Update

Human Skeletal Muscle Fiber Type Classifications

Human skeletal muscle is composed of a heterogenous collection of muscle fiber types.[1–3] This range of muscle fiber types allows for the wide variety of capabilities that human muscles display. In addition, muscle fibers can adapt to changing demands by changing size or fiber type composition. This plasticity serves as the physiologic basis for numerous physical therapy interventions designed to increase a patient's force development or endurance. Changes in fiber type composition also may be partially responsible for some of the impairments and disabilities seen in patients who are deconditioned because of prolonged inactivity, limb immobilization, or muscle denervation.[2] Over the past several decades, the number of techniques available for classifying muscle fibers has increased, resulting in several classification systems. The objective of this update is to provide the basic knowledge necessary to read and interpret research on human skeletal muscle.

Muscle fiber types can be described using histochemical, biochemical, morphological, or physiologic characteristics; however, classifications of muscle fibers by different techniques do not always agree.[1] Therefore, muscle fibers that may be grouped together by one classification technique may be placed in different categories using a different classification technique. A basic understanding of muscle structure and physiology is necessary to understand the muscle fiber classification techniques.

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Review of Muscle Fiber Anatomy and Physiology

Muscle fibers are composed of functional units called sarcomeres.[3] Within each sarcomere are the myofibrillar proteins myosin (the thick filament) and actin (the thin filament). The interaction of these 2 myofibrillar proteins allows muscles to contract (Fig. 1).[4] Several classification techniques differentiate fibers based on different myosin structures (isoforms) or physiologic capabilities.[1,2,5] The myosin molecule is composed of 6 polypeptides: 2 heavy chains and 4 light chains (2 regulatory and 2 alkali). A regulatory and an alkali light chain are associated with each of the heavy chains. The heavy chains contain the myosin heads that interact with actin and allow muscle to contract (Fig. 1).[4] The myosin heavy chain in the head region also contains an adenosine triphosphate (ATP) binding site and serves as the enzyme (adenosinetriphosphatase [ATPase]) for hydrolyzing ATP into adenosine diphosphate (ADP) and inorganic phosphate (P_I), which provides the energy necessary for muscle contraction. The thin filament is made of actin and 2 regulatory proteins, troponin and tropomyosin.[3] When the muscle fiber receives a stimulus in the form of an action potential, Ca^{2+} is released from the sarcoplasmic reticulum. The calcium then binds to troponin and, through tropomyosin, exposes a myosin binding site on the actin molecule (Fig. 1).[4] In the presence of ATP, the myosin head binds to actin and pulls the thin filament along the thick filament, allowing the sarcomere to shorten. As long as Ca^{2+} and ATP are present, the myosin heads will attach to the actin molecules, pull the actin, release, and reattach. This process is known as cross-bridge cycling. The speed at which cross-bridge cycling can occur is limited predominantly by the rate that the ATPase of the myosin head can hydrolyze ATP.

Classifications of muscle fibers by different techniques do not always agree.

Muscle Fiber Typing

Initially, whole muscles were classified as being fast or slow based on speeds of shortening.[3] This division also corresponded to a morphological difference, with the fast muscles appearing white in some species, notably birds, and the slow muscles appearing red. The redness is the result of high amounts of myoglobin and a high capillary content.[3] The greater myoglobin and capillary content in red muscles contributes to the greater oxidative capacity of red muscles compared with white muscles. Histological analysis shows that there is a correla-

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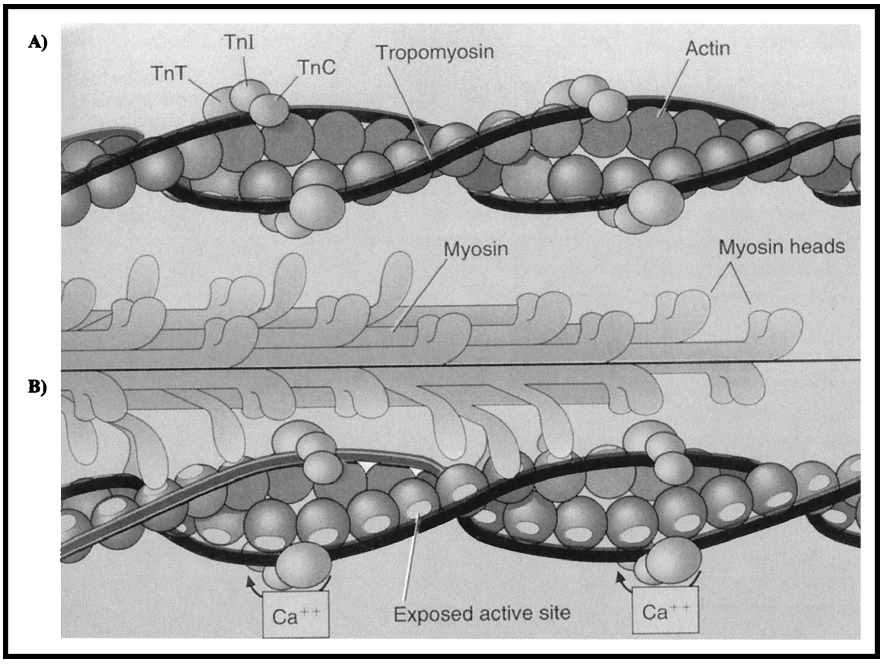

Figure 1.
Regulatory function of troponin and tropomyosin. Troponin is a small globular protein with 3 subunits (TnT, TnI, TnC). (A) Resting condition: Tropomyosin under resting conditions blocks the active sites of actin, preventing actin and myosin from binding. (B) Contraction: When troponin binds with Ca^{2+}, it undergoes a conformational change and pulls tropomyosin from the blocking position on the actin filament, allowing myosin heads to form cross-bridges with actin. From Plowman SA, Smith DL. *Exercise Physiology for Health, Fitness, and Performance*. Boston, Mass: Allyn & Bacon; 1997:433. Copyright 1997 by Allyn & Bacon. Reprinted/adapted by permission.

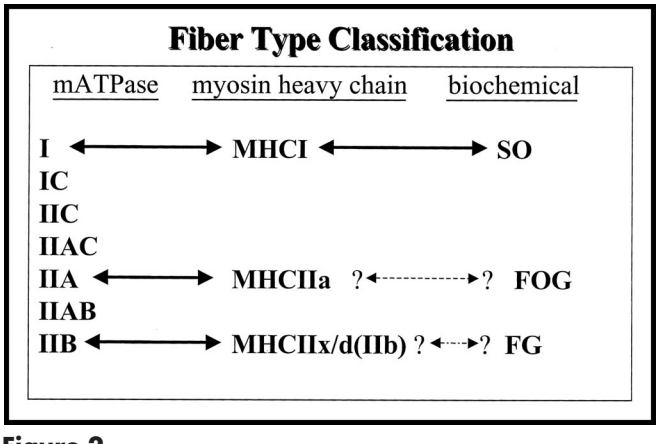

Figure 2.
Comparison of 3 different skeletal muscle fiber type classifications: histochemical staining for myosin adenosinetriphosphatase (mATPase), myosin heavy chain identification, and biochemical identification of metabolic enzymes. Note: in humans, MHCIIb are now more accurately referred to as MHCIIx/d. The question marks indicate the poor correlation between biochemical and myosin heavy chain or mATPase fiber type classification schemes.

tion between myosin ATPase activity and the speed of muscle shortening.[6] This histochemical analysis led to the original division of muscle fibers into type I (slow) and type II (fast). Currently, muscle fibers are typed using 3 different methods: histochemical staining for myosin ATPase, myosin heavy chain isoform identification, and biochemical identification of metabolic enzymes.

Myosin ATPase Staining

In humans, myosin ATPase hydrolysis rates for fast fibers are 2 to 3 times greater than those of slow fibers.[7] However, myosin ATPase histochemical staining, which is widely used for classifying muscle fibers, does not evaluate myosin ATPase hydrolysis rates.[1] Fibers are separated based solely on staining intensities because of differences in pH sensitivity, not because of the relative hydrolysis rates of ATPases.[1] Advances in the histochemical staining technique used to evaluate myosin ATPase have led to 7 recognized human muscle fiber types (Fig. 2).[1] Originally, fibers were identified as type I, IIA, or IIB.[1,5] More recently, types IC, IIC, IIAC, and IIAB, which have intermediate myosin ATPase staining characteristics, have been identified. The slowest fiber, type IC, has staining characteristics more like those of type I fibers, whereas the fastest fiber, type IIAC, stains more like type IIA. Type IIAB fibers have intermediate staining characteristics between type IIA and IIB fibers. Because these delineations are based on qualitative analysis of stained fibers, it remains possible that more fiber types will be identified in the future. In summary, the 7 human muscle fiber types, as identified by myosin ATPase histochemical staining are (from slowest to fastest): types I, IC, IIC, IIAC, IIA, IIAB, and IIB (Fig. 2).[1,3,5] These divisions are based on the intensity of staining at different pH levels, and, as such, any given fiber could be grouped differently by different researchers. Furthermore, not all studies use all 7 fiber types. Some researchers place all muscle fibers into just the original 3 fiber types.

Myosin Heavy Chain Identification

Identification of different myosin heavy chain isoforms also allows for fiber type classification (Fig. 2).[1] The different myosin ATPase-based fibers correspond to different myosin heavy chain isoforms.[1,8] This is not surprising because the myosin heavy chains contain the site that serves

as the ATPase. The fact that each muscle fiber can contain more than one myosin heavy chain isoform explains the existence of myosin ATPase fiber types other than the pure type I, type IIA, and type IIB fibers. Although the human genome contains at least 10 genes for myosin heavy chains, only 3 are expressed in adult human limb muscles.[1] Myosin heavy chain isoforms can be identified by immunohistochemical analysis using antimyosin antibodies or by sodium dodecyl sulfate–polyacrylamide gel electrophoretic (SDS-PAGE) separation.[5]

The 3 myosin isoforms that were originally identified were MHCI, MHCIIa, and MHCIIb, and they corresponded to the isoforms identified by myosin ATPase staining as types I, IIA, and IIB, respectively.[1,3,5] Human mixed fibers almost always contain myosin heavy chain isoforms that are "neighbors" (ie, MHCI and MHCIIa or MHCIIa and MHCIIb).[2] Consequently, the histochemical myosin ATPase type IC, IIC, and IIAC fibers coexpress the MHCI and MHCIIa genes to varying degrees, whereas the type IIAB fibers coexpress the MHCIIa and MHCIIb genes.[1] Because of its quantitative nature, identifying myosin heavy chain isoforms using single-fiber electrophoretic separation (SDS-PAGE technique) probably represents the best method for muscle fiber typing. Electrophoretic separation allows for the relative concentrations of different myosin heavy chain isoforms to be detected in a mixed fiber.[5,8]

One point regarding human myosin heavy chain isoforms and fiber type identification may prove confusing to someone trying to read research literature in this area. In small mammals, a fourth myosin heavy chain isoform, MHCIIx or MHCIId, is present that has an intermediate contractile speed between the MHCIIa and MHCIIb isoform.[9] Based on several types of evidence, extending to the level of DNA analysis, what was originally identified in humans as MHCIIb is actually homologous to MHCIIx/d of small mammals.[2,5,9] As a result, what has been called MHCIIb in humans is actually MHCIIx/d, and humans do not express the fastest myosin heavy chain isoform (MHCIIb).[5] Because the histochemical myosin ATPase fiber type nomenclature was developed using human muscle, type IIB fibers, which we now know correspond to the MHCIIx/d myosin heavy chain isoform, are not likely to be renamed type IIX.[1] Consequently, depending on the author, histochemical myosin ATPase-based human type IIB fibers may be associated with either MHCIIb or MHCIIx/d isoforms. It is important to remember that in human limb muscles only 3 myosin heavy chain isoforms are present (from slowest to fastest): MHCI, MHCIIa, and MHCIIx/d (formerly erroneously identified as MHCIIb).[1] Humans do not express the fastest myosin heavy chain isoform, MHCIIb.[9] We will associate MHCIIx/d in humans with the histochemical myosin ATPase-based type IIB fiber in the remainder of this article.

Biochemical

A third classification scheme that is often used to classify muscle fibers combines information on muscle fiber myosin ATPase histochemistry and qualitative histochemistry for certain enzymes that reflect the energy metabolism of the fiber (Fig. 2).[2] Histochemical myosin ATPase fiber typing is used to classify muscle fibers as type I or type II, which are known to correspond to slow and fast muscle fibers, respectively.[2] The enzymes that are analyzed reflect metabolic pathways that are either aerobic/oxidative or anaerobic/glycolytic.[5] This classification technique leads to 3 fiber types: fast-twitch glycolytic (FG), fast-twitch oxidative (FOG), and slow-twitch oxidative (SO).[2,3] Although a good correlation exists between type I and SO fibers, the correlations between type IIA and FOG and type IIB and FG fibers are more varied.[3,10] Therefore, the type IIB fibers do not always rely primarily on anaerobic/glycolytic metabolism, nor do the type IIA fibers always rely primarily on aerobic/oxidative metabolism.[5] Although, in general, fibers at the type I end of the continuum depend on aerobic/oxidative energy metabolism and fibers at the type IIB end of the continuum depend on anaerobic/glycolytic metabolism, the correlation is not strong enough for type IIB and FG or type IIA and FOG to be used interchangeably.[2,5]

Myosin Light Chains

The light chains of the myosin molecule also exist in different isoforms, slow and fast, that affect the contractile properties of the muscle fiber.[3,11] Muscle fibers that are homogeneous for a myosin heavy chain isoform (ie, a pure fiber) may be heterogenous in regard to myosin light chain isoforms, although, in general, *fast* myosin heavy chain isoforms associate with *fast* myosin light chain isoforms and *slow* myosin heavy chain isoforms associate with *slow* myosin light chain isoforms.[2,5,12] There is good evidence that additional proteins in muscle fibers are coexpressed so that the various "fast" proteins are expressed with one another and the various "slow" proteins are expressed with one another, which suggests "a fiber type specific program of gene expression."[2,11,12]

Motor Unit Classification

Although we have been discussing fiber types, the true functional unit of the neuromuscular system is the motor unit.[13,14] A motor unit is an alpha motoneuron (originating in the spinal cord) and all of the muscle fibers that it innervates. Based on myosin ATPase histochemistry and qualitative histochemistry for enzymes that reflect the energy metabolism of the fiber, all of the muscle fibers of a motor unit have similar characteristics.[15] Motor units can be divided into groups based on

the contractile and fatigue characteristics of the muscle fibers.[3,14] Based on contractile speed, motor units are classified as either slow-twitch (S) or fast-twitch (F).[14] The F motor units are further subdivided into fast-twitch fatigue-resistant (FR), fast-twitch fatigue-intermediate (Fint), and fast-twitch fatigable (FF).[16,17]

Motor Unit/Muscle Fiber Plasticity

Regardless of the classification scheme used to group muscle fibers, there is overwhelming evidence that muscle fibers—and therefore motor units—not only change in size in response to demands, but they can also convert from one type to another.[2,18,19] This plasticity in contractile and metabolic properties in response to stimuli (eg, training and rehabilitation) allows for adaptation to different functional demands.[2] Fiber conversions between type IIB and type IIA are the most common, but type I to type II conversions are possible in cases of severe deconditioning or spinal cord injury (SCI).[2,20] Less evidence exists for the conversion of type II to type I fibers with training or rehabilitation, because only studies that use denervated muscle that is chronically activated with electrical stimulation have consistently demonstrated that such a conversion is possible.[21]

Changes in the muscle fiber types are also responsible for some of the loss of function associated with deconditioning.[2] Experiments in animals involving hind-limb suspension, which unloads hind-limb muscles, and observations of humans and rats following microgravity exposure during spaceflight have demonstrated a shift from slow to fast muscle fiber types.[2] In addition, numerous studies on animals and humans with SCI have demonstrated a shift from slow to fast fibers.[2,20] In humans, detraining (ie, a decrease in muscle use from a previously high activity level) has been shown to lead to the same slow to fast conversion, with shifts from MHCIIa to MHCIIx/d and possibly MHCI to MHCIIa.[2] There is also a concomitant decrease in the enzymes associated with aerobic-oxidative metabolism.[2] In summary, decreased use of skeletal muscle can lead to a conversion of muscle fiber types in the slow to fast direction.

Interestingly, some of the loss of muscle performance (eg, decreased force production) due to aging does not appear to be only due to the conversion of muscle fibers from one type to another, but largely due to a selective atrophy of certain populations of muscle fiber types.[22,23] With aging, there is a progressive loss of muscle mass and maximal oxygen uptake, leading to a reduction in muscle performance and presumably some of the loss of function (eg, decreased ability to perform activities of daily living) seen in elderly people.[1,22,23]

Age-related loss of muscle mass results primarily from a decrease in the total number of both type I and type II fibers and, secondarily, from a preferential atrophy of type II fibers.[22,24] Atrophy of type II fibers leads to a larger proportion of slow type muscle mass in aged muscle, as evidenced by slower contraction and relaxation times in older muscle.[25,26] In addition, the loss of alpha motoneurons with age results in some reinnervation of "abandoned" muscle fibers by adjacent motor units that may be of a different type.[22,27] This may facilitate fiber type conversion, as the reinnervated muscle fibers take on the properties of the new "parent" motor unit.[3,22] Recent evidence in aged muscle suggests that fiber type conversion may occur, because there is a much larger coexpression of myosin heavy chain in older adults as compared with young individuals.[28] Older muscle was found to have a greater percentage of fibers that coexpress MHCI and MHCIIa (28.5%) compared with younger muscle (5%–10%).[28]

Fortunately, physical therapy interventions can affect muscle fiber types leading to improvements in muscle performance. In the context of this update, physical therapy interventions can be broadly divided into those designed to increase the patient's resistance to fatigue and those designed to increase the patient's force production. It has been known for some time that training that places a high metabolic demand on the muscle (endurance training) will increase the oxidative capacity of all muscle fiber types, mainly through increases in the amount of mitochondria, aerobic/oxidative enzymes, and capillarization of the trained muscle.[29,30] Using the metabolic enzyme–based classification system, this would lead to a transition from FG to FOG muscle fibers without, necessarily, a conversion of myosin heavy chain isoforms.[2]

The myosin heavy chain composition of a muscle fiber can change when subjected to endurance training.[19] Within type II fibers there is a transformation from IIB to IIA, with more MHCIIa being expressed, at the expense of MHCIIx/d.[2,19] Consequently, the percentage of pure type IIB fibers decreases and the percentages of type IIAB and pure type IIA fibers increase. Evidence is lacking to demonstrate that type II fibers convert to type I with endurance training,[19] although there does appear to be an increase in the mixed type I and IIA fiber populations.[2] Researchers have found that type I fibers become faster with endurance exercise and slower with deconditioning in humans.[31,32] This change in contractile speed is not because of a conversion of fiber types, but rather because of changes in the myosin light chain isoforms from slow to fast isoforms and from fast to slow isoforms, respectively.[31,32] Because this change in muscle contractile speed does not occur by altering the myosin ATPase, it would not be detectable by histochemical

fiber typing.[2] The shift from slow to fast myosin light chain isoforms allows the slow fibers to contract at a rate fast enough for the given exercise (eg, running, cycling), yet retain efficient properties of energy use.[30] In summary, muscle fiber adaptations to endurance exercise depend on fiber type, although the oxidative capacity of all fibers is increased. Type I fibers may become faster through myosin light chain conversion, whereas type II fibers convert into slower, more oxidative types.

High-intensity resistance training (eg, high-load–low-repetition training) results in changes in fiber type similar to those seen with endurance training, although muscle hypertrophy also plays an essential role in producing strength gains.[33] Initial increases in force production with high-intensity resistance training programs are largely mediated by neural factors, rather than visible hypertrophy of muscle fibers, in adults with no pathology or impairments.[34] Even so, changes in muscle proteins, such as the myosin heavy chains, do begin after a few workouts, but visible hypertrophy of muscle fibers is not evident until training is conducted over a longer period of time (>8 weeks).[33]

Most researchers have found that high-intensity resistance training of sufficient duration (>8 weeks) causes an increase in MHCIIa composition and a corresponding decrease in MHCIIx/d composition.[35–37] In many studies of high-intensity resistance training, researchers have also reported concomitant increases in MHCI composition,[37] although some researchers report no changes in MHCI composition.[38,39] Both endurance training and resistance training result in similar reductions in myosin heavy chain coexpression, such that a greater number of "pure" fibers are present.[40] Although the trends in fiber type conversions are similar for endurance training and resistance training, differences in physiological changes that occur with each type of exercise are also important. Endurance training increases the oxidative capacity of muscle, whereas training to increase force production of sufficient intensity and duration promotes hypertrophy of muscle fibers by increasing the volume of contractile proteins in the fibers.

Knowing the differences between human skeletal muscle fiber types allows clinicians to understand more completely the morphological and physiological basis for the effectiveness of physical therapy interventions, such as endurance training and resistance training. In addition, this knowledge also offers some explanation for the changes in muscle that occur with age, deconditioning, immobilization, and muscle denervation. Such knowledge is helpful for the optimal design of rehabilitation programs that target deficits in muscle morphology and physiology.

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