37 STRUCTURAL SUPPORT AND MOVEMENT

Pumping Up Muscles

Want to be more muscular and stronger, with a lot more endurance? Just use our pills or powders and be like the guy in Figure 37.1. That is the message in advertisements for many dietary supplements that target body builders and other athletes. The supplements are easily purchased from health food stores and through the Internet. They have not been classified as drugs by the Food and Drug Administration (FDA), so testing for their effectiveness and long-term side effects has been negligible. Independent monitoring of quality control over their commercial production ranges from little to none.

Consider androstenedione, or "andro." Sales of this drug got a big boost in 1998, after Mark McGwire admitted that he used it during his successful attempt to break Major League Baseball's home-run record. Androstenedione forms naturally in the body as an intermediate in the synthesis of testosterone. Testosterone *does* have tissue-building, anabolic effects that are well documented. Andro is said to raise the blood level of testosterone, which in turn boosts the rate of protein synthesis in muscles.

Does andro work? Probably not. In controlled studies, males of an experimental group used an andro supplement. They did not gain any more muscle mass or strength than males of a control group who were given a placebo. At most, the andro supplement raised the testosterone level in blood for a few hours.

Andro also forms as an intermediate during the synthesis of estrogen. This sex hormone has feminizing effects. Its known side effects on males include shrunken testicles, the development of female-like breasts, and hair loss. The users in both sexes risk liver damage, acne, and a lower blood level of "good" cholesterol (HDL). Females commonly develop masculinized patterns of hair growth and speech, and they can expect menstrual cycle disruptions.

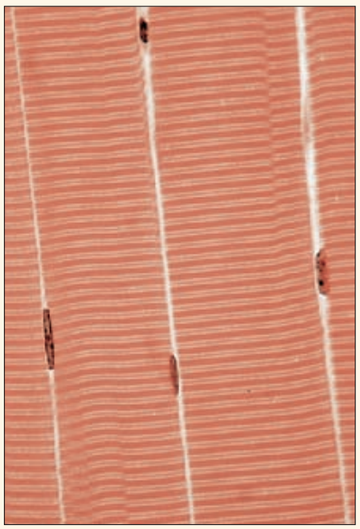
In early 2004, the FDA issued an advisory that andro supplements have serious side effects. Companies were ordered to stop distributing the drug immediately.

Creatine is another supplement that is being touted as a performance enhancer. Creatine is only a short chain of amino acids. The body normally makes some creatine and takes in more from food. When muscles are called upon to contract hard and fast, they use phosphorylated creatine as an instant energy source.

Unlike andro, creatine supplements might work. In several controlled studies, they improved performance during brief, high-intensity exercise. Clinical trials are under way to determine whether creatine may benefit individuals affected by muscular dystrophy and other

Figure 37.1 Overabundance of contractile tissue, of the sort shown on the facing page.

IMPACTS, ISSUES



muscle disorders. Nevertheless, excessive creatine intake does put a strain on the kidneys, and it is too soon to know whether it has long-term side effects. No regulatory agency checks to see how much creatine is actually present in any commercial product.

With this chapter, we turn to the point of having muscles in the first place. To most of us, the directional movement of the body or portions of it is one of the hallmarks of nearly all animals. Regardless of the species, movement requires contractile cells and some enclosed fluid or skeletal element against which the contractile force can be applied. The structure of skeletal and muscular systems, and how they work, has an evolutionary history—one that can help you evaluate how far both systems can be pushed in the pursuit of enhanced performance.

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? See BiologyNow for details, then vote online.

Key Concepts

HOW ANIMALS MOVE

Animals apply contractile force against a hydrostatic skeleton, exoskeleton, or endoskeleton. Muscle cells evolved from epithelial cells in which contractile filaments became organized in functional arrays. Section 37.1

DIVERSE INVERTEBRATE SKELETONS

In hydrostatic skeletons, a volume of fluid confined in a body chamber accepts the force of contraction. Muscles and often hydraulic pressure move exoskeletons. Endoskeletons are internal skeletons partnered with muscles. Section 37.2

VERTEBRATE SKELETONS

Bones are collagen-rich, mineralized organs that function in movement, protection and support of soft organs, and mineral storage. Blood cells form in some bones. Cartilage or ligaments connect bones at joints. Tendons attach skeletal muscles to bones. Sections 37.3, 37.4

THE MUSCLE–BONE PARTNERSHIP

Skeletal muscles are bundles of muscle fibers that interact with bones and with one another. Some cause movements by working as pairs or groups. Others oppose or reverse the action of a partner muscle. Section 37.5

HOW SKELETAL MUSCLE CONTRACTS

Inside a skeletal muscle, many myofibrils are transversely divided into sarcomeres, the basic unit of contraction. ATP energy forces parallel arrays of actin and myosin filaments in each sarcomere to interact. The interactions shorten the sarcomeres, which collectively accounts for contraction. Sections 37.6–37.8

VARIATIONS IN MUSCLE FUNCTION

Cross-bridges in sarcomeres collectively exert tension. A whole muscle shortens only when this mechanical force exceeds other, opposing forces. Exercise enhances the properties of whole muscles, and aging diminishes them. Sections 37.9, 37.10

Links to Earlier Concepts

In this chapter you will return to the contractile proteins actin and myosin (Sections 4.10, 4.11, 5.4). You will look once more at some skeletons of invertebrates (17.7, 25.4, 25.17) and vertebrates (26.2, 26.11–26.13), and at the fine structure of bone tissue and muscle tissues (33.2, 33.3). You will draw on your knowledge of active transport (5.4), ATP function (6.2), and pathways of organic metabolism (8.1, 8.6). You also will take a closer look at hormonal control of the body's calcium levels (36.4) and at how ACh triggers contraction (34.4).

37.1 An Evolutionary Heritage

LINKS TO SECTIONS 4.9, 17.7, 25.6, 25.17, 34.1

All animals make directional movements in response to stimuli. Even a mussel that lives out its adult life glued to a rock moves some of its structures. Animal larvae use cilia or flagella as motile structures. Larvae also change direction through interactions between their contractile cells and skeletal elements.

Where did animals get the capacity to move and bend the body in different directions? It started in epithelia of ancient invertebrates. Remember actin and myosin, the contractile proteins that are part of cytoskeletons (Section 4.9)? They became organized in longitudinal, circular, and diagonal arrays at the base of epithelial cells that formed the body's outer tissue layer. Being anchored to the rest of the body, the cells could change its shape by contracting and relaxing. They also could bend the body one way or another by contracting on one side at a time. Such "epitheliomuscular" cells are still present in cnidarians. Section 34.1 has a sketch of a few that are part of the body wall.

In other lineages, epitheliomuscular cells sank a bit below the free epithelial surface. Cells like this still occur in sweat glands, mammary glands, and the eye's iris. In many animals, however, these contractile cells sank completely into connective tissue. They evolved into muscle cells and became bundled in connective tissue inside muscles. Muscles are structural units that function exclusively in contraction.

Regardless of how they are organized in animals, contractile cells must interact with parts of a skeleton. A skeleton is a structural framework that functions in maintaining body shape, supporting and protecting cells, and accepting the force of contraction that can bring about movements. Three types are common.

With a **hydrostatic skeleton**, muscle cells apply the force of contraction against a body fluid and thereby redistribute it within a confined space. Try squeezing the middle of a long, skinny, water-filled balloon and you can see that fluid can offer considerable resistance to compression. With an **exoskeleton**, rigid or flexible structures at the body surface accept the applied force of contraction. An insect cuticle is one example. With an **endoskeleton**, *internal* body parts, such as bones, receive the applied force of muscle contraction.

The capacity of animals to move and bend in specific directions started with epithelial cells in which organized arrays of actin and myosin filaments evolved. Contractile cells of different animal groups are at different levels of organization. They direct their contractile force against a hydrostatic skeleton, exoskeleton, or endoskeleton.

37.2 Invertebrate Skeletons

Different kinds of skeletons show up among the 1.3 million or so named species of invertebrates. Some species apply contractile force to a skeleton that is no more than a confined body of water. Others apply contractile force to an exoskeleton or endoskeleton, either of which may be rubbery or rigid.

HYDROSTATIC SKELETONS

Sea anemones, worms, and many other soft-bodied animals have a hydrostatic skeleton and a tubular or cylindrical body. Many contractile cells are oriented side by side, longitudinally, in the body wall. Others are oriented like rings around the body cavity. Stiff fibers often form a mesh in the body wall. They help prevent uncontrollable bulges when contraction makes fluid move inside their gut cavity.

Think about a sea anemone (Figure 37.2). When the longitudinal cells in its body wall contract and radial ones relax (lengthen), its body collapses and is squat. When the longitudinal cells relax, radial ones contract and force fluid out of the gut, so the body lengthens into an upright feeding position. A nerve net controls the directional movements (Section 34.1).

An earthworm, recall, has a hydrostatic skeleton. It applies contractile force against fluid-filled coelomic chambers of its highly segmented body. Muscles in

Figure 37.2 Directional movement of a sea anemone, which has a hydrostatic skeleton. Epitheliomuscular cells in this cnidarian's body wall run longitudinally to the main body axis and radially around the gut. *Left*, radial epitheliomuscular cells are relaxed; longitudinal ones are contracted. *Right*, the radial cells have contracted and longitudinal ones are relaxed. The body extends upward, to its feeding position.

Fluid confined by the body wall accepts the contractile force.

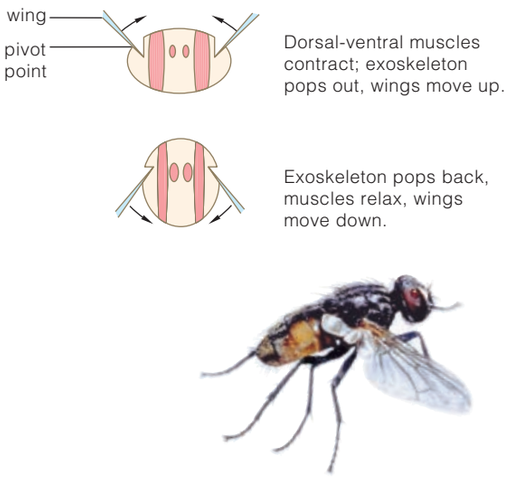


Figure 37.3 ***Animated!*** Motion of a fly wing. An insect cuticle forms a pliable hinge across gaps between body segments. Contracting muscles change the shape of the thorax, causing wings attached to it to move up and down.

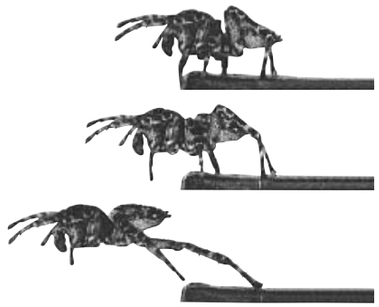


Figure 37.4 Jumping spiders. A large muscle in their cephalothorax contracts and forces blood into the hind legs. The surge of high fluid pressure extends the legs outward. As you might predict, these spiders have enormous eyes relative to their body size, which comes in handy for looking before they leap.

each segment's body wall contract and relax one after another as many anchoring bristles (setae) alternately plunge into soil and pull away from it. Section 25.6 shows how the contractions are coordinated to bring about forward motion.

EXOSKELETONS

Lobsters, spiders, insects, and other arthropods have a hinged exoskeleton with attachment sites for sets of muscles that move hard parts like levers. Consider a winged insect's cuticle. It thins where it extends over gaps between body segments on both sides of each wing. Being pliable at certain gaps, the cuticle acts as a hinge when contracting muscles alter the angle of the wing's attachment site (Figure 37.3 and Section 17.7). Small contractions can bring about large movements.

Arthropods move by a combination of muscles and hydraulic pressure. *Hydraulic* refers to fluid pressure within a tube. For instance, all spiders have an open circulatory system; their heart pumps blood directly into body tissues. Muscles attached to an exoskeleton contract and pull the spider legs inward, but there are no opposing muscles to push them out again. Instead, a large muscle of the spider cephalothorax contracts, which causes blood to surge into the hind legs (Figure 37.4). It is a bit like squeezing a rubber glove partially filled with water to make the limp fingers become erect. The hydraulic pressure helps a jumping spider leap twenty-five times its own length.

DO ANY INVERTEBRATES HAVE ENDOSKELETONS?

Glasslike spicules enclosed in the two cellular linings of a sponge's body are sort of like an endoskeleton, not that sponges do much with it. Contractile cells of some species close pores in the body wall in response to stimuli. Some freshwater sponges can expel water, like a slow sneeze, by coordinated contractions.

Echinoderms have an endoskeleton located within their dermis. It consists of arrays of structural elements called ossicles, which are made of tiny calcite crystals. Ossicles are shaped like spines, rods, and plates, and they form a honeycombed framework that is strong yet light in weight. Epidermis covers the ossicles that project above the body surface and give the group its name. Echinoderm means spiny-skinned.

All echinoderms move by a combination of muscles and hydraulic pressure. Their water-vascular system includes fluid-filled bulbs and sucker-bottomed tube feet (Section 25.17). Contracting muscles squeeze the bulbs and force water into the feet, which extend and attach to substrates or prey. Each tube foot retracts as its longitudinal muscles contract and thereby squeeze fluid back into the bulb above it (Figure 37.5).

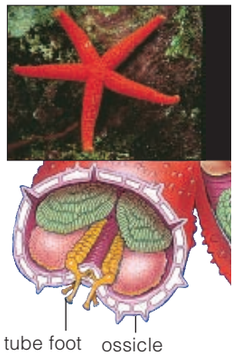


Figure 37.5 Ossicles of an echinoderm's endoskeleton

Cnidarians, worms, and many other invertebrates have a hydrostatic skeleton. Arthropods use muscles and hydraulic pressure to move their exoskeleton. Echinoderms have a honeycombed endoskeleton within their dermis.

37.3 Evolution of Vertebrate Skeletons

LINKS TO SECTIONS 26.2, 26.11–26.13

From notochord to vertebral column, from supports for gills to jaws, from structural elements inside lobed fins to limbs—Section 26.2 introduced these evolutionary trends, which occurred among vertebrates that invaded land. Many other modifications helped those lineages make the transition to life in a new medium—air.

Your skeleton, like those of other vertebrates on land, holds evidence of a time when body weight became deprived of water's buoyancy. Remember, the pelvic and pectoral girdles of a fish function as a stable base for moving fins that propel, guide, and stabilize the body in water. Among early four-legged vertebrates, those girdles transferred the weight of the main body mass to limbs, and they developed more surface area to which muscles became attached. The limbs became repositioned closer to the body mass and helped hold the body above the ground, so that forward thrusting motions became easier. A cage of hard bones became connected to the backbone. It helped keep the heart, lungs, and other soft organs from collapsing under the weight of a body no longer supported by water.

Figure 37.6 shows the cartilaginous skeleton of a fish and the kinds of bony endoskeletons that evolved among the amphibians and, later, among reptiles and mammals on land. Many thousands of variations on the original skeletal plan show up in the fossil record.

For example, compare Figure 37.6 with 37.7, and you see some of the same structural elements in the human skeleton, which has 206 bones. First, notice its pectoral girdle (at the shoulders), pelvic girdle (at the hips), and the paired arms, hands, legs, and feet. This is the *appendicular* portion—a legacy from an ancient tetrapod. Notice the shoulder blades, which are easy to dislocate. Also notice the long, thin collarbones, which are the bones broken most often. These vulnerabilities of the human skeleton are one outcome of an ancient aquatic heritage, of being a "fish out of water."

What about the *axial* portion of the human skeleton? The jaws and other skull bones, twelve pairs of ribs, a breastbone, and twenty-six vertebrae are the legacies of early craniates and jawed vertebrates. The **vertebrae** (singular, vertebra), or bony segments of a backbone, extend from the base of the skull to the pelvic girdle.

What does the vertebral column do now? Its bony parts offer attachment sites for paired muscles and a protective canal for the spinal cord. The column itself transmits your torso's weight to the lower limbs of a two-legged walker (Sections 26.11 through 26.13).

Our hominid ancestors started walking on two legs at least 4 million years ago. Over time, the backbone had to curve into an S shape to keep the body's main axis in vertical alignment. It helps that its bones are

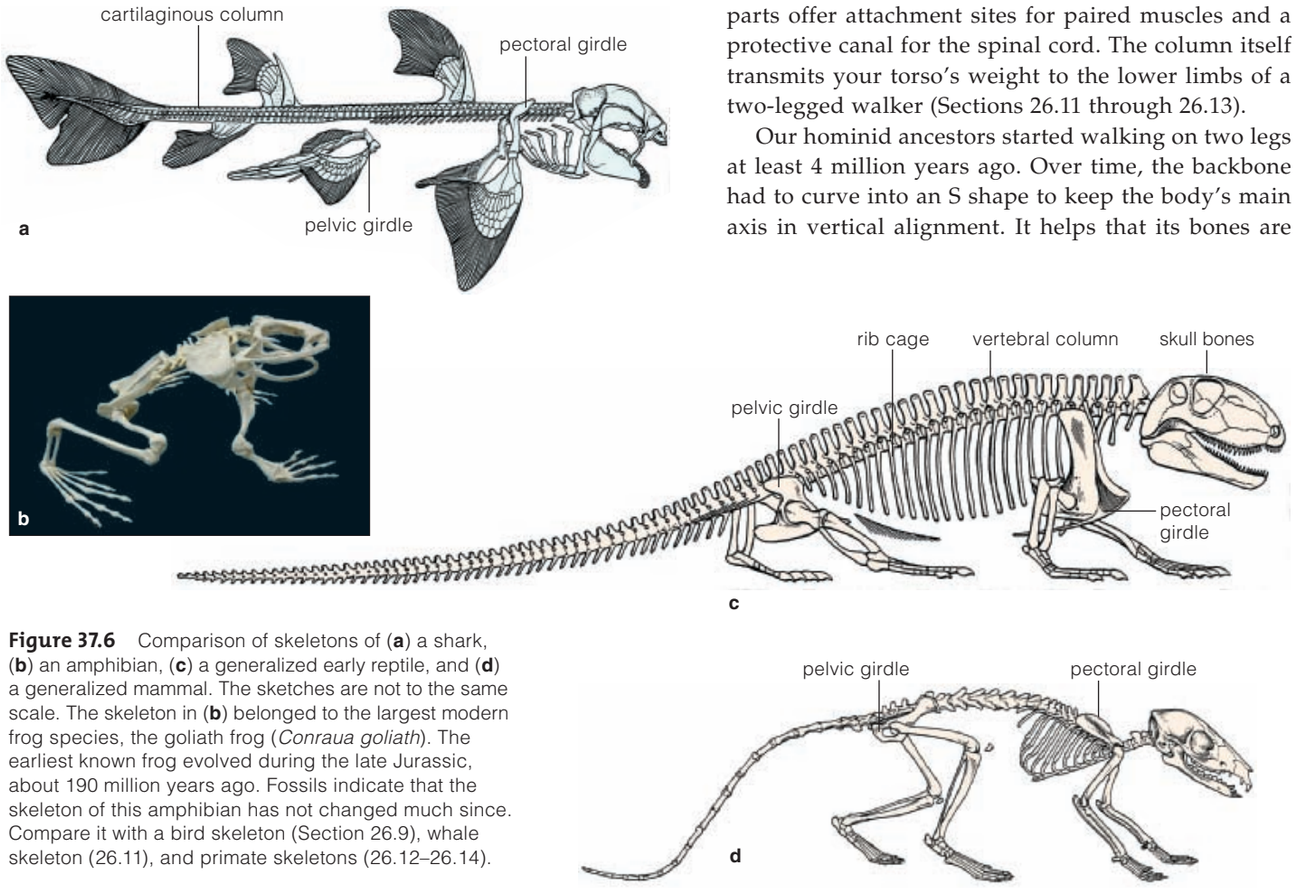


Figure 37.6 Comparison of skeletons of (**a**) a shark, (**b**) an amphibian, (**c**) a generalized early reptile, and (**d**) a generalized mammal. The sketches are not to the same scale. The skeleton in (**b**) belonged to the largest modern frog species, the goliath frog (*Conraua goliath*). The earliest known frog evolved during the late Jurassic, about 190 million years ago. Fossils indicate that the skeleton of this amphibian has not changed much since. Compare it with a bird skeleton (Section 26.9), whale skeleton (26.11), and primate skeletons (26.12–26.14).

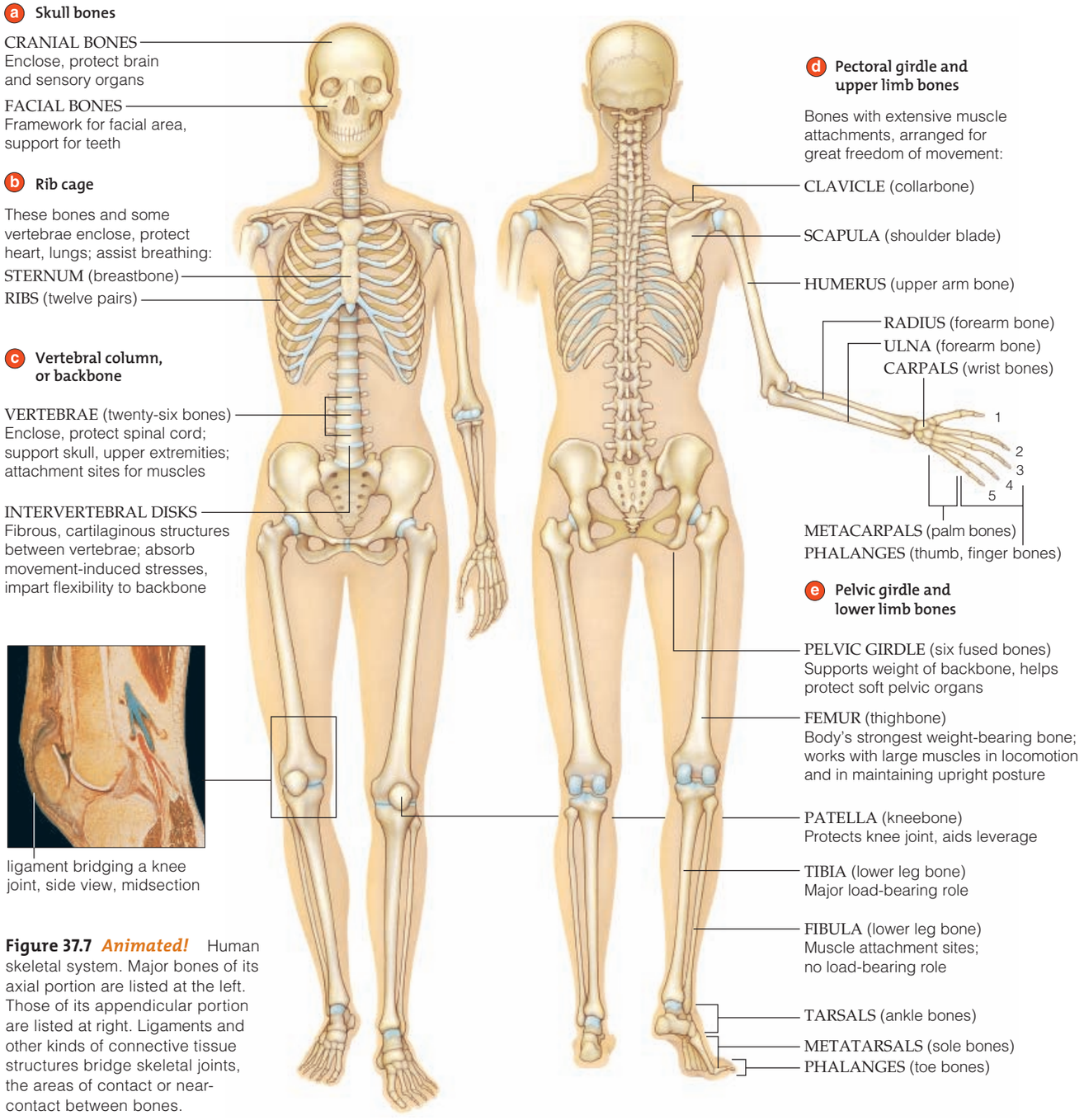


Figure 37.7 ***Animated!*** Human skeletal system. Major bones of its axial portion are listed at the left. Those of its appendicular portion are listed at right. Ligaments and other kinds of connective tissue structures bridge skeletal joints, the areas of contact or near-contact between bones.

separated from one another by **intervertebral disks**—cartilaginous shock absorbers and flex points. But the bones and disks are stacked against gravity. A rapid shock can force a disk to slip out of place or rupture—and this *herniated* disk can cause chronic back pain.

No longer aquatic, no longer tetrapods, the most recent representatives of the human species must live with the costs as well as benefits of getting around in the world with the two legs of an upright walker.

Skeletons of land vertebrates reflect early adaptations to life in water and later modifications to a life deprived of water's buoyancy.

37.4 Zooming In on Bones and Joints

LINKS TO SECTIONS 33.2, 36.4

Bones of a vertebrate skeleton are organs with diverse roles. They function in movement, in protection of soft internal organs, and as reservoirs for mineral ions. In certain bones, stem cells give rise to the body's blood cells (Table 37.1).

BONE STRUCTURE AND FUNCTION

Human bones range in size from middle ear bones smaller than lentils to clublike thighbones or femurs (Figure 37.7). Bone tissue, recall, consists of bone cells and collagen fibers in a calcium-hardened, organic matrix (Section 33.2). It has three types of bone cells. *Osteoblasts* are the bone-forming cells; they secrete the components of the matrix. Huge populations of them are present on the outer surfaces and internal cavities of the bones of adults. *Osteocytes* are osteoblasts that became imprisoned in small chambers after secreting matrix material around themselves. They are the most common bone cells in bony tissue of adults. *Osteoclasts* are bone cells that break down bone tissue by secreting acids and enzymes into the hardened matrix.

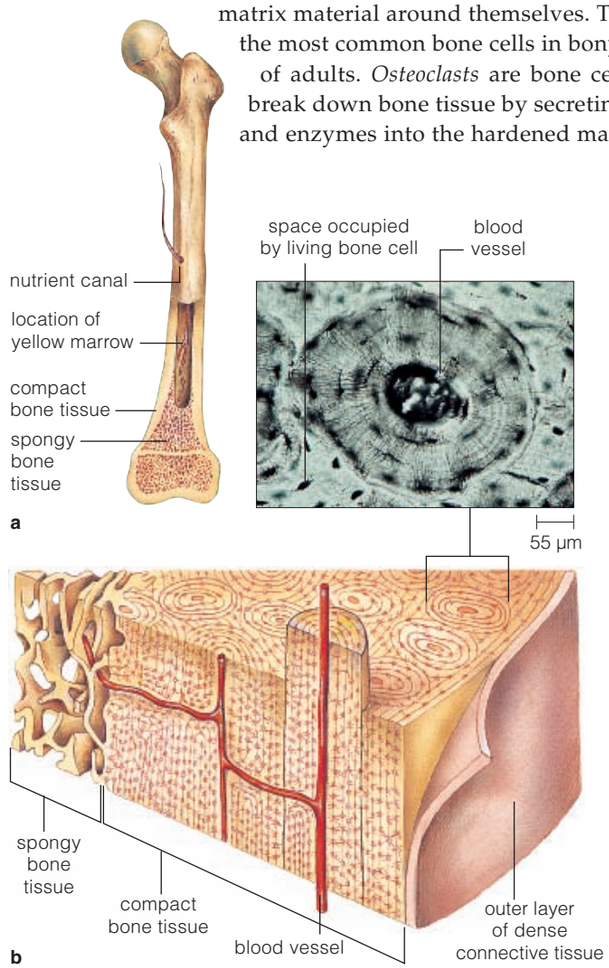


Figure 37.8 ***Animated!*** (**a**) Structure of a human femur, or thighbone, and (**b**) a section through its spongy and compact bone tissues.

Figure 37.8 shows the two types of bone tissue in a femur. *Compact* bone tissue resists mechanical shock. This tissue's matrix is laid down as dense concentric rings around tiny canals for nerves and blood vessels. Osteocytes reside in narrow clefts between the rings. *Spongy* bone tissue is present in the femur's shaft and knobby ends. It is strong but does not weigh much; its hardened matrix is pocketed with open spaces.

Red marrow, the major site of blood cell formation, fills the spaces in spongy bone. The central cavity of the femur and most mature bones of adults is filled with **yellow marrow**. This marrow is mostly fat, but it can be converted to blood cell-producing red marrow in times of severe blood loss.

BONE FORMATION AND REMODELING

The first skeleton to form in all vertebrate embryos is made of cartilage. Adult cartilaginous fishes retain it. In all other vertebrates, the cartilage is just the model. Osteoblasts infiltrate the model and transform it into bone. The cartilage in bone shaft breaks down, and a marrow cavity opens up inside (Figure 37.9).

In healthy young adults, the total bone mass does not change much even though osteocytes and mineral ions are being removed and replaced all the time. The removals and deposits help maintain required blood levels of calcium and phosphorus while keeping bones strong. In an ongoing process called **bone remodeling**, osteoblasts help form new bone tissue, which makes up for bone tissue that osteoclasts are breaking down. The osteoclast action releases mineral ions that enter interstitial fluid and the bloodstream, which transports them to metabolic reaction sites all through the body.

Calcium ions are the most prevalent mineral ions stored in and released from bones. Neural function, muscle contraction, and many other activities would end without them. The blood calcium level is a tightly

Table 37.1 Functions of Bone

1. *Movement.* Bones interact with skeletal muscle and change or maintain the position of the body and its parts.
2. *Support.* Bones support and anchor muscles.
3. *Protection.* Many bones are organized as hard compartments that enclose and protect soft internal organs.
4. *Mineral storage.* Bones are a reservoir for calcium and phosphorus ions. Deposits and withdrawals of these minerals help maintain essential ion concentrations in body fluids.
5. *Blood cell formation.* Only certain bones contain regions where blood cells form.

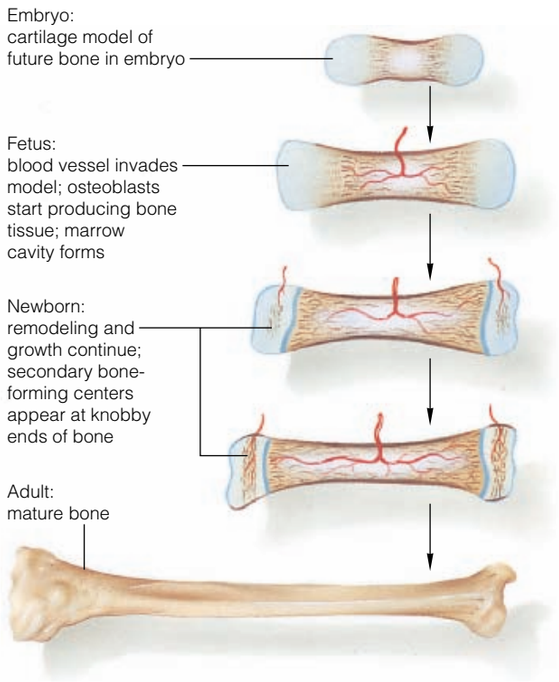


Figure 37.9 Long bone formation, starting with osteoblast activity in a cartilage model formed earlier in the embryo. The bone-forming cells are active first in the shaft region, then at the knobby ends. In time, cartilage is left only at the ends.

controlled aspect of metabolism. The bones and teeth store all but about 1 percent of the body's calcium.

Remember, negative feedback loops help regulate calcium release and uptake (Section 36.4). When there is too much calcium in the blood, the thyroid gland secretes calcitonin. By inhibiting osteoclast action, this hormone slows calcium release into blood. When there is too little calcium in blood, the parathyroid glands release parathyroid hormone, or PTH. This hormone stimulates calcium release from bones and calcium reabsorption from both kidneys. It enhances osteoclast action and also activates vitamin D, which stimulates calcium absorption from the gut lumen.

Until humans are about twenty-four years old, the osteoblasts are secreting more matrix than osteoclasts can break down, and so the bone mass increases. Bones become denser and stronger. Later in life, osteoblast activity declines, and bones gradually weaken.

Significant loss in bone density is called *osteoporosis* (Figure 37.10). Deficient calcium or vitamin D intake, parathyroid problems, and physical inactivity add to the risk. Declining sex hormone levels in menopause, smoking, excessive alcohol intake, and prolonged use of steroids also can slow bone deposition.

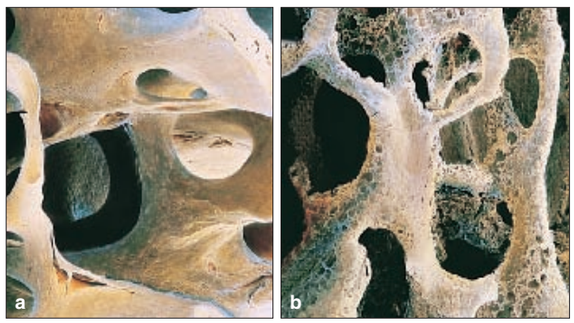


Figure 37.10 (**a**) Normal bone tissue. (**b**) Bone affected by osteoporosis.

WHERE BONES MEET—SKELETAL JOINTS

Connective tissue bridges **joints**, the areas of contact or near-contact between bones. **Ligaments** are straps of dense connective tissue at many joints, such as the knees. They attach one bone to another and let both move freely. The breastbone, vertebrae, and ribs move only a bit. Their joints of cartilage are pliable enough to cushion abutting bones and help absorb shocks.

For instance, knee joints let you swing, bend, and turn the long bones below them while absorbing the mechanical force of your weight. They also are easily injured. Abruptly twist a knee joint too far and you *strain* it. Tear its ligaments or tendons and you *sprain* it. Moving the wrong way might dislocate the attached bones. If a blow to the knee during football or another collision sport severs its ligaments, the ligaments must be surgically put together within ten days. Phagocytic white blood cells patrol fluid that bathes knee joints, where they clean up debris from daily wear and tear. Their action can turn torn ligaments to mush.

Joint inflammation and degenerative disorders are collectively called arthritis. In *osteoarthritis*, cartilage at freely movable joints wears away. Joints in fingers, knees, hips, and the backbone are affected most. With *rheumatoid arthritis*, joint membranes become inflamed. They thicken, cartilage degenerates, and bone deposits accumulate as a result of an autoimmune response. A bacterial or viral infection might be the trigger for the disorder, but genetics and smoking probably increase the risk. Rheumatoid arthritis can develop at any age, although symptoms usually appear before age fifty.

Bones are collagen-rich, mineralized organs that function in movement, support, protection, storage of calcium and other minerals, and blood cell formation.

Balancing ongoing calcium deposits with the withdrawals maintains bone mass and calcium concentrations in blood.

37.5 Skeletal–Muscular Systems

LINKS TO SECTIONS 17.4, 23.11, 33.3

Skeletal muscles are the functional partners of bones. They contract (shorten) in response to stimulation, then passively return to their resting position (lengthen).

Skeletal muscle cells are not your typical cells. Before they can differentiate and mature in embryos, groups of them fuse together into one multinucleated **muscle fiber**. Bundles of muscle fibers are sheathed in dense connective tissue, which extends past them. The entire array is a skeletal muscle. The extension, a cordlike or straplike **tendon**, attaches it to bone.

Most of these attachment sites are like a gearshift. *They act as a lever system, in which a rigid rod is attached to a fixed point and moves about it*. Muscles connect to bones (rigid rods) near a joint (fixed point). When they contract, they transmit force that makes bones move.

The skeletal muscles also interact with one another. Some work in pairs or groups in ways that promote a movement. Others work in opposition; the action of one opposes or reverses the action of another. As an example, extend your right arm forward, then place your left hand over the biceps in your upper right arm and slowly bend the elbow, as in Figure 37.11. Feel the biceps contract? Even when a biceps contracts just a bit, it causes a large motion of the bone connected to it. This is the case for most leverlike arrangements, as the frog in Figure 37.12 obligingly illustrates.

Bear in mind, only *skeletal* muscle is the functional partner of bone. As you read in Section 33.3, smooth muscle is mainly a component of soft internal organs, such as the stomach. Cardiac muscle forms only in the heart wall. Later chapters will consider the structure and function of smooth muscle and cardiac muscle.

The human body has close to 700 skeletal muscles, some near the surface, others deep in the body wall (Figure 37.13). The trunk has muscles of the thorax, backbone, abdominal wall, and pelvic cavity. Other muscles attach to the upper and lower limb bones. Later chapters describe how skeletal muscles assist in respiration and circulation. For now, turn to some of the mechanisms that bring about their contraction.

Cordlike or straplike tendons of dense connective tissue attach skeletal muscles to bones. Only skeletal muscles transmit contractile force to bones, and many groups function in opposition to one another.

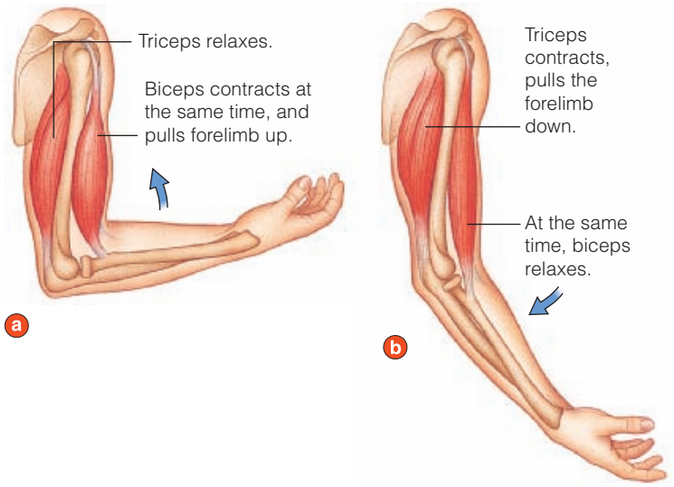
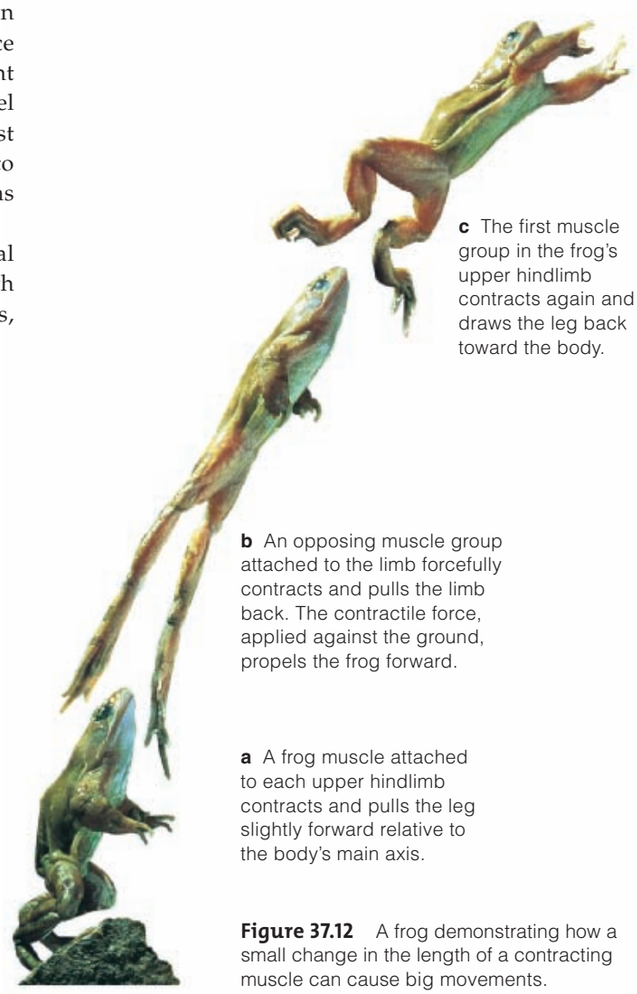


Figure 37.11 ***Animated!*** Two opposing muscle groups in human arms. (**a**) When the triceps relaxes and its opposing partner (biceps) contracts, the elbow joint flexes and the forearm is pulled upward. (**b**) When the triceps contracts and the biceps relaxes, the forearm is extended down.



c The first muscle group in the frog's upper hindlimb contracts again and draws the leg back toward the body.

b An opposing muscle group attached to the limb forcefully contracts and pulls the limb back. The contractile force, applied against the ground, propels the frog forward.

a A frog muscle attached to each upper hindlimb contracts and pulls the leg slightly forward relative to the body's main axis.

Figure 37.12 A frog demonstrating how a small change in the length of a contracting muscle can cause big movements.

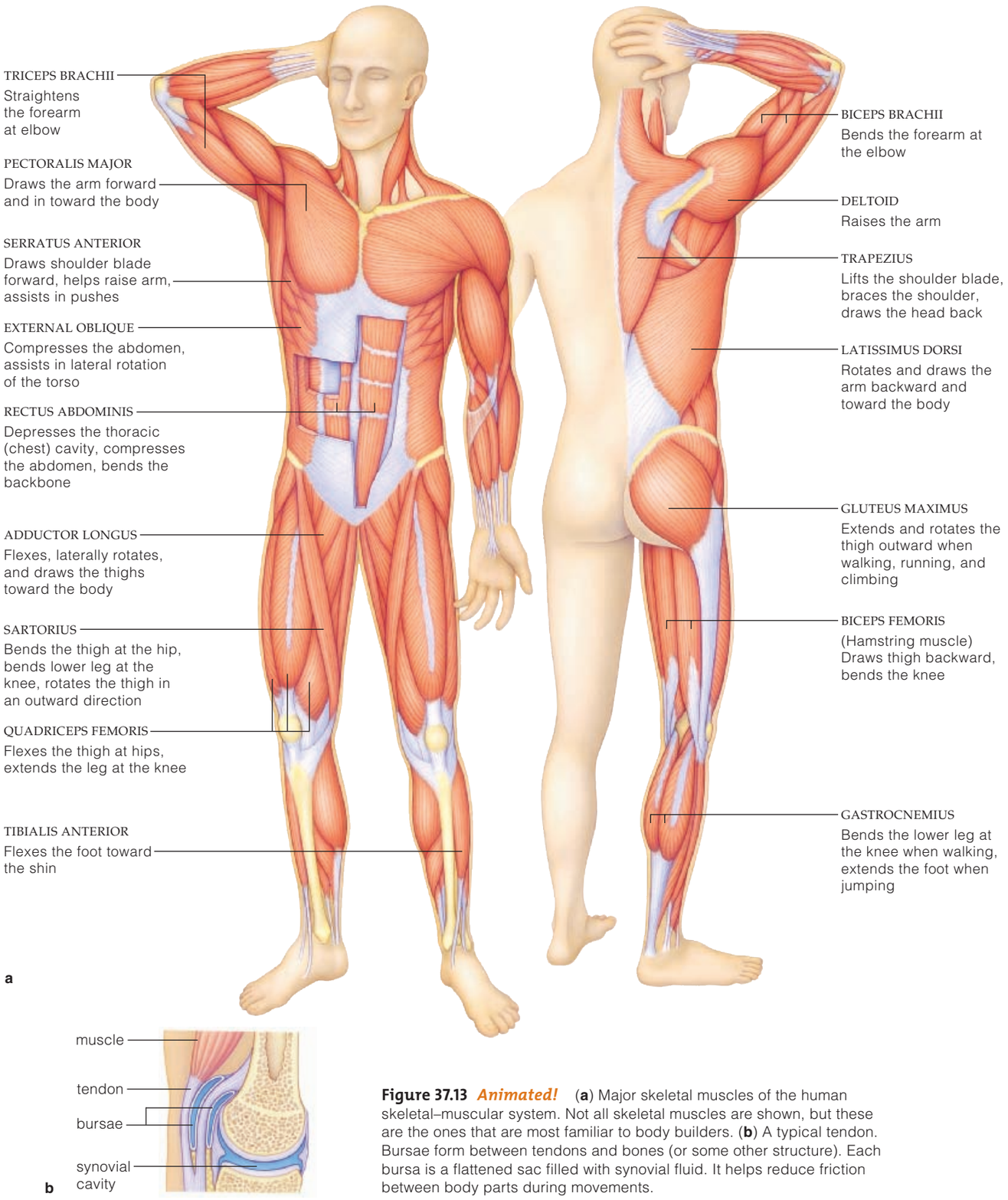


Figure 37.13 ***Animated!*** (**a**) Major skeletal muscles of the human skeletal–muscular system. Not all skeletal muscles are shown, but these are the ones that are most familiar to body builders. (**b**) A typical tendon. Bursae form between tendons and bones (or some other structure). Each bursa is a flattened sac filled with synovial fluid. It helps reduce friction between body parts during movements.

37.6 How Does Skeletal Muscle Contract?

LINKS TO SECTIONS 4.10, 6.2, 33.3

Bones of a dancer or any other human in motion move in some direction when skeletal muscles attached to them shorten. A muscle shortens when its muscle fibers, and individual contractile units inside the fibers, shorten.

FINE STRUCTURE OF SKELETAL MUSCLE

A skeletal muscle's function arises from its internal organization. Long, slender muscle fibers run parallel with the muscle's long axis. The fibers are packed with **myofibrils**, each a bundle of contractile filaments that run from one end of the fiber to the other. Staining the myofibrils for microscopy reveals repeats of light-to-dark crossbands along their entire length. The bands give the muscle fiber a striated, or striped, appearance (Figures 37.1 and 37.14).

The banding corresponds to units of contraction, or **sarcomeres**, that are repeated one after another along the length of the myofibril. Each end of a sarcomere is anchored to its neighbor at a Z band, a dense mesh of cytoskeletal elements (Figure 37.14*c*).

Parallel arrays of thin filaments extend from both Z bands toward the sarcomere center but stop short of it. The thin filaments are mainly repeating units of the globular protein **actin** (Section 4.10 and Figure 37.14*d*).

An array of thicker filaments starts at the center of the sarcomere. It runs parallel with the thin filaments but does not extend all the way to the Z bands (Figure 37.15*a*). The thick filaments consist of **myosin**, a motor protein with a clublike head (Section 4.10 and Figure 37.14*e*). Each myosin head is positioned no more than a few nanometers away from a thin actin filament.

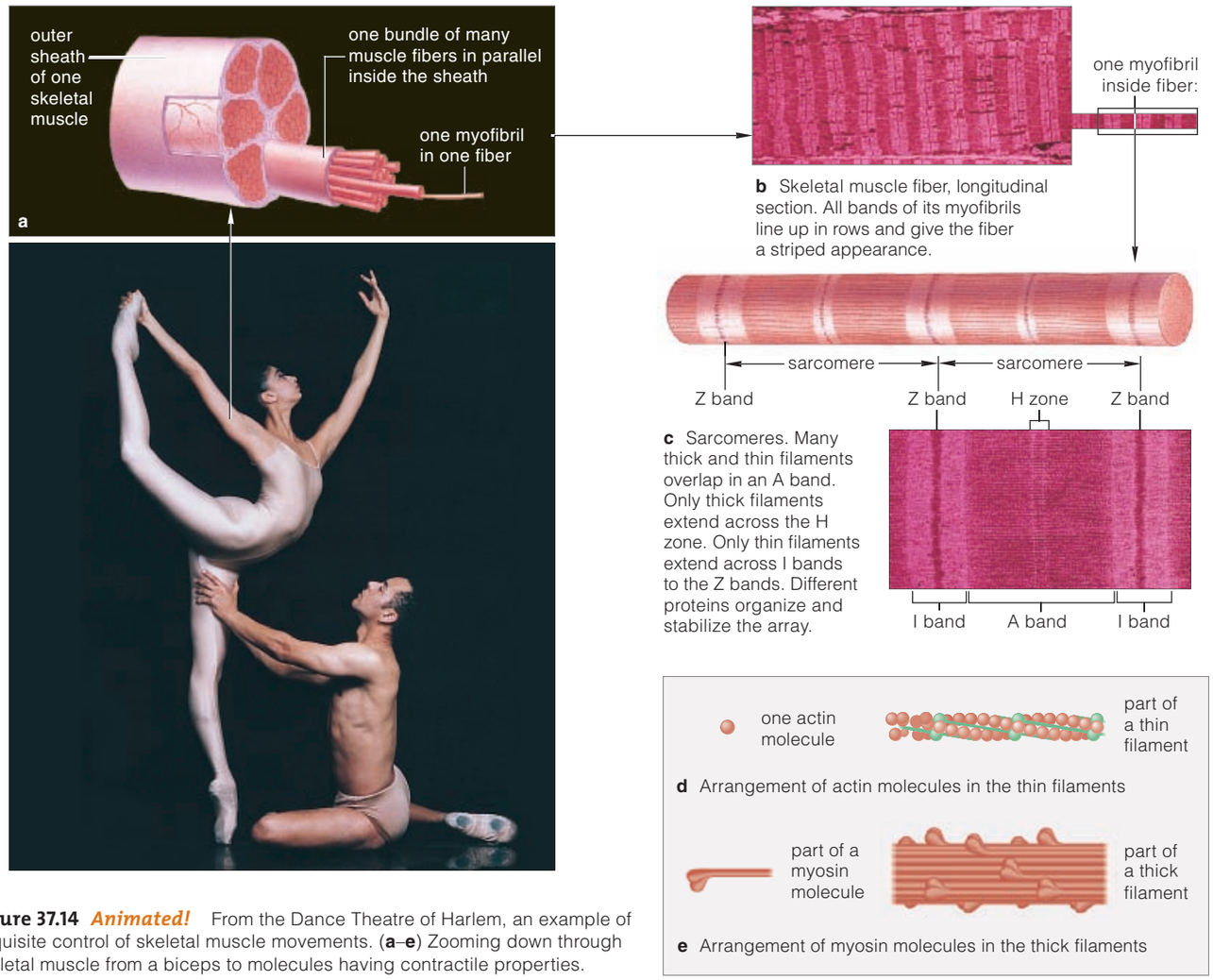


Figure 37.14 ***Animated!*** From the Dance Theatre of Harlem, an example of exquisite control of skeletal muscle movements. (**a**–**e**) Zooming down through skeletal muscle from a biceps to molecules having contractile properties.

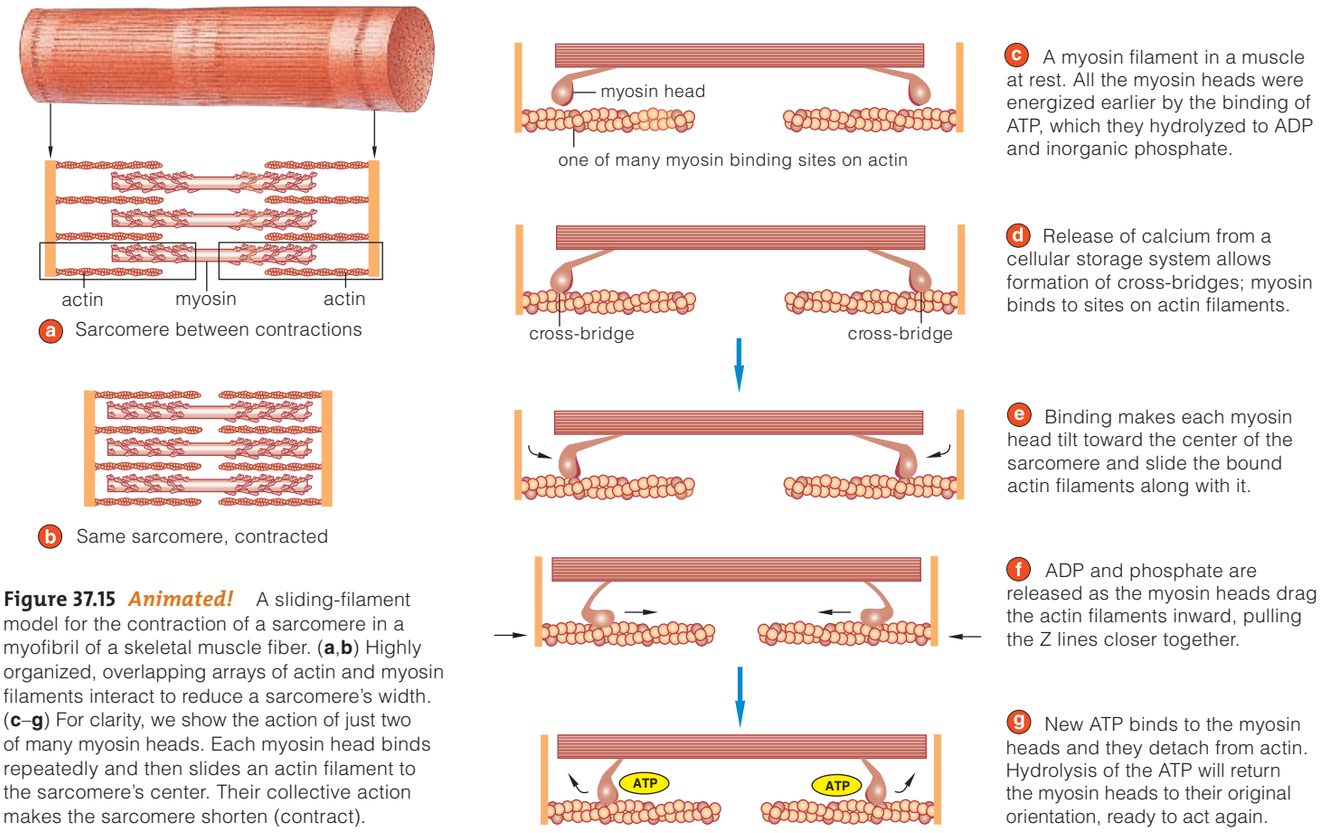


Figure 37.15 ***Animated!*** A sliding-filament model for the contraction of a sarcomere in a myofibril of a skeletal muscle fiber. (**a**,**b**) Highly organized, overlapping arrays of actin and myosin filaments interact to reduce a sarcomere's width. (**c**–**g**) For clarity, we show the action of just two of many myosin heads. Each myosin head binds repeatedly and then slides an actin filament to the sarcomere's center. Their collective action makes the sarcomere shorten (contract).

Muscle fibers, myofibrils, thin filaments, and thick filaments have the same orientation; they run parallel with the muscle's long axis. What is the point? *The repetitive orientation focuses the force of contraction, so that all sarcomeres in all fibers of a muscle work together to pull a bone in the same direction.*

SLIDING-FILAMENT MODEL FOR CONTRACTION

How do sarcomeres, and a skeletal or cardiac muscle, shorten? By a **sliding-filament model**, myosin heads move actin filaments toward the sarcomere's center by short, repetitive, ATP-driven power strokes. The myosin filaments stay in place, but the actin filaments slide past them. Both Z bands, which are connected to the actin filaments, are pulled inward with them, and that shortens the sarcomere (Figure 37.15*b*).

Each myosin head latches on to one binding site after another along an actin filament. Part of the head is enzymatic. It catalyzes a phosphate-group transfer from ATP, which is the energy that drives contraction. As you will read in the next section, myosin forms a cross-bridge to actin when the local concentration of calcium ions rises and a binding site for the myosin's head is exposed. Once the head binds, it tilts toward the sarcomere's center, and the actin slides along with it. When another ATP boost breaks the grip on actin, the myosin head reverts to its resting position (Figure 37.15*c*–*e*). Depending on the calcium and ATP levels, the myosin head may attach to the next binding site on the actin filament, tilt in another stroke, and so on. It takes hundreds of myosin heads, all performing a rapid series of short strokes along the actin filaments, to bring about a single contraction of a sarcomere.

Sarcomeres are the basic units of contraction in skeletal (and cardiac) muscle. The parallel orientation of all of the muscle components focuses contractile force on a bone that is to be moved.

By energy-driven interactions between myosin and actin filaments, the many sarcomeres of a muscle cell shorten and collectively bring about the muscle's contraction.

37.7 From Signal to Response: A Closer Look at Contraction

LINKS TO SECTIONS 4.6, 5.4, 34.2, 34.4

Section 34.4 introduced you to the idea that acetylcholine (ACh) can stimulate muscle contraction. Here is a closer look at what goes on after the nervous system commands a motor neuron to release this neurotransmitter into the synaptic cleft of a neuromuscular junction.

All cells at rest show a voltage difference across their plasma membrane, in that interstitial fluid has a slight positive charge relative to the cytoplasm next to the membrane. Only in muscle fibers, neurons, and some other *excitable* cells can the voltage difference reverse abruptly and briefly in response to stimulation. Such a reversal is an **action potential**.

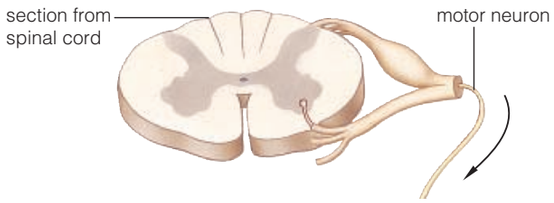
As Section 34.2 explains, an action potential arises as certain ions flow across the plasma membrane in an ever accelerating way. The disturbance self-propagates from the point of stimulation without diminishing.

Suppose signals from the nervous system strongly spread rapidly from the stimulation site, then along T tubules. The small tubes are extensions of the plasma membrane. Actin filaments in sarcomeres are attached to them, and so is a system of membranous chambers. That system, the **sarcoplasmic reticulum**, wraps lacily around the myofibrils. It takes up, stores, and releases calcium ions in controlled ways (Figure 37.16).

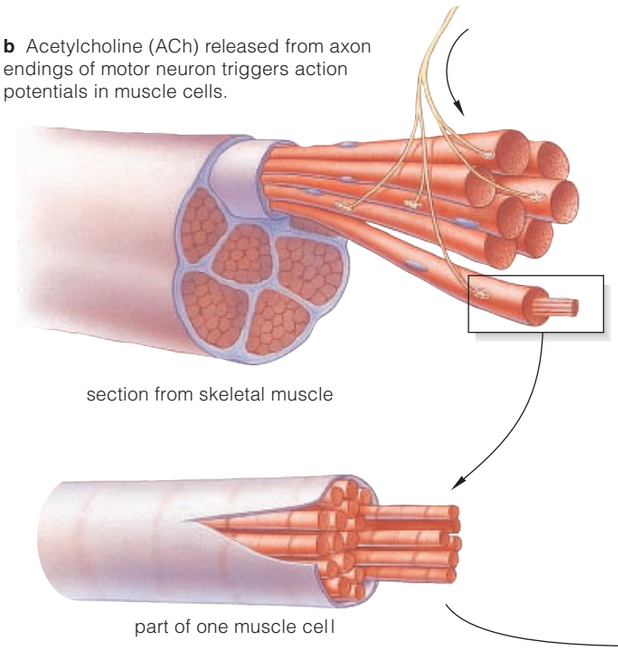
The arrival of action potentials causes calcium ions to flow out of the chambers. The released ions diffuse into the myofibrils and reach actin filaments. The actin binding sites for myosin heads are blocked in resting muscle fibers, but calcium ions clear them.

Figure 37.17 shows a cross-bridge binding site that is blocked by proteins. The proteins, tropomyosin and troponin, are positioned in or near grooves at the actin filament surface. When the calcium level is low, they are joined so tightly that the tropomyosin is forced out of the groove. It moves every so slightly, but that is enough to block the cross-bridge binding site.

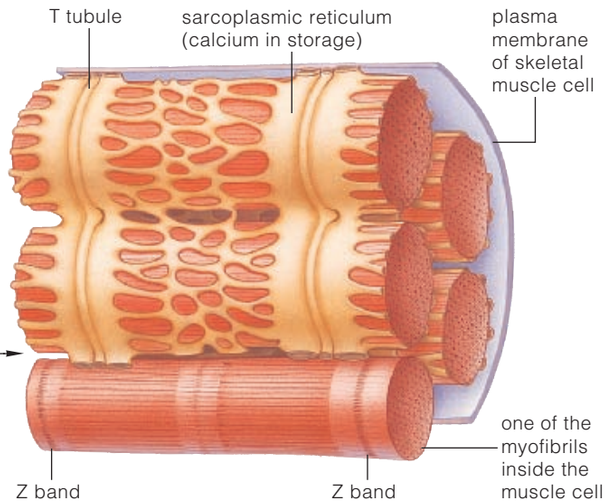
When signals from the nervous system trigger an inflow of calcium into the sarcomere, enough calcium ions bind with troponin to cause its shape to change. The troponin now has a different molecular grip on the tropomyosin filament, which is free to slip back into the groove. The binding site is now exposed.



a Messages generated in nervous system trigger action potentials in a motor neuron extending from the spinal cord to a skeletal muscle.



c Action potentials spread along muscle cell plasma membrane and reach the sarcoplasmic reticulum.



d Action potentials trigger release of calcium ions from sarcoplasmic reticulum threading among myofibrils.

Figure 37.16 Pathway by which the nervous system stimulates or inhibits skeletal muscle contraction. The plasma membrane of each muscle cell encloses myofibrils. Tubular extensions of it, the T tubules, have anchor points for actin filaments at the Z bands of all sarcomeres inside.

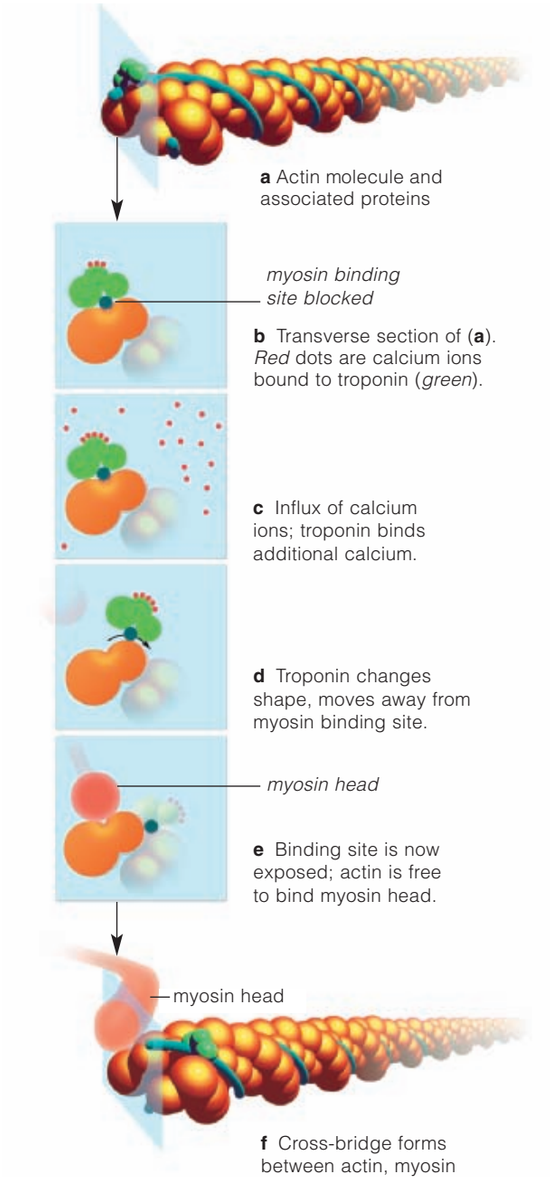


Figure 37.17 ***Animated!*** The interactions among actin, tropomyosin, and troponins in a skeletal muscle cell.

When contraction ends, membrane proteins actively transport the calcium ions back into the sarcoplasmic reticulum. The muscle fiber is ready for another signal.

Certain commands from the nervous system initiate action potentials in muscle cells. These action potentials are signals for cross-bridge formation, hence for contraction.

37.8 Energy for Contraction

When a muscle fiber at rest is called upon to contract, the demand for phosphate donations from ATP increases 20 to 100 times. Where does all the energy come from?

LINKS TO SECTIONS 6.2, 8.5, 8.6, 36.6

A muscle fiber has only a small supply of ATP when it starts contracting. It can produce more very quickly by transferring one phosphate group from **creatine phosphate** to ADP (Figure 37.18). It has about six times as much creatine phosphate as ATP, but the supply only fuels 15 seconds or so of contraction. After the call to contract ends, the supply of creatine phosphate is restored; ATP donates phosphate to creatine.

During prolonged but moderate exercise, aerobic respiration typically provides most of the energy for contraction. In the first five to ten minutes of activity, a muscle fiber converts stored glycogen to glucose, the starting substrate. Glucose and fatty acid arriving from the bloodstream sustain the next half hour or so of activity. Fatty acids become the main energy source for further contraction (Section 8.6).

Not all fuel is burned in aerobic respiration. Even in a resting muscle, some pyruvate from glycolysis is converted to lactate by a fermentation route. Lactate production rises with exercise. Remember, not much ATP forms by this anaerobic pathway. But lactate is a transportable form of energy. Skeletal muscle fibers can give it up, and the bloodstream can deliver it to other muscle fibers or tissues. There, it can be used as energy or stored as glycogen (Sections 8.5 and 36.6).

Muscle contraction requires high concentrations of ATP. Muscle fibers make ATP by dephosphorylation of creatine phosphate, by aerobic respiration, and by glycolysis.

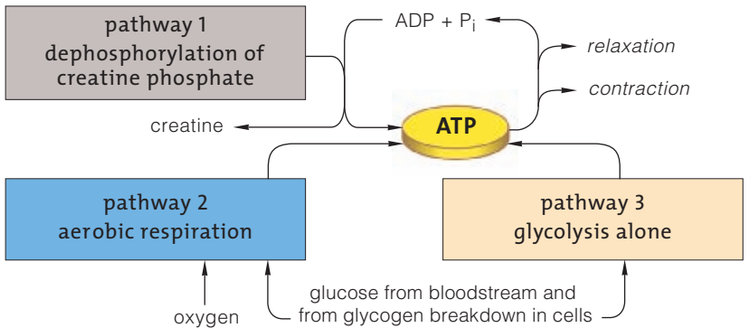


Figure 37.18 ***Animated!*** Three metabolic pathways by which ATP forms inside muscles in response to the demands of physical exercise.

37.9 Properties of Whole Muscles

LINKS TO SECTIONS 12.7, 34.2, 34.4

Muscle contractions vary in duration and intensity. Exercise can strengthen or fatigue muscles. Age and certain genetic disorders can weaken them.

TYPES OF CONTRACTIONS

A motor neuron has many axon endings that synapse on different fibers in each muscle (Sections 34.2 and 34.4). A motor neuron and all of the muscle fibers that are functionally connected to it constitute one **motor unit**. When a motor neuron is briefly stimulated, all fibers in the motor unit contract. A fleeting contractile force is generated that lasts just a few milliseconds. That contraction is a **muscle twitch** (Figure 37.19*a*).

Applying a new stimulus before a response ends makes a muscle twitch again. Repeatedly stimulating a motor unit during a short interval makes all of the twitches run together, and it results in the sustained contraction called **tetanus**. This contraction generates three or four times the force of a single twitch. Figure 37.19*c* shows a recording of tetanic contraction.

Muscle tension is the mechanical force exerted by a muscle on an object. The number of fibers recruited into action influences it. Opposing this force is a load, either the weight of an object or gravity's pull on the muscle. Only when muscle tension exceeds opposing forces does a stimulated muscle shorten. *Isotonically* contracting muscles shorten and move a load (Figure 37.20*a*). *Isometrically* contracting muscles do develop tension, as when you attempt to lift something that is too heavy, but they cannot shorten (Figure 37.20*b*).

WHAT IS MUSCLE FATIGUE?

When ongoing, strong stimulation keeps a muscle in a state of tetanic contraction, muscle fatigue follows. **Muscle fatigue** is a decrease in a muscle's capacity to generate force, a decline in tension.

After a few minutes of rest, a fatigued muscle will contract again in response to stimulation. The extent of recovery depends largely upon how long and how often the muscle was stimulated previously. Muscles trained by a pattern of brief, intense exercise fatigue and recover fast. This happens during weight lifting. Muscles used in prolonged, moderate exercise fatigue slowly. They take longer to recover, often up to a day. Exactly what causes muscle fatigue is unknown, but glycogen depletion is one factor.

A *muscle cramp* is an abrupt involuntary, and often painful contraction that resists release. Any skeletal muscle can cramp, but calf and thigh muscles cramp most often. Motion usually aggravates the cramping; gentle stretching, massage, and heat often may relieve it. To avoid muscle cramps, stretch muscles regularly and avoid overexertion and dehydration.

WHAT ARE MUSCULAR DYSTROPHIES?

Muscular dystrophies are a class of genetic disorders in which muscles progressively weaken and degenerate. Duchenne muscular dystrophy is the most common form among children. Myotonic muscular dystrophy is the most common form among adults.

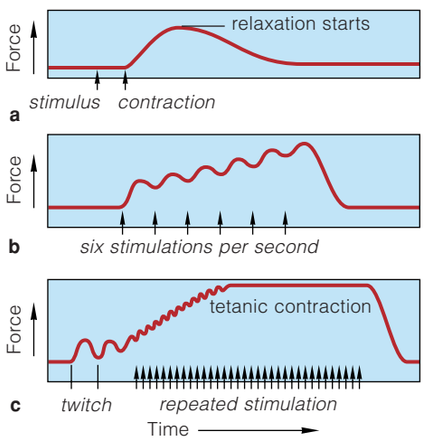


Figure 37.19 ***Animated!*** Three recordings of twitches in muscles exposed to artificial stimulation of different frequencies. (**a**) A single twitch. (**b**) Summation of twitches after six stimulations per second. (**c**) Tetanic contraction after twenty stimulations per second.

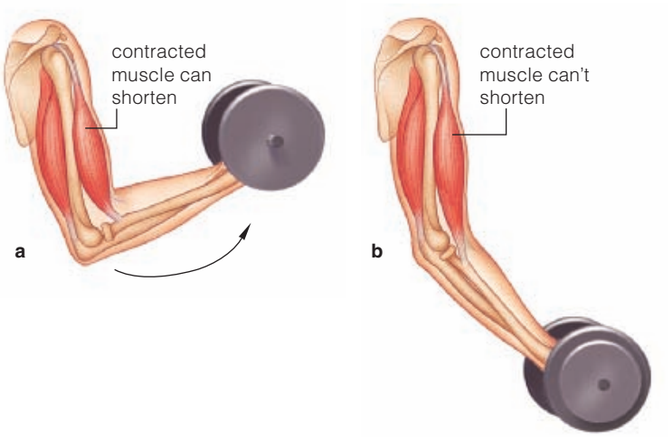


Figure 37.20 (**a**) Isotonic contraction. The load is less than a muscle's peak capacity to contract. The muscle can contract, shorten, and lift the load. (**b**) Isometric contraction. The load exceeds a muscle's peak capacity, so the muscle contracts but cannot shorten.

A single mutant gene on the X chromosome causes Duchenne muscular dystrophy. The gene encodes one of a group of proteins found in extensions of a muscle fiber's plasma membrane. The protein group attaches the plasma membrane to actin filaments at Z bands of sarcomeres. Unless actin binds firmly to proteins at the bands, sarcomeres cannot contract. Like all other X-linked disorders (Section 12.7), muscular dystrophy primarily affects males, at a frequency of about 1 in 3,500. Generally, affected individuals are confined to a wheelchair by their teens. They can expect to survive only into their twenties. There is no cure.

MUSCLES, EXERCISE, AND AGING

No matter how much you exercise, you will not make any more muscle fibers. Existing ones just get bigger, more active, and more resistant to fatigue. Think of *aerobic exercise*—not intense, but long in duration. It increases the number of mitochondria in muscles and the number of blood capillaries servicing them. These physiological changes improve endurance.

Strength training (brief but intense exercise, such as weight lifting) causes muscle fibers to thicken. Also, it stimulates the synthesis of certain enzymes necessary for glycolysis. Bulging muscles do form, but they do not have much endurance. They fatigue fast.

As people age, the number and size of their muscle fibers decline. Tendons that attach muscles to bone stiffen and are more likely to tear. Older people may exercise intensely for long periods, but their muscle mass no longer can increase as much. Even so, aerobic exercise does improve blood circulation, and modest strength training can slow the loss of muscle tissue.

As a beneficial side effect, aerobic exercise among the middle-aged and elderly can lift major depression as well as drugs do. It may improve memory and the capacity to plan and carry out complex tasks. Also, exercise increases blood flow and helps nerves make new connections. In short, exercise is good for more than muscles. It is good for the brain.

Cross-bridges in sarcomeres collectively exert a mechanical force called muscle tension. Muscles shorten only if this tension exceeds other opposing forces.

Muscle tension is related to the number of fibers that have been stimulated. A signal from a motor neuron stimulates all the fibers of the same motor unit.

Exercise, aging, and some genetic disorders influence the capacity of muscles to generate tension.

37.10 Oh, *Clostridium!*

FOCUS ON HEALTH

We conclude this chapter with a look at two bacterial infections that interfere with how muscles work.

LINKS TO SECTIONS 21.4, 34.4, 34.5

In Section 21.4, you read about *Clostridium botulinum*. This anaerobic soil bacterium can cause disease in humans. Food that has been stored in unsterilized cans or jars may contain its endospores. When the spores germinate, they start making an odorless botulinum toxin.

Now think about what happens in a person who ate toxin-tainted food. The poison enters motor neurons and stops the release of acetylcholine (ACh). Muscles cannot contract without this signal from the nervous system. The muscles become more and more flaccid and paralyzed. These are symptoms of *botulism*, a type of food poisoning. Affected people must be treated with antitoxin as quickly as possible, especially to safeguard their cardiac muscle as well as skeletal muscles with roles in breathing.

A related bacterium, *C. tetani*, lives in the gut of horses, cattle, and other grazing animals, and many people. Its endospores can survive for years in soil—manure-rich soils especially—as long as sunlight and oxygen do not reach them. The endospores resist strong disinfectants, heat, and boiling water. When they enter the body through a deep puncture or cut, they can germinate in necrotic (dead) tissues. Bacterial cells do not spread away from anaerobic, dead tissues. However, they make a toxin that blood or nerves deliver to the spinal cord and brain.

In the spinal cord, the toxin blocks the release of GABA and glycine from certain neurons. These neurotransmitters exert inhibitory control over motor neurons. In the absence of the controls, nothing can put the brakes on signals to contract, and so symptoms of the disease *tetanus* begin.

After four to ten days, the overstimulated muscles stiffen and cannot be released from contraction; they go into spasm. Prolonged, spastic paralysis follows. The fists and the jaws may remain clenched; lockjaw is a common name for the disease. The backbone may become locked in an abnormally arching curve. When respiratory and cardiac muscles become paralyzed, death nearly always follows.

Vaccines were not available for soldiers of early wars, when *C. tetani* lurked in dead cavalry horses and manure on battlefields (Figure 37.21). Vaccines have all but eradicated tetanus in the United States. Worldwide, the annual death toll is over 200,000, mostly due to unsanitary childbirths.

Figure 37.21 Painting of a casualty of a contaminated battle wound as he lay dying of tetanus in a military hospital.

Summary

Sections 37.1, 37.2 Various kinds of contractile cells occur in different animal groups. They all evolved from epithelial cells that contained filaments of contractile proteins—actin and myosin. Some evolved into muscle cells when they detached from epithelium and sank into connective tissue below it. In vertebrate muscles, muscle fibers are bundled in connective tissue sheaths.

Muscles cannot act alone. The force of contraction must be applied to skeletal elements to move the body or parts of it. Nearly all animals have a hydrostatic skeleton, an exoskeleton, or an endoskeleton.

In a hydrostatic skeleton, fluid confined inside a body chamber accepts the force of contraction. Sea anemones and earthworms have this type of skeleton.

Muscles and often hydraulic pressure interact with exoskeletons. Arthropods have this type of skeleton.

An endoskeleton is inside the body. In echinoderms, it consists of honeycombed ossicles inside the dermis. In vertebrates, the endoskeleton consists of cartilage or of bones and restricted regions of cartilage.

Biology Now

Learn how a fly moves its wings with the animation on BiologyNow.

Section 37.3 Humans, like other land vertebrates, have an endoskeleton with skull bones, a pelvic girdle, a pectoral girdle, and paired limbs. The move of ancestral vertebrates from water onto land involved modifications to fins, which evolved into the four limbs of amphibious tetrapods. Other modifications occurred in response to the challenges imposed on a body deprived of water's buoyancy. Benefits as well as costs were incurred when humans became upright, two-legged walkers.

Biology Now

Explore the vertebrate skeleton with the animation on BiologyNow.

Section 37.4 Bones are collagen-rich, mineralized organs. They function in mineral storage, movement, and protection and support of soft organs. Blood cells form in bones having red marrow. Ongoing mineral deposits and removals help maintain blood levels of mineral ions and also adjust bone strength. Parathyroid hormones induce bone breakdown and calcium release. Ligaments and cartilage hold bones together at joints.

Biology Now

Look inside a human femur with the animation on BiologyNow.

Section 37.5 Before they differentiate and mature in embryos, groups of muscle cells fuse together into one multinucleated muscle fiber. A skeletal muscle consists of bundles of muscle fibers sheathed in dense connective tissue that extends past them. The extensions, tendons, attach skeletal muscles to bones.

When skeletal muscles contract, they transmit force that moves bones. Many muscles work as opposing pairs. The internal organization of skeletal and cardiac muscle promotes strong, directional contraction.

Biology Now

Learn about the location and action of human skeletal muscles with the animation on BiologyNow.

Sections 37.6, 37.7 A skeletal muscle fiber runs parallel with the long axis of a muscle and extends to both ends of it. This fiber contains many myofibrils, or transversely banded filaments. Each myofibril is divided repeatedly along its length into sarcomeres, the basic unit of contraction in skeletal and cardiac muscle.

Each sarcomere has two arrays of actin filaments that are anchored to Z bands, or dense arrays of cytoskeletal elements that define the width of the sarcomere. Each sarcomere has an array of myosin filaments at its center that overlap with the actin filaments. All of the filaments run parallel with the muscle's long axis.

Cumulatively, the repetitive orientations focus the force of contraction, so that all sarcomeres in a muscle work together to pull a bone in the same direction.

Motor neurons relay signals from the nervous system to muscle fibers. When they release ACh, they call for action potentials in the sarcoplasmic reticulum. This is a membrane system of calcium-storing chambers around the fibers. Calcium floods out, and binding sites on actin filaments become exposed to the myosin heads close by. The sites are blocked in muscles at rest.

By a sliding-filament model, the myosin heads move actin filaments toward the sarcomere's center by short, repetitive, ATP-driven power strokes. Myosin filaments stay in place, but actin filaments slide past them. The Z bands, being connected to the actin filaments, are pulled inward with them, which shortens the sarcomere.

Biology Now

Investigate the molecular structure and function of human skeletal muscles with the animation on BiologyNow.

See how the nervous system controls skeletal muscle contraction with the animation on BiologyNow.

Section 37.8 Muscle fibers can get ATP energy for contraction by dephosphorylation of creatine phosphate, aerobic respiration, and glycolysis.

Biology Now

Compare different sources of energy for muscle contraction with the animation on BiologyNow.

Section 37.9 Muscle tension is a mechanical force caused by cross-bridge formation. A muscle shortens when muscle tension exceeds an opposing load. A motor neuron and all muscle fibers that form junctions with its endings are a motor unit. Repeated stimulation of a motor unit results in tetanus, or sustained contraction.

Biology Now

Observe what happens when a muscle is stimulated with the animation on BiologyNow.

Section 37.10 Bacterial toxins can interfere with nervous signals that stimulate muscle contraction.

Self-Quiz

Answers in Appendix II

1. Bones are _____ .
   a. mineral reservoirs
   b. skeletal muscle's partners
   c. sites where blood cells form (some bones only)
   d. all of the above

2. Bones move when _____ muscles contract.
   a. cardiac
   b. skeletal
   c. smooth
   d. all of the above

3. The _____ is the basic unit of contraction.
   a. osteoblast
   b. sarcomere
   c. muscle fiber
   d. myosin filament

4. In sarcomeres, phosphate-group transfers from ATP activate _____ .
   a. actin b. myosin c. both d. neither

5. A sarcomere shortens when _____ .
   a. thick filaments shorten
   b. thin filaments shorten
   c. both thick and thin filaments shorten
   d. none of the above

6. ATP for muscle contraction can be formed by _____ .
   a. aerobic respiration
   b. glycolysis
   c. creatine phosphate breakdown
   d. all of the above

7. Match the words with their defining feature.
   ____ osteoblast — a. actin's partner
   ____ muscle twitch — b. all in the hands
   ____ muscle tension — c. blood cell production
   ____ joint — d. decline in tension
   ____ myosin — e. bone-forming cell
   ____ red marrow — f. motor unit response
   ____ metacarpals — g. force exerted by cross-bridges
   ____ myofibrils — h. area of contact between bones
   ____ muscle fatigue — i. muscle fiber's threadlike parts

Additional questions are available on Biology Now™

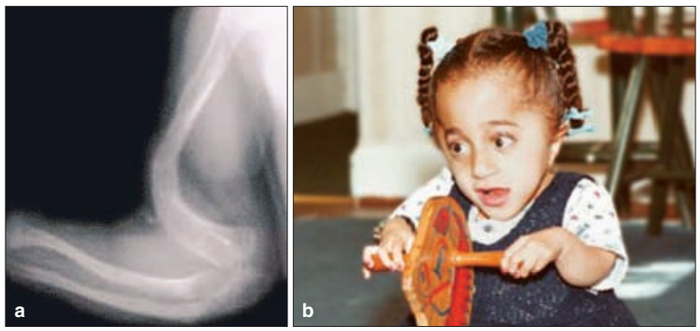


Figure 37.22 (**a**) X-ray of an arm bone deformed by *osteogenesis imperfecta* (OI). (**b**) Tiffany is affected by OI. She was born with multiple fractures in her arms and legs. By age six, she had undergone surgery to correct more than 200 bone fractures and to place steel rods in her legs. Every three months she receives intravenous infusions of an experimental drug that may help strengthen her bones.

Figure 37.23 Polar huskies warming up for an Arctic crossing.

Critical Thinking

1. The genetic disorder *osteogenesis imperfecta* (OI) arises from a mutant form of collagen. As bones develop, this protein forms a scaffold for the deposition of mineralized bone tissue. The scaffold forms improperly in children with OI, who end up with fragile, brittle, easily broken bones (Figure 37.22). OI is rare; most doctors never come across it. Parents are often accused of child abuse when they take an OI child with multiple bone breaks to an emergency room. Prepare a handout about OI that could be used to alert emergency room staffs to the condition. You may wish to start with resources on the Osteogenesis Imperfecta Foundation web site at www.oif.org.

2. Lydia is training for a marathon. While training, she plans to take creatine supplements because she heard that they give athletes extra energy. She asks you whether you think this is a good idea. What is your response?

3. Compared to most people, long-distance runners have far more mitochondria in skeletal muscles. In sprinters, skeletal muscle fibers have more of the enzymes required for glycolysis but not as many mitochondria. Suggest why.

4. In adults, the nuclei of muscle fibers are stuck in G1 of the cell cycle and cannot undergo division. Lots of adults increase the size of muscles by lifting weights. What accounts for the increased bulk of the muscles?

5. Botulinum toxin can kill you if you eat it, but some people pay hundreds of dollars to have it injected. These *botox injections* prevent facial muscles from contracting and pulling on the skin in ways that cause wrinkling. Most commonly, botox is injected into muscles between the eyebrows. It makes the eyelids droop for weeks in 3 percent of the patients. Explain why.

6. In 1989, Will Steger and his dogsled team walked on ice for seven months, enduring temperatures of –113°F and a seven-week blizzard. They crossed Antarctica, all 6,023 kilometers (3,741 miles) of it. Steger called his polar huskies the members of the team that worked hardest and pulled all the weight (Figure 37.23). Husky leg bones are sturdy yet lightweight. The forelegs move freely, thanks to a deep but not-too-broad rib cage. By contrast, human legs are adapted for long-distance walking, not long-distance, load-pulling motion. Compared to human legs, what kind of muscle fibers and muscle mass would you expect to find in a husky's hind legs?