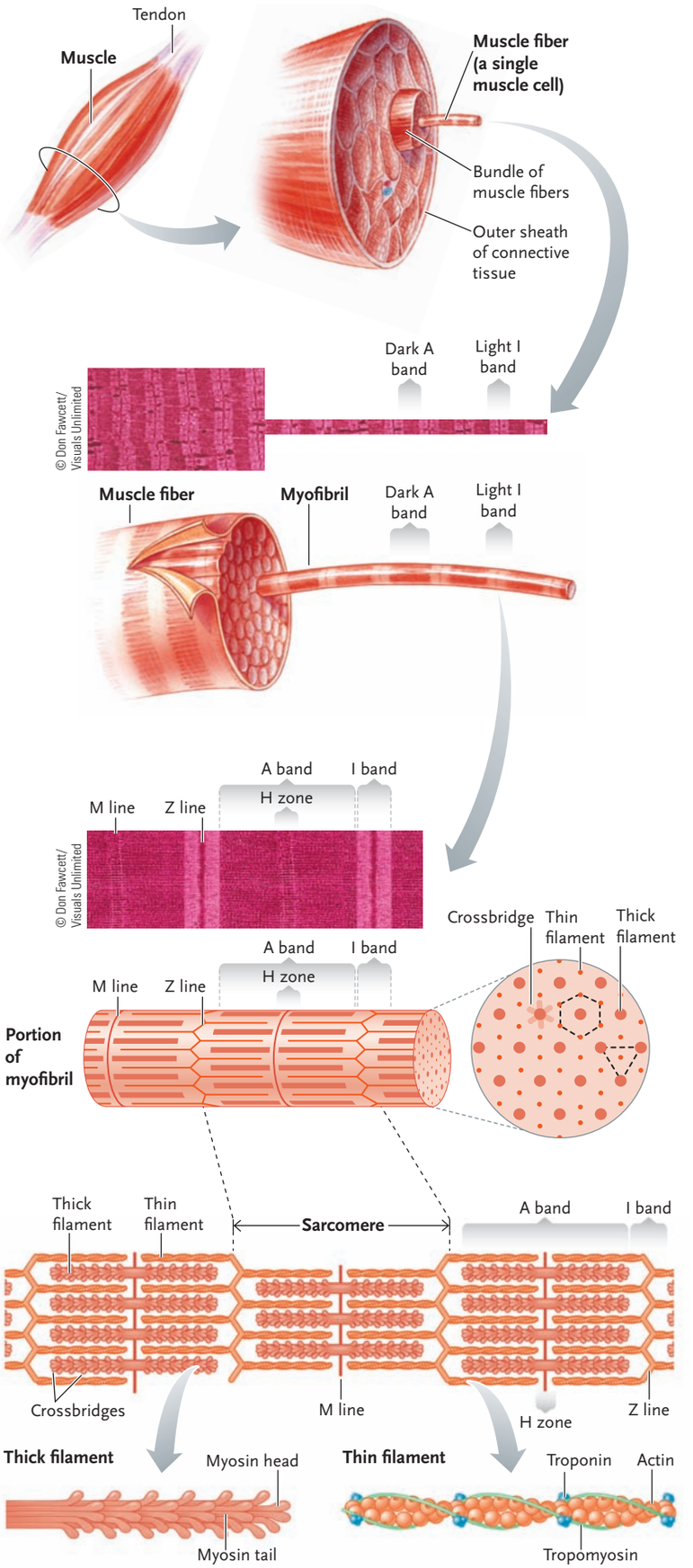
Muscle fibers are packed with **myofibrils,** cylindrical contractile elements about 1 μm in diameter that run lengthwise inside the cells. Each myofibril consists of a regular arrangement of **thick filaments** (13–18 nm in diameter) and **thin filaments** (5–8 nm in diameter) (see Figure 41.2). The thick and thin filaments alternate with one another in a stacked set.

The thick filaments are parallel bundles of myosin molecules; each myosin molecule consists of two protein subunits that together form a *head* connected to a long double helix forming a *tail*. The head is bent toward the adjacent thin filament to form a *crossbridge*. In vertebrates, each thick filament contains some 200 to 300 myosin molecules and forms as many crossbridges. The thin filaments consist mostly of two linear chains of actin molecules twisted into a double helix, which creates a groove running the length of the molecule. Bound to the actin are *tropomyosin* and *troponin* proteins. Tropomyosin molecules are elongated fibrous proteins that are organized end to end next to the groove of the actin double helix. Troponin is a three-subunit globular protein that binds to tropomyosin at intervals along the thin filaments.

The arrangement of thick and thin filaments forms a pattern of alternating dark bands and light bands, giving skeletal muscle a striated appearance under the microscope (see Figure 41.2). The dark bands, called *A bands,* consist of stacked thick filaments along with the parts of thin filaments that overlap both ends. The lighter-appearing middle region of an A band, which contains only thick filaments, is the *H zone*. In the center of the H zone is a disc of proteins called the *M line,* which holds the stack of thick filaments together. The light bands, called *I bands,* consist of the parts of the thin filaments not in the A band. In the center of each I band is a thin *Z line,* a disc to which the thin filaments are anchored. The region between two adjacent Z lines is a **sarcomere** (*sarco* = flesh; *meros* = segment); sarcomeres are the basic units of contraction in a myofibril.

At each junction of an A band and an I band, the plasma membrane folds into the muscle fiber to form a **T (transverse) tubule (Figure 41.3).** Encircling the sarcomeres is the **sarcoplasmic reticulum**, a complex system of vesicles modified from the smooth endoplasmic reticulum. Segments of the sarcoplasmic re-

Figure 41.2
Skeletal muscle structure. Muscles are composed of bundles of cells called muscle fibers; within each muscle fiber are longitudinal bundles of myofibrils. The unit of contraction within a myofibril, the sarcomere, consists of overlapping myosin thick filaments and actin thin filaments. The myosin molecules in the thick filaments each consist of two subunits organized into a head and a double-helical tail. The actin subunits in the thin filaments form twisted, double helices, with tropomyosin molecules arranged head-to-tail in the groove of the helix and troponin bound to the tropomyosin at intervals along the thin filaments.



ticulum are wrapped around each A band and I band, and are separated from the T tubules in those regions by small gaps.

An axon of an efferent neuron leads to each muscle fiber. The axon terminal makes a single, broad synapse with a muscle fiber called a **neuromuscular junction** (see Figure 41.3). The neuromuscular junction, T tubules, and sarcoplasmic reticulum are key components in the pathway for stimulating skeletal muscle contraction by neural signals—which starts with action potentials traveling down the efferent neuron—as will be described next.

During Muscle Contraction, Thin Filaments on Each Side of a Sarcomere Slide over Thick Filaments

The precise control of body motions depends on an equally precise control of muscle contraction by a signaling pathway that carries information from nerves to muscle fibers. An action potential arriving at the neuromuscular junction leads to an increase in the concentration of Ca^{2+} in the cytosol of the muscle fiber. The increase in Ca^{2+} triggers a process in which the thin filaments on each side of a sarcomere slide over the thick filaments toward the center of the A band, which brings the Z lines closer together, shortening the sarcomeres and contracting the muscle **(Figure 41.4).** This *sliding filament mechanism* of muscle contraction depends on dynamic interactions between actin and myosin proteins in the two filament types. That is, the myosin crossbridges make and break contact with actin and pull the thin filaments over the thick filaments—the action is similar to rowing, or a ratcheting process. A model for muscle contraction is shown in **Figure 41.5.**

Conduction of an Action Potential into a Muscle Fiber. Like neurons, skeletal muscle fibers are *excitable,* meaning that the electrical potential of their plasma membrane can change in response to a stimulus. When an action potential arrives at the neuromuscular junction, the axon terminal releases a neurotransmitter, *acetylcholine,* which triggers an action potential in the muscle fiber (see Figure 41.5, step 1). The action potential travels in all directions over the muscle fiber's

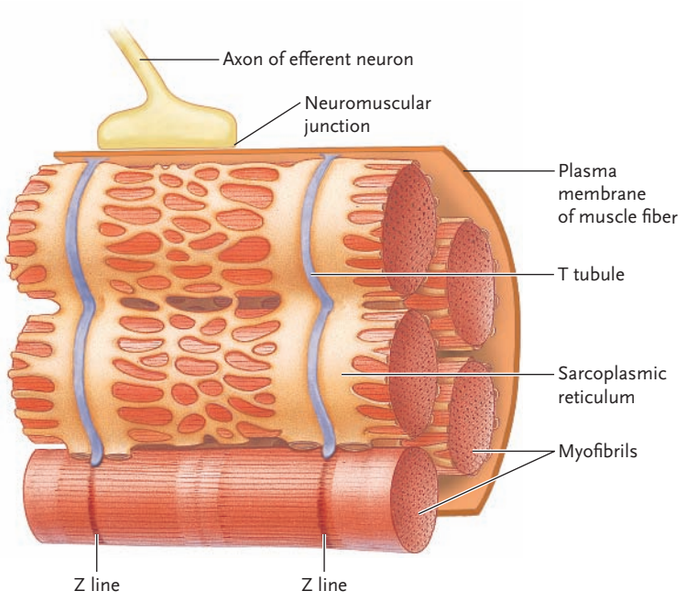


Figure 41.3
Components in the pathway for the stimulation of skeletal muscle contraction by neural signals. T (transverse) tubules are infoldings of the plasma membrane into the muscle fiber originating at each A band–I band junction in a sarcomere. The sarcoplasmic reticulum encircles the sarcomeres and segments of it end in close proximity to the T tubules.

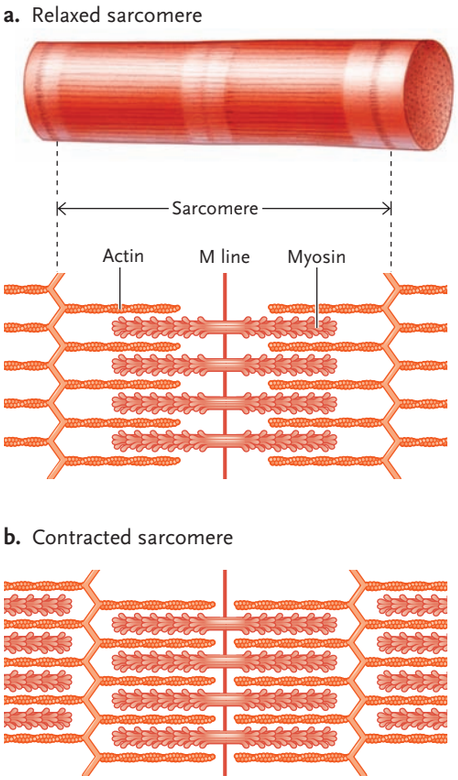


Figure 41.4
Shortening of sarcomeres by the sliding filament mechanism, in which the thin filaments are pulled over the thick filaments.

surface membrane, and also penetrates into the interior of the fiber through the T tubules.

Release of Calcium into the Cytosol of the Muscle Fiber. In the absence of a stimulus, the Ca^{2+} concentration is kept high inside the sarcoplasmic reticulum by active transport proteins that continuously pump Ca^{2+} out of the cytosol and into the sarcoplasmic reticulum. (The active transport proteins are Ca^{2+} pumps, discussed in Section 6.4.) When an action potential reaches the end of a T tubule, it opens ion channels in the sarcoplasmic reticulum that allow Ca^{2+} to flow out into the cytosol (see Figure 41.5, step 2).

When Ca^{2+} flows into the cytosol, the troponin molecules of the thin filament bind the calcium and undergo a conformational change that causes the tropomyosin fibers to slip into the grooves of the actin double helix. The slippage uncovers the actin's binding sites for the myosin crossbridge (see Figure 41.5, step 3). At this point in the process, the myosin crossbridge has a molecule of ATP bound to it, and is not in contact with the thin filament.

The Crossbridge Cycle. Using the energy of ATP hydrolysis, the myosin crossbridge bends away from the tail and binds to a newly exposed myosin crossbridge binding site on an actin molecule (see Figure 41.5, step 4). In effect, this bending compresses a molecular spring in the myosin head. The binding of the crossbridge to actin triggers release of the molecular spring in the crossbridge, which snaps back toward the tail producing the power stroke (motor) that pulls the thin filament over the thick filament (step 5).

The crossbridge now binds another ATP and myosin detaches from actin (see Figure 41.5, step 6). The cycle repeats again, starting with ATP hydrolysis (step 4). Contraction ceases when action potentials stop: Ca^{2+} is pumped back into the sarcoplasmic reticulum, and its effect on troponin is reversed, leading to tropomyosin again blocking myosin crossbridge binding sites on actin. Contraction ceases and the actin thin filaments slide back over the myosin thick filaments to their original relaxed positions (step 7). Crossbridge cycles based on actin and myosin power movements in all living organisms, from cytoplasmic streaming in plant cells and amoebae to muscle contractions in animals.

Although the force produced by a single myosin crossbridge is comparatively small, it is multiplied by the hundreds of crossbridges acting in a single thick filament, and by the billions of thin filaments sliding in a contracting sarcomere. The force, multiplied further by the many sarcomeres and myofibrils in a muscle fiber, is transmitted to the plasma membrane of a muscle fiber by the attachment of myofibrils to elements of the cytoskeleton. From the plasma membrane, it is transmitted to bones and other body parts

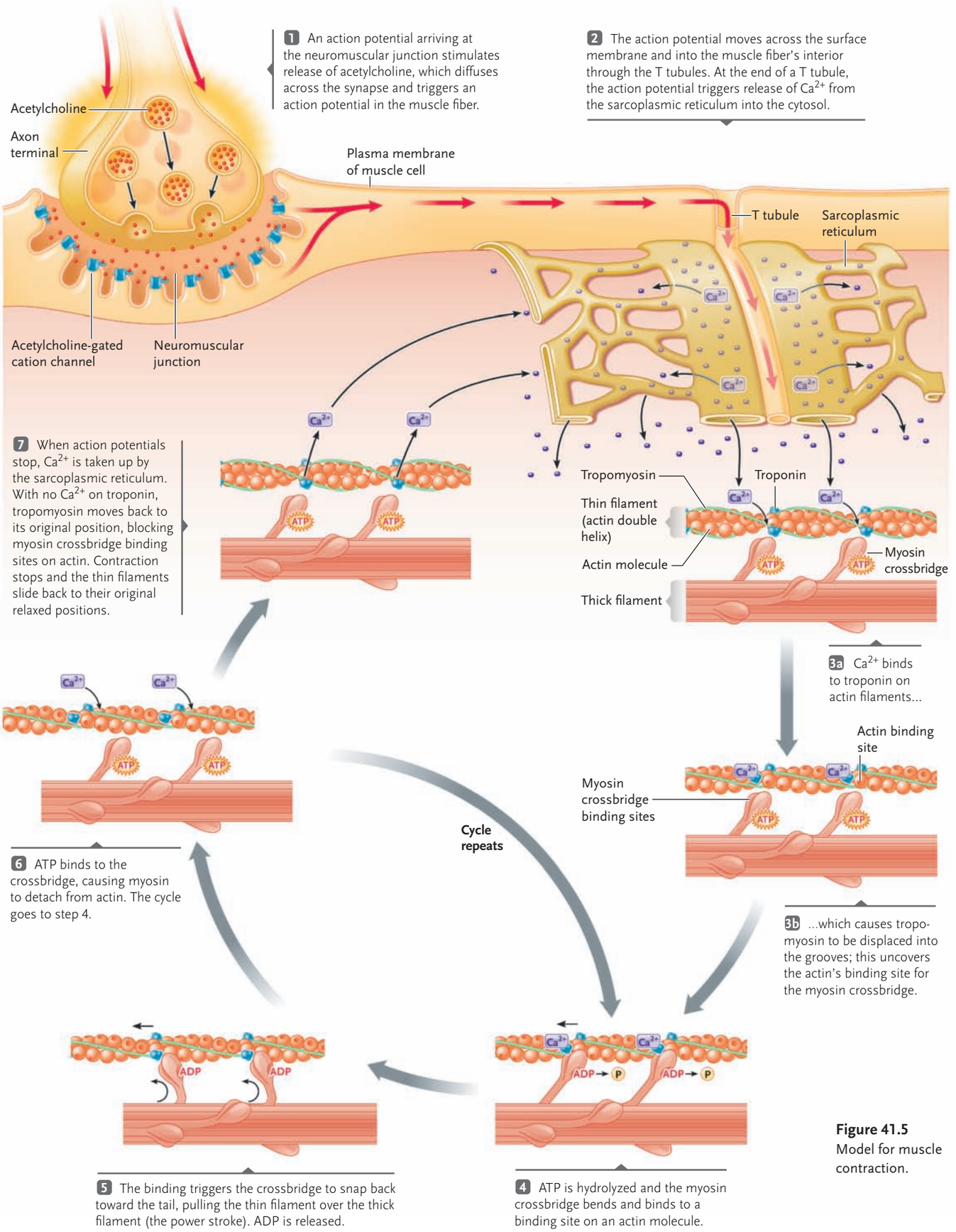


Figure 41.5
Model for muscle contraction.

Insights from the Molecular Revolution

A Substitute Player That May Be a Big Winner in Muscular Dystrophy

Duchenne muscular dystrophy (DMD) is an inherited disease, characterized by progressive muscle weakness, that primarily affects males—about 1 out of every 3500 males is born with the disease. When DMD patients are 3 to 5 years old, their muscle tissue begins to break down, and by the time they are in their teens most can walk only with braces. They usually die of complications from degeneration of the heart and diaphragm muscle by their early 20s. Currently, there is no effective treatment for DMD.

The gene that causes DMD, which is located on the X chromosome, was isolated and identified in 1985. In its normal form, the gene encodes the protein *dystrophin,* which anchors a glycoprotein complex in the plasma membrane of a muscle fiber to the underlying actin cytoskeleton (see Section 5.3). In most people with DMD, segments of DNA are missing from the coding sequence of the gene, so the protein cannot function. Without functional dystrophin, the plasma membrane of the muscle fibers is susceptible to tearing during contraction, which leads to muscle destruction. Creatine kinase (CK), an enzyme found predominantly in muscles and in the brain, leaks out of the damaged muscles and accumulates in the blood, which normally contains little CK. Elevated CK in the blood, then, is diagnostic of muscle damage such as that found in DMD.

Many researchers are working to develop a gene-therapy cure for DMD. For example, Kay E. Davies and her colleagues at Oxford University in England have identified a protein that is structurally similar to dystrophin and appears to have a highly similar function. That protein, called *utrophin,* is made in small quantities in muscle fibers and normally functions only in neuromuscular junctions. The utrophin gene and its protein function normally in DMD patients.

The Davies team reasoned that utrophin might be able to substitute for the missing dystrophin in DMD patients if a means could be found to increase its quantity in muscle cells. For their research, they used *mdx* mice, a strain that has the dystrophin gene deleted and is therefore a mouse model of human DMD. First, they introduced an artificial gene (consisting of the mouse utrophin gene under the control of a strong promoter) into fertilized oocytes; the resulting transgenic mice produced much more than the usual amount of utrophin. The researchers were excited to find that CK levels in the blood of the transgenic mice were reduced to 25% of the level in *mdx* mice without the added gene, indicating that muscle damage was markedly decreased. This was confirmed by microscopic examination. Other techniques showed that utrophin, instead of being concentrated in neuromuscular junctions as it is normally, was now distributed throughout the muscle plasma membranes. In short, in these experiments the elevated level of utrophin was able to substitute for dystrophin, and decreased significantly the onset of disease symptoms. Moreover, no deleterious side effects from the overproduction of utrophin could be detected in the genetically engineered mice.

Promising as these results are, germline gene therapy of humans is not allowed, so this approach cannot be used with human patients. Davies's group looked for another way to increase utrophin production, and suggested that upregulating the utrophin gene in all cells of the body could be a strategy to treat DMD. In experiments again using transgenic *mdx* mice, they showed that moderate overproduction of utrophin beginning as late as 10 days after birth caused improvements in muscle appearance compared with controls. Overall, the results show that utrophin overproduction therapy, initiated after birth, can be effective, but that both the timing of therapy and the amount of utrophin expressed are important. Davies's group is now searching for a chemical compound that would increase the levels of utrophin already present in DMD patients. However, much work remains before this can be an effective therapy in humans.

by the connective tissue sheaths surrounding the muscle fibers and by the tendons.

Several mutations affecting muscle and nerve tissues interrupt the transmission of force and cause severe disabilities. Duchenne muscular dystrophy (DMD), for example, is caused by a mutation that weakens the cytoskeleton of the muscle fiber, causing the cells to rupture when contractile forces are generated. *Insights from the Molecular Revolution* describes experiments that may lead to a cure for this debilitating disease.

From Contraction to Relaxation. As long as action potentials continue to arrive at the neuromuscular junction, Ca^{2+} is released in response, and ATP is available, the crossbridge cycle continues to run, shortening the sarcomeres and contracting the muscle fiber.

When action potentials stop, excitation of the T tubules ceases, and the Ca^{2+} release channels in the sarcoplasmic reticulum close. The active transport pumps quickly remove the remaining Ca^{2+} from the cytosol. In response, troponin releases its Ca^{2+} and the tropomyosin fibers are pulled back to cover the myosin binding sites in the thin filaments. The crossbridge cycle stops, and contraction of the muscle fiber ceases. In a muscle fiber that is not contracting, ATP is bound to the myosin head and the crossbridge is not bound to the actin filament (see Figure 41.5, step 7).

Deadly Interruptions of the Crossbridge Cycle. The mechanism controlling vertebrate muscle contraction can be blocked by several toxins and poisons. For example, the bacterium *Clostridium botulinum,* which grows in improperly preserved food, produces a toxin that blocks acetylcholine release in neuromuscular junctions. Many of the body muscles are unable to contract, including the diaphragm, the muscle that is essential for inflating the lungs. As a result, the victim dies from respiratory failure. The toxin is so poisonous that 0.0000001 g is enough to kill a human; 600 g could wipe out the entire human population. This same toxin, under the brand name Botox, is injected in low doses as a cosmetic treatment to remove or reduce wrinkles—if muscles cannot contract, then wrinkles cannot form.

The venom of black widow spiders (genus *Latrodectus*) causes massive release of acetylcholine, leading to convulsive contractions of body muscles; the diaphragm becomes locked in position, causing respiratory failure. Curare, extracted from the bark and sap of some South American trees, blocks acetylcholine from binding to its receptors in muscle fibers. The body muscles, including the diaphragm, become paralyzed and the victim dies of respiratory failure. Some native peoples in South America took advantage of these effects by using curare as an arrow and dart poison.

In a natural process, within a few hours after an animal dies, Ca^{2+} diffuses into the cytoplasm of muscle cells and initiates the crossbridge cycle, producing *rigor mortis,* a strong tension of essentially all the skeletal muscles that stiffens the entire body. As part of rigor mortis, the crossbridges become locked to the thin filaments because ATP production stops (remember that ATP is required to release the crossbridges from actin). The stiffness reverses as actin and myosin are degraded.

The Response of a Muscle Fiber to Action Potentials Ranges from Twitches to Tetanus

A single action potential arriving at a neuromuscular junction usually causes a single, weak contraction of a muscle fiber called a **muscle twitch (Figure 41.6a).** After a muscle twitch begins, the tension of the muscle fiber increases in magnitude for about 30 to 40 ms, and then peaks as the action potential runs its course through the T tubules and the Ca^{2+} channels begin to close. Tension then decreases as the Ca^{2+} ions are pumped back into the sarcoplasmic reticulum, falling to zero in about 50 ms after the peak.

If a muscle fiber is restimulated after it has relaxed completely, a new twitch identical to the first is generated (see Figure 41.6a). However, if a muscle fiber is restimulated before it has relaxed completely, the second twitch is added to the first, producing what is called *twitch summation,* which is basically a summed, stronger contraction **(Figure 41.6b).** And, if action potentials arrive so rapidly (about 25 ms apart) that the fiber cannot relax at all between stimuli, the Ca^{2+} channels remain open continuously and twitch summation produces a peak level of continuous contraction called **tetanus (Figure 41.6c).** (This is not to be confused with the disease of the same name, in which a bacterial toxin causes uncontrolled and con-

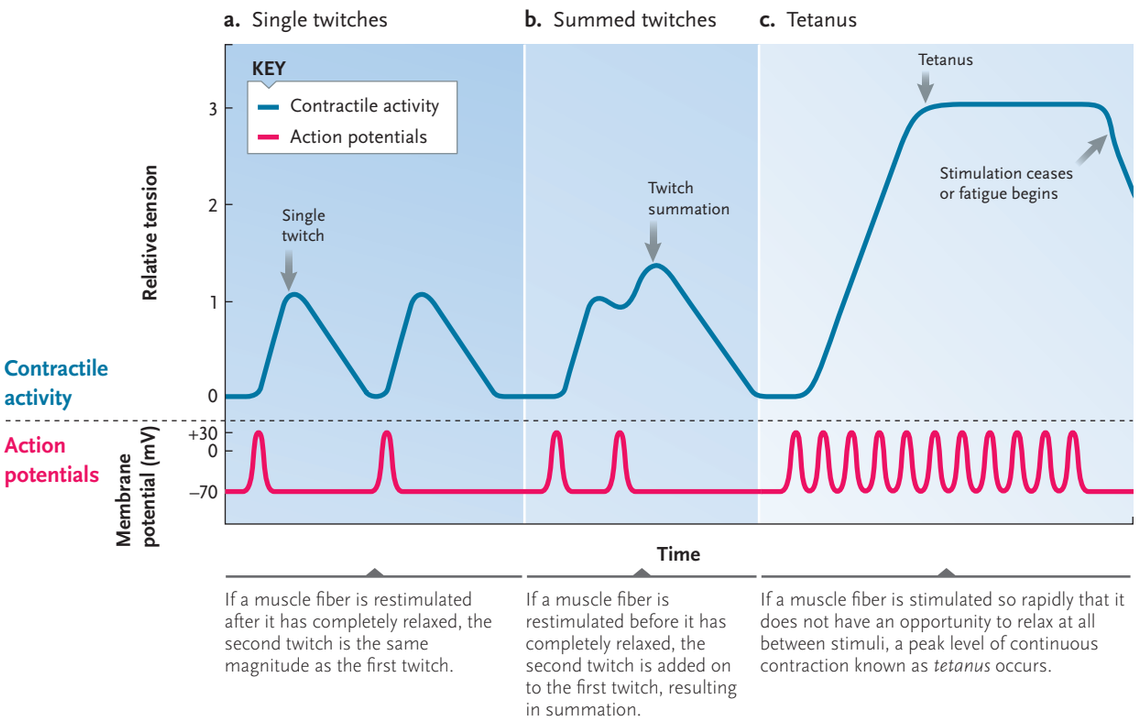


Figure 41.6
The relationship of the tension produced in a muscle fiber to the frequency of action potentials.

tinuous muscle contraction.) Contractile activity will then decrease if either the stimuli cease or the muscle fatigues.

Tetanus is an essential part of muscle fiber function. If we lift a moderately heavy weight, for example, many of the muscle fibers in our arms enter tetanus and remain in that state until the weight is released. Even body movements that require relatively little effort, such as standing still but in balance, involve tetanic contractions of some muscle fibers.

Muscle Fibers Differ in Their Rate of Contraction and Susceptibility to Fatigue

Muscle fibers differ in their rate of contraction and resistance to fatigue, and thus can be classified as slow, fast aerobic, and fast anaerobic muscle fibers. Their properties are summarized in **Table 41.1.** The proportions of the three types of muscle fibers tailor the contractile characteristics of each muscle to suit its function within the body.

Slow muscle fibers contract relatively slowly and the intensity of contraction is low because their myosin crossbridges hydrolyze ATP relatively slowly. They can remain contracted for relatively long periods without fatiguing. Slow muscle fibers typically contain many mitochondria and make most of their ATP by oxidative phosphorylation (aerobic respiration). They have a low capacity to make ATP by anaerobic glycolysis. They also contain high concentrations of the oxygen-storing protein **myoglobin**, which greatly enhances their oxygen supplies. Myoglobin is closely related to hemoglobin, the oxygen-carrying protein of red blood cells. Myoglobin gives slow muscle fibers, such as those in the legs of ground birds such as quail, chickens, and ostriches, a deep red color. In sharks and bony fishes, strips of slow muscles concentrated in a band on either side of the body are used for slow, continuous swimming and maintaining body position.

Fast muscle fibers contract relatively quickly and powerfully because their myosin crossbridges hydrolyze ATP faster than those of slow muscle fibers. Fast aerobic fibers have abundant mitochondria, a rich blood supply, and a high concentration of myoglobin, which makes them red in color. They have a high capacity for making ATP by oxidative phosphorylation, and an intermediate capacity for making ATP by anaerobic glycolysis. They fatigue more quickly than slow fibers, but not as quickly as fast anaerobic fibers. Fast aerobic muscle fibers are abundant in the flight muscles of migrating birds such as ducks and geese.

Fast anaerobic fibers typically contain high concentrations of glycogen, relatively few mitochondria, and a more limited blood supply than fast aerobic fibers. They generate ATP mostly by anaerobic respiration (glycolysis) and have a low capacity to produce ATP by oxidative respiration. Fast anaerobic fibers produce especially rapid and powerful contractions but are more susceptible to fatigue. Because their myoglobin supply is limited and they contain few mitochondria, they are pale in color. Some ground birds have flight muscles consisting almost entirely of fast anaerobic muscle fibers. These muscles can produce a short burst of intensive contractions allowing the bird to escape a predator, but they cannot produce sustained flight. Most muscles of lampreys, sharks, fishes, amphibians, and reptiles also contain fast anaerobic muscle fibers, allowing the animals to move quickly to capture prey and avoid danger.

The muscles of humans and other mammals are mixed, and contain different proportions of slow and fast muscle fibers, depending on their functions. Muscles specialized for prolonged, slow contractions, such as the postural muscles of the back, have a high proportion of slow fibers and are a deep red color. The muscles of the forearm that move the fingers have a higher proportion of fast fibers and are a paler red than the back muscles. These muscles can contract rapidly and powerfully, but they fatigue much more rapidly than the back muscles.

The number and proportions of slow and fast muscle fibers in individuals are inherited characteristics. However, particular types of exercise can convert some fast muscle fibers between aerobic and anaerobic types. Endurance training, such as long-distance running, converts fast muscle fibers from the anaerobic to the aerobic type, and regimes such as weight lifting induce the reverse conversion. If the training regimes stop, most of the fast muscle fibers revert to their original types.

Table 41.1 **Characteristics of Slow and Fast Muscle Fibers in Skeletal Muscle**

	Fiber Type		
Property	**Slow**	**Fast Aerobic**	**Fast Anaerobic**
Contraction speed	Slow	Fast	Fast
Contraction intensity	Low	Intermediate	High
Fatigue resistance	High	Intermediate	Low
Myosin–ATPase activity	Low	High	High
Oxidative phosphorylation capacity	High	High	Low
Enzymes for anaerobic glycolysis	Low	Intermediate	High
Mitochondria	Many	Many	Few
Myoglobin content	High	High	Low
Fiber color	Red	Red	White
Glycogen content	Low	Intermediate	High

Skeletal Muscle Control Is Divided among Motor Units

The control of muscle contraction extends beyond the simple ability to turn the crossbridge cycle on and off. We can adjust a handshake from a gentle squeeze to a strong grasp, or exactly balance a feather or dumbbell in the hand. How are entire muscles controlled in this way? The answer lies in activation of the muscle fibers in blocks called **motor units.**

The muscle fibers in each motor unit are controlled by branches of the axon of a single efferent neuron **(Figure 41.7).** As a result, all those fibers contract each time the neuron fires an action potential. All the muscle fibers in a motor unit are of the same type—either slow, fast aerobic, or fast anaerobic. When a motor unit contracts, its force is distributed throughout the entire muscle because the fibers are dispersed throughout the muscle rather than being concentrated in one segment.

For a delicate movement, only a few efferent neurons carry action potentials to a muscle, and only a few motor units contract. For more powerful movements, more efferent neurons carry action potentials, and more motor units contract.

Muscles that can be precisely and delicately controlled, such as those moving the fingers in humans, have many motor units in a small area, with only a few muscle fibers—about 10 or so—in each unit. Muscles that produce grosser body movements, such as those moving the legs, have fewer motor units in the same volume of muscle but thousands of muscle fibers in each unit. In the calf muscle that raises the heel, for example, most motor units contain nearly 2000 muscle fibers. Other skeletal muscles fall between these extremes, with an average of about 200 muscle fibers per motor unit.

Invertebrates Move Using a Variety of Striated Muscles

Invertebrates also have muscle cells in which actin-based thin filaments and myosin-based thick filaments produce movements by the same sliding mechanism as in vertebrates. Muscles that are clearly striated, which occur in virtually all invertebrates except sponges, have thick and thin filaments arranged in sarcomeres remarkably similar to those of vertebrates, except for variations in sarcomere length and the ratio of thin to thick filaments.

In invertebrates, an entire muscle is typically controlled by one or a few motor neurons. Nevertheless, invertebrate muscles are capable of finely graded contractions because individual neurons make large numbers of synapses with the muscle cells. As action potentials arrive more frequently at the synapses, more Ca^{2+} is released into the cells, and the muscles contract more strongly.

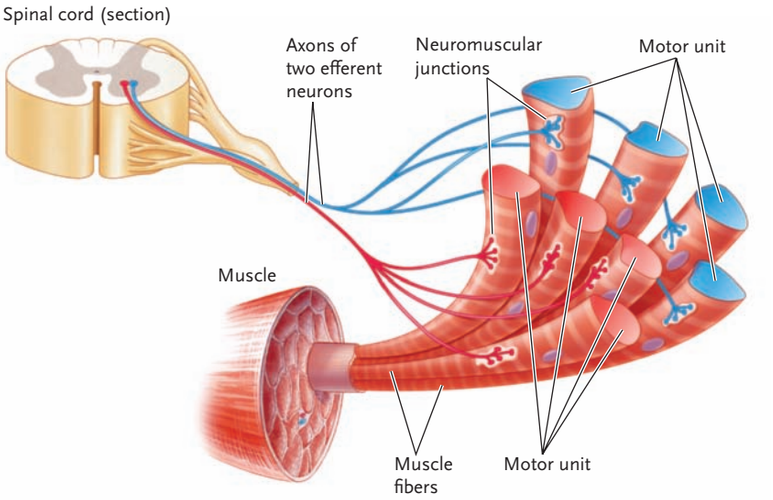


Figure 41.7
Motor units in vertebrate skeletal muscles. Each motor unit consists of groups of muscle fibers activated by branches of a single efferent (motor) neuron.

Study Break

1. Muscle contraction occurs in response to a stimulus from the nervous system. How does this occur?
2. Outline the molecular events that take place in the sliding filament mechanism of muscle contraction.

41.2 Skeletal Systems

Animal skeletal systems provide physical support for the body and protection for the soft tissues. They also act as a framework against which muscles work to move parts of the body or the entire organism. There are three main types of skeletons found in both invertebrates and vertebrates: hydrostatic skeletons, exoskeletons, and endoskeletons.

A Hydrostatic Skeleton Consists of Muscles and Fluid

A **hydrostatic skeleton** (*hydro* = water; *statikos* = causing to stand) is a structure consisting of muscles and fluid that, by themselves, provide support for the animal or part of the animal; no rigid support, like a bone, is involved. A hydrostatic skeleton consists of a body compartment or compartments filled with water or body fluids, which are incompressible liquids. When the muscular walls of the compartment contract, they pressurize the contained fluid. If muscles in one part of the compartment are contracted while muscles in another part are relaxed, the pressurized fluid will

Linda Pitkin/Planet Earth Pictures

Figure 41.8
Sea anemones in **(a)** the resting and **(b)** the feeding position. In **(a),** longitudinal muscles in the body wall are contracted, and circular muscles are relaxed. In **(b),** the longitudinal muscles are relaxed, and the circular muscles are contracted. Both sets of muscles work against a hydrostatic skeleton.

move to the relaxed part of the compartment, distending it. In short, the contractions and relaxations of the muscles surrounding the compartments change the shape of the animal.

Hydrostatic skeletons are the primary support systems of cnidarians, flatworms, roundworms, and annelids. In all these animals, compartments containing fluids under pressure make the body semirigid and provide a mechanical support on which muscles act. For example, sea anemones have a hydrostatic skeleton consisting of several fluid-filled body cavities. The body wall contains longitudinal and circular muscles that work against that skeleton. Between meals, longitudinal muscles are contracted (shortened), while the circular ones are relaxed, and the animal looks short and squat **(Figure 41.8a).** It lengthens into its upright feeding position by contracting the circular muscles and relaxing the longitudinal ones **(Figure 41.8b).** In flatworms, roundworms, and annelids, striated muscles in the body wall act on the hydrostatic skeleton to produce creeping, burrowing, or swimming movements. Among these animals, annelids have the most highly developed musculoskeletal systems, with an outer layer of circular muscles surrounding the body, and an inner layer of longitudinal muscles running its length **(Figure 41.9).** Contractions of the circular muscles reduce the diameter of the body and increase the length of the worm; contractions of the longitudinal muscles shorten the body and increase its diameter. Annelids move along a surface or burrow by means of alternating waves of contraction of the two muscle layers that pass along the body, working against the fluid-filled body compartments of the hydrostatic skeleton.

Many arthropods have hydrostatic skeletal elements. In the larvae of flying insects, internal fluids held under pressure by the muscular body wall provide some body support. In spiders, the legs are extended from the bent position by muscles exerting pressure against body fluids.

Some structures of echinoderms are supported by hydrostatic skeletons. The tube feet of sea stars and sea urchins, for example, have muscular walls enclosing the fluid of the water vascular system (see Figure 29.46).

In vertebrates, the erectile tissue of the penis is a fluid-filled hydrostatic skeletal structure.

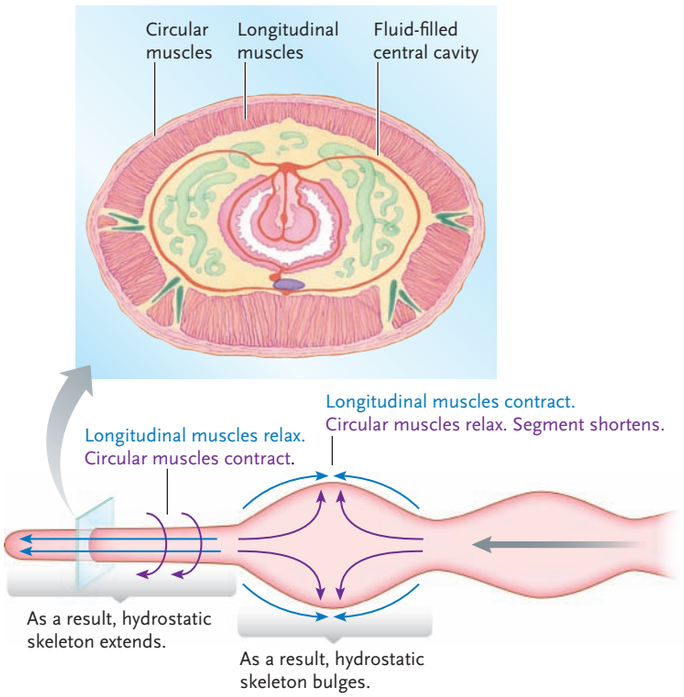


Figure 41.9
Movement of an earthworm, showing how muscles in the body wall act on its hydrostatic skeleton. Contraction of the circular muscles reduce body diameter and increase body length, while contraction of the longitudinal muscles decrease body length and increase body diameter.

An Exoskeleton Is a Rigid External Body Covering

An **exoskeleton** (*exo* = outside) is a rigid external body covering, such as a shell, that provides support. In an exoskeleton, the force of muscle contraction is applied against that covering. An exoskeleton also protects delicate internal tissues such as the brain and respiratory organs.

Many mollusks, such as clams and oysters, have an exoskeleton consisting of a hard calcium carbonate shell secreted by glands in the mantle. Arthropods, such as insects spiders, and crustaceans, have an external skeleton in the form of a chitinous cuticle, secreted by underlying tissue, that covers the outside surfaces of the animals. Like a suit of armor, the arthropod exoskeleton has movable joints, flexed and extended by muscles that extend across the inside surfaces of the joints **(Figure 41.10).** The exoskeleton protects against dehydration, serves as armor against predators, and provides the levers against which muscles work. In many flying insects, elastic flexing of the exoskeleton contributes to the movements of the wings.

In vertebrates, the shell of a turtle or tortoise is an exoskeletal structure, as are the bony plates, abdominal

ribs, collar bones, and most of the skull of the American alligator.

An Endoskeleton Consists of Supportive Internal Body Structures Such as Bones

An **endoskeleton** (*endon* = within) consists of internal body structures, such as bones, that provide support. In an endoskeleton, the force of contraction is applied against those structures. Like exoskeletons, endoskeletons also protect delicate internal tissues such as the brain and respiratory organs.

In mollusks, the mantle of squids and cuttlefish is reinforced by an endoskeletal element commonly called a "pen" (in squid) or the "cuttlebone" in cuttlefish (see Figure 29.22). Squids also have an internal case of cartilage that surrounds and protects the brain; other segments of cartilage support the gills and siphon in squids and octopuses.

Echinoderms have an endoskeleton consisting of *ossicles* (*ossiculum* = little bone), formed from calcium carbonate crystals. The shells of sand dollars and sea urchins are the endoskeletons of these animals.

The endoskeleton is the primary skeletal system of vertebrates. An adult human, for example, has an endoskeleton consisting of 206 bones arranged in two structural groups **(Figure 41.11).** The **axial skeleton**, which includes the skull, vertebral column, sternum, and rib cage, forms the central part of the structure (shaded in red in Figure 41.11). The **appendicular skeleton** (shaded in green) includes the shoulder, hip, leg, and arm bones.

Bones of the Vertebrate Endoskeleton Are Organs with Several Functions

The vertebrate endoskeleton supports and maintains the overall shape of the body and protects key internal organs. In addition, the skeleton is a storehouse for calcium and phosphate ions, releasing them as required to maintain optimal levels of these ions in body fluids. Bones are also sites where new blood cells form.

Bones are complex organs built up from multiple tissues, including bone tissue with cells of several kinds, blood vessels, nerves, and in some, stores of adipose tissue. Bone tissue is distributed between dense, compact bone regions, which have essentially no spaces other than the microscopic canals of the osteons (see Figure 36.5d), and spongy bone regions, which are opened by larger spaces (see Figure 41.11). Compact bone tissue generally forms the outer surfaces of bones, and spongy bone tissue the interior. The interior of some flat bones, such as the hip bones and the ribs, are filled with *red marrow,* a tissue that is the primary source of new red blood cells in mammals and birds. The shaft of long bones such as the femur is opened by a large central canal filled with adipose tissue called *yellow marrow,* which is a source of some white blood cells.

Throughout the life of a vertebrate, calcium and phosphate ions are constantly deposited and withdrawn from bones. Hormonal controls maintain the concentration of Ca^{2+} ions at optimal levels in the blood and extracellular fluids (see Figure 40.10), ensuring that calcium is available for proper functioning of the nervous system, muscular system, and other physiological processes.

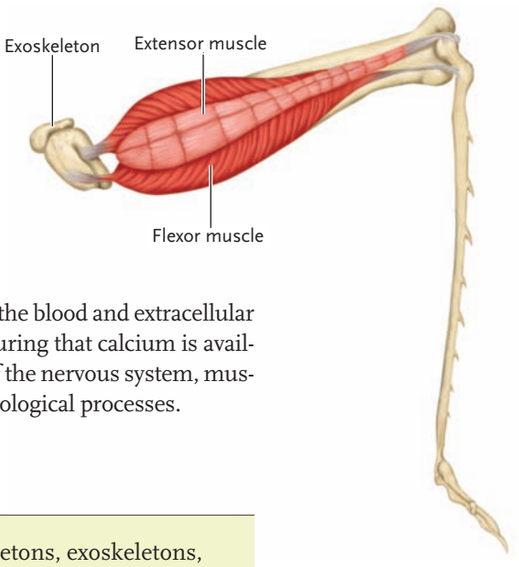


Figure 41.10
Muscles are attached to the inside surfaces of the exoskeleton in a typical insect leg such as this one.

Study Break

1. How do hydrostatic skeletons, exoskeletons, and endoskeletons provide support to the body? Give an example of each of these types in echinoderms and vertebrates.
2. What are the functions of the bones of the vertebrate endoskeleton?

41.3 Vertebrate Movement: The Interactions between Muscles and Bones

The skeletal systems of all animals act as a framework against which muscles work to move parts of the body or the entire organism. In this section, the muscle–bone interactions that are responsible for the movement of vertebrates are described.

Joints of the Vertebrate Endoskeleton Allow Bones to Move and Rotate

The bones of the vertebrate skeleton are connected by joints, many of them movable. The most-movable joints, including those of the shoulders, elbows, wrists, fingers, knees, ankles, and toes, are *synovial joints,* consisting of the ends of two bones enclosed by a fluid-filled capsule of connective tissue **(Figure 41.12a).** Within the joint, the ends of the bones are covered by a smooth layer of cartilage and lubricated by synovial fluid, which makes the bones slide easily as the joint moves. Synovial joints are held together by straps of connective tissue called *ligaments,* which extend across the joints outside the capsule **(Figure 41.12b).** The ligaments restrict the motion of the joint and help prevent it from buckling or twisting under heavy loads.

In other, less movable joints, called *cartilaginous joints,* the ends of bones are covered with layers of cartilage, but have no fluid-filled capsule surrounding them. Fibrous connective tissue covers and connects

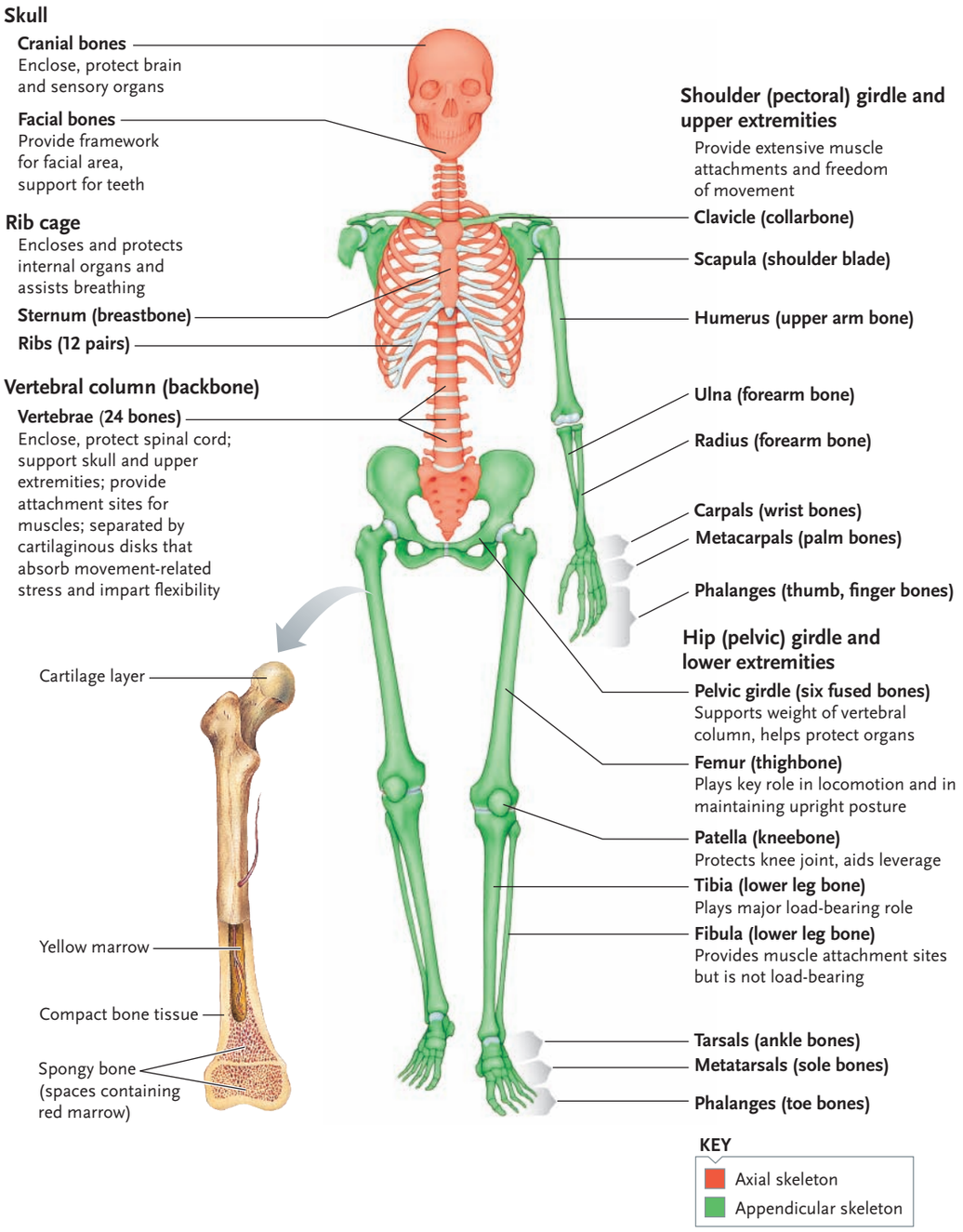


Figure 41.11
Major bones of the human body. The inset shows the structure of a limb bone, with the location of red and yellow marrow. The internal spaces lighten the bone's structure. The cartilage layer forms a smooth, slippery cushion between bones in a joint.

the bones of these joints, which occur between the vertebrae and some rib bones.

In still other joints, called *fibrous joints,* stiff fibers of connective tissue join the bones and allow little or no movement. Fibrous joints occur between the bones of the skull and hold the teeth in their sockets.

The bones connected by movable joints work like levers. A lever is a rigid structure that can move around a pivot point known as a *fulcrum*. Levers differ with respect to where the fulcrum is along the lever and where the force is applied. The most common type of lever system in the body—exemplified by the elbow joint—has the fulcrum at one end, the load at the opposite end, and the force applied at a point between the ends **(Figure 41.13).** For this lever, the force applied must be much greater than the load, but it increases the distance the load moves as compared with the distance over which the force is applied. This allows small mus-

cle movements to produce large body movements, and also allows movements such as running or throwing to be carried out at high speed.

At a joint, a muscle that causes movement in the joint when it contracts is called an **agonist**. In many cases, other muscles that assist the action of an agonist are involved in the movement of a joint. For instance, deltoid and pectoral muscles assist the biceps brachii muscle in lifting a weight.

Most of the bones of vertebrate skeletons are moved by muscles arranged in **antagonistic pairs:** *extensor muscles* extend the joint, meaning increasing the angle between the two bones, while *flexor muscles* do the opposite. (Antagonistic muscles are also used in invertebrates for movement of body parts—for example, the limbs of insects and arthropods.) In humans, one such pair is formed by the biceps brachii muscle at the front of the upper arm and the triceps brachii muscle at the back of the upper arm **(Figure 41.14).** When the biceps muscle contracts, the bone of the lower arm is bent (flexed) around the elbow joint, and the triceps muscle is passively stretched (see Figure 41.14a); when the triceps muscle contracts, the lower arm is straightened (extended) and the biceps muscle is passively stretched (see Figure 41.14b).

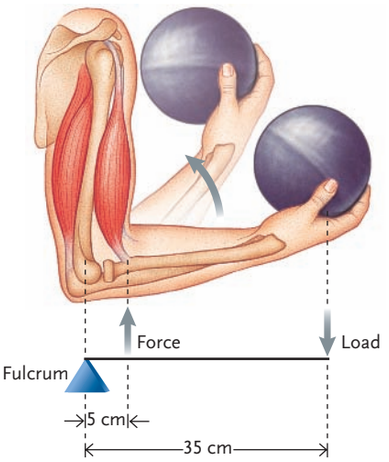


Figure 41.13
A body lever: The lever formed by the bones of the forearm. The fulcrum (the hinge or joint) is at one end of the lever, the load is placed on the opposite end, and the force is exerted at a point on the lever between the fulcrum and the load.

Vertebrates Have Muscle–Bone Interactions Optimized for Specific Movements

Vertebrates differ widely in the patterns by which muscles connect to bones, and in the length and mechanical advantage of the levers produced by these connections. These differences produce limbs and other body parts that are adapted for either power or speed, or the most advantageous compromise between these characteristics. Among burrowing mammals such as the mole, for example, the limb bones are short, thick, and heavy, and the point at which muscles attach produce levers that are slow to move

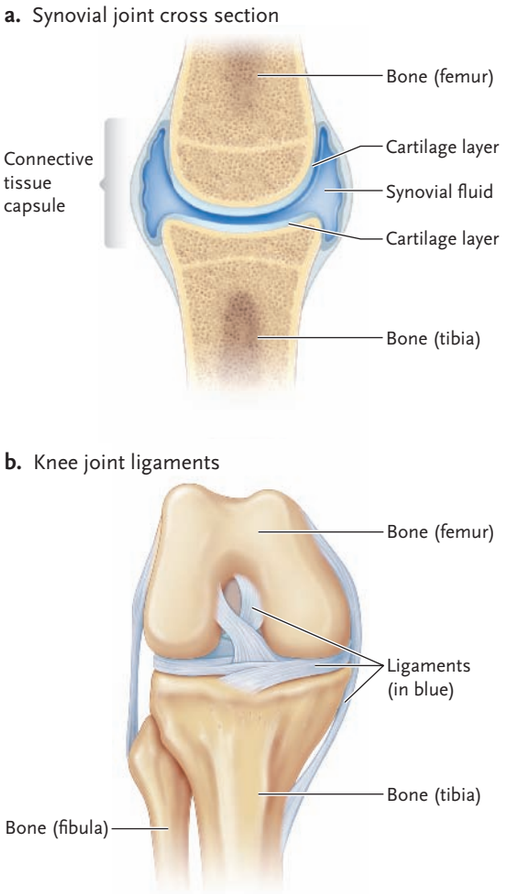


Figure 41.12
A synovial joint. (a) Cross section of a typical synovial joint. **(b)** Ligaments reinforcing the knee joint.

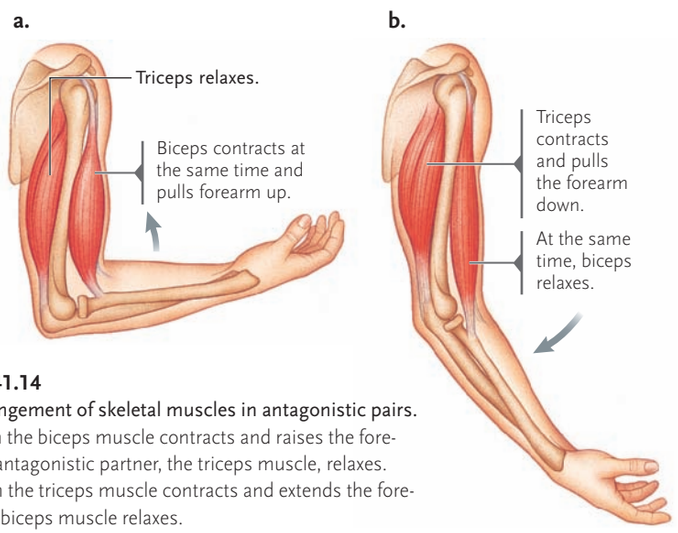


Figure 41.14
The arrangement of skeletal muscles in antagonistic pairs. (a) When the biceps muscle contracts and raises the forearm, its antagonistic partner, the triceps muscle, relaxes. **(b)** When the triceps muscle contracts and extends the forearm, the biceps muscle relaxes.

but that need to apply smaller forces to move a load compared with a human biceps. In contrast, a mammal such as the deer has relatively light and thin bones with muscle attachments producing levers that can produce rapid movement, moving the body easily over the ground.

Study Break

1. Distinguish synovial joints, cartilaginous joints, and fibrous joints.
2. What are antagonistic muscle pairs?

Unanswered Questions

How can muscle growth processes be controlled to improve the clinical treatment of muscular dystrophy and related disorders?
The mechanisms by which different muscle types develop and their impact on organismal metabolism is not well known. However, we do know that one of the proteins produced in muscle cells, myostatin, is a growth factor that inhibits skeletal muscle growth and development. Thus, myostatin is a potential therapeutic target for treating some of the most debilitating types of muscular dystrophy, which is a degenerative and fatal disease associated with the progressive loss of skeletal muscle mass. Animals with mutations that knock out myostatin gene function completely (for example, the Belgian Blue and Piedmontese cattle breeds) or myostatin knockout mice, in which the gene has been removed experimentally (see Section 18.2 and *Focus on Research* in Chapter 43), have significantly enhanced musculature that is commonly referred to as *double muscling*. Such mutations and enhanced skeletal muscle mass have also been described in a racing dog breed, the whippet, and recently in a young boy.

Our laboratory studies focus on developing novel technologies that introduce protein inhibitors of myostatin activity—in essence, inhibiting the inhibitor—and thereby stimulate skeletal muscle growth in both clinical and agricultural settings. We have recently determined that myostatin can also negatively regulate cardiac muscle growth. Thus, disrupting myostatin production or availability may also help heart attack patients. Replacing damaged skeletal and cardiac muscle using adult or embryonic stem cells engineered to match either tissue type is another highly promising technique for treating these disorders; it could be improved by using "antimyostatin" technologies that enhance growth of the transplanted cells.

How much do the metabolic processes of skeletal muscle specifically contribute to energy storage and whole body form?
Complications associated with obesity, particularly type 2 diabetes mellitus, have reached near-epidemic proportions worldwide. Type 2 diabetes differs from type 1 and is caused not by a lack of the pancreatic hormone insulin but rather by insulin resistance, in which an individual's physiological levels of insulin are inadequate to produce a normal insulin response in the tissues. Both types, however, result in the body's inability to properly process and store metabolites, mostly glucose. Type 2 diabetes can be a debilitating and fatal disease if poorly managed and often aggravates other diseases as well. Scientists now recognize that growth and metabolic processes are integrated and controlled by the same hormones, growth factors, and cytokines. Indeed, skeletal muscle is the largest consumer of metabolites and has the greatest potential to impact their circulating levels. Recent studies suggest that increasing muscle mass can significantly reduce fat mass as growing muscle is supported by energy from fat metabolism. Enhancing skeletal muscle growth and/or the ability of the tissue to consume blood metabolites in obese patients with type 2 diabetes could therefore improve treatments for both. The same antimyostatin technologies used to treat muscle growth disorders could also be used to treat severe cases of obesity and type 2 diabetes with the goals of increasing muscle mass, decreasing fat mass, and improving insulin sensitivity.

How do the extremely complex electrical properties of cardiac muscle develop, and how can they be controlled for biomedical purposes?
An ischemic event that blocks blood flow to a region of the heart and ultimately deprives the muscle of oxygen often results in a heart attack and can damage or destroy significant amounts of cardiac muscle. The surviving muscle, however, compensates by increasing the specific force generated by individual myofibers. Scientists have recently determined that these changes are due to the remodeling of electrical properties—changes in the amount and relative distribution as well as the activity of different classes of ion channels—within the surviving muscle itself. Although the specific channels and the mechanisms of regulation are unknown, a better understanding and ultimately control of these processes could help heart attack patients survive.

Buel (Dan) Rodgers is an assistant professor and assistant animal scientist at Washington State University, studying molecular endocrinology and animal genomics, specifically skeletal muscle growth and development. To learn more about his research, visit http://www.ansci.wsu.edu/People/rodgers/faculty.asp.

Review

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41.1 Vertebrate Skeletal Muscle: Structure and Function

- Skeletal muscles move the joints of the body. They are formed from long, cylindrical cells called muscle fibers, which are packed with myofibrils, contractile elements consisting of myosin thick filaments and actin thin filaments. The two types of filaments are arranged in an overlapping pattern of contractile units called sarcomeres (Figure 41.2).
- Infoldings of the plasma membrane of the muscle fiber form T tubules. The sarcomeres are encircled by the sarcoplasmic reticulum, a system of vesicles with segments separated from T tubules by small gaps (Figure 41.3).

- In the sliding filament mechanism of muscle contraction, the simultaneous sliding of thin filaments on each side of sarcomeres over the thick filaments shortens the sarcomeres and the muscle fibers, producing the force that contracts the muscle (Figure 41.4).
- The sliding motion of thin and thick filaments is produced in response to an action potential arriving at the neuromuscular junction. The action potential causes the release of acetylcholine, which triggers an action potential in the muscle fiber that spreads over its plasma membrane and stimulates the sarcoplasmic reticulum to release Ca^{2+} into the cytosol. The Ca^{2+} combines with troponin, inducing a conformational change that moves tropomyosin away from the myosin-binding sites on thin filaments. Exposure of the sites allows myosin crossbridges to bind and initiate the crossbridge cycle in which the myosin heads of thick filaments attach to a thin filament, pull, and release in cyclic reactions powered by ATP hydrolysis (Figure 41.5).
- When action potentials stop, Ca^{2+} is pumped back into the sarcoplasmic reticulum, leading to Ca^{2+} release from troponin, which allows tropomyosin to cover the myosin-binding sites in the thin filaments, thereby stopping the crossbridge cycle (Figure 41.5).
- A single action potential arriving at a neuromuscular junction causes a muscle twitch. Restimulation of a muscle fiber before it has relaxed completely causes a second twitch, which is added to the first, causing a summed, stronger contraction. Rapid arrival of APs causes the twitches to sum to a peak level of contraction called tetanus. Normally, muscles contract in a tetanic mode (Figure 41.6).
- Muscle fibers occur in three types. Slow muscle fibers contract relatively slowly, but do not fatigue rapidly. Fast aerobic fibers contract relatively quickly and powerfully, and fatigue more quickly than slow fibers. Fast anaerobic fibers can contract more rapidly and powerfully than fast aerobic fibers, but fatigue more rapidly. The fibers differ in their number of mitochondria and capacity to produce ATP (Table 41.1).
- Skeletal muscles are divided into motor units, consisting of a group of muscle fibers activated by branches of a single motor neuron. The total force produced by a skeletal muscle is determined by the number of motor units that are activated (Figure 41.7).
- Invertebrate muscles contain thin and thick filaments arranged in sarcomeres, and contract by the same sliding filament mechanism that operates in vertebrates.

Animation: Structure of skeletal muscle

Animation: Sliding filament model

Animation: Nervous system and muscle contraction

Animation: Troponin and tropomyosin

Animation: Energy sources for contraction

Animation: Types of contractions

41.2 Skeletal Systems

- A hydrostatic skeleton is a structure consisting of a muscle-surrounded compartment or compartments filled with fluid under pressure. Contraction and relaxation of the muscles changes the shape of the animal (Figures 41.8 and 41.9).
- In an exoskeleton, a rigid external covering provides support for the body. The force of muscle contraction is applied against the covering. An exoskeleton can also protect delicate internal tissues (Figure 41.10).
- In an endoskeleton, the body is supported by rigid structures within the body, such as bones. The force of muscle contraction is applied against those structures. Endoskeletons also protect delicate internal tissues. In vertebrates, the endoskeleton is the primary skeletal system. The vertebrate axial skeleton consists of the skull, vertebral column, sternum, and rib cage, while the appendicular skeleton includes the shoulder bones, the forelimbs, the hip bones, and the hind limbs (Figure 41.11).
- Bone tissue is distributed between compact bone, with no spaces except the microscopic canals of the osteons, and spongy bone tissue, which has spaces filled by red or yellow marrow (Figure 41.11).
- Calcium and phosphate ions are constantly exchanged between the blood and bone tissues. The turnover keeps the Ca^{2+} concentration balanced at optimal levels in body fluids.

Animation: Vertebrate skeletons

Animation: Human skeletal system

Animation: Structure of a femur

Animation: Long bone formation

41.3 Vertebrate Movement: The Interactions between Muscles and Bones

- The bones of a skeleton are connected by joints. A synovial joint, the most movable type, consists of a fluid-filled capsule surrounding the ends of the bones forming the joint. A cartilaginous joint, which is less movable, has smooth layers of cartilage between the bones with no surrounding capsule. The bones of a fibrous joint are joined by connective tissue fibers that allow little or no movement (Figure 41.12).
- The bones moved by skeletal muscles act as levers, with a joint at one end forming the fulcrum of the lever, the load at the opposite end, and the force applied by attachment of a muscle at a point between the ends (Figure 41.13).
- At a joint, an agonist muscle, perhaps assisted by other muscles, causes movement. Most skeletal muscles are arranged in antagonistic pairs, in which the members of a pair pull a bone in opposite directions. When one member of the pair contracts, the other member relaxes and is stretched (Figure 41.14).
- Vertebrates have a variety of patterns in which muscles connect to bones, giving different properties to the levers produced. Those properties are specialized for the activities of the animal.

Animation: Opposing muscle action

Animation: Human skeletal muscles

Questions

Self-Test Questions

1. Vertebrate skeletal muscle:
   a. is attached to bone by means of ligaments.
   b. may bend but not extend body parts.
   c. may rotate one body part with respect to another.
   d. is found in the walls of blood vessels and intestines.
   e. is usually attached at each end to the same bone.

2. In a resting muscle fiber:
   a. sarcomeres are regions between two H zones.
   b. discs of M line proteins called the A band separate the thick filaments.
   c. I bands are composed of the same thick filaments seen in the A bands.
   d. Z lines are adjacent to H zones, which attach thick filaments.

e. dark A bands contain overlapping thick and thin filaments with a central thin H zone composed only of thick filaments.

3. The sliding filament contractile mechanism:
   a. causes thick and thin filaments to slide toward the center of the A band, bringing the Z lines closer together.
   b. is inhibited by the influx of Ca^{2+} into the muscle fiber cytosol.
   c. lengthens the sarcomere to separate the I regions.
   d. depends on the isolation of actin and myosin until a contraction is completed.
   e. uses myosin crossbridges to stimulate delivery of Ca^{2+} to the muscle fiber.
4. During contraction of skeletal muscle:
   a. ATP stimulates Ca^{2+} to move out of the cytosol, which allows tropomyosin to bind myosin causing contraction of the thin filament.
   b. myosin crossbridges use ATP to relax the molecular spring in the myosin head, which pulls the thick filaments away from the thin actin filaments.
   c. actin binds ATP, allowing troponin in the thick filaments to form the myosin crossbridge.
   d. action potentials cause the release of Ca^{2+} into the sarcoplasmic reticulum allowing tropomyosin fibers to uncover the actin binding sites needed for the myosin crossbridge.
   e. botulinum toxin could increase the release of acetylcholine at the contracting muscle site.
5. When a trained marathoner is running, most likely his:
   a. muscles have low concentrations of myoglobin.
   b. slow muscle fibers will do most of the work for the run.
   c. slow muscle fibers will remain in constant tetanus over the length of the run.
   d. fast muscle fibers will be employed in the middle of his run.
   e. slow muscle fibers are using ATP obtained primarily by anaerobic respiration.
6. Which description is characteristic of a motor unit?
   a. A single motor unit's muscle fibers vary among the slow/fast aerobic and slow/fast anaerobic forms.
   b. When receiving an action potential, a motor unit is controlled by a single efferent axon that causes all its fibers to contract.
   c. When a motor unit contracts, certain sections of the muscle as a whole remain relaxed.
   d. If a motor unit controls walking, it is found in large numbers in the same volume of muscle.
   e. If a motor unit controls finger movement, it contains a large number of muscle fibers that are stimulated over a large area.
7. Which of the following is *not* an example of a hydrostatic skeletal structure?
   a. the tube feet of sea urchins
   b. the body wall of annelids
   c. the trunk of an elephant
   d. the body wall of cnidarians
   e. the penis of mammals
8. Endoskeletons:
   a. protect internal organs and provide structures against which the force of muscle contraction can work.
   b. differ from exoskeletons in that endoskeletons do not support the external body.
   c. cannot be found in mollusks and echinoderms.
   d. are composed of appendicular structures that form the skull.
   e. compose the arms and legs, which are part of the axial skeleton.
9. Connecting the vertebrate skeleton are:
   a. nonmovable synovial joints.
   b. ligaments holding together connective tissue of fibrous joints.
   c. cartilaginous joints found in the shoulders and elbows.
   d. synovial joints lubricated by synovial fluid.
   e. fibrous joints that move around a fulcrum allowing small muscle movements to produce large body movements.
10. The movement of vertebrate muscles is:
    a. agonistic when it extends the joint.
    b. antagonistic when it causes movement in the joint.
    c. caused by extensor muscles that flex the joint.
    d. caused by flexor muscles that extend the joint.
    e. most efficient when the biceps and triceps contract simultaneously.

Questions for Discussion

1. A coach must train young athletes for the 100-meter sprint. They need muscles specialized for speed and strength, rather than for endurance. What kinds of muscle characteristics would the training regimen aim to develop? How would it be altered to train marathoners?
2. What kind of exercise program might the coach in question 1 recommend to an older person developing osteoporosis? Why?
3. Based on material in this chapter and in Chapter 40 on endocrine controls, outline some possible causes and physiological effects of calcium deficiency in an active adult.

Experimental Analysis

Design an experiment with rats to determine whether endurance training alters the proportion of slow, fast aerobic, and fast anaerobic muscle fibers.

Evolution Link

What characteristics of vertebrate muscle suggest that the genes for muscle structure were inherited from invertebrate ancestors?

How Would You Vote?

Dietary supplements are largely unregulated. Should they be placed under the jurisdiction of the Food and Drug Administration, which could subject them to more stringent testing for effectiveness and safety? Go to www.thomsonedu.com/login to investigate both sides of the issue and then vote.