Marquette University

# e-Publications@Marquette

Biological Sciences Faculty Research and Publications

Biological Sciences, Department of

4-2003

# Effects of regular exercise training on skeletal muscle contractile function

Robert Fitts
*Marquette University*, robert.fitts@marquette.edu

## Recommended Citation

# Effects of Regular Exercise Training on Skeletal Muscle Contractile Function

Robert H. Fitts
Department of Biology, Marquette University, Milwaukee, Wisconsin

## Keywords:
Fiber Types; Endurance; Strength; Aging

## Abstract
Skeletal muscle function is critical to movement and one's ability to perform daily tasks, such as eating and walking. One objective of this article is to review the contractile properties of fast and slow skeletal muscle and single fibers, with particular emphasis on the cellular events that control or rate limit the important mechanical properties. Another important goal of this article is to present the current understanding of how the contractile properties of limb skeletal muscle adapt to programs of regular exercise.

Skeletal muscle function is critical to movement and one's ability to perform daily tasks, such as eating and walking. Neuromuscular diseases and aging can lead to deleterious alterations in the functional capacity of muscle, making such tasks difficult if not impossible to achieve. Before the mechanisms responsible for age- or disease-induced changes in muscle can be elucidated, the basic cellular and molecular properties of muscle need to be understood. The fundamental contractile properties of limb skeletal muscle, such as, twitch tension ($P_t$), peak tetanic tension ($P_0$), and the maximal unloaded shortening velocity ($V_0$) have been known for some time. [1] However, despite considerable advancement in the last few decades, our knowledge of excitation-contraction coupling and cross-bridge cycling is still incomplete. [2,3] Specifically, how depolarization of the surface (sarcolemma) and t-tubular membranes leads to the release of $Ca^{2+}$ from the adjacent $Ca^{2+}$ release channel of the sarcoplasmic reticulum (SR), and the interaction of the contractile proteins actin and myosin with the subsequent generation of force and filament movement (cross-bridge kinetics) are two important muscle cell functions for which we currently lack a complete understanding. One objective of this article is to review the contractile properties of fast and slow skeletal muscle and single fibers, with particular emphasis on the cellular events that control or rate limit the important mechanical properties.

Skeletal muscle is known to be a very adaptable tissue that atrophies during conditions of unloading, such as bed rest or exposure to microgravity, and hypertrophies in response to heavy resistance exercise. [4–6] Muscle wasting is also known to be associated with aging and neuromuscular diseases. [7,8] An important goal of this article is to present the current understanding of how the contractile properties of limb skeletal muscle adapt to programs of regular exercise. This knowledge is important to our ultimate understanding of what type and how much exercise should be prescribed to slow muscle deterioration associated with aging and neuromuscular diseases. In this context, I will compare endurance with strength exercise training in rats and humans. It is not my intention to present a complete review of this topic; for this, the reader is referred to the article of Fitts and Widrick. [9]

## Isometric (Static) Contractile Properties

By definition, an isometric or static contraction involves the generation of force in the absence of any length change. Thus, theoretically, the sarcomere length remains constant. An isometric contraction in response to a single stimulus (action potential) is defined as an isometric twitch. The characteristics of the twitch are dependent on fiber type and the temperature, but in general, the force transient of the twitch reflects the amplitude and duration of the $Ca^{2+}$ transient. Thus, fast fibers and muscles, such as the rat plantaris, are characterized by short duration twitches compared with slow fibers or muscles, such as the rat soleus (Fig. 1). The isometric twitch contraction time (sometimes referred to as the time to peak tension) and relaxation time for the fast plantaris were approximately 2.5-fold faster than the slow soleus (Fig. 1). This difference is reflective of the fact that fast muscles contain a higher SR content and SR pump activity compared with slow muscles. [10] One of the primary reasons for recording isometric twitches is that they provide information about the activity of the SR. Conditions that shorten the twitch, such as hindlimb unloading in the rat, do so by increasing the content and activity of the SR, which leads to a shorter $Ca^{2+}$ transient and reduced twitch duration. [11,12] Similarly, muscle fatigue leads to a prolonged twitch duration by reducing the activity of the SR pump. [13]
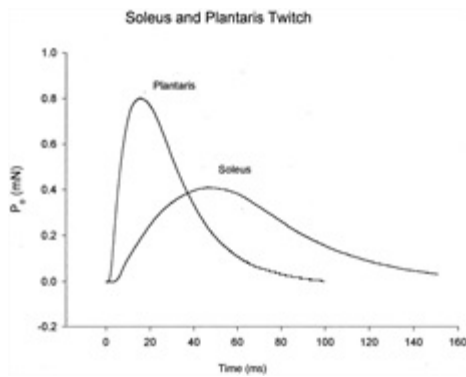
**Figure 1:** Representative isometric twitch contractions for the fast-twitch plantaris and the slow-twitch soleus muscles of the rat measuredin situ at 34°C. The twitch contractions were elicited by a single supramaximal electrical stimulation of the sciatic nerve.

A second obvious feature of the isometric twitch is the rate of tension rise, which is 4- to 5-fold higher in fast *vs.* slow muscles (Fig. 1). In nonfatigued fibers, the rate of force development is thought to be limited by the rate of transition from the low-force actin-myosin cross-bridge state ($AM.ADP.P_i$) to the high-force generating state of the cross-bridge ($AM^*.ADP.P_i$). [14] The functional correlate of this transition is best measured experimentally by monitoring the rate of tension redevelopment following a rapid release and re-extension of a fully activated single fiber. With this experimental technique, Metzger and Moss [15] demonstrated that fast type II fiber has a 7-fold higher tension redevelopment compared with the slow type I fiber. The mean tension redevelopment obtained with maximal $Ca^{2+}$ activation was $22.9 \pm 0.5$ $sec^{-1}$ and $3.0 \pm 0.1$ $sec^{-1}$ in fast and slow fibers, respectively. Although the authors did not type the fibers, the fast fibers were isolated from the superficial region of the rat vastus lateralis, which is known to be 100% type IIb.

The number of cross-bridges acting in parallel determines the peak force generating capacity of a muscle or single fiber. The latter is measured experimentally by eliciting a peak tetanic contraction. The frequency of stimulation required depends on the temperature and fiber type of the muscle. To elicit $P_0$ at 34°C requires a stimulation rate of 120 and 180 Hz for slow and fast muscles, respectively. Representative tetanus for the fast rat plantaris and slow soleus are shown in Figure 2. The higher force for the plantaris compared with the soleus was explained by the greater plantaris cross-sectional area (CSA), as no significant differences in relative $P_0$ (force/mg of tissue) was noted. The rate of tetanic tension development was identical to that observed for the twitch, and the differences between muscles remained 4- to 5-fold. In contrast, the rate of relaxation following the tetanus was considerably faster than that observed for the twitch. For the plantaris, the rate of relaxation was 10-fold higher than that observed for twitch relaxation, whereas for the soleus, the difference was 5-fold. The initial portion of relaxation was linear and then exponential. This has also been observed in single fibers in which the break from linearity coincided with and was likely caused by sarcomere nonuniformity that developed, as some regions of the fiber inactivated faster than others. [16]
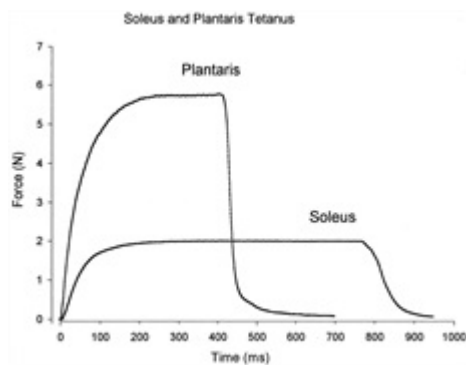
**Figure 2:** Representative isometric tetanic contractions for the fast-twitch plantaris and the slow-twitch soleus muscles of the rat measuredin situ at 34°C. The contractions were elicited by supramaximal electrical stimulation of the sciatic nerve at 120 Hz and 180 Hz for the soleus and plantaris, respectively.

## Dynamic Contractile Properties

Maximal shortening velocity can be determined using either the slack test technique or from extrapolation of the Hill force-velocity relation to zero load. [17,18] The slack test value-abbreviated $V_0$ is generally higher than the value obtained from the Hill plot-abbreviated $V_{max}$. Figure 3 shows a representative slack test experiment for the slow type I, fast type IIa, and fast type IIb fiber type. The latter was likely a type IIx fiber, because recently the human type IIb fiber was shown to have homology with the rat type IIx fiber. [19] To determine $V_0$ by the slack test, the fiber is activated to produce peak force and then rapidly slacked to a shorter length, such that the force falls to zero. The process is repeated five or six times using different slack distances (none >20% of fiber length), and the duration of unloaded shortening is plotted against the slack distance (Fig. 3). $V_0$ is determined from the slope of the fitted straight line. For individual fibers, the hierarchy for $V_0$ is type IIb > IIx > IIa > I. [9] Rat fast-twitch muscles have been reported to be 3- to 4-fold faster than slow muscles. [11] At the single-fiber level, the fast type IIa fiber is at the low end of this range, whereas the $V_0$ of the fast IIb fiber is 5- to 6-fold higher than the slow type I fiber. [9] Similar differences in fiber $V_0$ have been noted in humans, where the type IIa and IIx fibers are between 3- to 4- and 6-fold higher, respectively, than the type I fiber. [20,21]
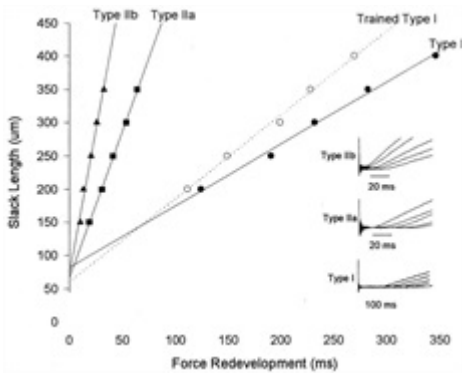


**Figure 3:** Slack test methodology for the determination of maximal unloaded shortening velocity. Theinserts in the lower right display original force-time recordings obtained from three single human gastrocnemius fibers. Each insert consists of five superimposed force-time recordings, illustrating the redevelopment of force after imposed slack lengths, ranging from 150 to 400 μm. Note the different time scale in the type I fiber recordings. All fibers were obtained from sedentary, middle-aged males. In the main figure, the time required for force redevelopment is plotted against slack length for each individual fiber. The points are fit by a least-squares regression line (R2 ≥ .995 for all fibers). The slope of each line is the maximal unloaded shortening velocity, or V0, of the individual fiber in mm·sec−1. The velocity is expressed in terms of fiber lengths·sec−1 (FL·sec−1). V0 values for type IIb, type IIa, and type I fibers were 4.46, 2.31. and 0.45 FL·sec−1, respectively. The opensymbols and dottedline show the data for a slow type I fiber from the gastrocnemius of an elite middle-aged male distance runner. Note that at each imposed slack distance, this fiber required less time to redevelop force, in comparison with the sedentary type I fiber. The fiber V0 was 0.55 FL·sec−1, which is approximately 20% greater than the V0 of the sedentary type I fiber (adapted from Reference 9).

From the standpoint of physical performance or one's ability to move about, the important functional property is peak power, which can be determined from force-velocity curves. Figure 4 shows force-velocity and force-power curves for all three-fiber types in human gastrocnemius muscle. [20] The average peak power was 8, 42, and 80 μN·fiber lengths·sec$^{-1}$ or a ratio of 1:5:10 for the type I, IIa, and IIx fiber type, respectively. This is consistent

with the observation that in humans, peak power during knee extension was correlated with the percentage of fast-twitch fibers. [22] Animal [23] and human studies demonstrated that peak power is obtained at loads considerably below 50% of $P_0$, and in our experiments, the load at peak power was 14%, 20%, and 25% of $P_0$ for the type I, IIa, and IIx fiber, respectively. [20]
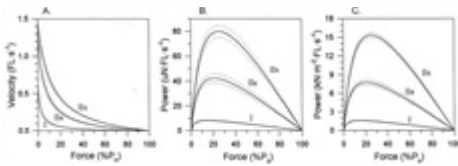


**Figure 4:** Composite force-velocity and force-power curves for types I, IIa, and IIx gastrocnemius fibers from sedentary human subjects. Thedottedlines represent ±1 standard error of estimation (SEE). In several instances, lines representing ±1 SEE are obscured by composite curve. A, force-velocity relationship;B, absolute force-power relationship;C, normalized force-power relationship (adapted from Reference 20).

## Effect of Exercise Training Programs on Fiber Type Distribution

Despite considerable research, the question of whether programs of regular exercise training can induce slow-to-fast or fast-to-slow fiber type transitions is still controversial. Clearly, extreme alterations in either loading or activation frequency can cause the ratio of slow/fast fibers to be altered. For example, in rats and humans, space flight has been shown to increase the number of fast fibers and hybrid fibers that express both fast and slow myosin and decrease the percentage of slow type I fibers. [5] The fiber shifts presumably result from an increased expression of fast type myosins, with a corresponding decrease in the transcription and translation of slow type I myosin. The reverse switch from fast to slow fiber type can be induced by chronic electrical stimulation of fast muscles. [24] Regarding programs of regular exercise training, all types of exercise (endurance, sprint, and heavy resistance) have been shown to reduce the percentage of type IIb and increase the percentage of type IIa fibers. [25–29] In physically active individuals, there seems to be a total lack of type IIb (likely IIx) fibers. Staron et al. [29] found a significant decline in type IIb fibers following a 20-wk strength training program and an increase in this fiber type with detraining. After 13 wk of retraining, no type IIb fibers and only a small percentage of hybrid type IIab fibers could be found. [29]

Regarding slow to fast transitions, Esbjörnsson et al. [25] observed 6 wk of sprint training (Wingate cycle ergometry) to increase the percentage of type IIa fibers and to decrease the percentage of both type I and IIb fiber. From this data, the authors concluded that sprint training induced fiber transitions toward IIa from both IIb and I. These results were supported by the data of Andersen et al., [26] who studied male sprinters before and after a 3-mo period of intensive interval training. Posttraining, individual fibers from the vastus lateralis of the sprinters contain significantly less myosin heavy chain (MHC) type I and hybrid IIa/IIb and significantly more MHC type IIa than fibers from the pretraining biopsy. In the study by Esbjörnsson et al., [25] the authors found that 1 additional week of intensified sprint training increased the number of type I fibers and further decreased the percentage of type IIb fibers. The authors interpreted this result as an exercise-induced fiber transition from IIb to IIa to I. Endurance exercise has also been reported to increase the number of slow type I fibers in both humans [27] and rats. [30] In the latter study, by the 15th wk of training, the rats were running for 3.5 hr per day at 27 m/min and at a 15-degree grade. The authors found a significant decrease in fast IIb fibers and a corresponding increase in fast IIa fibers in the plantaris muscle, whereas the deep region of the vastus lateralis showed a significant increase in the type I fiber number. The studies by Andersen et al., [26] Esbjörnsson et al., [25] and Green et al. [30] support the concept that exercise training can induce fiber type shifts from fast to slow or vice versa. Other studies have failed to observe transitions between slow and fast fiber types. For example, Adams et al. [28] reported that a 19-wk heavy resistance exercise program decreased type IIb fiber number and the percentage of type IIb MHC and, correspondingly, increased type IIa fiber number and MHC content in the

vastus lateralis of male subjects. However, neither the type I MHC composition nor the type I fiber percentage changed. We repeated the training paradigm of Green et al. [30] and evaluated the structural and functional properties of individual fibers isolated from control, 2, and 3.5 hr per day endurance-trained male rats. Fibers were isolated from the soleus, the superficial region of the medial head of the gastrocnemius (primarily type IIx and IIb fibers, thus referred to as white gastrocnemius), and the deep region of the lateral head of the gastrocnemius (primarily type I and IIa fibers, thus referred to as the red gastrocnemius). The study was primarily designed to assess the effects of endurance training on single-fiber function. In the 200–300 fibers studied per group, we observed no change in the percentage of slow and fast fibers between groups. The control white gastrocnemius contained only fast fibers, and this distribution remained unchanged in both the 2- and 3.5-hr per day trained groups. However, fiber type changes were observed within the fast fiber subgroups. In the control white gastrocnemius, type IIb and hybrid type IIb/IIx fibers made up 76% and 22% of the total fiber distribution, whereas only 1% of the fibers were type IIx. Following 2 and 3.5 hr of daily running, IIb fibers declined to 44% and 31%, respectively. In the 2-hr group, the major increase was in the hybrid IIb/IIx fibers (41% of total), whereas in the 3.5-hr group, the primary increase was in type IIx fibers (31% of total). In summary, any type of exercise training will decrease the percentage of the fastest fiber type (IIb in rats and IIx in humans) and cause an increase in IIx or IIa fibers. It seems considerably more difficult to switch slow to fast or fast to slow with regular programs of exercise, and when such a switch does occur, the percent change is small. [27,30]

## Effect of Endurance Exercise Training on Contractile Function

The effects of regular endurance exercise training on the metabolic and contractile properties of limb skeletal muscle have been extensively characterized. [9,31] Since the mid 1970s, it has been know that endurance exercise training increases the myofibrillar ATPase activity of muscles composed of predominately type I fibers, while reducing the activity of this enzyme in fast-twitch muscles. [32] Because this enzyme is thought to be rate limiting in the control of the cross-bridge cycle rate, regular endurance exercise should increase slow and decrease fast muscle $V_0$. This premise was difficult to prove because most limb skeletal muscles are composed of both slow and fast fibers. We were able to establish that regular endurance exercise increased the soleus $V_0$ in rats by 20% and that this increase was correlated to the increased myofibrillar ATPase activity. Subsequently, it was established in single-fiber studies that endurance exercise training increased the $V_0$ of the slow type I fiber and that this effect occurred in both rats (treadmill running) [33] and humans (swim training). [34] In the human study, the type II fibers showed a significant decline in fiber $V_0$. [34] The mechanisms of the exercise-induced increase in slow fiber and decrease in fast fiber $V_0$ have not been established. Because exercise training of any type is known to shift the fast fiber profile from type IIb to IIx or IIx to IIa (reviewed above), the type II fiber decline in $V_0$ in the human swim-training study may have been caused by a shift from IIx to IIa fibers. In the case of the slow type I fiber, we demonstrated that the increased type I fiber $V_0$ following endurance running in rats was correlated with an increased fiber myofibrillar ATPase activity. The type I fibers from the trained rats contained only slow MHC and, thus, the increased $V_0$ could not be attributed to co-expression of a small amount of fast MHC. However, the fibers did show an increased expression of the fast myosin light chains (LC), with the greatest increase in $LC_{2f}$ (Fig. 5). Because myosin light chains are known to influence fiber-shortening speed, we hypothesized that the exercise-induced increase in the velocity and myofibrillar ATPase activity of the type I fiber could have resulted from the incorporation of the fast LCs. [33] We observed a qualitatively similar pattern when type I fibers from elite endurance-trained master runners were compared with an aged-matched sedentary group. The average type I fiber $V_0$ from the runners was 19% greater than the type I fibers from the sedentary counterparts. [35] The type I fibers from the runners had a greater amount of myosin $LC_3$, and this resulted in an increased $LC_3/LC_2$ ratio compared with fibers from the sedentary group. In addition, the frequency distribution of both fiber $V_0$ and the $LC_3/LC_2$ ratio was skewed toward higher values in the running group. [35] Myosin $LC_3$ seems to be particularly important in modulating $V_0$. [36] A 10% increase in $LC_3$/total $LC_1$ was

associated with a 0.4 fiber length/sec increase in the $V_0$ of rabbit plantaris fibers, [37] and although it was not statistically significant, Larrson and Moss [38] found that type I fibers expressing $LC_3$ have a mean $V_0$ that was 29% higher than fibers that did not contain $LC_3$. Collectively, these data suggest that the endurance exercise training-induced increase in type I fiber $V_0$[33–35] might be caused by an increased expression of fast myosin LCs in the slow type I fiber.
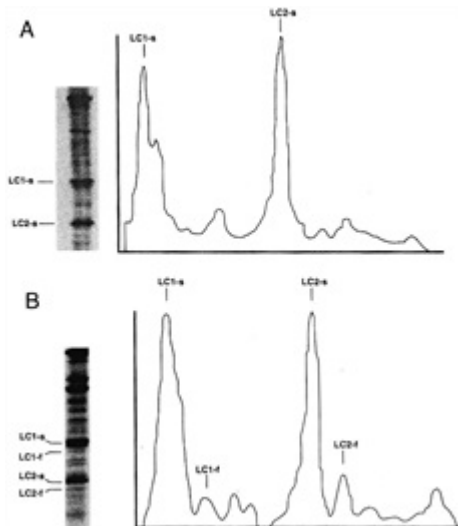


**Figure 5:** Myosin light-chain (LC) profile of rat single fibers run on 12% sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Fibers were isolated from the soleus of control (A) and exercise-trained (B) rats. Beside each gellane is a densitometric scan, illustrating the relative concentration of the LC species. s, slow;f, fast. The exercise-trained fiber contained significantly more fast type myosin light chains (adapted from Reference 33).

Endurance exercise training generally has no effect on the size of either type I [33,34] or type II [34] fibers. However, the elite master runners did have significantly smaller type I and IIa fibers compared with their sedentary counterparts. [35] This suggests that endurance exercise training over many years may yield somewhat smaller fibers. This adaptation would be desirable to an endurance athlete, as it would facilitate oxygen diffusion into the center of the cell. Because of their smaller size, the type I and IIa fibers of the elite master runners generated less peak force. [35] This can be clearly seen in Figure 6, where the diameter and peak force of type I fibers are compared for the sedentary and master athlete groups. The reduced fiber size also led to a lower peak power for the type I and IIa fibers of the elite runners compared with the sedentary group (Fig. 7). The reduced power of the elite runners was entirely explained by the fiber size because no differences were observed in peak power when the data were expressed relative to the fiber cross-sectional area (Fig. 7).
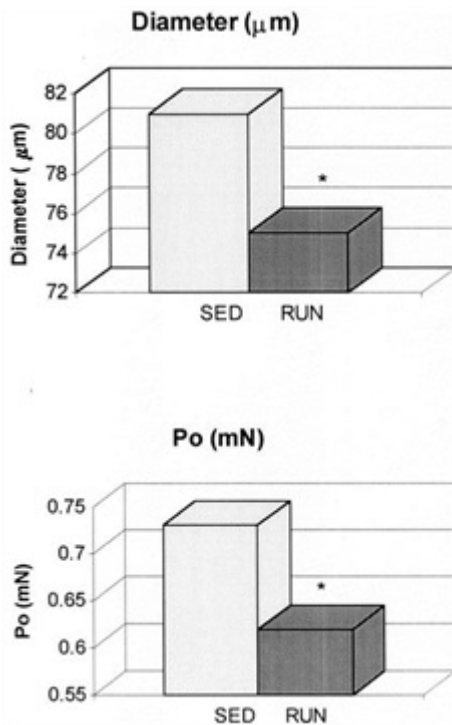
**Figure 6:** Comparison of the fiber diameter (µm) and peak force (mN) of gastrocnemius type I fibers from sedentary control (SED) and elite master athletes (RUN). *P < .05 (adapted from data published in Reference 35).
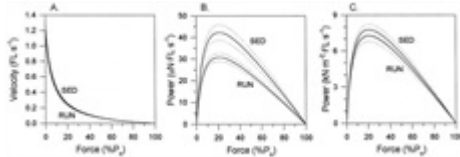


**Figure 7:** Composite force-velocity and force-power curves for type I gastrocnemius fibers from sedentary (SED) and endurance-trained (RUN) human subjects. The dottedlines represent ±1 standard error of estimation. A, force-velocity relationships; curves for SED and RUN subjects virtually overlap;B, absolute force-power relationships;C, normalized force-power relationships.

In rats that are exercise trained on a treadmill for 2 hr, 5 days per wk for 6 wk, we observed no change in fiber diameter or peak force in slow or fast fibers. [33] Recently, we extended this work to include a group that trained for 3.5 hr per day, 5 days per wk. In agreement with our published work, fiber size (both slow and fast fibers) was unaffected by the exercise program. However, the 3.5 hr per day program caused a significant decline in the peak force ($kN/m^2$) of both type I and IIa fibers (unpublished observations). Our interpretation is that prolonged daily endurance exercise leads to a reduction in the total amount of myofibrillar protein and that this in turn reduces peak force and power. Because a high-power output is not required in endurance sports, the decline in peak force and power is not likely to be problematic for an endurance athlete. Nonetheless, these data do point to the need for heterogeneous programs of training that include endurance, flexibility, and strength components.

## Effect of Strength Exercise Training on Contractile Function

Although heavy resistance weight training is generally acknowledged to be the most effective way of increasing skeletal muscle fiber size and strength, [29] there is, compared with endurance exercise, a paucity of data regarding the cellular and molecular adaptations to strength training. An increase in muscle strength is a consistent observation after high-intensity exercise training in humans. The strength gains begin to occur before muscle fiber hypertrophy, and thus, the early gains have been attributed to an elevated neural drive. [9] Heavy

resistance training causes hypertrophy of all fiber types. Recently, Widrick et al. [39] studied the cellular effects of a 12-wk strength exercise program in young males. Single fibers were isolated from biopsy samples obtained from the vastus lateralis, and their functional properties were determined. Both slow type I and fast type IIa fibers showed a 30% increase in CSA and 30%–40% increases in peak force and power. There was no change in the force per cross-sectional area (kN/m2) or in the unloaded shortening velocity. Thus, the exerciseinduced increase in force and power was attributed entirely to the fiber hypertrophy. These results suggest that the strength exercise program did not increase the packing density of the myofilaments. This agrees with the findings of Claassen et al. [40] who found strength exercise training to have no effect on the spacing between myosin filaments or the ratio of actin to myosin filaments.

An important question is whether one type of strength exercise training may be superior to others. What, if any, are the advantages of isometric *vs.* isotonic or isokinetic contractions, and should eccentric contractions be employed? The answer will likely depend on the individual and their reason for training. An individual with primarily slow-twitch fibers would likely benefit from a different program of exercise than one with primary fast-twitch fibers, whereas strength programs for athletes and senior citizens would be different. A general observation is that peak isometric torque is increased more by isometric or eccentric programs of exercise than concentric isotonic contractions. [41,42] Häkkinen et al. [43] observed a 27% increase in maximal isometric leg extension force after a 24-wk training program that included an eccentric component, and Hather et al. [44] reported higher strength gains in subjects trained by concentric/eccentric exercise than by concentric alone. The greater increase in peak torque elicited by eccentric exercise training is thought to be related to the higher forces obtained during training compared with exercise involving isometric or concentric contractions. [45] This hypothesis is supported by a study in which power outputs performed during eccentric-only and concentric-only exercise were matched, such that the same amount of work was done during a 4-wk period. The concentric-only group showed greater gains in peak isometric torque and in the hypertrophy of type II fibers. [46]

As pointed out by Izquierdo et al., [47] the ability to develop force rapidly (measured by the rate of tension development, dP/dt) and peak muscle power are likely more important to the functional capacity of limb skeletal muscle than peak torque. Häkkinen et al. [48] reported that the increase in dP/dt after exercise training was correlated with an elevation in the integrated electromyography activity and the increased CSA of the fast-twitch, as compared with the slow-twitch fibers. Additionally, McCall et al. [49] found heavy resistance weight exercise training to significantly increase the type II/I fiber area ratio in the biceps brachii from 1.53 pretraining to 1.63 posttraining. These data support the hypothesis that at least part of the exercise training-induced increase in whole muscle dP/dt and the maximal shortening velocity (measured from the force-velocity relationship and thus abbreviated $V_{max}$) can be attributed to the increased fast-twitch fiber area and the relative contribution of this fiber type to the functional capacity of the whole muscle. The published literature is inconsistent in regard to whether one type of exercise training is more effective than others in increasing muscle $V_{max}$. Although eccentric and isometric contractions seem more effective in increasing peak torque than dynamic concentric contractions, only dynamic contractions have been shown to increase $V_{max}$. [41] This adaptation cannot be explained by fiber type shifts because all forms of exercise training have been shown to reduce the percentage of type IIb and increase the percentage of type IIa fibers. [9] A shift of this nature would reduce, not increase, muscle $V_{max}$. These data could be reconciled if heavy resistive exercise training increased the $V_{max}$ of the type IIa fiber type. Although this is an attractive hypothesis, Widrick et al. [39] failed to observe any change in type IIa fiber maximal unloaded $V_0$ following a 12-wk dynamic resistance training program in young males.

Besides strength and velocity, peak power will be influenced by the curvature of the force-velocity relationship. In the study of Widrick et al., [39] resistance training had a relatively small effect on the curvature of the force-

velocity relationship, as reflected by the $a/P_0$ ratio. Consequently, there was no pre- to posttraining difference in the fractional force or velocity that elicited peak power for either the slow type I or fast type IIa fiber. Izquierdo et al. [47] recently suggested that muscle performance might be best improved by combining high-velocity explosive types of exercise with heavy resistance training. However, Duchateau and Hainaut [41] found that isometric training increased peak power by 51% compared with only 19% following dynamic training. The isometric training-induced increase in peak power could be attributed entirely to improved strength, because muscle $V_{max}$ was unaltered. Additionally, subjects who trained at slow or intermediate isokinetic velocities improved peak power at all isokinetic testing velocities, whereas subjects trained at relatively high velocities displayed improvements only at the high test velocities.

Rat data suggest that the "ideal" heavy resistance exercise program may depend on the fiber type distribution of the muscles being trained. Tamaki et al. [50] observed a highresistance dynamic weight training program in rats to increase the fast-twitch gastrocnemius and plantaris muscle to body weight ratios by 18% and 31%, respectively, while having no effect on the slow-twitch soleus. One possible explanation is that the slow soleus may become unloaded during the rapid contractions of the fast gastrocnemius and plantaris, such that the force generated by the soleus would be low. Additionally, because the soleus is a one-joint muscle, it undergoes considerable shortening during plantar flexion of the ankle. Thus, during a dynamic contraction, the muscle would operate at continually shorter sarcomere lengths, which would further reduce the force capacity of the soleus. In contrast, an isometric contraction would allow the soleus to contract at an optimal sarcomere length and allow high forces to be maintained for the duration of the contraction. Consequently, isometric contractions may be more effective than dynamic contractions in inducing an increased strength and power of slow muscles, such as the soleus. We have preliminary data that this is indeed the case when one uses exercise to prevent muscle atrophy during hindlimb unloading in the rat (Hurst JE, Fitts RH, unpublished observations).

## Importance of Regular Exercise Programs for the Elderly

It is well established that aging is associated with significant declines in limb skeletal muscle mass (sarcopenia) and losses in peak force, velocity, and power. [51,52] One consequence of these changes is a reduced mobility and balance and an increased risk of falls that result in bone fractures. The latter leads to further declines in activity, muscle mass, and physical work capacity. Studies have shown that the elderly are capable of enhanced limb muscle performance following both endurance and highresistance programs of exercise and that this adaptive capacity exists, even in the very old. [53–55] An important question that is yet unanswered is what is the ideal exercise training program for the elderly. Should the program be primarily aerobic in nature or should it focus primarily on strength training or contain an equal balance of both? Endurance exercise training programs administered to elderly men and women have been shown to elicit adaptations in aerobic capacity similar to those observed in young people. For example, Coggan et al. [53] observed an average 23% increase in maximal aerobic capacity in older adults (64 ± 3 yr) who trained by exercising at 80% of maximal heart rate for 45 min per day, 4 days per wk, for 9–12 mo. Similar to young subjects, these older adults showed a significant decline in the fast type IIb and an increase in the fast type IIa fiber type, and an increase in capillary density and in muscle enzymes of both the Krebs cycle and β-oxidative pathways. Surprisingly, the training also induced a significant increase in the cross-sectional area of both type I and IIa fibers in older adults. [53] This result is not consistent with adaptations observed in rats [33] or master atheletes, [35] in whom fiber size was either unaffected or reduced by endurance exercise training. In the studies showing no change in fiber CSA with endurance exercise training, measurements were made on isolated single fibers maintained at the optimal sarcomere length, whereas in the study of Coggan et al., [53] the analyses were made on histochemical crosssections. With the latter technique, differences in fiber CSA can be caused by variability in the plane of sectioning and/or fiber length. In a subsequent study, we obtained biopsies from the gastrocnemius muscle of a similarly aged group of subjects (64 ± 1 yr) who underwent the same program of exercise as the subjects in the Coggan et al. [53] study. Individual

fibers were isolated from the biopsy and suspended between a force transducer and a position motor, and the fiber diameters were determined at a constant sarcomere length of 2.5 μ. These results showed that the endurance exercise program caused a significant 12% decline in the diameter of the slow type I fiber, whereas the type II fiber size was unaltered (Fig. 8). [56] As a result of the fiber atrophy, peak force of the type I fiber was also significantly depressed by the training program. Although the aerobic training program had clear, positive results in that it increased aerobic capacity, the type I fiber atrophy could lead to a reduced power and increased muscle weakness. For these reasons, it is clear that the elderly should not depend entirely on endurance exercise programs.
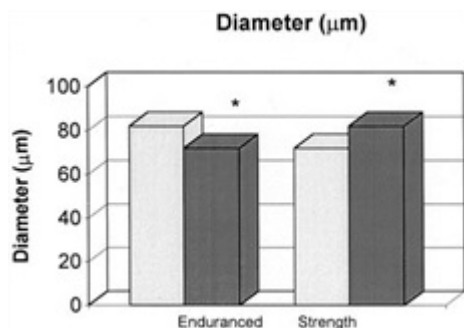


**Figure 8:** Comparison of the type I fiber diameter (μm) of 60- to 70-yr-old subjects before and after endurance (fibers from gastrocnemius muscle) and strength (fibers from vastus lateralis) exercise training programs. *P < .05.

Since high-resistance weight training has been shown to be effective in causing fiber hypertrophy in young subjects, we studied the effects of a 16-wk strength-training program in 60–70 yr olds. [54] Biopsy samples were obtained from the vastus lateralis of six men before and after the exercise program. The strength training consisted of two sessions/wk of four sets of resistive leg exercises at 75%–90% of maximal strength. The exercise program induced a 14% increase in the type I fiber diameter and a small but significant increase in peak force (Fig. 8). [57] The type II fibers were unaffected by the exercise program. Collectively, these data suggest that elderly individuals should be encouraged to participate in regular programs of exercise that contain both aerobic and strength components. In addition, because of the loss of flexibility and balance with age, the ideal exercise program should also contain flexibility exercises.

## SUMMARY

Considerably information can be obtained from an analysis of the isometric and isotonic contractile properties of limb skeletal muscle. For example, the isometric twitch duration is a reflection of the intracellular $Ca^{2+}$ transient, which in turn depends on the content and activity of the SR, whereas the rate of force development is dependent on the rate of transition of the cross-bridge from a low- to a high-force state. The peak isometric force provides an index of the number of cross-bridges acting in parallel; this value is generally slightly higher in fast compared with slow fibers. Maximal unloaded shortening velocity ($V_0$, fiber length/sec) is fiber type dependent with the hierarchy for $V_0$ IIb > IIx > IIa > I. In humans, the $V_0$ of the type IIa and IIx fibers are between 3- to 4- and 6-fold higher, respectively, than the type I fiber. Fiber $V_0$ is a reflection of the overall cross-bridge cycle speed, which is dependent on the myofibrillar ATPase activity. From a physical performance perspective, an important functional property is peak power, which can be determined from the force-velocity relationship.

Skeletal muscle is a very mutable tissue, and it shows multiple adaptations to programs of regular exercise. It is clear that all exercise programs (endurance and strength) reduce the number to type IIb and IIx fibers and increase the percentage of type IIa fibers. Despite the fact that they have the highest power, type IIb fibers

would be of little use during exercise of any duration because of their rapid fatigability. A not fully resolved question is whether programs of regular exercise can shift fibers from fast to slow or vice versa. When such shifts were observed, the percent change was small.

Regular endurance exercise is known to increase the myofibrillar ATPase activity and the $V_0$ of the slow soleus. At the single-cell level, individual slow fibers showed an approximate 20% increase in $V_0$ and ATPase activity, and this change occurred without any detectable change in the MHC. The exercise-induced increase in the slow fiber $V_0$ was associated with an increase in the content of fast myosin light chains. In contrast to the observed affects on slow muscle, endurance exercise training reduced the myofibrillar activity of primarily fast-twitch muscles and the velocity of single fast type II fibers isolated from fast muscles. The mechanism of this change has not been established, but the reduced type II fiber velocity may have resulted from a fiber shift from type IIx to IIa.

Fiber CSA was generally not altered (or somewhat reduced) by regular endurance exercise, whereas strength exercise training induced hypertrophy in both slow- and fast-twitch fibers. Although strength training increased peak force in all fiber types, the force per CSA and $V_0$ were unaltered. This indicates that the functional effects of the strength training could be entirely attributed to the fiber hypertrophy. The increase in whole muscle dP/dt and $V_{max}$ observed following some strength training programs was likely a result of an increased fast-twitch fiber area and the relative contribution of this fiber type to the functional capacity of the whole muscle. Rat studies suggest that the optimal training for improving force and power may be fiber type dependent. Fast muscles may benefit more from high-resistance dynamic weight training, whereas slow muscles adapt best with isometric contractions. Finally, it is clear from the published data that the optimal exercise programs for both young and old, men and women, require a combination of both endurance and strength exercises and a flexibility component. Additional research is needed to determine if the optimal ratio of these components may vary with sex or age.

## ACKNOWLEDGMENTS

## REFERENCES
1. Close RI: Dynamic properties of mammalian skeletal muscles. *Physiol Rev* 1972; 52: 129–97
2. Greeves MA, Holmes KC: Structural mechanism of muscle contraction. *Ann Rev Biochem* 1999; 68: 687–728
3. Franzini-Armstrong C, Protasi F: Ryanodine receptors of striated muscles: A complex channel capable of multiple interactions. *Physiol Rev* 1997; 77: 699–729
4. Fitts RH, Riley DA, Widrick JJ: Functional and structural adaptations of skeletal muscle to microgravity. *J Exp Biol* 2001; 204: 3201–8
5. Fitts RH, Riley DA, Widrick JJ: Physiology of a microgravity environment invited review: Microgravity and skeletal muscle. *J Appl Physiol* 2000; 89: 823–39
6. Green H, Goreham C, Ouyang J, et al: Regulation of fiber size, oxidative potential, and capillarization in human muscle by resistive exercise. *Am J Physiol* 1999; 276: R591–6
7. Frontera WR, Suh D, Krivickas LS, et al: Skeletal muscle fiber quality in older men and women. *Am J Physiol* 2000; 279: C611–8
8. Straub V, Campbell KP: Muscular dystrophies and the dystrophin-glycoprotein complex. *Curr Opin Neurol* 1997; 10: 168–75
9. Fitts RH, Widrick JJ: Muscle mechanics: Adaptations with exercise-training. *Exerc Sports Sci Rev* 1996; 24: 427–73

10. Fitts RH, Courtright JB, Kim DH, et al: Muscle fatigue with prolonged exercise: Contractile and biochemical alterations. *Am J Physiol* 1982; 242: C65–73

11. Fitts RH, Metzger JM, Riley DA, et al: Models of disuse: A comparison of hindlimb suspension and immobilization. *J Appl Physiol* 1986; 60: 1946–53

12. Schulte LM, Navarro J, Kandarian SC: Regulation of sarcoplasmic reticulum calcium pump gene expression by hindlimb unweighting. *Am J Physiol* 1993; 264: C1308–15

13. Fitts RH: Cellular mechanisms of muscle fatigue. Physiol Rev 1994; 74: 49–94

14. Metzger JM: Mechanism of chemomechanical coupling in skeletal muscle during work. *Perspect Exerc Sci Sports Med* 1992; 5: 1–51

15. Metzger JM, Moss RL: Calcium-sensitive cross-bridge transitions in mammalian fast and slow skeletal muscle fibers. *Sci* 1990; 247: 1088–90

16. Westerblad H, Lännergren J: Slowing of relaxation during fatigue in single mouse fibres. *J Physiol* 1991; 434: 323–36

17. Edman KAP: The velocity of unloaded shortening and its relation to sarcomere length and isometric force in vertebrate muscle fibres. *J Physiol* 1979; 291: 143–59

18. Hill AV: The heat of shortening and dynamic constants of muscle. *Proc R Soc Lond Ser B* 1938; 126: 136–95

19. Smerdu V, Karsch-Kizrachi I, Campione M, et al: Type IIx myosin heavy chain transcripts are expressed in type IIb fibers of human skeletal muscle. *Am J Physiol* 1994; 267: C1723–8

20. Widrick JJ, Trappe SW, Costill DL, et al: Force-velocity and force-power properties of single muscle fibers from elite master runners and sedentary men. *Am J Physiol* 1996; 271: C676–83

21. Krivickas LS, Suh D, Wilkins J, et al: Age- and gender-related differences in maximal shortening velocity of skeletal muscle fibers. *Am J Phys Med Rehabil* 2001; 80: 447–455

22. Coyle EF, Costill DL, Lesmes GR: Leg extension power and muscle fiber composition. *Med Sci Sports* 1979; 11: 12–5

23. Caiozzo VJ, Haddad F, Baker MJ, et al: Microgravity-induced transformations of myosin isoforms and contractile properties of skeletal muscle. *J Appl Physiol* 1996; 81: 123–32

24. Pette D, Vrbová; G: Invited review: Neural control of phenotypic expression in mammalian muscle fibers. *Muscle Nerve* 1985; 8: 676–89

25. Esbjörnsson M, Hellsten-Westing Y, Balsom PD, et al: Muscle fibre type changes with sprint training: Effect of training pattern. *Acta Physiol Scand* 1993; 149: 245–6

26. Andersen JL, Klitgaard H, Saltin B: Myosin heavy chain isoforms in single fibres from m. vastus lateralis of sprinters: Influence of training. *Acta Physiol Scand* 1994; 151: 135–42

27. Howald H, Hoppeler H, Claassen H, et al: Influences of endurance training on the ultrastructural composition of the different muscle fiber types in humans. *Pflugers Arch* 1985; 403: 369–76

28. Adams GR, Hather BM, Baldwin KM, et al: Skeletal muscle myosin heavy chain composition and resistance training. *J Appl Physiol* 1993; 74: 911–5

29. Staron RS, Leonardi MJ, Karapondo DL, et al: Strength and skeletal muscle adaptations in heavy-resistance-trained women after detraining and retraining. *J Appl Physiol* 1991; 70: 631–40

30. Green HJ, Klug GA, Reichmann H, et al: Exercise-induced fibre type transitions with regard to myosin, paravalbumin, and sarcoplasmic reticulum in muscles of the rat. *Pflugers Arch* 1984; 400: 432–8

31. Holloszy JO, Booth FW: Biochemical adaptations to endurance exercise in muscle. *Annu Rev Physiol* 1976; 38: 273–91

32. Baldwin KM, Winder WW, Holloszy JO: Adaptations of actomyosin ATPase in different types of muscle to endurance exercise. *Am J Physiol* 1975; 229: 422–6

33. Schluter JM, Fitts RH: Shortening velocity and ATPase activity of rat skeletal muscle fibers: Effect of endurance exercise training. *Am J Physiol* 1994; 266: C1699–713

34. Fitts RH, Costill DL, Gardetto PR: Effect of swim exercise training on human muscle fiber function. *J Appl Physiol* 1989; 66: 465–75

35. Widrick JJ, Trappe SW, Blaser CA, et al: Isometric force and maximal shortening velocity of single muscle fibers from elite master runners. *Am J Physiol* 1996; 271: C666–75

36. Sweeney HL, Kushmerick MJ, Mabuchi K, et al: Myosin alkali light chain and heavy chain variations correlate with altered shortening velocity of isolated skeletal muscle fibers. *J Biol Chem* 1988; 263: 9034–9

37. Greaser ML, Moss RL, Reiser PJ: Variations in contractile properties of rabbit single muscle fibres in relation to troponin T isoforms and myosin light chains. *J Physiol* 1988; 406: 85–98

38. Larsson L, Moss RL: Maximum velocity of shortening in relation to myosin isoform composition in single fibres from human skeletal muscles. *J Physiol* 1993; 472: 595–614

39. Widrick JJ, Stelzer JE, Shoepe TC, et al: Functional properties of human muscle fibers after short-term resistance exercise training. *Am J Physiol* 2002;283 (in press)

40. Claassen H, Gerber C, Hoppeler H, et al: Muscle filament spacing and short-term heavy-resistance exercise in humans. *J Physiol* 1989; 409: 491–5

41. Duchateau J, Hainaut K: Isometric or dynamic training: Differential effects on mechanical properties of a human muscle. *J Appl Physiol* 1984; 56: 296–301

42. Jones DA, Rutherford OM: Human muscle strength training: The effects of three different regimes and the nature of the resultant changes. *J Physiol* 1987; 391: 1–11

43. Häkkinen K, Alén M, Komi PV: Changes in isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of human skeletal muscle during strength training and detraining. *Acta Physiol Scand* 1985; 125: 573–85

44. Hather BM, Tesch PA, Buchanan P, et al: Influence of eccentric actions on skeletal muscle adaptations to resistance training. *Acta Physiol Scand* 1991; 143: 177–85

45. Westing SH, Seger JY, Karlson E, et al: Eccentric and concentric torquevelocity characteristics of the quadriceps femoris in man. *Eur J Appl Physiol* 1988; 58: 100–4

46. Mayhew TP, Rothstein JM, Finucane SD, et al: Muscular adaptation to concentric and eccentric exercise at equal power levels. *Med Sci Sports Exerc* 1995; 27: 868–73

47. Izquierdo M, Häkkinen K, Ibañez J, et al: Effects of strength training on muscle power and serum hormones in middle-aged and older men. *J Appl Physiol* 2001; 90: 1497–507

48. Häkkinen K, Alén M, Komi PV, et al: Effect of explosive type strength training on isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. *Acta Physiol Scand* 1985; 125: 587–600

49. McCall GE, Byrnes WC, Dickinson A, et al: Muscle fiber hypertrophy, hyperplasia, and capillary density in college men after resistance training. *J Appl Physiol* 1996;81F

50. Tamaki T, Akatsuka A, Tokunaga M, et al: Morphological and biochemical evidence of muscle hyperplasia following weight lifting exercise in rats. *Am J Physiol* 1997; 273: C246–56

51. Frontera WR, Hughes VA, Lutz KJ, et al: A cross-sectional study of muscle strength and mass in 45- to 78-yr-old men and women. *J Appl Physiol* 1991; 71: 644–50

52. Larsson L, Grimby G, Karlsson J: Muscle strength and speed of movement in relation to age and muscle morphology. *J Appl Physiol* 1979; 46: 451–6

53. Coggan AR, Spina RJ, King DS, et al: Skeletal muscle adaptations to endurance training in 60- to 70- yr- old men and women. *J Appl Physiol* 1992; 72: 1780–6

54. Yaraskeski KE, Zachwieja JJ, Campbell JA, et al: Effect of growth hormone and resistive exercise on muscle growth and strength in older men. *Am J Physiol* 1995; 268: E268–76

55. Fiatarone MA, Marks EC, Ryan ND, et al: High-intensity strength training in nona-genarians: Effects on skeletal muscle. *JAMA* 1990; 263: 3029–34

56. Thompson LV, Romatowski J, Spina RJ, et al: Effect of endurance exercise on the contractile properties of slow and fast fibers in 60–70 yr olds. *Med Sci Sports Exerc* 1994; 26: S215

57. Romatowski J, Thompson LV, Yarasheski KE, et al: Effect of strength training on the contractile function of slow and fast fibers in 60–70 yr olds. *Med Sci Sports Exerc* 1994; 26: S215