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Introduction

Skeletal muscle is necessary for every movement of the human body. The physiology of skeletal muscle is the foundation for understanding human movement and muscle injuries. Skeletal muscle has one primary task; namely, to transform chemical energy into mechanical energy. Simply stated, muscle converts the chemical energy of our diet into fuel for muscle action and then, when stimulated by the nervous system, skeletal muscle uses this fuel to produce mechanical energy in the form of muscle action.

In this chapter, we review each of these components of skeletal muscle physiology and how they relate to practical applications in nutrition, rehabilitation, and sport. We begin with metabolism and the process of skeletal muscle energetics. The details of the skeletal muscle machinery and control of muscular action are the focus of the remaining sections of this chapter.

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Metabolism

Macronutrient Use, ATP Production, and Energy Pathways

In order to fully appreciate the physiology of skeletal muscle, you must know how muscle transforms the food we eat into the muscular action that produces force and motion. This flow of energy is called skeletal muscle energetics and it focuses on the creation and use of adenosine triphosphate (ATP). ATP is the energy currency of the body. In skeletal muscle, ATP is responsible for contraction and relaxation of skeletal muscle.

Skeletal muscle energetics begins with macronutrient consumption. When ingesting macronutrients, we start to convert food into energy—a process called metabolism. Macronutrients, such as a combination of carbohydrate, fat, and protein, are reduced to their basic form primarily of glucose, fatty acids, and amino acids, respectively, when used in metabolic processes to produce ATP, the energy currency of the body. Importantly, glucose and fatty acids are used more commonly than protein for sustained muscular action. This preserves protein and its constituent amino acids as the building blocks of skeletal muscle. Because protein serves in a lesser capacity as energy precursor, the focus of skeletal muscle energetics is on carbohydrate and fats.

To more clearly understand how macronutrients promote the production of ATP and subsequent performance in various activities, such

as endurance exercise, let us consider a classic study [1]. In this experiment, participants were fed a high-fat diet (5% or less carbohydrate intake), normal diet (met the recommended daily allowance of carbohydrate, lipid, and protein), and a high-carbohydrate diet (82% of calories from carbohydrates). When tested in a repeated-measures design, all subjects underwent all feeding conditions for 3 days with recovery days in between conditions and bouts on a cycle ergometer to exhaustion. Not only did the high-carbohydrate diet produce the best glycogen storage (i.e., stored glucose in skeletal muscle that was six times greater than the high-fat diet), but time to exhaustion when cycling was also three times longer than the high-fat diet. The normal diet fell in between. Therefore, when thinking about optimizing endurance performance, carbohydrate stored in skeletal muscle and even in the liver is paramount to sustaining high-intensity muscular actions, especially when exercising or competing longer than 1 h. Other studies have since supported this [2, 3].

On the other hand, high-fat *meals* may help boost endurance exercise as well, but only as long as an adequate carbohydrate *diet* is followed. For instance, ingesting a high-fat *meal* (1007 ± 21 kcal/meal in total calories; 30% carbohydrates, 55% fat, and 15% protein) 4 h before an exercise bout after 3 days of a high-carbohydrate *diet* (2562 ± 19 kcal/day in total calories: 71% carbohydrates, 19% fat, and 10% protein) and with tapered training, demonstrated the greatest time until exhaustion versus ingesting a high-carbohydrate *meal* (1007 ± 21 kcal, 71% CHO, 20% F and 9% P) after the 3 days of high-carbohydrate intake [4]. Interestingly, just prior to exercise, in the high-fat meal group, participants ingested a maltodextrin jelly or placebo. In the high-carbohydrate meal condition, participants ingested only a placebo. From a practical viewpoint, the above study underscored the need for sufficient carbohydrate intake prior to exhaustive exercise to maintain strong skeletal muscle action with the addition of a high-fat meal showing some benefit. Thus, skeletal muscle macronutrient needs during sustained muscular action are nicely supported by both carbohydrate

and fat, with a preference for carbohydrate as the exercise intensity increases. This is excellent information to remember in view of optimizing skeletal muscle physiological properties. Note that because high-intensity, short-duration exercise is usually much less than 1 h, a reliance on carbohydrate is the norm with almost no risk of glycogen depletion.

The production of ATP from various macronutrients follows three primary energy pathways that use various substrates (e.g., carbohydrate, fat, phosphocreatine (PCr), among others) to produce ATP. These energy pathways, from fastest to slowest rate of ATP production, are: (1) ATP-PCr or phosphagen, (2) glycolytic or lactic acid, and (3) aerobic processes [5]. Said another way, these three energy-producing pathways are preferentially favored during differing durations of intense exercise of approximately 0–15 s, 16–90 s, and >90 s, respectively. Keeping these pathways and associated timeframes in mind during the following discussion about skeletal muscle physiology, helps maintain a helpful perspective on human performance.

On elaborating individual energy pathways, we find that, in short, the ATP-PCr pathway, or “fast” ATP-producing process, utilizes a coupled reaction in the cytoplasm whereby the breakdown of PCr by creatine kinase to creatine (C) + inorganic phosphate (P_i) yields energy used to synthesize ATP from the recombination of adenosine diphosphate (ADP) + P_i . The glycolytic system, also housed in the cytoplasm and a “fast” ATP-producing process that may be described as anaerobic or aerobic, relies mostly on glucose degradation from blood or stored skeletal muscle glycogen with the end products of lactic acid (which spontaneously becomes lactate + H^+ if anaerobic ATP production is favored) and/or pyruvic acid (which spontaneously becomes pyruvate if aerobic ATP production is feasible). To summarize, if exercise is “slow” or low intensity in nature, then pyruvate production is favored whereas, the buildup of lactate + H^+ in tissue and blood occurs when exercise is “fast” or high intensity. Not surprisingly, various glycolytic enzymes, some rate limiting, control the glycolytic process and are namely: hexokinase

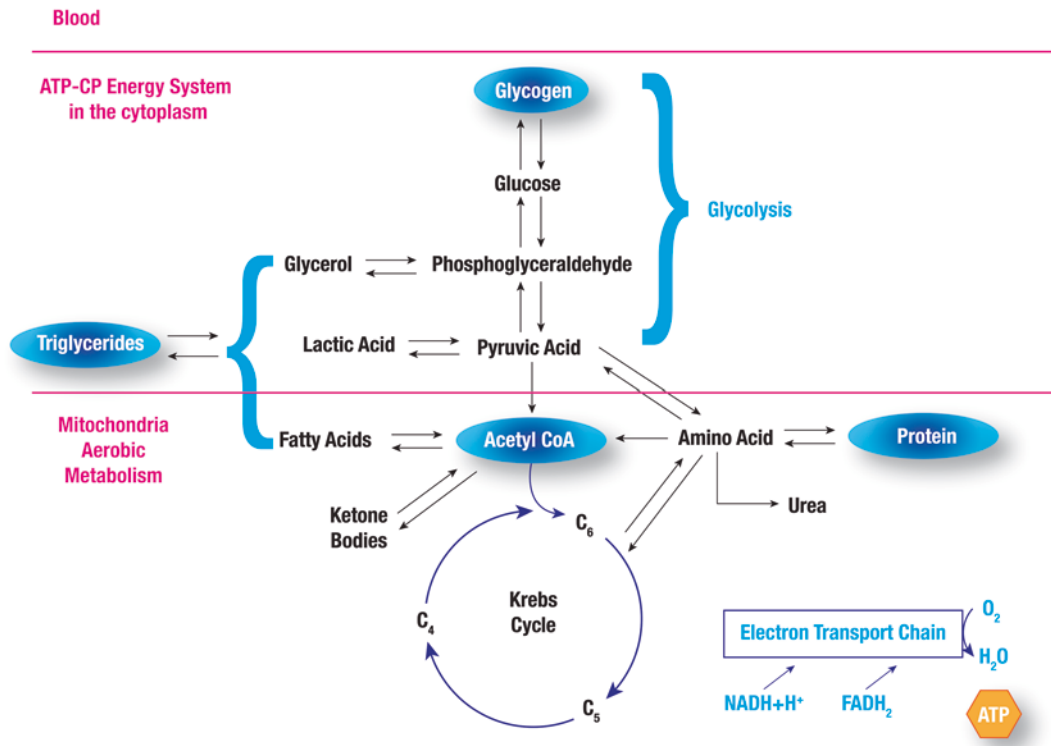


Fig. 2.1 The interaction of carbohydrates, triglycerides, and proteins, termed macronutrients, in the metabolic pathway. Note that all macronutrients have the capability to become Acetyl CoA, the common entryway to the

oxidative energy cycle. Likewise, each macronutrient has the potential to become a different substrate in the metabolic process. Thus, metabolic precursors are highly inter-related. (Courtesy of Elizabeth A. Drum)

(HK), phosphofructokinase (PFK), and lactate dehydrogenase (LDH).

The aerobic energy system takes place in the mitochondrion of a skeletal muscle cell and begins when pyruvate (from aerobic glycolysis) is converted to Acetyl CoA and combines with oxaloacetate (OOA) within the organelle as part of the Krebs or citric acid cycle [5]. Both carbohydrate and fat (with negligible input from protein sources) are utilized at rest and during exercise, depending on intensity (i.e., low to moderate), to “run” aerobic processes. ATP creation, also known as oxidative phosphorylation, occurs in the electron transport chain (ETC) via H⁺s shuttled from the Krebs cycle to the ETC via electron shuttles (i.e., nicotinamide adenine dinucleotide, NAD⁺; and flavin adenine dinucleotide, FAD). Therefore, ATP is synthesized by transferring electrons from NADH and FADH₂ to oxygen in the ETC [5]. Keep in mind that as a person

exercises on a regular basis, more mitochondria, denser muscle mass, improved blood flow, and greater aerobic (and anaerobic) enzymes are upregulated. With cessation of exercise these changes reverse. Figure 2.1, below, depicts the interaction of the aforementioned energy pathways and the use of carbohydrates, fats, and proteins in the metabolic synthesis of ATP.

Muscle Fiber Types

Having reviewed macronutrient utilization in skeletal muscle cells in accordance with the energy pathways to produce ATP for sustained muscular action, we now transition to a discussion of muscle fiber types. Skeletal musculature is composed of slow- and fast-twitch (ST and FT, respectively) fibers or cells. ST are also known as type I or slow-oxidative (SO) cells and rely

Table 2.1 Skeletal muscle/fiber classification

System 1	Type I	Type II	Type Iix
System 2	Slow twitch (ST)	Fast twitch a (FTa)	Fast twitch x (FTx)
System 3	Slow oxidative (SO)	Fast oxidative/glycolytic (FOG)	Fast glycolytic (FG)
<i>General attributes of fiber types</i>			
Oxidative ability	Excellent	Good	Poor
Glycolytic ability	Poor	Excellent	Excellent
Contractile speed	Slow	Fast	Fast
Fatigue resistant	Excellent	Good	Poor
Unit strength	Poor	Excellent	Excellent

primarily on the aerobic energy system to produce ATP for muscle action. FT fibers, also known as type II fibers, are further categorized as FTa and FTx or type IIa and type Iix, respectively. FT fibers rely more on the ATP-PCr and anaerobic glycolytic energy pathways to sustain high intense action with a high rate of ATP production.

In general, type II muscle fibers are larger, transmit action potentials faster, have greater activity of the ATP splitting enzyme myosin ATPase, release and uptake calcium ions (Ca^{2+}) more rapidly, and exhibit a high-rate crossbridge turnover (between actin and myosin or skeletal muscle myofibrils) than type I fibers, which exhibit all the aforementioned characteristics albeit at a protracted rate. Additionally, type II fibers tend to store more carbohydrate and are favored during high-intensity and/or power-strength exercises. Type I fibers, on the other hand, favor aerobic or low- to moderate-intensity exercise and rely on both carbohydrate and fat as fuel (note, both of these macronutrients are stored in type I fibers). Additionally, type I fibers have a greater concentration of mitochondria and capillaries versus type II fibers [5]. See Table 2.1 for a side by side comparison of the various fiber types.

Skeletal Muscle Force Production

We have seen that skeletal muscle physiology creates the opportunity for a plethora of muscle actions based on the combination of fiber types and energy pathways available. With the varied pathways to manufacture ATP, there is balance

between low-energy requirements (e.g., rest or low-moderate-intensity exercise) and high-energy needs (e.g., high-intensity exercise). Notably, skeletal muscle can be selectively recruited to the muscular action needed based on fiber type and/or size of the fibers. Generally, as alluded above, type I fibers are smaller and therefore innervated by smaller alpha motor neurons, the primary neurons responsible for initiating muscular action. The size principle implies that during force development of progressively increasing magnitude, type I fibers are recruited first, followed by type IIa, and finally type Iix, the most powerful fiber set [6].

Therefore, the discussion about force production begins with acknowledging that the process of developing force in muscle is efficiently regulated, especially by increasing motor unit recruitment and increasing the frequency of motor unit discharge [5]. Recognize that a motor unit is, by definition, a single motor neuron and all the fibers it innervates—from a few to thousands [5]. The following section introduces the properties of skeletal muscle that allow it to exert force, namely the sliding filament theory (SFT). Underlying the mechanics of force production are the omnipresent metabolic processes that maintain the ATP supply for sustainable muscle action and, therefore, force output. Said another way, you get work (W) output, which equals force (F) times distance (D) or $W = F \times D$. This is a useful equation to remember when discussing muscular force production in that internal muscular force normally equals an external result, such as the movement of a weight stack or self-propulsion during walking or running.

A Few Muscle Action Definitions

A muscle fiber contraction or shortening of the muscle belly is a complex process that involves cellular and chemical interactions. The result is movement within the myofibrils in which thin (actin) and thick (myosin) filaments slide past and over one another. A muscular action may cause a shortening of a sarcomere, the smallest contractile unit of a muscle fiber, under tension (and ultimately a shortening of the muscle or contraction). This is termed as concentric muscle action. When the filaments slide in a direction causing a lengthening of the sarcomere under tension, an eccentric muscle action is created. When concentric and eccentric muscle actions are performed continuously, one after the other, such as during a full range of motion movement during a bench press exercise using free weights, this is termed isotonic exercise. Isometric muscle action, another relatable term, is created when thick and thin filaments attempt to slide in either direction, but a fixed or static length is held for a period of time. In other words, there is no lengthening or shortening of the sarcomere in a static action, even though energy is continuously being utilized to fuel the isometric event; maintaining your posture while sitting is a good example of static torso action. Another pertinent muscle movement to mention is isokinetic action in which muscle fibers exert a near constant, maximal force throughout a preset constant speed as seen in the use of an isokinetic Cybex machine in the rehabilitation setting.

Collectively, all the muscular actions mentioned above provide a brief overview of how specificity in resistance training methods can be incorporated with the principle of overload to create a thoughtful training or rehabilitation plan. Taken together, overload is the progressive increase in resistance on a particular muscle group over time in a systematic and specific fashion to elicit muscular force application and adaptation changes. Discussion of the role of resistance training in rehabilitation and performance training plans is covered in the later chapters of this book.

Sliding Filament Theory (SFT)

Working independently in 1954, H. E. Huxley [7] and A. F. Huxley [8] were involved in establishing the SFT. The theory explains how fixed-length thick (myosin) and thin (actin) filaments move in relation to each other, resulting in a change in muscle length and subsequently force production. In 1957, A. F. Huxley [9] further elaborated this model with a theory of crossbridge behavior which offered insights into how muscular force application is accomplished via internal, microscopic force application mechanisms. We have already established that metabolically force cannot be produced and sustained without macro-nutrient- or substrate-derived ATP. This primary energy structure is what interacts with muscle fiber myofilaments, actin and myosin, to allow the sliding filament model to “run” and cross-bridge formation to unfold. Force happens when muscular action occurs (either concentric or eccentric). However, neurological input, discussed later in this chapter, is necessary to the initiation of muscle action via Ca^{2+} release into the sarcoplasm via specific structures. With that in mind, we turn to a few more characteristics of muscular force generation before returning to a more detailed discussion of the SFT.

Types of Muscular Action Relationships

Having introduced the SFT, where microscopic force application occurs, let us discuss a bit more about types of muscular interactions. Keep in mind that the term contraction can be misleading when discussing muscle properties and force-generating capabilities. Contraction of skeletal muscle seems to denote primarily a shortening of the fibers. However, in this chapter, we want to underscore that it indicates a shortening, lengthening, retention of the same length, or a combination of these depending on external loads and the desired activity. Therefore, a muscular *contraction* more accurately reflects a muscular action, than a contraction per se. Following are a few

muscle fiber relationships that will help round out a review of muscular force production or the lack thereof.

Length–Tension Relationship

The length of a muscle fiber relative to its optimal length is critical in determining the amount of force or tension that can be developed. The optimal length can be defined as the length of the sarcomere, the smallest contractile unit of a fiber, that provides ideal overlap of thick and thin filaments [10]. Therefore, when optimal length is obtained in a sarcomere, normally around 90° in a joint, the greatest amount of force production is possible because of optimal actin–myosin interaction. Hence, when a muscle is shorter or longer than *optimal* length, there is impairment in maximal or optimal force production. For additional information concerning this type of relationship, especially in active muscle, the reader is directed to review Rassier et al.'s paper [11] titled *Length dependence of active force production in skeletal muscle*. Figure 2.2, below, illustrates various knee angles and the angular position that elicit the greatest muscular tension or strength.

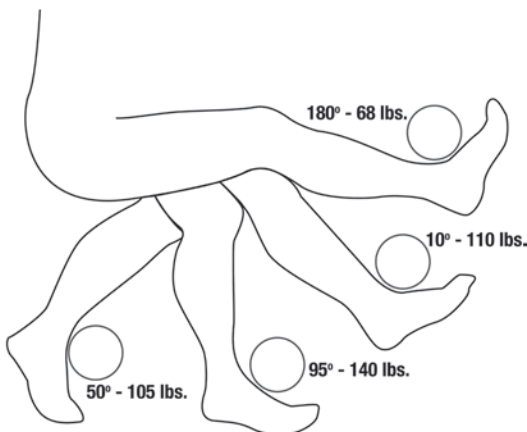


Fig. 2.2 Length–tension relationship of various joint angles, such as the knee joint, where at an angle of 95° , tension within fibers is highest or strength mobilization is greatest. Said another way, range of motion provides a basis for suboptimal and optimal crossbridge interaction. (Courtesy of Elizabeth A. Drum)

Force–Velocity Relationship

In accordance with the length–tension discussion, force–velocity variables also change the dynamics of skeletal muscle interaction. Simply stated, the force–velocity relationship indicates that as the speed of a muscular action increases, force production drops and vice versa [10]. This can be important during the rehabilitation of a patient or performance training of a client to insure that force development is optimized for whatever the outcome goal might be. For instance, to maximize force production in skeletal muscle, slower and controlled movement is probably warranted to optimize progressive overload manifested as greater force development in the musculature. On the other hand, if the goal is to improve speed or endurance, then it might be necessary to add a speed component to the performance of muscular actions along with understanding that force in the form of resistance needs to decline. In other words, the force–velocity relationship should guide training and rehabilitation to reflect that sport-specific or even every-day life activities are not slow and controlled but rather quick and sometimes unanticipated. Figure 2.3 showcases the force–velocity relationship.

Motor Innervation

Neuromotor System Arrangement

Recall that to initiate the SFT, sufficient neuromotor stimulus had to be realized. Let us look at this in a bit more detail but still from a big picture perspective. Most efferent (“output”) electrical signals begin with central nervous system (CNS) involvement and end with peripheral nervous system (PNS) activation, while afferent (“input”) signals relay sensory information from peripheral areas to the spinal cord and CNS. Recall, then, that the human nervous system is made up of primarily the CNS (i.e., brain and spinal cord) and PNS (i.e., nerves that transmit electrical waves to and from the CNS) [5]. In relation to skeletal muscle physiology, CNS and PNS input both travel to the muscle fiber to influence muscular

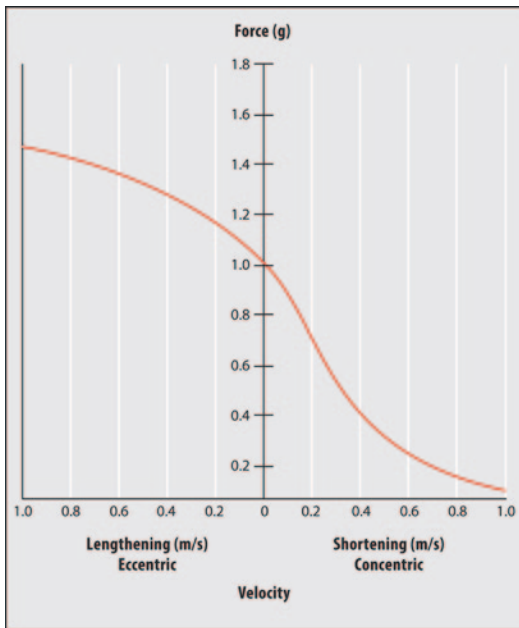


Fig. 2.3 The force–velocity relationship of muscle action. During eccentric action, as the speed of fiber lengthening under tension increases, so does muscle tension. The opposite is true during concentric action or shortening of the fiber under tension whereby tension decreases as the speed of fiber shortening increases. (Courtesy of Elizabeth A. Drum)

action at the neuromuscular junction effecting the action potential (AP) and motor unit properties.

The SFT—Action Potential (AP) and Muscle Fiber Motor Unit Integration

The primary mechanism, physically responsible for force production, in the SFT is the filaments themselves. This process of sliding filaments is primarily regulated by afferent and efferent neurological signals via APs, where sodium (Na^+) and potassium (K^+) are exchanged across neuronal and muscle cell membranes. Eventually, the AP is propagated deep inside a muscle fiber, via transverse tubules (T-tubules), and triggers Ca^{2+} release from the sarcoplasmic reticulum (SR) into the sarcoplasm. Ca^{2+} then binds with troponin, a protein on actin. Suddenly troponin, through a conformational change, pulls another protein, tropomyosin—a long, winding, and thin

stranded structure—off actin’s active sites. Myosin’s energized heads interact with actin and excitation–contraction coupling ensues [10]. What follows are definitions and phases of muscle action related to the SFT—with all its moving parts in a regulated and force-producing sequence.

Constituent Structures of the SFT

Myofibrils

Each muscle fiber contains several hundred to several thousand myofibrils. Myofibrils are arranged in a three-dimensional mosaic pattern and contain the basic contractile structures or proteins—actin and myosin (also known as thin and thick filaments, respectively, a common theme we identified prior)—of a muscle fiber. Additionally, recall that the smallest contractile unit of a muscle fiber is termed the sarcomere which is made up of thin and thick filaments.

Microscopically, a distinctive striped or striated appearance is observed when viewing myofibrils. These striations are apparent due to the thin and thick structural appearance of muscle. The mid-portion of a sarcomere that appears to be dark is known as the A-band and is formed because both thick and thin filaments overlap in this region. The lighter areas on the outer ends are known as I-bands. The lighter appearance occurs because only thin filaments are located in this range. The H-zone is located within the central region of the A-band, where no thick and thin filament overlap is evident. A dark line in the middle of the H-zone is termed the M-line. The M-line is composed of proteins that help aid the sarcomere in maintaining spatial orientation as the fiber lengthens and shortens (i.e., relaxes and contracts, respectively). The I-band is interrupted by a dark stripe, another protein structure, referred to as the Z-disk or Z-line, which serves as an anchor or attachment point for actin.

As the sarcomere is the basic fundamental contractile unit of a muscle cell, each cell is composed of a plethora of sarcomeres joined end to end. To summarize, each sarcomere contains the following aforementioned regions: I-bands (light

zone), A-band (dark zone), H-zone, and M-line. Furthermore, sarcomeres are joined together at the Z-disks with the help of two proteins, titin and nebulin, which help provide points of attachment and stability for thin filaments. Interestingly, titin is now considered a possible third filament, serving as a “spring-like” attachment to the Z-line along with actin [12]. Following are more detailed descriptions of the individual types of myofilaments.

Thick Filament (Myosin)

The principal protein of the thick filament is myosin. Myosin accounts for nearly one half to two thirds of the total myofibrillar protein. Each myosin filament is typically formed by 200 or more myosin molecules. Myosin is a hexameric molecule consisting of one pair of myosin heavy chains and two pairs of light chains. These proteins twist together with one end forming a globular head, called the myosin head. Within each thick filament, many myosin heads are formed and protrude to form crossbridges that interact with specialized activation sites on the thin filament during muscular contraction. An array of fine filaments composed of titin extend from Z-disk to M-line that aid in stabilizing the myosin filaments along the longitudinal axis [10].

Thin Filament

Each thin filament is composed of three different proteins—actin, tropomyosin, and troponin. However, thin filament is often referred to simply as an actin filament. Thin filament has one end inserted into the Z-disk with the aid of nebulin anchoring the filament in place and the opposite end extending to the center of the sarcomere. Also, keep in mind that titin also interacts with actin and quite possibly helps anchor it to the Z-line [12]. Furthermore, thin filament is located in the space between thick filaments [5].

Individual actin molecules are globular proteins (known as G-actin) that join together to form

the backbone of thin filaments. Two strands of actin-derived filaments twist in a helical pattern. Twisting around the actin strands is a tube-shaped protein called tropomyosin. Yet another protein, a more complex protein called troponin, is attached at regular intervals to both actin and tropomyosin. Troponin and tropomyosin work together along with Ca^{2+} ions to maintain relaxation or initiate an action of the myofibril. Keep in mind that actin and myosin work in unison to help promote muscle movement, such as an isotonic action, which is the cornerstone of how you do “work” (remember $W=F \times D$) when lifting weights or simply walking, among many other tasks.

Sarcoplasm

Within the plasma membrane is the sarcoplasm, a muscle fiber’s cytoplasm. This area is rich in soluble proteins, minerals, high-energy intermediates, substrates, enzymes of metabolism, mitochondrial protein, and other necessary organelles. Additionally, Ca^{2+} flux will occur into and out of this medium as part of the SFT. The sarcoplasm differs from cytoplasm of other cells due to the high quantity of stored glycogen as well as myoglobin, an oxygen-binding compound (or protein) similar to hemoglobin [5, 10].

Transverse Tubules (T-tubules)

T-tubules are an extensive network of extensions of the plasma membrane that pass laterally through and deep into the muscle fiber. Due to the interconnected pathways of these tubules through the myofibrils, APs can be rapidly transmitted to individual myofibrils from superficial to deep. The tubules also act as a pathway for external substances to enter the cell and waste products to leave the interior of the muscle fiber. Each T-tubule lies between two enlarged portions of the SR called cisternae. These three structures (SR, T-tubules, and cisternae) form a triad near the region where actin and myosin overlap [10].

Sarcoplasmic Reticulum (SR)

The SR, which corresponds to the endoplasmic reticulum of other cells, is a longitudinal network of tubules that parallel myofibrils and wrap around them. The SR is a storage site for Ca^{2+} which is a critical component for muscular contraction as introduced above [5].

Along with depolarizing the muscle fiber membrane, the AP travels over the T-tubule network and directly into the fiber toward the SR. The SR has a high concentration of Ca^{2+} ions compared to the sarcoplasm due to an active (i.e., ATP requiring) calcium pump in the SR membrane. In response to the muscle impulse (i.e., AP), the SR cisternae or terminal sacs housing Ca^{2+} become more permeable and the Ca^{2+} ions diffuse into the sarcoplasm.

Phases of the SFT—The Big Picture of Microscopic Muscle Action

Muscle Fiber Resting Phase

During resting conditions, most Ca^{2+} is stored in the SR, so little Ca^{2+} is in the myofibril. Therefore, very few myosin crossbridges are attached to actin and little to no tension is maintained in the musculature. Hence, at this time the muscle is considered to be in a resting state with actin's active sites covered by tropomyosin.

Excitation Coupling Phase

Subsequently, when Ca^{2+} ions are released from the SR into the sarcoplasm, they bind to troponin on actin filaments, effectively changing the conformation of troponin. Troponin, therefore, initiates the contraction process by moving tropomyosin strands, through a chemical, conformational change, from the myosin-binding sites on actin. The fiber's contractile proteins have now gone from a resting state to an active state. Furthermore, once tropomyosin has been shifted off of actin's binding sites (i.e., myosin-binding sites),

the myosin heads are able to attach, allowing crossbridge (i.e., the attachment of the myosin head to actin's active site) flexion to occur [13]. The force produced at any time is directly related to the number of myosin crossbridge heads bound to actin filaments at that instant [14]. This might explain genetic differences in strength potential or development and, therefore, variances in sport performance based on variable training adaptations. Conversely, keep in mind that a loss of muscle fibers, such as during deconditioning, injury, or aging, is associated with a loss of force production and, therefore, inadequate myosin crossbridge formation, among other degradations.

Recall that every muscle fiber is functionally connected to an axon of a motor neuron. Specifically, an α motor neuron connects and innervates many muscle fibers. Notably, as mentioned previously, a motor unit is an α motor neuron and all the muscle fibers it innervates. The site of functional connection, not physical connection, occurs at the synapse.

To review, in order for a muscular action to occur, an AP or electrical signal must be initiated from the brain or spinal cord to an α motor neuron. The AP arrives at the dendrites of the α motor neuron and eventually travels down the axon to the axon terminal. After the AP reaches the axon terminal, acetylcholine (ACh) secretion is initiated. ACh is a neurotransmitter synthesized in the cytoplasm of the motor neuron and in the distal end of the axon. ACh is then released into the synaptic cleft and binds to specific receptors in the muscle fiber membrane (on the postsynaptic membrane). This causes an increase in the permeability of Na^+ ions and stimulates a muscle impulse due to the entry of these charged particles into the muscle fiber. This process is known as depolarization. Repolarization occurs when K^+ effluxes from inside to outside a cell, such as with a fiber or dendrite. Na^+/K^+ membrane potentials are maintained, then, via the Na^+/K^+ ATPase pumps, which continuously facilitate active transport of the aforementioned ions across the muscle membrane to maintain a polarized state.

Contraction Phase

With the above protein interactions in mind, recall that there needs to be a sufficient amount of energy available in the form of ATP for muscular action to occur and/or be maintained. For crossbridge flexion or action, energy is derived from the hydrolysis of ATP to ADP and P_i . This reaction is catalyzed by the enzyme myosin adenosine triphosphatase (ATPase) in the myosin head, where energy release is captured and held until “contraction” or crossbridge shortening occurs. After “contraction,” an ATP molecule must replace the ADP molecule on the myosin crossbridge or head in order for the myosin head to detach from the actin site and be able to re-cock (and to attach again as needed). Thus, during the muscle action, the ADP structure detaches so that ATP is able to bind to the myosin head. Next, the myosin head detaches from actin and falls back into a re-cocked position. Spontaneously the ATP molecule is then enzymatically split to $ADP + P_i + \text{energy}$, whereby the energy is “held” in the myosin head. Now the energized crossbridge or myosin head is ready to attach to actin’s active sites and pull actin toward the center of the sarcomere as needed. This causes a muscular contraction (or action) by creating greater overlap of the actin and myosin filaments and decreasing the length of the sarcomere. This process continues as long as ATP is present, nerve impulses release ACh, and enough Ca^{2+} is available in the sarcoplasm to bind to troponin; otherwise, relaxation occurs and very few crossbridges remain. Collectively this cyclical process is termed excitation–contraction coupling [15].

Relaxation Phase

As suggested prior, relaxation in the muscle fiber occurs primarily when nerve impulses cease to exist and Ca^{2+} is actively taken up into the SR. Thus, there are a few main events that occur during relaxation. First, the remaining ACh in the synapse is rapidly broken down by acetylcholinesterase. This enzyme prevents the continuous stimulation of a muscle fiber from a nerve im-

pulse. Second, ACh is no longer a stimulus for the sarcolemma. Moreover, active Ca^{2+} pumps rapidly move Ca^{2+} back into the SR, causing greater Ca^{2+} concentrations in the SR than the sarcoplasm. As the myosin crossbridge links break, tropomyosin again covers actin’s active sites, effectually blocking and preventing crossbridge attachment. It is important to note that ATP is required for *relaxation* to occur. At this point, the muscle fiber remains relaxed and energized awaiting the opening of actin’s active site (i.e., from an appropriate nerve impulse and Ca^{2+} release into the sarcoplasm). Figure 2.4 succinctly summarizes the SFT.

Proprioceptors in Muscles, Joints, and Tendons

Last but not least, let us explore a brief flurry of information derived from the musculoskeletal system itself. These proprioceptors provide information to protect or enhance a muscular action and our awareness of limb movement. In short, muscles spindles, Golgi tendon organs (GTO), and Pacinian corpuscles are the main receptor we will discuss. They all serve to enhance our ability to move or mobilize muscle in a coordinated and protected fashion. “Proprioception can be defined as the cumulative neural input to the central nervous system from specialized nerve endings called mechanoreceptors,” according to Ribeiro et al. [16].

With that in mind, let us discuss the primary proprioceptor, muscle spindles, which provide mechanosensory feedback regarding alterations in skeletal muscle fiber length and tension [5]. Foremost, the spindles, which are wrapped around intrafusal fibers surrounded by extrafusal fibers, provide feedback about muscle stretch and, therefore, will initiate a counteraction if too much stretch is “sensed.” This opposing action is in the form of a contraction whereby the muscle belly shortens, avoiding a potential “over-stretch” of the fiber. Interestingly, this might be advantageous to power development (e.g., plyometric-type exercise) in that a greater force-producing effort may be realized if the “stretch reflex” is

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