Exercise Physiology and Body Systems
After reading this chapter, you should be able to:

1. Explain how skeletal muscle produces force and creates movement in the body
2. Describe the structural anatomy of skeletal muscle, including the different components of the sarcomere and the phases of muscle action
3. List histochemical techniques that are used to identify muscle fiber types
4. List the different muscle fiber types using the myosin ATPase histochemical analysis scheme
5. Discuss the role of muscle fiber types as it relates to different types of athletic performances
6. Discuss the force production capabilities of muscle, including types of muscle actions
7. Explain proprioception in muscle and kinesthetic sense, including the roles of muscle spindles and Golgi tendon organs
8. List the training-related changes in skeletal muscle, including specific training effects related to endurance and resistance exercise on muscle hypertrophy and muscle fiber subtype transition
9. Explain the effects of simultaneous high-intensity endurance and strength training on adaptations specific to each type of training

The ability of skeletal muscle to mediate human performance is impressive. From the ability to lift more than 1,000 pounds (453.5 kg) in the squat lift to the ability to run a marathon in less than 2 hours and 4 minutes, the human species demonstrates a dramatic range of physical performance capabilities (Fig. 4-1). We might ask, “How can such functional variability be possible in a single species?” As we will continue to discover throughout this textbook, there are many physiological functions that contribute to exercise performance. One such contributor is the skeletal muscle system, which is covered in this chapter. The structure and function of skeletal muscle, which is muscle that is attached to a bone at both ends, profoundly affects the ability to perform exercise. Moreover, because of the very close functional relationship between skeletal muscles and nerves (covered in the next chapter), together they are known as the
neuromuscular system, which profoundly influences athletic ability. Thus, different exercise training programs can be designed to favor neuromuscular adaptations for improving strength or endurance. It may be interesting to see how mathematical calculations have tried to predict the limits of human performance, but no doubt the capacity of human performance will always be influenced by a combination of an individual’s genetics, sports equipment, motivation, and exercise training programs.  

To aid in understanding these concepts, this chapter presents the structure of skeletal muscle, sliding filament theory, muscular activity, and the types of muscle action. It also covers muscle fiber types, force production capabilities, and proprioception as applied to kinesthetic sense. Finally, it introduces the classical training adaptations in muscle to endurance and resistance exercise training.

FIGURE 4-1. Examples of exceptional human performance. (A) The elite endurance runner. (B) The elite strength athlete. Each of these athletes brings a specific set of genetic capabilities to their sport. This includes the type and number of muscle fibers they have in their muscles. Elite competitive capabilities require an underlying neuromuscular system that can meet the physiological demands of the sport as evidenced by these two elite performances of running a marathon in just more than 2 hours or squatting several times one’s body mass.

BASIC STRUCTURE OF SKELETAL MUSCLE

Remarkably, despite the dramatic diversity in exercise performance capabilities in humans, each person’s neuromuscular system is similar in its basic structure and function. Every exercise training program will influence to some extent each of the components of muscle function (see Box 4-1). We will now examine the fundamental structures of skeletal muscle and gain some insight as to how muscles produce force and movement.

In order to understand the structure of skeletal muscle, we start with the intact muscle and continue to break it down to smaller and smaller organizational components. These basic organizational components of skeletal muscle structure are shown in Figure 4-2. The intact muscle is connected to bone at each end by tendons, which are bands of tough, fibrous connective tissue. The actions of muscle exerting force through the tendons to move the bones cause human movement. The intact muscle is made up of many

BOX 4-1

Applying Research
Training Specificity

It is important to keep in mind that with exercise training each of the organizational components of muscle, from the myofibrils to the intact muscle, will undergo changes, or adaptations, to meet the specific exercise demands. Furthermore, the forces generated by the muscle will translate into adaptations in tendons and bones. Therefore, the development of optimal exercise training programs is not trivial, as the specificity of the demands placed on muscle results in very specific adaptations or training outcomes. This has become known as the principle of training specificity.
fasciculi. Each fasciculus is a small bundle of muscle fibers, which are long multinucleated cells that generate force when stimulated. Each muscle fiber is made up of myofibrils or the portion of muscle composed of the thin and thick myofilaments called actin and myosin, respectively, which are also known as the “contractile proteins” in muscle.

Connective Tissue

Connective tissue in muscle plays a very important role in helping to stabilize and support the various organizational components of skeletal muscle. When connective tissue is lost due to injury or exercise-induced damage (e.g., microtrauma to muscle resulting from overuse injuries), muscle strength and power are reduced. Connective tissue surrounds muscle at each of its organizational levels, with the epimysium covering the whole muscle, the perimysium covering the bundles of muscle fibers (fasciculi), and the endomysium covering the individual muscle fibers (see Fig. 4-3).

The connective tissue is vital for physical performance for several reasons. First, the muscle’s connective tissue sheaths coalesce to form the tendons at each end of the muscle, helping to ensure that any force generated by the muscle will be transferred through the tendon and ultimately to the bone. Second, the endomysium helps prevent the signal for muscle activation from spreading from one muscle fiber to an adjacent fiber. This is necessary to allow fine control of the activation of specific groups of fibers, allowing the body to specifically control force generation and match it to the task at hand (see Chapter 5). Third, the muscle’s connective tissue sheaths make up the elastic component of muscle, which contributes to force and power production. It has been shown that static stretching right before a strength or power event may in fact reduce the elastic components’ power capability and thus inhibit “explosive” muscle performance (Box 4-2).

The elastic component of connective tissue is a vital contributor to the stretch-shortening cycle, which consists of controlled muscle elongation (eccentric action) followed by a rapid muscle shortening (concentric action). The force produced by the elastic component is analogous to the force involved with the recoil of a rubber band after being stretched and released. However, movements in which the prior eccentric action or elongation of the muscle is not followed immediately by the rapid shortening or concentric muscle action (e.g., starting a vertical jump from the squat position) do not take advantage of this added force production, resulting in a reduced level of performance (Box 4-3). Taking advantage of this connective tissue feature of muscle in a training program (e.g., plyometrics; Box 4-4) can contribute to improved force and power production.

Quick Review

- The organizational structure of muscle is as follows: Whole muscle → fasciculi → muscle fibers → myofibrils → myofilaments (actin, myosin).
- Connective tissue of the muscle is important in that it helps stabilize and support all portions of the muscle from the whole muscle to the muscle fibers.

The Sarcomere

The sarcomere is the smallest or most basic contractile unit of skeletal muscle capable of force production and
shortening. Skeletal muscle is also called striated muscle because the arrangement of protein filaments in the muscle’s sarcomere gives it a striped or striated appearance under a microscope (Fig. 4-4).

At each end of a sarcomere are Z lines. At rest, there are two distinct light areas in each sarcomere: the H zone in the middle of the sarcomere, which contains myosin but not actin, and the I bands located at both ends of the

**Apply Research**

**Think Before You Stretch**

A host of studies have demonstrated that static stretching may be detrimental to force production. It now appears that this loss of function is due to the fact that static stretching may elongate the elastic component in muscle, thereby reducing the recoil forces of the muscle. This may be especially true if the stretching is performed immediately before an event (e.g., high jump). Thus, if maximal power, speed, and even strength can be reduced when static stretching is performed immediately prior to the effort, we should carefully consider when we should stretch. From a practical perspective, prior to an athletic event an individual should perform a dynamic warm-up with low-level cycling or jogging; static stretching right before an event requiring maximal force or power development should be eliminated. Flexibility training should be undertaken well before efforts requiring maximal force development in cool-down periods or at another time so that speed, strength, and power performances will not be negatively affected.

**Apply Research**

**Prove It to Yourself**

You can see the effect of the stretch-shortening cycle by doing a simple experiment. Which movement allows you to jump the highest? First, get into a squat position, hold the position, and jump as high as you can. Next, begin from a standing position and drop into a downward countermovement before you jump as high as you can. Try it. You will feel right away that a jump with a countermovement is higher, and in the laboratory, a difference in power on a force plate can be seen between the two types of jumps.
More to Explore

Plyometric Exercise

Interestingly, plyometric training is actually based on the fundamental principle of the "stretch-shortening cycle," which is eccentric (plyometric) action followed by a concentric (myometric) muscle action; when performed at high intensity, it becomes a potent power training modality. In addition, such movements are used to help prevent injury. It has been shown that the stretch-shortening cycle can contribute up to 20% to 30% of the power in a stretch-shortening-type activity, such as a maximal vertical jump needed for high-jump performances. By performing plyometric training, improvements in speed and power production can be achieved. Plyometrics range from low-intensity (standing hops) to high-intensity drills (drop or depth jumps from different heights).

Examples of plyometric exercises include the following:

- Standing vertical jumps
- Long jumps
- Hops and skips
- Standing hops
- Depth or drop jumps from different heights

Further Readings


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sarcotome, which contain only actin filaments. These two areas appear light in comparison with the A band, which contains overlapping actin and myosin filaments. The A band represents the length of the myosin filaments. The M line, found in the middle of the H zone, is important as its proteins hold the myosin filaments in place.

As the sarcomere shortens, the actin filaments slide over the myosin filaments. This causes the H zone to decrease in size as actin filaments slide into it and give it a darker appearance. The I bands become shorter as the actin and myosin slide over each other, bringing myosin into the I band as the Z lines come closer to the ends of the myosin filaments. When the sarcomere relaxes and returns to its original length, the H zone and I bands return to their original size and appearance, as there is less overlap of myosin and actin. The A band does not change in length during either shortening or lengthening of the sarcomere, indicating that the length of the myosin filaments does not change during the process of shortening and returning to resting length when the fiber relaxes. This is also true of the actin filaments.

Noncontractile Proteins

As we have already discussed, the role of noncontractile proteins is vital for muscle function. Even at the level of the sarcomere, noncontractile proteins are needed to provide the lattice work or structure for the positioning of the actin and myosin protein filaments. The contractile proteins of actin and myosin are secured in a very close proximity by noncontractile proteins (Fig. 4-5). These noncontractile proteins in the sarcomere also contribute to the elastic component of the muscle fiber, as previously discussed. For example, titin, also known as connectin, connects the Z line to the M line in the sarcomere and stabilizes myosin in the longitudinal axis. Titin also limits the range of motion of the sarcomere and therefore contributes to the passive stiffness of muscle, which in turn can affect the force produced by that muscle. Another noncontractile protein, nebulin, which extends from the Z line and is localized to the I band, stabilizes actin by binding with the actin monomers (small molecules that can bond to other monomers and become a chain of molecules or a polymer, such as the actin filament).

Before we can explain how muscle contracts, it is important to understand the basic structures of the actin and myosin filaments as they are found within muscle fibers.

Actin Filament

The actin, or thin filament, is composed of two helices of actin molecules intertwined. The actin filaments are attached to the Z lines and stick out from each Z line toward the middle of the sarcomere. Each actin molecule has an active site on it (Fig. 4-6). The active site is the place where the heads of the myosin crossbridges can bind to the actin filament that is needed to cause shortening of muscle. Wrapped around the actin filament are troponymosin and troponin, two regulatory protein molecules. Troponymosin is a tube-shaped molecule that wraps around the actin filament, fitting into a groove created by the intertwining of the actin molecule helices. Troponin protein complexes are found at regular intervals along the troponymosin molecule. Troponin is made up of three regulatory protein subunits.
**FIGURE 4-4.** The sarcomere is the functional contractile unit of muscle. (A) A graphic depiction of a sarcomere. (B) An ATPase-stained micrograph. The myosin filaments (also called thick filaments) and the actin filaments (also called thin filaments) make up the sarcomere. One complete sarcomere runs from one Z line to the next Z line. When shortening occurs, the myosin and actin filaments slide over each other, causing the two Z lines of a sarcomere to come closer together.

**FIGURE 4-5.** Noncontractile proteins. Noncontractile proteins are named such because they are not involved with the contraction process but hold the contractile proteins in place so that they are in close proximity with each other for optimal myosin–actin binding.
Troponin I (which binds to actin) has an affinity for actin and holds the troponin–tropomyosin complex to the actin molecules. Troponin T (which adheres to tropomyosin) has an affinity for tropomyosin and holds the troponin to the tropomyosin molecule. Troponin C (which binds for calcium) has an affinity for calcium ions, and the binding of calcium ions is the stimulus within the muscle fiber that causes muscle activation, due to its role in causing the active site on the actin molecule to be exposed.

Myosin Filament

For the myosin and actin filaments to slide past each other, their molecular structure must allow them to interact in some way and develop a force that pulls them over each other. Each myosin molecule has a globular head, a hinged pivot point, and a fibrous tail (Fig. 4-7). Crossbridges are made up of two myosin molecules. Thus, when the myosin heads stick out from the myosin filament, notice that each crossbridge has two globular myosin heads. The dual heads of the myosin crossbridge are made up of the enzyme myosin ATPase. The fibrous tails of the myosin molecules making up the crossbridges intertwine to form the myosin filament. The crossbridge is the part of the myosin filament that will interact with actin and develop force to pull the actin filaments over the other myosin filaments. There are different isoforms, or kinds, of myosin ATPase found at the crossbridge. The specific isoform expressed by a fiber in many ways determines the type, and thus contractile characteristics, of that fiber.

Muscle Fiber Types

Skeletal muscle is a heterogeneous mixture of several types of muscle fibers, with each fiber type possessing different metabolic, force, and power capabilities. Several different fiber-type classification systems have been developed over the years (Table 4-1) on the basis of the different histochemical, biochemical, and physical characteristics of the muscle fiber.

Table 4-1. The Primary Muscle Fiber Type Classification Systems

<table>
<thead>
<tr>
<th>Classification System</th>
<th>Theoretical Basis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red and white fibers</td>
<td>Based on fiber color; the more myoglobin (oxygen carrier in a fiber), the darker or redder the color; used in early animal research; the oldest classification system.</td>
</tr>
<tr>
<td>Fast-twitch and slow-twitch</td>
<td>Based on the speed and shape of the muscle twitch with stimulation; fast-twitch fibers have higher rates of force development and a greater fatigue rate.</td>
</tr>
<tr>
<td>Slow oxidative, fast oxidative glycolytic, fast glycolytic</td>
<td>Based on metabolic staining and characteristics of oxidative and glycolytic enzymes.</td>
</tr>
<tr>
<td>Type I and type II</td>
<td>Stability of the enzyme myosin ATPase under different pH conditions; the enzyme myosin ATPase has different forms; some forms result in quicker enzymatic reactions for ATP hydrolysis and thus higher cycling rates for that fiber’s actin–myosin interactions; most commonly used system to type muscle fibers today.</td>
</tr>
</tbody>
</table>
staining method. Recall that myosin ATPase is an enzyme that is found on the globular heads of the myosin cross-bridges. From this assay method, type I and type II muscle fibers and their subtypes are classified on the basis of a histochemical reaction of myosin ATPase with ATP supplied in the staining procedure. Each myosin isoform catalyzes this reaction at a unique rate, resulting in different staining intensities among the different fiber types. Using imaging software, the staining intensity can actually be quantified, and the range of staining intensity can be divided into different categories so that each fiber can be assigned to a specific fiber type based on its reaction with ATP.43

As the isoform of myosin ATPase present is directly related to the speed with which the myosin heads bind to

**Practical Questions from Students**

**Can You Explain What Is Involved with a Muscle Biopsy Procedure?**

In order to fiber type an individual, a biopsy sample must be obtained from the muscle. This has been called the **percutaneous muscle biopsy technique**. In this procedure, the skin area where the biopsy will be obtained is first bathed with a disinfectant. Then, several injections of a local anesthetic using a small-gauge needle and syringe are made around the biopsy site. A scalpel is then used to make a small incision through the skin and epimysium of the muscle from which the biopsy will be obtained. Then, a hollow, stainless steel needle is inserted through the incision and into the muscle and used to obtain about 100 to 400 mg of muscle tissue (typically from a thigh, calf, or arm muscle). A biopsy needle consists of a hollow needle and a plunger that fits inside the needle (see the figure below). The needle has a window that is closed when the plunger is pushed to the end of the needle but open when it is not. The needle is inserted with the window closed. The plunger is then withdrawn slightly, opening the window, and suction is applied with a syringe attached to the back of the needle using plastic tubing. The suction creates a vacuum in the needle, pulling the muscle sample into the needle. The plunger is then pushed to the end of the needle, cutting off the muscle sample. The biopsy needle is withdrawn, and the sample is removed from the needle, orientated, processed, and then frozen. After withdrawing the biopsy needle, the incision is dressed. The muscle sample is then cut (using a cryostat, which is a cutting device called a microtome set in a freezer case that keeps the temperature about –24°C) into consecutive (serial) sections and placed on cover slips for histochemical assay staining to determine the various muscle fiber types. Other variables (e.g., glycogen content of the fibers, receptor numbers, mitochondria, capillaries, other metabolic enzymes) can also be analyzed from serial sections of the biopsy sample.
an actin filament’s active site and swivels to generate force, it provides a functional classification representative of a muscle fiber’s shortening velocity. **Type I fibers** are also termed slow-twitch fibers, meaning that not only do they reach peak force production at a slow rate, but also once achieved, their peak force is low. Yet type I muscle fibers possess a high capacity for oxidative metabolism since they receive a rich blood supply and are endowed with excellent mitochondrial density. As such, type I slow fibers have the greatest aerobic capacity and are recruited first and, therefore, most often. As intensity and/or duration increases, the fast fibers are recruited as needed. If a maximal effort is required (e.g., attempting a maximal lift for one repetition), the nervous system will attempt to recruit all the muscle fibers (both fast and slow) in the working muscles.

The percentage of each of these major types in a given muscle appears to be genetically determined. Although a few muscles in everyone contain a predominance of either fast (e.g., triceps brachii) or slow (e.g., soleus), most muscles in the average person contain approximately a 50–50 mixture. Research has shown that the percentage of these two major fiber types and the percentage area occupied by each are two factors that have an impact on performance. The muscles of elite strength/power athletes tend to have a high percentage of fast fibers, whereas elite endurance athletes tend to have a predominance of slow fibers. These two extremes demonstrate the importance of fiber composition in determining athletic excellence on the two ends of the strength–endurance continuum. Obviously, not everyone will be able to reach an elite level. In addition, other factors such as motivation, pain tolerance, biomechanics, diet, rest, and skill all play a role in separating the very best from the very good.

Although the percentages of the major fiber types appear to be established early in life, significant adaptations to enhance performance can still occur. Regardless of the fiber-type composition, dramatic improvements in performance can occur with training. Specific training regimens can increase force output (increase in the cross-sectional area) or aerobic capacity (quantitative and qualitative changes in metabolic enzyme activity levels) in specific muscles. For example, a strength/power athlete with a predominance of slow fibers is at a disadvantage competing against individuals with a predominance of fast fibers. However, through training, significant increases in the cross-sectional areas of the fast fibers can help to overcome this disadvantage. As such, a muscle containing, for example, 50% fast fibers can undergo hypertrophic changes so that after training the fast fiber population makes up more than 60% to 70% of the total fiber area. Although under extreme conditions (e.g., paralysis, long-term electrical stimulation), muscle fibers do have the capability to transform from slow-to-fast or fast-to-slow, exercise does not appear to be enough of a stimulus. Most research has shown that training is capable of eliciting transformations within the fast fiber population (fast subtype transitions), but not between fast and slow (i.e., a complete transition all the way from fast to slow or slow to fast).

**Figure 4-8. Muscle twitch characteristics.** Fast-twitch (type II) fibers have a more rapid force production, produce higher amounts of force, and relax more rapidly than slow-twitch (type I) fibers.
(or type II) fibers do not display an abundance of mitochondria or a generous blood supply, resulting in a tendency to fatigue easily. Characteristics of type I and type II muscle fibers are overviewed in Table 4-2. In addition to those major characteristics, it has been shown that type I and type II muscle fibers have subtypes, so a continuum of muscle fiber types exists within each fiber type. This continuum, and how the muscle fiber types are distinguished, will be described next.

**Myosin ATPase Histochemical Analysis**
The analysis used to differentiate among different muscle fiber subtypes involves a histochemical staining procedure that makes each subtype stain at a slightly different intensity, resulting in a unique shade of gray. To begin the process, a thin cross-section of muscle is obtained from the biopsy sample and is placed into different pH conditions, with one alkaline bath (pH 10.0) and two acid baths (pH 4.6 and 4.3). When taken out of the baths, the fibers on the section can be classified according to each fiber’s staining intensity under the various pH conditions, as shown in Figure 4-9. The standard fiber types in humans span from the most oxidative fiber type to the least oxidative fiber type, or from type I, type IC, type IIC, type IIAC, type IIA, type IIAX, and type IIX. It should be noted that

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**Table 4-2. Characteristics of Type I and Type II Muscle Fibers**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Type I</th>
<th>Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Force per cross-sectional area</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Myofibrillar ATPase activity (pH 9.4)</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Intramuscular ATP stores</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Intramuscular phosphocreatine stores</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Contraction speed</td>
<td>Slow</td>
<td>Fast</td>
</tr>
<tr>
<td>Relaxation time</td>
<td>Slow</td>
<td>Fast</td>
</tr>
<tr>
<td>Glycolytic enzyme activity</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Endurance</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Intramuscular glycogen stores</td>
<td>No difference</td>
<td>No difference</td>
</tr>
<tr>
<td>Intramuscular triglyceride stores</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Myoglobin content</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Aerobic enzyme activity</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Capillary density</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Mitochondrial density</td>
<td>High</td>
<td>Low</td>
</tr>
</tbody>
</table>

FIGURE 4-9. Myosin ATPase delineation of muscle fiber types. Histochemical assay used for the delineation of skeletal muscle fiber types. The separation of fiber types is based on differences in the pH stability of the ATPase molecule; i.e., the presence or absence of ATPase activity after exposure of the tissue to solutions of varying pH: (a) pH 4.3, (b) pH 4.6, and (c) pH 10.0. In human muscle, the array of fiber types that can be delineated includes type I, type IC, type IIC, type IIAC, type IIA, type IIAX, and type IIX. (Courtesy of Dr. Jenny Herman, Rocky Vista College of Osteopathic Medicine, Parker, CO.)
the oxidative capacity of a fiber is inversely proportional to its contractile velocity. That is, type I fibers, which are highly oxidative, are the slowest to develop peak force, while at the opposite extreme, type IIX fibers, which show the poorest oxidative potential, have the fastest contractile velocity. In animals (rat, mouse, cat, etc.), a greater array of muscle fiber types exists, again going from the most oxidative to the least oxidative types, from type I, type IC, type IIC, type IIAC, type IIA, type IIAX, type IIX, type IIXB, and type IIB. The greater array of muscle fiber types in lower mammals is thought to be due to a less sophisticated nervous system, requiring a greater array of muscle fiber types. Fiber type influences muscular performance in that type I fibers and its subtypes are conducive to endurance performance, while possessing a high percentage of type II fibers and related subtypes would be favorable to speed and power performance (Box 4-7).

**Myosin Heavy Chains**

The head of the myosin filament is made up of two heavy chains and two pairs of light chains. Each heavy chain has a molecular weight of about 230 kDa and is associated with two light chains (the essential light chain and the regulatory light chain) (Fig. 4-10). Some investigators prefer using the myosin heavy chain (MHC) composition of muscle, which can be determined with electrophoresis to separate out proteins or by using protein-specific antibodies, to profile a muscle sample’s fiber type composition. In the muscles of humans, three major types of MHCs exist: I, IIa, and IIx. In animals, four heavy chains have been identified: I, IIa, IIx, and IIb. If one collapses the multiple variations of fiber subtypes in humans into the three basic muscle fiber types of I, IIA, and IIX, and compares them to the MHC I, Ia, and IIx subtypes, a high correlation is found, suggesting that the two procedures provide similar results concerning a muscle’s fiber type profile. The muscle fiber subtyping discussed above, particularly as assessed by myosin ATPase staining, which allows greater detail of fiber types compared to MHCs, allows greater understanding of the adaptations of muscle to exercise training and the transitions of the major types, and their subtypes, to that stimulus.
**Quick Review**

- The sarcomere is the smallest or most basic contractile unit of skeletal muscle.
- Noncontractile proteins provide a lattice work for the organization of the actin and myosin filaments.
- Muscle fibers contain both actin and myosin filaments that interact with each other to produce shortening and force generation.
- Performance will be influenced by the type of muscle fibers in a muscle.
- ATPase histochemical staining techniques can determine if a muscle fiber is a type I or type II fiber, and even more specifically in humans, it can determine which subtype is being expressed (e.g., type IIA or IIX).
- Myosin heavy chains reflect the fiber type of the muscle fiber.

**SLIDING FILAMENT THEORY**

Whether walking across campus, lifting a heavy weight, or running a marathon, movement is produced by the contracting muscle fibers. Exactly how muscle contracts to produce force remained a mystery until an interesting theory was proposed by two groups of scientists in the middle of the 20th century. This theory, referred to as the sliding filament theory, was proposed in two papers published in 1954 in *Nature*, one by Andrew Huxley and Rolf Niedergerke, and another by Hugh Huxley (no relation to Andrew Huxley) and Jean Hanson. These articles provided experimental evidence revealing how the muscle shortens and develops force. The sliding filament theory of muscle contraction remains the most insightful explanation of how muscle proteins interact to generate force.

The essence of the sliding filament theory requires changes in the length of the muscle to be caused by the actin filaments and the myosin filaments sliding over each other to produce force without these filaments themselves changing in length (think of opening and closing the sliding doors leading onto the back deck of your home). At rest, the arrangement of actin and myosin filaments results in a repeating pattern of light (actin or myosin filaments alone) and dark (overlapped actin and myosin filaments) areas. The change in the striated pattern in muscle indicates the interaction between the two myofilaments. In the contracted (fully shortened) state, there are still striations, but they have a different pattern. This change in the striation pattern occurs due to the sliding of the actin over the myosin.

But before this contractile process can happen, Ca++ must be released into the cytosol of the muscle fiber so that it can interact with the regulatory protein troponin. The number of interactions between the actin and myosin filaments, or actomyosin complexes formed, dictates how much force is produced. In the next sections, we will learn in more detail how this shortening of muscle is accomplished at the molecular level of the muscle.

**Steps Mediating the Contraction Process**

At rest, crossbridges of the myosin filaments are in close proximity to the actin filaments, but they cannot interact to cause shortening because the active sites on the actin filaments are covered by tropomyosin protein strands. To create an interaction with the actin filament, the heads of the myosin crossbridges must be able to bind to the active sites of the actin protein. This means that the tropomyosin protein strands, which cover actin’s active sites under resting conditions, need to be moved to expose the active sites. This essential displacement of tropomyosin is triggered by an increase in the muscle fiber’s cytosolic Ca++ concentration. To understand how this occurs, we must recognize that the initial excitation of the muscle fiber begins with an electrical impulse that is initiated at the neuromuscular junction, the synapse joining the motor neuron to the muscle fiber, when the neurotransmitter binds to its receptors on the muscle fiber’s surface (see Box 4-8). (This process is discussed in detail in Chapter 5.)

**More to Explore**

**What is Postactivation Potentiation?**

Postactivation potentiation (PAP) is an increase in force generation after exposure to brief, nonfatiguing conditioning stimuli such as a maximal isometric contraction or submaximal dynamic contractions. This augmentation of force has been attributed to phosphorylation of myosin regulatory light chains, in turn increasing the myofilaments’ sensitivity to Ca++. But before this contractile process can happen, Ca++ must be released into the cytosol of the muscle fiber so that it can interact with the regulatory protein troponin. The number of interactions between the actin and myosin filaments, or actomyosin complexes formed, dictates how much force is produced. In the next sections, we will learn in more detail how this shortening of muscle is accomplished at the molecular level of the muscle.

**Further Readings**


Spread of the Electrical Impulse
This electrical impulse, first detected at the neuromuscular junction, spreads across the muscle fiber’s membrane, or sarcolemma, down into the transverse tubules (T-tubules), which penetrate into the core of the fiber, reaching the sarcoplasmic reticulum. The sarcoplasmic reticulum is a membrane-bound structure that surrounds each myofibril within the muscle fiber and acts as a depot that stores Ca\(^{2+}\) (Fig. 4-11). When the electrical impulse travels down the T-tubules, it excites proteins called DHP (dihydropyridine) receptors, which act as voltage sensors. Upon this excitation, the voltage sensors interact with ryanodine receptors located in the membrane of the sarcoplasmic reticulum. These ryanodine receptors are actually channels that, when stimulated by the voltage sensors of the T-tubules, open to allow a sudden release of Ca\(^{2+}\) from the sarcoplasmic reticulum into the cytosol of the muscle fiber.

Uncovering the Active Sites
The released Ca\(^{2+}\) then binds to the troponin C subunit of the troponin protein complex, and this interaction is what ultimately triggers a conformational change in the tropomyosin, thus preventing it from covering the active sites on the actin filament, leaving them exposed. The process by which, under resting conditions, tropomyosin blocks the active sites on the actin filament is called the steric

FIGURE 4-11. Sarcoplasmic reticulum. Muscular contraction is mediated by the electrical charge disruption of the calcium pump in the sarcoplasmic reticulum, which turns off the pump, allowing Ca\(^{2+}\) to be released. The release of Ca\(^{2+}\) from the sarcoplasmic reticulum into the cytosol results in the binding of Ca\(^{2+}\) with the troponin C component of the troponin molecule, which in turn initiates a conformational change of the troponin–tropomyosin complex, pulling the tropomyosin off the active site. This allows the heads of the myosin crossbridge to bind, and the ratcheting movement of the head inward pulls the Z lines toward each other. ACh, acetylcholine.
blocking model in muscle. With the active sites of actin now exposed, the heads of the myosin crossbridges can start a binding process with actin that will ultimately result in muscle fiber shortening and force production. This binding process features two distinct phases. First there is a weak state, which under nonfatiguing conditions is followed by a phase of strong binding, which allows greater and faster force production. Under fatiguing conditions, however, the transition from the weak to the strong binding state does not occur, resulting in less, and slower, force production.

**Interaction of Actin and Myosin Filaments**

When the actin and myosin filaments combine, an actomyosin complex is formed. Once this reaction occurs, the myosin crossbridge heads pull actin toward the center of the sarcomere and force is produced. This movement of the myosin crossbridges is called the power stroke, which has been described as a type of ratchet movement. In other words, the myosin head swivels at its hinged pivot point and pulls the actin filament over the myosin filament, causing the sarcomere to shorten, bringing the Z lines closer to each other (Fig. 4-12). Adenosine triphosphate (ATP), which is generated by the different energy pathways discussed in Chapters 2 and 3, is vital to the contraction process. The myosin head goes through the same cycle of events each time it binds to an active site. Let us start with the myosin head bound to an active site after a power stroke has taken place. In order for the myosin head to detach from the active site, an ATP molecule binds to the myosin head, breaking the actomyosin complex. Following this, the myosin ATPase found on the myosin crossbridge head hydrolyzes the ATP, and the energy is used to cock the myosin head back so it is over a new active site closer to the Z line. The adenosine diphosphate (ADP) and inorganic phosphate (Pi) formed from the breakdown of ATP remain bonded to the myosin head. In this energized state, the crossbridge head is ready for its next interaction with another exposed active site closer to the Z line. After weakly binding to the new active site on actin to initiate the next power stroke, the Pi is released from the myosin head. At the end of the power stroke, the ADP is released from the myosin and the myosin head is again tightly bound to the active site where it remains until a new ATP molecule (it cannot simply be a rephosphorylation of the already present ADP molecule) binds to the myosin head, separating the myosin head from the active binding site. This cycle is repeated, resulting in the ratcheting movement of repetitive power strokes. This crossbridge cycling sequence will continue to be repeated until the muscle fiber no longer is excited by the nervous system. At that point, no further release of Ca++ from the sarcoplasmic reticulum occurs, enabling the Ca++ pump located in the membrane of that organelle to return cytosolic Ca++ levels to those seen at rest by moving Ca++ back into the sarcoplasmic reticulum. Due to the drop in Ca++ concentration in the cytosol, the troponin C subunit is no longer bound to Ca++, which causes the troponin to stop the tugging on the tropomyosin strand, allowing it to once again cover the active sites on the actin filament. As a result, the myosin crossbridge heads cannot bind to the active sites to form the actomyosin complexes necessary to execute the power stroke.

**Return to Resting Muscle Length**

When bound to an active site, the crossbridges of the myosin molecule can swivel only in the direction that pulls actin over the myosin so that the Z lines come closer together, resulting in a shortening of the muscle. More to the point, myosin crossbridges are designed to cause muscle shortening. So the muscle fiber cannot by itself return to its
Neural activation causes a release of Ca++ from the sarcoplasmic reticulum. Ca++ binds to troponin, altering its shape and causing tropomyosin to shift, exposing the active sites. Myosin filaments can now attach to actin. ATP binds to myosin head causing detachment from actin active site, ATPase breaks down ATP to ADP + Pi which repositions myosin head at 90° position as in step 1. Steps 1 to 5 are repeated to produce further shortening.
Part II  Exercise Physiology and Body Systems

Applying Research
Muscular Contraction

The basic steps in the contractile process of skeletal muscle are as follows:

Excitation
1. An action potential takes place in an alpha motor neuron axon.
2. The neurotransmitter acetylcholine (ACh) is released from the axon terminal.
3. ACh binds to receptors on the muscle fiber membrane.
4. Channels open on the muscle fiber membrane, generating an ionic current.
5. The ionic current sweeps through the T-tubules and stimulates the DHP receptors, which act as voltage sensors, in the T-tubules.
6. The stimulated voltage sensors activate the ryanodine receptors, which are Ca++ channels, located on the membrane of the sarcoplasmic reticulum.
7. Upon opening of the ryanodine receptors, the sarcoplasmic reticulum releases Ca++ into the cytosol.

Contraction or Shortening
1. Ca++ binds to the troponin C.

When excited by the electrical impulse, DHP receptors activate ryanodine receptors located on the membrane of the sarcoplasmic reticulum.
Ryanodine receptors are actually Ca++ channels embedded in the membrane of the sarcoplasmic reticulum, and upon activation, they open to release Ca++ stored in the sarcoplasmic reticulum into the cytosol of the muscle fiber.
The released Ca++ can then bind to troponin, causing a shift in positioning of tropomyosin, thus exposing active sites on actin.
This allows myosin crossbridge heads to bind to exposed actin active sites, forming actomyosin complexes.
A swiveling movement of the myosin crossbridge head occurs, resulting in a power stroke, which creates a shortening of the sarcomere and, ultimately, the muscle.
When electrical impulses are no longer delivered to the muscle fiber’s surface, there is a cessation in the release of Ca++ from the sarcoplasmic reticulum, allowing the Ca++ pump of the sarcoplasmic reticulum to return cytosolic Ca++ back to resting concentrations.
With no Ca++ to bind to the troponin, tropomyosin again blocks the active sites on actin, causing muscle contraction to stop.
The intact muscle is returned to its resting length by an outside force such as gravity or an actively contracting antagonistic muscle.

2. A conformational change in troponin causes the tropomyosin to move, exposing active sites on actin.
3. Myosin crossbridges bind to the exposed active sites.
4. Myosin heads swivel, pulling the actin filaments over the myosin filaments.
5. Myosin heads acquire a new ATP and release from the active site.
6. ATPase on myosin head hydrolyzes the ATP, energizing the crossbridge and “cocking” it back to its starting position so that it is ready to bind another active site.
7. As long as sufficient cytosolic calcium ions are present, the cycle continues.

Relaxation
1. Alpha motor neuron axon action potential stops.
2. Ca++ is actively pumped back into sarcoplasmic reticulum.
3. Ca++ is unbound from troponin C.
4. Active sites are covered by tropomyosin and troponin.
5. External force is needed to return the muscle to resting length.

Quick Review
- The proposed theory to explain muscular contraction is the sliding filament theory.
- This theory holds that changes in muscle length are caused by the actin and myosin filaments sliding over each other without either of those filaments changing its own length.

Steps of the sliding filament theory include:
- At rest, actin and myosin crossbridges are in near contact with each other, but no binding occurs.
- An electrical impulse crosses the neuromuscular junction and goes down the T-tubules where the impulse is detected by the DHP receptors (voltage sensors).

- When excited by the electrical impulse, DHP receptors activate ryanodine receptors located on the membrane of the sarcoplasmic reticulum.
- Ryanodine receptors are actually Ca++ channels embedded in the membrane of the sarcoplasmic reticulum, and upon activation, they open to release Ca++ stored in the sarcoplasmic reticulum into the cytosol of the muscle fiber.
- The released Ca++ can then bind to troponin, causing a shift in positioning of tropomyosin, thus exposing active sites on actin.
- This allows myosin crossbridge heads to bind to exposed actin active sites, forming actomyosin complexes.
- A swiveling movement of the myosin crossbridge head occurs, resulting in a power stroke, which creates a shortening of the sarcomere and, ultimately, the muscle.
- When electrical impulses are no longer delivered to the muscle fiber’s surface, there is a cessation in the release of Ca++ from the sarcoplasmic reticulum, allowing the Ca++ pump of the sarcoplasmic reticulum to return cytosolic Ca++ back to resting concentrations.
- With no Ca++ to bind to the troponin, tropomyosin again blocks the active sites on actin, causing muscle contraction to stop.
- The intact muscle is returned to its resting length by an outside force such as gravity or an actively contracting antagonistic muscle.
PROPRIOEPTION AND KINESTHETIC SENSE

For the body to optimally perform everyday activities (e.g., going down stairs) or sport skills (e.g., triple jump), feedback, or a constant flow of information about our body position, needs to occur within the neuromuscular system. We can appreciate how important this feedback is when we witness the complex skills exhibited by gymnasts, divers, figure skaters, basketball players, or almost any other athlete performing in his or her sport. The importance of this constant stream of neural feedback is highlighted during injury to the peripheral receptors and proprioceptive organs found in muscles and other tissues. Following this sort of injury, our sense of body position and orientation among different body segments is disturbed, making coordinated movements difficult to accomplish. How the body senses where it is in space is achieved through the neuromuscular system’s proprioceptive capabilities.

This sense of the body’s position is monitored by feedback as to the length of the muscle and force being produced. Such monitoring is achieved by proprioceptors, which are receptors located within the muscles and tendons. The information that proprioceptors gather is constantly being relayed to conscious and subconscious portions of the brain. Such information is also important for learning motor tasks, especially when repeated over and over again to create a learning effect, which is the ability to repeat a specific motor unit recruitment pattern that results in successful performance of a skill, such as making a jump shot in basketball. The reason coaches have athletes repeatedly practice their sport skill is to learn specific motor patterns that can be accurately reproduced during competition. Because of proprioceptive mechanisms, one can perform complex skills, such as the pole vault or a gymnastics maneuver, and just “feel it” as being right. Proprioceptors keep the central nervous system constantly informed as to what is going on with the body’s movements, many times at the subconscious level. Many movements are conducted so quickly that the individual may not even think about the performance of the activity or skill except before it begins (e.g., visualizing a sport skill or seeing a long set of steps before descending them). Such continuous information is vital for normal human movement as well as any sport performance. This ability of knowing where the body’s position is in space has been called kinesthetic sense.

Muscle Spindles

The proprioceptors in skeletal muscle are called the muscle spindles. The two functions of muscle spindles are to monitor stretch or length of the muscle in which they are embedded and to initiate a contraction when the muscle is stretched. The stretch reflex, in which a quickly stretched muscle initiates an almost immediate contraction in response to being stretched, is attributed to the response of the muscle spindles.34

Spindles are located in modified muscle fibers that are arranged parallel to the other fibers within the whole muscle (Fig. 4-14). The modified muscle fibers containing spindles are called intrafusal fibers. These intrafusal fibers are composed of a stretch-sensitive central area (or sensory area), embedded in a muscle fiber capable of contraction. If a muscle is stretched, as in picking up an unexpectedly heavy suitcase, the spindles are also stretched. The sensory nerve of the spindle carries an impulse to the spinal cord where the sensory neuron synapses with alpha motor neurons. The alpha motor neurons relay a reflex nerve impulse to the muscle, causing a contraction, or shortening, of the stretched muscle, relieving pressure on its spindles (more on reflexes in Chapter 5). Concurrently, other neurons inhibit activation of antagonistic muscles of the stretched muscle so that they do not interfere with the desired reflexive shortening of the agonist muscle. From a practical perspective, performing exercises with a prestretch (e.g., stretching your pectoral chest muscles in a bench press by taking a wide grip and pulling your clavicles together) takes

![FIGURE 4-14. Muscle spindles. Muscle spindles send information on the length and tension of the muscle fibers to the higher brain centers. This is very important to patterned skills, where position of the muscles and precise force development determine the effectiveness of the skill being performed (e.g., touch in a basketball jump shot).]
advantage of this stretch reflex. This reflex is one explanation for the greater force output with a prestretch before an activity. For example, throw a ball as far as you can using a wind-up, which acts as a prestretch, and then throw a ball by stopping at the end of the wind-up for several seconds before throwing it. You will definitely throw the ball farther with a wind-up, which causes a prestretch, in part because of the reflex action of stretching the muscle spindles.

Alpha motor neurons innervate muscle fibers that do not contain spindles (called extrafusal fibers), and gamma motor neurons innervate the intrafusal fibers. Because muscle spindles are found in functioning muscle fibers, the nervous system can regulate the length and therefore the sensitivity of the spindles to changes in the length of the muscle fibers. Adjustments of the spindles in this fashion enable the spindle to more accurately monitor the length of the muscles in which they are embedded. Such adjustments appear to take place with trained athletes, making them more capable of performing highly complex, well-practiced movements.

**Golgi Tendon Organs**

The proprioceptor in the tendon that connects muscle to bone is called the *Golgi tendon organ*. The Golgi tendon organ’s main function is to respond to tension (force) within the tendon (Fig. 4-15). If the forces exerted on the tendon are too high, injury might occur and the Golgi tendon organ is activated. Because of their location in the tendon, these proprioceptors are well positioned to monitor tension developed by the whole muscle and not just individual fibers.  

The sensory neuron of each Golgi tendon organ travels to the spinal cord where it synapses with the alpha motor neurons of both the agonist and antagonist muscles. As an activated muscle develops force, the tension within the muscle’s tendon increases and is monitored by the Golgi tendon organs. If the tension becomes great enough to damage the muscle or tendon, the Golgi tendon organ inhibits the activated muscle. The tension within the muscle is alleviated so that damage to the muscle and/or tendon can be avoided.

The proprioceptive sense of body position is affected by fatigue, demonstrating the importance of exercise training and conditioning to avoid, or at least reduce, fatigue during athletic competitions. Interestingly, new methods of training are being added to many exercise programs with the goal of enhancing the flow of information from muscles to the central nervous system and back to the muscles. By effectively training these neuromuscular pathways of information flow, they can be expected to be more resistant to the onset of fatigue, allowing the performance of complex athletic movements to remain at their optimal level for a longer period of time in a competition or training session. In some cases, the goal is to increase the excitatory inflow from muscle spindles to the motor neuron pools and decrease inhibition by Golgi tendon organs, resulting in greater force production by the muscle(s).

**Quick Review**

- Proprioceptors sense the body’s position by monitoring the length of the muscle and force being produced.
- Proprioceptors relay important information regarding body position and orientation to the central nervous system.
- Muscle spindles monitor stretch and length of the muscle and initiate a contraction to reduce the stretch in the muscle.
- Golgi tendon organs respond to tension (force) within the tendon.

**FORCE PRODUCTION CAPABILITIES**

Because of its capacity to generate the force necessary to cause movement of limbs and the whole body, skeletal
muscle plays, perhaps, the most important role in determining an individual’s performance during exercise and sports. The muscle's capacity to generate force is critical not only to sports performance, but also to the ability to carry out the tasks of daily living. With this in mind, it is important to understand the different types of muscle actions, modes of exercise, and how force, power, and contractile velocity are related to each other.

Types of Muscle Actions

When muscle is activated and generates force, it can shorten, remain the same length, or resist elongation. These three types of muscle actions are typically referred to as concentric, isometric, and eccentric, respectively (Fig. 4-16). If we think about what occurs when lifting weights, we can easily see the differences among these muscle actions. Normally, when a weight is being lifted, the muscles involved are shortening (concentric muscle action), hence the term “contraction.” During such concentric actions, the force produced by the muscle exceeds that imposed by the resistance, or load. So if one successfully completes a 200-lb (90.9-kg) bench press, it is because the muscles were able to generate more than 200 lb of force. In an isometric muscle action, the myosin heads keep attaching and detaching at the same or close to the same active site on the actin filament. Thus, no visible movement occurs, but force is developed by the attempt to shorten. In this case, the force produced by the active muscle is equal to the resistance opposing its movement. An example would be when in the middle of a repetition of a bench press, the weight is held stationary. An example of an eccentric action would be when a weight is being lowered in a controlled manner from the arms extended position to the chest touch position in the bench press. As the weight is lowered, the muscles involved are lengthening while producing force. During an eccentric muscle action, the load, or resistance, is greater than the force produced by the muscle. This can occur either when the muscle is exerting its maximal force, but the force is inadequate to overcome the resistance, or when an individual deliberately reduces muscle force production to allow the muscle to gradually lengthen. In either case, the myosin heads interact with the actin filament’s active site to slow the elongation of the muscle by binding, but do not complete the normal ratchet shortening movement discussed above.

Terms Used to Describe Resistance Exercise

The term isotonic is the most popular term used to describe types of resistance exercise and, often, any exercise movement. It infers that the muscle generates the same amount of force throughout the entire range of motion (iso meaning the same and tonic referring to tension, or force, produced by the muscle). Since the amount of force produced over the muscle’s range of motion varies (more on this later in the chapter), an isotonic muscle action would not typically occur unless using an advanced computerized resistance system that modulates the force and velocity of movement. Therefore, in place of the term isotonic, dynamic constant external resistance has been used to describe this typical type of muscle activity when exercising with external resistances such as free weights, or weight stacks on a machine, when the resistance remains the same throughout the range of motion. Isoinertial is another term used in place of isotonic to describe an exercise movement with variable velocity and a constant resistance throughout a movement’s range of motion. Variable resistance describes weight machines that produce a change in the resistance over the range of motion normally in an attempt to match the variation in the force produced by the muscle during an exercise. Hydraulic machines (concentric-only resistance) and pneumatic machines (concentric and eccentric resistances) using compressed fluids and air can also create external resistances that vary in attempts to match the resistance to the force production capabilities throughout the range of motion of an exercise. The term isokinetic is used to describe muscular actions in which the velocity of the limb’s movement throughout the range of motion is held constant using a specialized isokinetic dynamometer (iso again meaning the same and kinetic meaning motion). This type of sophisticated device (e.g., Biodex, Cybex, KinCom, Lido dynamometers) allows the speed of movement throughout a repetition to be set at a specific, constant rate and then measures the torque production to allow the muscle to gradually lengthen. In either case, the myosin heads interact with the actin filament’s active site to slow the elongation of the muscle by binding, but do not complete the normal ratchet shortening movement discussed above.

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(i.e., rotational force) produced at that specific velocity. These types of machines are typically found in athletic training rooms and physical therapy clinics and are used for clinical assessments of joint function in a concentric and/or eccentric joint motion. An isokinetic action requires the use of a dynamometer to produce the desired effect of constant velocity because this type of joint action is not found in normal physical activity. Training with isokinetic dynamometers was initially attractive because it allowed training at high velocities of movement (e.g., 300° s⁻¹) that mimic power movements, and the devices automatically kept computerized records of the results of training sessions. But most isokinetic dynamometers allow only single muscle groups to be trained in simple or isolated movements (i.e., knee extension, calf extensions) that generally do not occur in sports activities. Thus, translation of training with isokinetic muscle actions to normal muscular activity in everyday life or sports may be minimal because most of those activities involve multiple muscle groups contracting in highly coordinated sequences. Although their use as effective training devices for competitive athletes may be limited, isokinetic dynamometers can be used effectively for accurately assessing, or testing, various parameters of muscle function, including strength, rate to peak force, and endurance.

**Force–Velocity Curve**

The force–velocity curve demonstrates the influence of changing the velocity of movement on the muscle's maximal force production capabilities. This classic relationship was first described in experiments using isolated muscle by Nobel Laureate Professor Archibald Vivian (A. V.) Hill at University College in London. The relationship between the maximal force a muscle can produce and the velocity of movement depends on the type of muscle action used (i.e., eccentric, isometric, and concentric phases) and is shown in Figure 4-17.

As shown in the figure, there are distinct differences in the force–velocity relationships between concentric and eccentric muscle actions. As a starting point, we will use the maximal isometric force production, which by definition is at zero velocity. If we move with increasing velocity using a concentric muscle action, the force production declines at first very dramatically as movement velocity increases.

As velocity continues to increase, the decline in force becomes more moderate. But at any velocity, the maximal force produced by a concentric muscle action is less than that of a maximal isometric action. However, if we move with increasing velocity in an eccentric action, maximal force actually increases as the velocity increases; again, at first very markedly, but as velocity increases, elevations in eccentric force production become more moderate, eventually reaching a plateau. At any velocity of eccentric action, maximal force produced is always greater than that during maximal isometric actions. The increase in force production with increasing velocity during eccentric actions has been thought to be due to the elastic component of the muscle. However, complete understanding of the reasons for such a response remains elusive.

It is important to note that the high forces seen with maximal or near maximal eccentric muscle actions, which are much higher than those generated during maximal concentric or isometric actions, have been identified as one of the main contributors to muscle damage with exercise. Eccentric actions have been called the mechanical stressor of muscle. **Delayed-onset muscle soreness (DOMS)** is one of the major symptoms of muscle damage due to high eccentric loads (Box 4-10). Untrained individuals are

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**Practical Questions from Students**

**What Causes Delayed-Onset Muscle Soreness?**

Experts believe delayed-onset muscle soreness (DOMS) is due to tissue injury caused by mechanical stress on the muscle and tendon. Microtears occur in the muscle fibers, causing disarrangement of the normally aligned sarcomere. This structural damage likely triggers an immune response involving the release of histamines and prostaglandins (specific agents involved in the immune regulatory process) and edema (fluid accumulation in the tissue), which results in the sensation of pain. DOMS is typically related to the eccentric component of muscular contraction, appears 24 to 48 hours after strenuous exercise, and is more common in untrained people. One big coaching myth is that lactate causes DOMS; this is simply not the case, as no evidence has been presented to support this hypothesis.
especially sensitive to such high mechanical stresses, and therefore exercise programs featuring eccentric actions (e.g., negative weight training or downhill running) must start out at lower intensities or volumes and gradually progress to higher intensities or volumes to allow for adaptations to occur that will minimize muscle damage and soreness. This approach of gradually increasing the resistance or load used during exercise sessions, especially with resistance training, is known as progressive overload.

Most training programs focus on the concentric phase of the force–velocity curve to increase muscular power. **Power** is defined as force times the vertical distance a mass moves divided by time or, alternatively, as force times velocity. Proper strength and power training can move the entire force–velocity curve upward and to the right (Fig. 4-18). This movement of the concentric force–velocity curve has positive benefits for both activities of daily living and athletic performances, as power has increased. Improving power across the entire curve requires a training program that uses both heavy strength training (loads > 80% load of the 1-repetition maximum) and high-velocity ballistic training protocols (e.g., plyometrics). If only one training component, that is, either force or speed of contraction, is addressed, then changes will primarily occur over only part of the force–velocity curve. In other words, training heavy and slow will primarily improve force production at the slower velocities, whereas training light and fast results primarily in improvements at higher velocities of movement. From a practical perspective, many coaches use the term **speed-strength** to define training that is focused on force production at higher velocities and lighter resistances to improve power. Due to their interdependency in physical performance, optimal training should typically address the entire force–velocity curve.

**Strength Curves**

There are three basic types of strength curves, as shown in Figure 4-19. A strength curve is the amount of force that can be produced over a range of motion. Strength curves take on different patterns depending on the biomechanics of the exercise movement and the individual’s body structure. For example, in a resistance exercise with an ascending strength curve, such as a barbell squat, it is possible to produce more force toward the end of the concentric range of motion. If an exercise has a descending strength curve, such as with upright rowing, it is possible to produce less force near the completion of the concentric phase of a repetition. An exercise, such as arm curls, in which it is possible to produce more force in the middle portion rather than the beginning or end portions of the range of motion has a bell-shaped strength curve.

**Length–Tension Relationship**

The length–tension relationship shows that the length of a muscle has a direct influence on the total force or tension it is able to generate (Fig. 4-20). The total tension generated by a muscle is the sum of its passive and active tension. The **passive tension** reflects the contributions of the elastic elements of a muscle in the absence of neural stimulation. As the passive tension is produced in order to maintain the structural integrity of the muscle, increasing the degree of stretch results in a corresponding increase in its passive tension up to a physiological maximum. The **active tension** of a muscle is generated by the number of actomyosin complexes formed in response to neural stimulation. Therefore, active tension is greatest when the length of the muscle permits maximal overlap between the myosin and actin filaments. At any length above or below the length allowing maximal overlap of actin and myosin, less tension is developed because fewer actomyosin complexes can be formed.

Variable-resistance weight training equipment has been designed to take advantage of the change in strength
potential over a range of motion by varying the load during a repetition to help maximize strength development. Hypothetically, varying resistance at different points in the range of motion would allow the muscle to develop closer to its maximal strength throughout the range of motion, rather than be limited to what is possible when the muscle is at its weakest point in the strength curve. Although this type of training would seem to be desirable, individualizing the pattern of resistance variation would be essential, and many machines have not been very successful in the attempt to match the strength curve of a movement with the use of cams, rollers, or angle changes in the lever arm of the exercise machine. This is due to individual differences in limb length, point of attachment of the muscle’s tendon to the bones, and body size. It is hard to conceive of one mechanical arrangement that would match the strength curve of all individuals for a particular exercise.

**Force–Time Curves**

Just as the force–velocity curve helps us visualize force production at different velocities of movement, the force–time curve helps us visualize force production over different segments of time after the stimulation to contract. The ability to produce force quickly is an important quality of neuromuscular function, from an older person trying to prevent a fall after a momentary loss of balance, to a volleyball player spiking a ball over the net. The force–time curve also allows us to evaluate training programs directed at power development. In Figure 4-21, the amount of force produced over time is shown with three example curves, one normal curve for an untrained individual, one in which heavy strength and ballistic power training were included, and one in which only heavy strength training was performed. Using both strength and power training components not only results in greater strength but also translates into a reduction in the time it takes to reach peak force production, that is, greater power. Because most physical performances require power for their performance, one can easily see that enhancing the ability to produce force very rapidly could be considered an important attribute of any conditioning program, whether it is for health and fitness or sports performance.
Quick Review

- The force–velocity curve describes the influence of changing the velocity of movement on the muscle’s force production capabilities.
- The strength curve describes the amount of force that can be produced over a movement’s range of motion.
- The force–time curve helps us visualize force production over different segments of time during a complete movement and allows us to evaluate training programs directed at power development.
- The ability to produce force quickly is an important quality of neuromuscular function.
- Proper strength and power training can improve power across the entire force–velocity curve.

SKELETAL MUSCLE TRAINING ADAPTATIONS THAT IMPROVE PERFORMANCE

Endurance and resistance training are the most prominent forms of exercise to enhance health and athletic performance. Understanding some of the basic adaptations of skeletal muscle to these two popular forms of exercise is essential if we are to appreciate how exercise can improve health and exercise performance. Each of these exercise modalities will be covered further in Chapter 13, but let us specifically look at skeletal muscle adaptations here. In addition, because few people perform one or the other training modalities exclusively, we will also take a closer look at exercise compatibility, or what happens when both forms of exercise are done concurrently with the same musculature.

Effects of Endurance Training

Looking at the effects of endurance training on skeletal muscle, we must remember that only those fibers that are recruited during exercise will adapt to the exercise stimuli. The primary adaptations to endurance training are related to the need to better utilize oxygen and enhance muscular endurance. Motor units with type I muscle fibers are recruited first, and then, as the intensity of the endurance activity increases, type II motor units are recruited as needed (this will be covered in more detail in Chapter 5). The more type II motor units that are recruited, the less efficient and effective the endurance performance will be, especially as the duration of activity increases. Perhaps this is presented best in the following sports scenario: It can be estimated that running a marathon race at about a 2:10:00 pace would require the athlete to run at a pace of about 4.84 m s⁻¹. This would require the runner to recruit about 80% of the motor neuron pool. If motor units containing type I muscle fibers are better able to meet the aerobic demands of such a task, we can see the benefit of having a fiber-type profile of about 80% type I muscle fibers in the motor neuron pool in the muscles involved in running. In fact, many elite endurance runners actually have such a fiber-type composition of the thigh muscles. Although the above example may be a bit oversimplified, because many factors go into making an elite endurance performance, remember that motor units containing type I muscle fibers are better suited to endurance performance because of their high mitochondrial density and blood supply. Nevertheless, endurance adaptations will occur in all of the muscle fibers recruited to perform the endurance exercise. For many of us who have a more even distribution of fiber types (e.g., 45% type I and 55% type II), a number of motor units containing type II muscle fibers will be used to run a 10-km race or even to go out for a noon-time run with friends and will also undergo adaptations to improve their aerobic capacity. But if elite performance is the objective (e.g., running a marathon under 2 hours and 10 minutes), the runner needs to genetically have a predominance of type I motor units to optimize performance, because although type II fibers will show training-induced improvements in aerobic capacity, they will never match the aerobic capacity that is inherent in type I fibers.

So, what happens to the muscle fibers, both type I and type II, when they are recruited as a part of a motor unit to perform an endurance exercise workout? First, in order to enhance the delivery of oxygen to the muscles, an increase in the number of capillaries per muscle fiber will take place. This will enhance the delivery of oxygen to the exercising muscle, as well as training-induced increase in capillary density (i.e., number per unit size of muscle tissue). These adaptations appear to be fiber-type specific, as type I fibers enjoy more pronounced improvements than type II fibers. In addition to these changes in capillarity, endurance training elicits an increase in the size
and number of mitochondria within muscle fibers (see Chapter 3). Mitochondria are the organelles that produce ATP via the aerobic pathway, and the increase in a fiber’s mitochondrial content is accompanied by an enhanced ability for aerobic metabolism. With the increase in mitochondrial content within the trained fiber comes a greater concentration of the enzymes of the Krebs cycle and of the cytochromes of the electron transport chain. Recall that these enzymes and cytochromes work together to synthesize ATP. As with changes in capillarity, gains in mitochondrial content stimulated by endurance training occur to a greater extent in type I muscle fibers, demonstrating the advantage of having more type I muscle fibers for optimal endurance performance. Keep in mind that capillaries are the vessels that exchange blood, oxygen, CO₂, nutrients, and waste products with the muscles, and mitochondria are the organelles inside the muscle cell where ATP is aerobically produced, thus linking increases in delivery of oxygen with greater capacity to use that oxygen to synthesize ATP.

In summary, with more capillaries surrounding each muscle fiber along with more myoglobin and mitochondria in each muscle fiber, the distance for diffusion to the mitochondria from the cell membrane or vice versa is shorter and the amount of time for the exchange of various substances to take place is reduced, which improves the efficiency and speed of the aerobic processes. This facilitates the exchange of oxygen, CO₂, nutrients, wastes, and heat between the muscle and the blood. With more oxygen and nutrients being delivered to the exercising muscle and more wastes and heat being removed, the muscle is better able to aerobically produce ATP to fuel the endurance energy demands, as well as remove potentially fatiguing metabolic byproducts. The overall result is improved endurance performance.

Interestingly, changes in fiber size may also contribute to improved aerobic function. More specifically, type I (slow-twitch) muscle fibers typically experience a decrease in their size with endurance training resulting in a reduction in the distances from the capillaries to the mitochondria and expediting the rate at which gases diffuse across the fiber. The percentages of type I and type II fibers do not significantly change with endurance exercise training, but some changes may take place in the percentages of the fiber subtypes to become more aerobic in nature (i.e., type IC to type I, type IIA to type IIC, and, if recruited, type IIX to type IIA).

Effects of Resistance Training

MuscLes come in different sizes and fiber-type distributions, both of which are related to a muscle’s function (Box 4-11). Yet all muscles, regardless of fiber type, composition, or function, are capable of enlarging in response to a resistance training program. This growth in the size of the whole muscle is primarily due to the increase in the size of its individual muscle fibers. In contrast, it has yet to be established whether muscles adapt to resistance training by increasing the number of their fibers, or what is referred to as hyperplasia. This may be because an increase in the number of muscle fibers would also result in an increase in total muscle size. Due to methodological difficulties (one cannot take out the whole muscle of a human for examination), the potential for hyperplasia in humans remains unresolved; however, it has been shown in response to various muscle overload protocols in birds and some nonhuman mammals.

Hypertrophy

Hypertrophy is the increase in the size of the muscle, or its constituent fibers, which occurs as a result of participating in an exercise program. Myofibrillar protein (i.e., actin and myosin) is added, which results in the addition of newly formed myofibrils to existing fibers, thereby increasing fiber size. It does not, however, appear that the size of preexisting myofibrils is altered as a result of resistance training. Despite the increase in myofibrillar number, the myofibrillar packing distance (the distance between myosin filaments) and the length of the sarcomere appear to remain constant following 6 weeks to 6 months of resistance training. Similarly, myofibrillar density, or the number of myofibrils within a given
volume of muscle tissue, is unaltered by resistance training even though muscle fiber size is increased. And although increases in myofilament number take place, the spatial orientation of the contractile proteins in the sarcomere appears to remain unchanged following resistance training. To increase muscle cross-sectional area during resistance training, sarcomeres are added in parallel to each other, resulting in muscle fiber hypertrophy (Box 4-12).

The remodeling of muscle tissue with heavy resistance exercise is a function of the program and the sequential changes in the synthesis/degradation of contractile proteins. All fibers appear to hypertrophy but not to the same extent. Conventional weight training in humans and animals elicits a greater degree of hypertrophy in type II fibers compared to type I fibers. Also, type I and type II fibers appear to hypertrophy using different mechanisms. In type II muscle fibers, the process involves an increase in the rate of protein synthesis, and with type I muscle fibers, a decrease in the rate of protein degradation.

Recent research has added much to our understanding of the mechanism(s) involved in muscle fiber hypertrophy. It is now understood that the acquisition of additional myonuclei or nuclei located with the muscle fiber is required to support an increase in the size of the muscle fiber. The source of the extra myonuclei is the satellite cells, which are located between the muscle fiber’s membrane and its thin, outer layer of connective tissue encasing the fiber, referred to as the basal lamina. Exercise stress, or other forms of damage to the connective tissue isolating these satellite cells, exposes them to agents called mitogens. As a result, the satellite cells undergo replication and newly produced satellite cells are fused into the muscle fiber. In this process, satellite cells contribute the needed increase in the number of myonuclei (i.e., DNA machinery). The added genetic machinery is necessary to manage the increased volume of protein and other cellular constituents (Box 4-13). A single myonucleus can only manage a specific volume of muscle protein, and therefore, without an appropriate increase in the number of myonuclei, increases in the amount of protein, which produce muscle fiber enlargement, would not be possible. The area within the fiber that each myonucleus is responsible for is called its nuclear domain.

With this in mind, Kadi and Thornell showed that 10 weeks of strength training can induce changes in the number of myonuclei and satellite cells in women’s trapezius muscles. These investigators found that their strength training program resulted in a 36% increase in the cross-sectional area of muscle fibers. This hypertrophy was accompanied by an approximately 70% increase in myonuclear number and a 46% increase in the number of satellite cells. Myonuclei number was positively correlated to satellite cell number, indicating that a muscle with an increased concentration of myonuclei will contain a correspondingly higher number of satellite cells.

Hyperplasia

Hyperplasia, or an increase in muscle fiber number, has been examined over the years as a possible mechanism for increasing the size of skeletal muscle. Interest in the viability of this concept was rekindled when several studies examining the muscles of body builders and power lifters concluded that the cross-sectional area of the body builders’ individual muscle fibers was not significantly larger than normal; however, whole muscle size of these athletes was larger than normal. About one decade later, a study reexamined the possibility of hyperplasia when McCall et al., using both magnetic resonance imaging (MRI) and biopsy techniques, demonstrated an increase in muscle fiber number in the biceps after a typical heavy resistance training program, again presenting some evidence for hyperplasia; however, muscle fiber hypertrophy accounted for the greatest amount of whole muscle hypertrophy. It is possible that only high-intensity resistance training can cause hyperplasia and that type II muscle fibers may be targeted for this type of adaptation. Power lifters have been shown to have higher numbers of myonuclei, satellite cells, and small-diameter fibers expressing markers for early myogenesis, thereby indicating hyperplasia or the formation of new muscle fibers. The effects appeared enhanced by anabolic steroid use, and thus one impact of anabolic drug use may be to increase the amount of hyperplasia that occurs.

Although limited data support hyperplasia in humans, there are indications that hyperplasia may occur as a consequence of resistance training. Due to these conflicting results, this topic continues to be controversial. Further
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Advances in molecular biology techniques have allowed us to evaluate alterations in gene expression and protein products in skeletal muscle during and following acute or multiple bouts of exercise. These advances have provided scientists with a better understanding of the molecular basis of skeletal muscle hypertrophy. This information is critical in the development of treatment interventions to promote hypertrophy, and possibly attenuate atrophy (see figure).

It is important to comprehend the role of genes and proteins in promoting hypertrophy. Fundamentally, each nucleus of every fiber (single muscle cell) contains combinations of four bases, known as nucleotides, which comprise deoxyribonucleic acid (DNA). These bases include adenine (A), guanine (G), cytosine (C), and thymine (T). Unique combinations of these nucleotides provide the genetic code necessary to make messenger ribonucleic acid (mRNA) and proteins. The translation of the DNA code into a protein involves two processes: transcription (DNA to mRNA) and translation (mRNA to protein). In response to exercise, genes are up- or downregulated. The extent and time course of these changes in gene expression are dependent on the duration, intensity, and frequency of exercise training. Depending on the magnitude and duration of increased or decreased gene expression, levels of mRNA can be affected to mirror those changes in DNA, resulting in a parallel change in the expression pattern of mRNA. Because the amount of mRNA dictates how much protein will be made, the magnitude of gene expression impacts the amount of protein made by each skeletal muscle fiber. Although the transcription:translation ratio is not 1:1, it is possible to modify stimuli to induce alterations in genes and gene products critical for hypertrophy.

Basic genetic mechanisms mediate the production of protein and the development of muscular hypertrophy. Early research ventures to decode the molecular basis of hypertrophy were daunting due to the lack of technology allowing the analysis of global changes in mRNA. Although it was clear that many genes were up- or downregulated in response to a single bout of exercise, it was not possible to examine all of the genes simultaneously in a time- and cost-efficient manner. At the same time, investigators were suggesting that the molecular events that stimulate hypertrophy might be as unique as the stimuli because there was a lack of continuity between specific signaling pathways. In other words, molecular events that regulate adaptations in skeletal muscle mRNA and protein in response to resistance training were different from those that regulate adaptations to plyometric or sprint training. Furthermore, results from this work identified a unique time course of alterations, indicating that molecular adaptations that occur immediately after exercise differ from those that occur days to weeks following the onset of training.

Advances in gene transcription and protein profiling techniques, including microarray technology and proteomics, have allowed scientists to examine global changes in thousands of genes and proteins simultaneously in a single muscle sample. These techniques have been important in many aspects of exercise science that require investigators to understand the behavior of the cell in response to a particular stimulus. For example, these techniques have allowed investigators to address specific questions such as the optimal time to ingest protein when exercising at different intensities. From a global health perspective, these techniques have allowed investigators to better understand why certain pharmacological drugs interfere with muscle growth or, in the case of statins, predispose individuals to conditions such as muscle wasting and rhabdomyolysis. These examples are just a few of the ways that scientists have leveraged technology to profile many genes and proteins simultaneously and subsequently perform targeted analysis of specific proteins that regulate phenotypical responses.

To do this, investigators take a multifaceted approach to understanding molecular alterations in response to exercise, nutrition, and/or injury. Recent investigations make use of several confirmational tools to measure what the cell is “thinking” at the DNA level, and also what the cell is “doing,” by exploring alterations in the amount and location of gene products (proteins).
Alterations in protein levels can be measured using Western blotting techniques. Western blotting is an assay that involves exposing an electrical current to a gel and separating proteins on the gel by their molecular weight. After transfer to a membrane, the membrane is exposed to an antibody, which recognizes the protein of interest, allowing the investigator to quantify the amount of protein in a sample. Immunohistochemistry is the technique used to identify the location of proteins in a muscle cell. Skeletal muscle samples are sectioned and probed with fluorescent-labeled antibodies that recognize proteins of interest or dyes that recognize specific structures, such as the nuclei. With these tools, investigators can visualize where a protein of interest is most active in a cell, revealing clues to its mechanism of action and relationship to other proteins.

The greatest challenge to date has been singling out the most critical genes and proteins involved in hypertrophy. To overcome this, scientists have used sophisticated techniques to attenuate the activity of single genes to better understand their roles in complex pathways. RNA interference (RNAi) is one such tool that allows the study of single genes in cell culture and in vivo experiments. This tool uses double-stranded RNA that is synthesized with a sequence complementary to the target gene and subsequently introduced into the cell or organism. Because this exogenous material is recognized as such by the cell or model system, the RNAi pathway is activated, causing a significant decrease in the expression level of the target gene. The effects of this decrease identify the physiological role of the protein product. By not completely abolishing the expression of target genes, RNAi is superior to knockout experiments, resulting in a more “physiologically accurate” system.

The field of epigenetics has grown remarkably over the past few years. Epigenetic traits are inherited phenotypes that result from a change in a chromosome without a change in the DNA sequence. Investigators have categorized those signals that act on chromosomes into three specific categories. These categories include environmental cues, responding signals in the cell that indicate the location of the affected chromosome, and third, the sustaining signal that perpetuates the chromatin change throughout generations. Epigenetics may help us to understand how to optimize nutritional and exercise programs for individuals to obtain the greatest success. Using epigenetics to understand how genes express themselves is a new and exciting field of research for the exercise scientist.

Collectively, great strides have been made in our understanding of the molecular basis of skeletal muscle adaptation and growth. However, as we learn more about the muscle cell and those processes that alter signaling and phenotypical adaptations, our ability to promote positive adaptations will continue to expand. While the field of molecular biology and skeletal muscle is rapidly evolving, it is critical to address each new research question with patience to ensure that data are valid and repeatable. This is particularly important when moving from in vitro to in vivo experimental designs. Continued advances and refinement in the available technology will be critical in completing our quest to fully understand the complex interactions involved in promoting skeletal muscle adaptation.

Research with elite competitive lifters and novel imaging techniques may help to resolve the controversy. Although hyperplasia in humans may not be the primary adaptational response to resistance training, it might represent one that is possible when certain muscle fibers reach a theoretical “upper limit” in cell size. It is possible that very intense long-term training may make some type II muscle fibers primary candidates for such an adaptational response. But even if hyperplasia does occur, it probably only accounts for a small portion (5–10%) of the increase in muscle size.

**Muscle Fiber Transition**

The quality of protein refers to the type of proteins found in the contractile machinery and the muscle’s ability to change its phenotype (i.e., actual protein expression) in response to resistance training, which, in turn, is based on the individual’s genetic profile (i.e., inherited DNA).

Much of the resistance training research focuses on the myosin molecule and examination of fiber types based on the use of the histochemical myosin adenosine triphosphatase (mATPase) staining activities at different pHs. Changes in muscle mATPase fiber types also give an indication of associated changes that are taking place in the MHC content. We now know that a continuum of muscle fiber subtypes exists in humans, ranging from type I, to type IIA to type IIX fibers with interspersing subtypes. In addition, we know that transformation (e.g., type IIX to type IIA) within a particular muscle fiber type is a common adaptation to resistance training. It appears that as soon as type IIX muscle fibers are recruited, they start a process of transformation toward the type IIA profile by changing the quality of proteins and expressing varying amounts of different types, or isoforms, of mATPase. For example, starting with type IIX, an initial transition might be to type IIXA, so that both types of mATPase are expressed in the muscle fiber. Such fibers that co-express more than a single type of myosin isoform are sometimes referred to as “hybrid” fibers. Minimal changes from type II to type I probably occur with exercise training unless mediated by damage and neural sprouting from another alpha motor neuron. For example, sprouting of a type I motor neuron may result in the innervation of a type II fiber that has been damaged with an exercise bout and has lost its neural connection to the fast motor neuron that had been innervating it. Thus, the fiber is said to have been re-innervated by a slow, or type I, neuron. However, the occurrence of such a phenomenon does not appear to be sufficiently frequent to alter absolute type I and type II fiber typing. Thus, a muscle’s basic fiber type profile is determined by genetics, and although it is possible to make transitions within the
type I and type II fiber subtypes due to performance of strength or endurance training, one’s fiber type distribution, as related to the broad categories of type I versus type II fibers, is pretty much set from birth.\textsuperscript{15,40}

**Compatibility of Exercise Training Programs**

The topic of exercise compatibility first came to the attention of the sport and exercise science community when Hickson\textsuperscript{15} showed that the development of dynamic strength may be compromised when both resistance training and high-intensity endurance training are included in a single training program. In contrast, improvements in cardiovascular fitness (VO\textsubscript{2} max) and endurance performance (time to exhaustion at a given submaximal intensity) did not suffer as a result of a combined strength and aerobic training program. In short, a program that includes both strength and aerobic training may limit strength gains, but improvements in cardiovascular fitness and performance are as impressive as those observed when endurance training is performed alone. Subsequent studies seem to confirm Hickson’s original results.\textsuperscript{16,13}

The understanding of exercise training compatibility has focused on what is called concurrent training or simultaneous training of both aerobic performance and strength development. The effects of concurrent training on skeletal muscle are of interest to athletes and exercise scientists alike, as the body tries to adapt to both exercise stimuli. The challenge appears to be directed primarily to the motor units that are used in both training styles.

Studies examining concurrent training using high levels of training frequency and/or intensity for endurance and strength present the following conclusions (Box 4-14):

- Strength can be compromised, especially at high velocities of muscle actions, due to the performance of endurance training.
- Muscular power may be compromised more than strength by the performance of both strength and endurance training.
- Anaerobic performance may be negatively affected by endurance training.
- Development of maximal oxygen consumption is not compromised when a heavy resistance training and aerobic training program are performed.
- Endurance capabilities (i.e., time to exhaustion at a given submaximal intensity) are not negatively affected by strength training.

Few cellular data are available to provide insights into changes at the muscle fiber level with concurrent training. The muscle fibers that are recruited for both activities are faced with the dilemma of trying to adapt to the oxidative stimulus to improve their aerobic function, and at the same time, to the strength training stimulus by adding contractile proteins to increase their contractile force. Recall that endurance training alone typically decreases contractile protein content and fiber size to better enable diffusion of gasses across the muscle fiber. So, what happens to the muscle fiber population when it is exposed to a program of concurrent training? Kraemer et al.\textsuperscript{25} examined changes in muscle fiber morphology over a 3-month training program in physically fit men. All training groups (endurance alone, strength alone, and combined endurance and strength) had a shift of muscle fiber types from type IIX to type IIA. In this study, the number of type IIX muscle fibers was lower after heavy strength training when compared with endurance training, which included both long-distance and interval training. The group completing a concurrent strength and endurance training program showed a sharp decrease in the percentage of type IIX fibers, much like the group performing strength training alone. This may be due to the greater recruitment of high-threshold motor units, those that contain type IIX fibers, with heavy resistance training performed by the strength only and the combined training groups.

Muscle fiber cross-sectional areas demonstrate that changes occur differentially across the continuum of exercise training modalities and are dictated by the type or combination of training stimuli to which the muscle is exposed. That is, when training only to develop strength, all muscle fiber types get larger. But when performing only cardiovascular endurance training, type I muscle fibers atrophy, whereas no size changes are observed in the type II muscle fibers. And when training to concurrently develop strength and

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**Applying Research Practical Applications**

Exercise prescription must take into consideration the demands of the total program and make sure the volume of exercise does not become counterproductive to optimal physiological adaptations and performance. This requires the following steps:

1. **Prioritize the training program and the goals of training.** Do not attempt to perform high-intensity and high-volume strength and endurance training together. Allow for adequate recovery from training sessions by using periodized training programs and planned rest phases.
2. **If you are a strength/power athlete, limit your high-intensity aerobic training.** One can perform lower intensity aerobic training, but high oxidative stress due to high-volume or high-intensity endurance training appears to negatively affect power development.
cardiovascular endurance, no changes are observed in the size of the type I muscle fibers, but increases are seen in the type II muscle fibers. Thus, attempting to train maximally for both muscle strength and cardiovascular endurance can result in different size adaptations in the type I and type II muscle fibers, compared with just a single mode of training.

A multitude of factors (e.g., exercise prescriptions, pre-training fitness levels, exercise modalities) can affect the exercise stimulus and therefore the subsequent adaptational responses. Such factors will affect the muscle cell’s signaling pathways for atrophy or hypertrophy. The majority of studies in the literature have used relatively untrained subjects to examine the physiological effects of simultaneous strength and endurance training. Little data are available regarding the effects of simultaneous strength and endurance training using previously active or fit individuals, who are able to tolerate much higher intensity exercise training programs. It appears that maximal simultaneous training may be particularly detrimental to optimal adaptations in muscle size, strength, and power, possibly due to overtraining with such high levels of work, volume of exercise, and intensity. Interestingly, aerobic capacity appears to be the least affected by such simultaneous training. If concurrent exercise training is properly designed, it may just require a longer time for the summation of physiological adaptations to occur; most of the studies to date have examined training programs lasting no more than 2 to 3 months. Based on the available data, it seems that one cannot have optimal adaptation to both modes of training. In addition to longer training program durations, other factors may be important to the successful concurrent development of strength and improved aerobic fitness. For example, both program periodization (varying the volume and intensity of the training) and prioritization (prioritizing what goals will be focused on in a training program) of training may allow individuals to adapt successfully.

**Quick Review**

- Only muscle fibers that are recruited with the exercise will adapt to exercise stimuli.
- Adaptations to endurance training are related to the need to better deliver and utilize oxygen, in order to enhance muscular endurance.
- The growth in the absolute size of a muscle that results from resistance training is primarily due to the increase in the size of the individual muscle fibers rather than an increase in the number of fibers.
- When endurance and strength training programs are performed concurrently, the body tries to adapt to both of the exercise stimuli, but findings suggest that the body will experience greater improvements in endurance performance than strength.

**CASE STUDY**

**Scenario**

You are a strength and conditioning specialist working in the weight room at the Olympic Training Center in Colorado Springs, CO. You are trying to improve your strength and conditioning program for aspiring Olympic-level women figure skaters.

The coach has asked you what can be done to help the women better land their jumps in their routines. With the scoring system requiring the skaters to perform triple and quad jumps to score high, more power training is needed. Currently, you have each athlete on an individualized resistance training program. The program is periodized to complement each skater’s on-ice training.

**Questions**

- What type of muscle action produces the force needed by skaters to perform jumps?
- What takes place in the muscle when a skater bends her knees in preparation for a jump?
- What provides the power for the upward movement in a jump?
- What type of muscle strength provides the skater with the braking system to absorb the load coming down on the leg during a landing?
- What types of training need to be included in the weight training program?

**Options**

Jumps are primarily a coordinated set of muscle actions that use the stretch-shortening cycle to produce the power output. The concentric action in the vertical jump that benefits from the stretch-shortening cycle propels the skater upward. Muscular power appears to be the primary muscular performance feature to propel the skater into the air, and the maximal eccentric strength determines the ability of the skater to brake upon landing on the ice. Thus, the training program needs to include stretch-shortening cycle or plyometric training to help with jump height and eccentric training to assist in landing of jumps.

**Scenario**

You are a personal trainer for a triathlete getting ready for the ironman competition. In the past she has had trouble...
Part II  Exercise Physiology and Body Systems

CHAPTER SUMMARY

Neural recruitment of skeletal muscle is what causes force production and movement in the human body. Several other systems of the body (e.g., skeletal, neural, immune) interact with skeletal muscle to sustain its health and assist it in force generation and movement. Skeletal muscle is highly organized, from the connective tissue that surrounds the intact whole muscle, to the connective tissue that keeps the contractile proteins of the sarcomere in place for optimal myofibril interactions. This connective tissue also contributes to the force and power production of muscle due to an elastic component, which upon stretch and recoil adds force to the muscle contraction. The elastic component in skeletal muscle provides the basis for plyometric training or for training using the stretch-shortening cycle. Skeletal muscle is a target of all training programs, whether for sport performance or health and fitness, and is highly plastic or adaptable to the exercise stimuli. Skeletal muscle is composed of different muscle fiber types, with each type being designed to accomplish different kinds of tasks, ranging from prolonged activities of low intensity (e.g., type I fibers), to short, burst activities requiring tremendous force production (e.g., type II fibers).

Resistance exercise typically results in muscle hypertrophy, whereas endurance training results in no change or even a decrease in muscle fiber size. Combining resistance training with endurance training will result in limited hypertrophy of the type I muscle fibers, with increases in size observed primarily in type II muscle fibers. Understanding skeletal muscle structure and function will allow a better understanding of the many methods of training and therapy that are used to enhance function, performance, and health. In the next chapter, we will examine how the nervous system controls muscle function and how it adapts to training.

Options

Although high-intensity endurance training has been shown to potentially interfere with power and strength development, few data are available to suggest that this will hurt endurance performances. This athlete is truly at a risk for connective tissue injury, and resistance exercise will strengthen the connective tissue, making it better able to cope with the high volume of endurance training needed to compete and train for an ironman competition. The changes in the muscle would likely result in more protein in both the contractile and connective tissues with the type I fibers remaining unchanged in size but more resistant to protein loss. Overall the performance of resistance training will not decrease endurance performance and will help to decrease the chance of injury.

Questions

• Is it true that lifting weights will hurt endurance performance?
• What might be the expected adaptations in muscle when lifting weights and performing high volumes of endurance exercise?
• What are the benefits of combining both types of training protocols for this athlete?

REVIEWS QUESTIONS

Fill-in-the-Blank

1. ________ proteins are those that are not involved with the contraction process, but hold contractile proteins in close proximity to each other for optimal myosin–actin binding interactions.
2. Tension receptors, called __________, which are located in the tendon of skeletal muscle, sense the amount of force in the tendon created by the skeletal muscle.
3. A form of exercise that utilizes the stretch-shortening cycle, termed __________, helps in the development of muscular power.
4. The acquisition of __________, formed from satellite cells that are found between the mature muscle fiber's membrane and its basal lamina, is required to support muscle fiber hypertrophy.
5. With endurance exercise training only, the size of type I (slow-twitch) muscle fibers __________ to reduce the distance between the capillaries and the mitochondria.

Multiple Choice

1. Which protein of the sarcomere has the heads that bind to active sites?
   a. Actin
   b. Myosin
   c. Troponin
   d. Tropomyosin
   e. Titin
2. A marathon runner at the Olympic Games would have a high percentage of what type of fiber?
   a. Type II
   b. Type IIC
3. What technique is used to obtain a small sample of muscle with a needle through the skin?
   a. Percutaneous muscle biopsy
   b. Subdermal musclectomy
   c. Myofibril biopsy
   d. Percutaneous sarcomere removal
   e. Incision musclectomy

4. What physiological adaptations occur when muscle fibers are recruited to perform an endurance exercise workout?
   a. The number of capillaries increase.
   b. The capillary density in the type I fibers increases.
   c. The number of mitochondria increases.
   d. The concentration of myoglobin increases.
   e. All of the above

5. Which of the following is an example of an isokinetic muscle action?
   a. Lifting a barbell in a biceps curl
   b. Lowering a barbell in a biceps curl
   c. Exerting force against an object that does not move
   d. A movement during which the velocity is held constant
   e. The action of the triceps during a biceps curl

True/False
1. At rest, the proteins troponin and tropomyosin cover the active sites on actin molecules, preventing the myosin heads from binding to the active sites.
2. When training for both strength and endurance at the same time, muscle fibers that are recruited improve their aerobic function as when training only for endurance.
3. Fast-twitch muscle fibers are characterized by the ability to resist fatigue and produce relatively small amounts of force.
4. The growth in the absolute size of a muscle due to resistance training is primarily due to the increase in the number of individual muscle fibers.
5. With endurance training, fiber types do not change from type I to type II.

Critical Thinking
1. During a normal weight training back squat, what types of muscle actions take place during a repetition, and what type of muscle action will limit the maximal amount of weight that can be lifted for one complete repetition?
2. Describe the skeletal muscle adaptations to endurance training.

KEY TERMS
A band: the area in a sarcomere where actin and myosin overlap; it represents the length of the myosin filaments
actin: a thin myofilament that has active sites on it capable of interacting with the myosin protein to produce muscle force
active site: place on the actin filament where myosin heads can bind
active tension: the amount of force generated by the actomyosin complexes formed within a muscle in response to neural stimulation
antagonistic muscle: a muscle that contracts and acts in physiological opposition with the action of an agonist muscle
concentric (contraction): muscle activation characterized by muscle shortening
concurrent strength and endurance training: training for both strength and endurance at the same time
connectin (titin): a noncontractile protein of the sarcomere that connects the Z line to the M line, stabilizes myosin in the longitudinal axis, contributes to the elastic component of the muscle fiber, and limits the range of motion of the sarcomere
crossbridges: small projections on the myosin filament that interact with actin to cause muscle contraction and force production
cytosol: the liquid portion of the contents inside living cells, including muscle fibers
delayed-onset muscle soreness (DOMS): pain several hours to several days after an exercise bout that is a symptom of muscle damage

DHP receptors: proteins found in the T-tubules that act as voltage sensors when the electrical impulse travels along the T-tubule
dynamic constant external resistance: isotonic muscle contraction; describes the type of muscle activity when exercising with external resistances in activities such as lifting weights
eccentric: muscle elongation while the muscle is activated and producing force
elastic component: the recoil force in the muscle after being stretched, due to noncontractile portions of muscle
endomysium: the connective tissue surrounding each individual muscle fiber
epimysium: the external layer of connective tissue surrounding the whole muscle
fasciculus (pl. fasciculi): a small bundle of muscle fibers
fast-twitch fibers: muscle fibers that develop force very rapidly, demonstrate high force production capability, are less resistant to fatigue than slow fibers, have a relatively small number of mitochondria, and have a limited capacity for aerobic metabolism
force–time curve: a graph illustrating the force production over different lengths of time
force–velocity curve: a graph illustrating the influence of changing the velocity of movement on the muscle’s force production capabilities
Golgi tendon organ: force receptors located in the tendon of skeletal muscle
H zone: the region in the middle of the sarcomere, which contains only myosin
heavy chains: protein components that form the myosin head and a portion of the tail of a myosin molecule
hyperplasia: an increase in the number of cells of a tissue
I bands: the light bands of the sarcomere that contain only actin
intrafusal fibers: modified muscle fibers arranged parallel to normal muscle fibers that contain muscle spindles
isoinertial: (isotonic) an exercise movement with a fixed resistance and variable velocity
isokinetic: a movement characterized by muscle force exerted at a constant velocity
isometric: a muscle contraction characterized by tension in the muscle with no change in muscle fiber length
isotonic: a muscle contraction characterized by muscle shortening against a constant external load, such as during the lifting of a barbell
kinesthetic sense: awareness of the body’s position in space
learning effect: mastery of the motor unit recruitment pattern for a specific skill or movement due to repeatedly performing the skill or movement
light chains: protein components of the myosin filament that form the hinge portion of a myosin molecule
M line: proteins in the middle of the H zone that hold the myosin filaments in place
muscle fibers: long multinucleated cells that contain myofibrils that contract when stimulated
muscle spindle: a stretch receptor arranged in parallel to muscle fibers that monitors stretch and length of the muscle
myofibril: the portion of the muscle containing the thin and the thick contractile filaments
myonuclei: nuclei located beneath the sarcolemma of the muscle fiber
myosin: the contractile protein in the myofibril that has crossbridges that can bind to actin to cause tension development
myosin ATPase: an enzyme found on the globular heads of the myosin crossbridges, which breaks down ATP to release energy needed for muscle contraction
myosin ATPase staining: a method for distinguishing type I and type II human muscle fibers and their subtypes based on a staining for the enzyme that hydrolyzes ATP
nebulin: a noncontractile protein that stabilizes actin; it is localized to the I band and extends from the Z line
neuromuscular system: the close functional relationship between the nerves and skeletal muscle
nuclear domain: the area within a muscle fiber that each myonucleus controls
passive tension: the force due to the elastic elements of a muscle in the absence of neural stimulation
percutaneous muscle biopsy technique: a technique in which a hollow needle is inserted through the skin to obtain a sample of muscle
perimysium: the fibrous connective tissue surrounding each fasciculus of skeletal muscle fibers
periodization: varying the volume and intensity of training in a planned manner
plyometric exercise: a form of exercise that uses the stretch-shortening cycle to help in the development of muscular power
power: product of force exerted by the muscle and the vertical distance the load is displaced divided by time, or force times velocity
power stroke: the movement of the myosin crossbridges that pulls the actin filaments during the shortening of muscle
prioritization: a principle of prioritizing what goals will be focused on in a training program
progressive overload: a gradual increase in the intensity or volume of exercise
proprioceptors: sensory receptors located within the muscles, tendons, and joints that provide information about the body’s position by monitoring the length of muscle, force produced by muscle, and joint position
ratchet movement: a movement in which the myosin head swivels at its hinged pivot point to a new angle, pulling the actin over the myosin filament and causing the sarcomere to shorten
ryanodine receptors: calcium channels located on the membrane of the sarcoplasmic reticulum, which open when activated by voltage sensors of the T-tubules
sarcomere: the smallest or most basic contractile unit of skeletal muscle capable of shortening
sarcoplasmic reticulum: membranous organelle found within the muscle fiber that stores the calcium needed for muscle force development
satellite cells: cells found under the basal lamina membrane of mature muscle fibers that are the source of new myonuclei
skeletal muscle: muscle that is connected at both ends to a bone
sliding filament theory: a theory of muscle contraction describing the sliding of the thin filaments (actin) past the thick filaments (myosin) by the attaching and detaching of the heads of myosin molecules to the actin filaments, drawing each end of a sarcomere toward each other
slow-twitch fibers: muscle fibers that contain large amounts of oxidative enzymes are highly fatigue-resistant and do not develop force as quickly as type II fibers

speed-strength: training that is focused on force production at higher velocities and lighter resistances to improve power

steric blocking model: the process of covering the active sites on the actin filament by tropomyosin preventing interaction with the myosin filament keeping the muscle fiber in a nonactivated condition

strength curve: a graph of the amount of force produced over a range of motion

stretch-shortening cycle: muscle elongation followed by a rapid muscle shortening

striated muscle: the striped appearance of muscle that is created by the arrangement of myofibrils in sarcomeres

T-tubules: a membrane-enclosed tunnel allowing electrical impulse to spread through the muscle fiber

tendons: a band of tough, inelastic fibrous tissue that connects a muscle with bone

titin (connectin): a noncontractile protein of the sarcomere that connects the Z line to the M line, stabilizes myosin in the longitudinal axis, contributes to the elastic component of the muscle fiber, and limits the range of motion of the sarcomere

tropomyosin: a protein covering the actin binding sites when muscle is at rest that prevents the myosin crossbridge from touching active sites on actin; when muscle is stimulated to contract, it moves, exposing the active sites so myosin and actin can interact to shorten

troponin: a protein, associated with actin and tropomyosin, that binds Ca²⁺ and initiates the movement of tropomyosin on actin to allow the myosin crossbridge to touch active sites on actin and initiate contraction

type I muscle fibers (slow-twitch fibers): muscle fibers that contain large amounts of oxidative enzymes are highly fatigue-resistant and do not develop force as quickly as type II fibers

type II muscle fibers (fast-twitch fibers): muscle fibers that develop force very rapidly, demonstrate high force production capability, are less resistant to fatigue than slow fibers, have a relatively small number of mitochondria, and have a limited capacity for aerobic metabolism

variable resistance: strength training equipment in which the resistance varies throughout the range of motion to better match the strength curve

Z line: a band delineating the ends of the sarcomere

REFERENCES


**SUGGESTED READINGS**


**CLASSIC REFERENCES**


