

Motor Unit Properties & Fiber Types: Speculations on Exercise Prescription

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There is little doubt that genetics play a major role in one's predisposition for muscular strength and hypertrophy. Almost all authors also recognize the need for a sufficient level of training intensity (i.e. the degree to which a muscle has been fatigued/inroaded per unit time) for optimizing muscle strength and hypertrophic adaptations. Precise quantification of training intensity, however, is relatively difficult to monitor; consequently, relatively uniform training guidelines are often prescribed (i.e. train to concentric muscular failure within a 45-90 sec. time interval). Many authors also acknowledge the importance of individual recovery ability and/or fiber type as important factors that need to be considered when implementing any exercise program. Precisely how and to what degree these factors affect exercise intensity and prescription is currently being debated and will be the focus of this paper.

There is also increasing awareness by many authors that individuals will need to change some aspect of their training, if long-term progress is to continue. One group of authors believe that, in order to ensure optimal and continued progress, all trainees (regardless of fiber-type, recovery ability, etc.), will need to continually down-regulate the volume and frequency of workouts as they get progressively stronger. It is argued that increases in recovery ability cannot keep pace with strength increases. Some of these same authors also contend that as one gets progressively stronger, there will also be a need to decrease the muscular time under load (TUL) or number of repetitions performed/exercise. Another group of authors disagree with this view. They argue for variety in training parameters as the key to long-term progress (21). They contend that only by continually varying the frequency, intensity, repetition speed, volume of workouts, etc., can one ensure optimal adaptation of all the different muscle components, fibers, enzymes, etc. By looking at some of the relevant exercise research/principles and their biological correlates (i.e. how these entities may be realized in physical mechanisms at a more "fundamental" cellular level), an attempt will be made to offer some speculative input on some of these issues and controversies.

Histochemical Properties of MUs

A MU includes a motor neuron and all the muscle fibers it innervates. Properties of MUs include the following:

1. The physiological and biochemical properties of MUs can be divided into distinct subtypes that show a variation in maximal force, isometric twitch-speed, and fatigue resistance (22, 26, 36, 58, 59). Furthermore, the transient appearance of hybrid forms, simultaneously expressing multiple forms suggests that there may exist a gradual transition between them. Based on contraction strengths, firing thresholds and fatigue resistance, they are usually divided into 3 or 4 types. These include:

a. S(slow-twitch or type I)-these fibers are generally innervated by smaller, slower conducting neurons. They have a lower firing threshold (i.e. recruited at lower force levels), produce less force but show greater fatigue resistance. They are rich in oxidative enzymes. Although quite variable, many of the postural muscles or muscles designed to support sustained periods of activation (diaphragm, spinal extensors, some leg muscles, soleus, abdominals) are particularly rich in these fibers. Such units can be found to be tonically or phasically active, firing at fairly low rates for up to 20-35% of the day (15). Endurance athletes often display a higher relative percentage of these fibers (15, 35).

b. FR (fast-twitch, fatigue resistant, or type IIA)-these fibers are generally innervated by medium diameter neurons, have medium firing threshold, produce medium or high force and are also rich in oxidative enzymes. Fatigue resistance tends to be moderate or high. Many bodybuilders seem to have high relative amounts of these fibers (33). This has led some researchers to suggest that these fibers are particularly adaptive to hypertrophic responses under appropriate training stimuli (33).

c. Flnt and FF (fast-twitch, intermediate fatigue and fast-twitch, fatigable or type IIAB/IIIX and IIB)-these fibers are generally innervated by the larger, fastest conducting neurons, have the highest firing thresholds (i.e. last to be recruited) and produce the greatest force. They fatigue quite rapidly and are poor in oxidative enzymes and rich in glycolytic enzymes. Major antigravity antagonists and/or muscles implicated in powerful phasic movements (biceps, hamstrings, etc.) are often rich in these fibers. These units typically fire in short, scarce, high frequency bursts. Sprinters and Olympic lifters often have a higher relative percentage of these fibers (2, 63). Surprisingly, a few studies show that bodybuilders often "possess significantly less type IIB Myosin Heavy Chain (MHC) isoforms than untrained controls or even endurance-trained...subjects." (33)

2. All fibers innervated by a given motor neuron have the same physiological and histochemical properties. This is not surprising, since it is known that the type of motor neuron and pattern of nerve impulses transmitted, plays a significant role in determining the mechanical and histochemical properties of the muscle fibers of a MU. In fact, cross-innervation of fast-twitch (FT) muscle fibers by a nerve that previously supplied slow-twitch (ST) fibers has been shown to transform those muscles to take on ST characteristics (3,4). Some studies show complete myosin isoform transformations (3, 8). In cross-reinnervation of ST fibers by FT nerves, there also appears to be changes in properties of muscle myosin towards those of FT, but the transformation is less complete. It has been suggested that "sensitivity to motor innervation increases from the glycolytic to the oxidative types of fibers, in the order IIB>IIIX/D>IIA>I (3)". Transformations, however, are not complete with respect to all parameters. For example, muscle satellite cells appear to maintain their original properties in cross-reinnervation studies (4).

3. There is experimental evidence to suggest that chronic long-term stimulation can transform type IIB fibers into IIA and even to type I (8, 15). In fact, "chronic stimulation of a FT muscle at a frequency resembling that in a nerve to a ST muscle causes as complete a transformation of the muscle fibers as cross-innervation." (8) This includes not only complete isozymic transformation but also "a marked increase in the time-to-peak and half-relaxation time of the isometric twitch, decrease in tetanus-to-twitch ratio, and a decrease in the rate of development of tetanic tension" (15). Thus, "indirect stimulation of a FT muscle with an impulse pattern similar to that normally delivered to a slow muscle results in an orderly sequence of changes affecting all functional elements of the muscle fibers: the contractile and regulatory proteins of the thick and thin filaments, the proteins of the Ca⁺-regulatory system, as well as enzyme activity and isozyme patterns of energy metabolism" (51). It has also been suggested that the use of particular training protocols (endurance, sprint or strength) can similarly result in fiber conversion via training-induced altered gene expression. In particular, resistance training has been shown to alter the MHC toward the FR or type IIA fibers (2, 33, 51). Likewise, sprint training has been shown to increase the ratio of type II/type I cross-sectional area with some studies demonstrating greater hypertrophy of the type IIB fibers (1, 39, 40).

Alternatively, many endurance training studies done in both animals and humans "have demonstrated by mATPase histochemistry, increases in the fraction of type IIA fibers with concomitant decreases in type IIB fibers" (51). Of course, the changes that occur with exercise, are nowhere as dramatic as those seen with stimulation or cross-reinnervation experiments; consequently, many authors dispute the concept of exercise-induced fiber conversion (30, 55). They are quick to point to twin studies which demonstrate that MZ and not DZ twins show identical fiber distribution (15). Thus, they argue, fibers cannot be altered by training and any "apparent" fiber conversion is the result of selective hypertrophy of one fiber-type combined with selective atrophy of different fiber type (due to disuse or overuse atrophy) (30). For example, while strength training will induce hypertrophic adaptation of the larger FT fibers (particularly, the type IIA), it will have a smaller effect on ST fibers. In fact, depending on the previous level of endurance training, there may even be a relative atrophy of ST fibers (52). Conversely, while endurance training and/or high volume, low intensity, resistance training may induce hypertrophy of the S units and some of the more fatigue-resistant FR units, it may result in atrophy of the larger fatiguable units. In fact, "a decrease in muscle fiber size has been demonstrated in the gastrocnemius muscle of marathon runners and a reduced fiber size after endurance training has also been demonstrated in rats and horses" (15). At this time, no conclusive proof exists for exercise-induced ST to FT conversion, although there are many studies suggesting FT sub-type interconversions (1, 2, 33, 40).

4. Statistical histological analysis also reveals that muscle fibers display a distinct tendency to be surrounded by fibers of a different type. Similar fiber subtypes are often not associated in the immediate vicinity of each other. There also appears to be a predominance of slower fibers in the deep layers of muscles while superficial layers tend to have a greater concentration of faster fibers (26, 36). It has been hypothesized that this layering allows for optimizing mechanical advantage during dynamic movements. Finally, as one ages, there seems to be a preferential atrophy of type II fibers (17, 58). Whether this is the result of disuse atrophy or is part of the "normal" biological aging process, is currently not known. There is, however, plenty of evidence demonstrating the usefulness of resistance training in reversing age-related muscular atrophy.

5. In general (although there are many exceptions), fast-twitch (FT) fibers are about 30-40% larger in cross-sectional area than the slow-twitch (ST) fibers. Thus, even in a muscle composed of a 50/50 mix, the overall % FT contribution to total cross-sectional area of the muscle can be up to 65% or more, depending on the level of hypertrophy (52, 62). In FT subjects and/or muscle groups, this is even higher.

Relevancy to Bodybuilding and Strength Training With regard to optimizing hypertrophy, will individual and/or muscle fiber-type distribution affect training prescription? Although there is some controversy, some authorities believe that fiber-type distribution is indeed, relevant to exercise prescription (30, 31, 52, 68). In particular, it has been argued that all other factors being equal, FT subjects and/or muscle groups usually require (for optimal strength progression):

1.Lower volume and/or frequency of exercise. 2.Lower repetition ranges and/or TUL. 3.Longer recovery periods.

ST subjects and/or muscles show completely opposite characteristics requiring:

1.Higher volume and/or frequency of exercise.

2.Higher repetition ranges and/or TUL. 3.Shorter recovery periods.

Intermediate fiber type individuals and/or muscle groups fall somewhere in between these two groups. A. Jones, in particular, has argued that an individual's muscle fiber recruitment and fatigue characteristics are largely genetically determined, so that there may exist an optimal TUL for each exercise, where one's musculature receives optimal growth stimulation (30). In fact, MedX technicians often incorporate the use of a "Fatigue Response Test" as a way of finding out one's particular fatigue and fiber-type characteristics. More recently, this theme has been repeated by several other authors who contend, "that (even) the concept of double progression (increasing weight and reps) is actually mistaken. Instead one should find the signature TUL for a given person in that movement and then carry out single progression. That is, progress weight at a fixed TUL as is determined by a particular fiber type and MU recruitment pattern. Once you know the ideal TUL, single progression (increasing resistance) appears to be the way to rapid gains." (44)

There are, however, relatively few (if any) properly controlled, peer-reviewed research studies looking at these parameters. Most studies involve the simultaneous variation of multiple variables, including different intensities, volumes and frequencies that make any conclusions highly questionable. There are, however, a number of non-formal studies, by at least one equipment manufacturer (A. Jones of MedX and Nautilus) and two other exercise researchers (T.V. Pipes and W.L. Westcott) suggesting differences in exercise prescriptions based on fiber type (30, 52, 68). Furthermore, one study looking at the relative effectiveness of a 50% of 1-RM (20 repetitions) protocol versus 80% of 1-RM (12 repetitions), showed statistically significant strength increases with the 80% protocol and not the 50% protocol (13). Somewhat similar (although, not universal) findings have been reported elsewhere (21, 43, 48). Furthermore, these researchers found that the increases in strength were positively correlated to the FT fiber content in the muscle being exercised. What is interesting, however (and not reported by authors), is that in Fig. 6 of their article, there seems to be a tendency for ST subjects to have a somewhat better response than FT subjects with the 50% protocol and some of the extremely high % FT subjects seem to display even a strength decrement with the 50% of 1-RM training protocol (13).

In the final analysis, we do know that FT subjects and/or muscles clearly display different levels of muscular endurance or fatigue in comparison to ST subjects and/or muscle groups. For example, many studies show that skeletal muscles with a predominance of FT fibers possess shorter contraction times, higher twitch and tetanic tensions, and greater susceptibility to fatigue than muscles with predominantly ST fibers (47, 61, 62, 64). Many studies involving human subjects have shown a positive correlation between % FT distribution and the level of muscle fatiguability (10, 15, 16, 28, 29, 34, 61, 65). It is also known that women (who often have significantly smaller FT fibers, and a lower type II/type I area ratio) display significantly longer muscular endurance times than men (45, 67). In some studies, the type I fibers in women were found to be larger than their type II fibers (23, 58). Whether this difference between the sexes is biologically dictated or the result of disuse atrophy in women, is not known; there is, however, one study suggesting the latter view since it was found that sprint training had a greater increase of type II fiber area (especially the type IIB fibers) in women than in the men. These authors "suggested that the smaller area of type II fibers generally found in muscle of women may in part be due to less frequent activation of their type II fibers, especially type IIB. It could be expected that less well-trained subjects (the women) would show a greater training response than subjects closer to their upper limit of performance (the men)" (39).

It is also known that individuals with congenital myopathies (central core disease and nemaline rod myopathy) who are characterized by type I fiber predominance, show greater levels of muscular endurance (and lower levels of strength) relative to controls (42). Likewise, "there is no decline in endurance time for older muscles when an isometric contraction is performed at a relative percentage of the maximum force. The absence of an increase in fatiguability with age is probably related to the greater proportion of ST muscle fibers" (17).

There are many reasons given for the greater fatiguability of FT fibers and/or muscles but "the preponderance of evidence suggests that the primary sites of fatigue lie within the muscle itself." (20) In particular, it is known that FT fibers have approximately a 50% lower lactate-H⁺ transport rate than ST fibers (32). Differences are especially pronounced in the type IIB fibers. Thus, it has been hypothesized that a greater level of exercised-induced acidosis (decrease in pH) in FT fibers results in greater rates of fatigue. Recently, however, the pH effects on muscle contractility have been shown to be less critical at normal (above 25 degrees C) physiological temperatures (54, 69). Even more recently, increases in the products of ATP hydrolysis (Pi, ADP, AMP) have been implicated in muscle fatigue (54, 69). High intensity exercise is known to induce a greater increase in Pi levels in FT fibers (20). Regardless, most researchers do not see muscular fatigue as the result of neuromuscular transmission failure since "fatigue (is) not associated with a substantial decline in the MU action potential (either between-or within train)" (16, see also 7, 12, 20, 49, 70).

Finally, some studies have demonstrated fiber-type differences with respect to decompensation/atrophy and length of detraining. For example, while "the muscle oxidative potential was shown already to be significantly reduced as early as after 1 week of detraining...the glycolytic enzyme activity remained stable even after 12 weeks of interruption in training...(Furthermore)...even after 7 weeks of detraining, the sprint training-induced hypertrophy in both extensor muscles and fibers was maintained or even increased" (41). With all the known differences in properties between the different classes of fiber types, it would, therefore, not be surprising to also find differences in adaptive training ranges among the different classes of fiber types. Whether the simultaneous optimal hypertrophy of all the different fibers is physiologically possible, is debatable. There are however, some data to suggest incompatibility and compromise between different modes of training. In fact, Stone et al. (1996) have argued that, "the function of the transforming myonuclei pool that is maintained in adapting fibers in response to overload may be limited such that they can adequately support the expression of proteins that enable a high endurance capability, i.e. mitochondria, at the expense of a high force-generating potential (contractile machinery) and vice versa" (14, 60).

MU Recruitment Properties

During graded voluntary muscular contractions, MUs are recruited in order of increasing size, increasing contraction strength and diminishing fatigue resistance. Thus, the smaller, less powerful, fatigue-resistant fibers are almost always found to be recruited before the larger, more powerful, fatigable fibers, regardless of speed of contraction (Henneman's size principle). Furthermore, "all MUs are recruited at successively lower force levels if the contractions are performed at increasingly greater velocities. (In fact)...during the fastest contraction of tested movements, the recruitment threshold (becomes) so low for all units, including the largest, that the motor neuron pool (is) virtually simultaneously activated." (22) Although, the threshold force of fiber recruitment of all MUs decreases at increasingly faster contractions of fiber recruitment (i.e. in brisk phasic or ballistic contractions), the same general fiber recruitment pattern (from smaller to larger) is still maintained. In some rare cases, however, if the movements are carried with sufficient velocity and the conduction is occurring along a fairly long axon (say 1 meter or so) even though the smaller motor neurons get recruited earlier, there will be an "apparent" reversal of fiber recruitment order, because of the slower conduction velocity of the smaller lower-threshold motor neuron (22).

We also know that highly motivated subjects are quite capable of achieving full, maximal voluntary contraction (MVC), since supramaximal electrical stimulations superimposed upon a MVC have been shown not to increase muscular tension (5, 6, 15). Thus, when one is using a high % of maximum muscular tension (as is likely to occur in most strength training protocols), there will come a point in any set (and often quite early in the set) where you will have effectively recruited all MUs available for that particular exercise movement. Subsequently, any further increases in force are generated by increasing the firing rate (i.e. pulse modulation) of all these recruited units. Since it is also recognized that a fused muscle contraction is 5-10 times higher than an unfused contraction then, it would seem reasonable to conclude that firing rate is the main regulator of force during tonic contraction (15). In fact, in some of the smaller muscles, only at relatively low levels of force, is the recruitment of fibers, the major mechanism of increasing the force of voluntary contractions. In these muscle groups, it was found that a large % of total MUs were already recruited at relatively low force levels and increasing firing rate tended to be the main mechanism at both intermediate and high force levels (15, 18, 22, 25, 46). Thus, pulse modulation "contributes the large majority of force if the entire physiological range is considered." (46) In fact, Grillnar and Udo found that about 90% of soleus MUs were already recruited at below 50% of maximal tension and no MUs were recruited beyond 75% of maximal tension (25). In testing the MU recruitment of hand muscles, Milner-Brown found that up to 50% of MUs were already active at a 200g force level of a total maximal tension of about 4 kg (i.e. at only 5% of maximal tension)(46). Similar findings have also been reported by others (15). It has been hypothesized that, "if recruitment were the only (or even principle) means by which additional force was developed, the muscle would be incapable of producing a smoothly increasing contraction. As force increased, the orderly addition of large MUs would produce a 'staircase' effect in the force output record" (11).

The tension at which new MUs are recruited, however, does seem to vary depending on muscle group studied. There also seems to be some inconsistencies in the research findings. While some studies report MU recruitment to play a major role in up to 80% of MVC in the deltoid and brachial bicep muscles, others report no new recruitment above 40% MVC for the same muscles (11, 18, 38). Whether these differences are the result of differences in fiber-types and/or muscle use is unclear. Many researchers have also suggested that during prolonged contractions involving a relatively large % of MVC (as seen in most strength training protocols), the larger, more fatigable units, will inevitably be de-recruited before the smaller units in the reverse order in which they were recruited (11, 18, 49). Thus, in most strength training protocols, which employ a relatively high % of 1-RM, the majority of the fibers (including the largest, fatigable, high threshold types), will eventually be recruited (and often quite early in the set,) especially if taken to concentric failure. In fact, even during prolonged low levels contractions (employing a relatively low percent of a 1-RM) "there was a successive recruitment of motor units to compensate for contractile fatigue, so that all motor units finally were depleted of glycogen" (15). Fallentin et al. (1993) contend that during prolonged low level contractions (i.e. <20% of MVC or so), newly recruited MUs replace previously active and fatiguing units so that "MU rotation" may be an important characteristic in such prolonged submaximal contractions (18).

Clearly such findings (if accurate) have some bearing on current debates in training philosophies.

Thus, as R. Carpinelli points out, "as fatigue increases throughout a set of repetitions, your brain recruits greater number of MUs and stimulates them more frequently. When you achieve maximal recruitment, further increases in force are generated by continuing to increase the frequency of stimulation of all the MUs. At the point of momentary muscular fatigue (and probably much sooner)...you are recruiting the maximal number of MUs available for that specific exercise." (9)

So the typical bodybuilding argument that multiple sets of the same exercise will recruit more MUs or muscle fibers, is very likely erroneous. In fact, the same level of fiber recruitment and a much greater level of fiber fatigue can be induced by "drop-sets" (i.e. "descending pyramid" training protocol) (43) or even single sets (carried to momentary failure) employing a longer set duration. One may, however, argue that multiple sets of the same exercise (assuming sufficient rest between sets) may provide greater hypertrophic stimulation by recruiting the same fibers more times; whether this is more effective in optimizing hypertrophic adaptations is currently being debated. The preponderance of scientific evidence, however, does not support the superiority of multiple sets (19, 50, 57). (For an extensive review see R. Carpinelli's article in the February, 1997 issue of Master Trainer).

Speculations & Recommendations

1. All other factors being equal (i.e. recovery ability, training volume, nutrition, etc.), those individuals and/or muscle groups that have a greater % of FT, tend to fatigue more rapidly because a greater % of total muscular force is generated by the more fatigable and powerful FT fibers. As each of these larger fibers fatigues (i.e. de-recruitment), one sees a fairly rapid drop in strength (greater inroad/time under tension). This makes sense on two counts. First, due to a greater level of fatiguability of the larger and more abundant fibers in FT subjects, there will be a more rapid decline in strength in these trainees's muscle groups. Secondly, each of these larger fibers contributes greater increments of tension than smaller fibers. Thus, as these more abundant and powerful fibers in FT subjects and/or muscle groups fatigue, a correspondingly greater decrement in strength will result. With ST subjects and/or muscle groups (where the majority of total muscular force is generated by smaller, more fatigue-resistant fibers), one sees the opposite pattern. Since these fibers are fatigue-resistant and contribute smaller increments to total force, then, there will be a correspondingly shallower drop in muscular force output and fatigue with each successive repetition or TUL. So, it's not surprising to find great variability among these two contrasting fiber types. As A. Jones has pointed out, "So far, out of several 100 subjects, the widest range we have encountered involved one subject that could perform only 1 rep with 80% of his positive strength...and another subject that performed 34 reps with more than 80% of her positive strength." (31)

2. Since selective or greater hypertrophy of FT fibers is usually observed in most strength training programs (27, 63), then, as an individual gets progressively stronger, a greater % of total muscular tension will be generated by the pool of FT fibers. It then follows that as one gets progressively stronger, one's muscle fiber characteristics will take on more "FT-like" characteristics. By this account, the recommendations put forth by some authors to progressively down-regulate the volume and frequency of workouts as one gets stronger, make sense (i.e. such long-term trainees are simply responding more and more like FT subjects). Whether this fully explains the disproportionality that exists between strength and recovery ability increases in long-term trainees, is debatable. It does, however, offer a fairly simple explanation. The need to decrease TUL as one grows stronger (a view put forth by some authors), also makes sense by this account. As one gets progressively stronger, and the adapting FT fibers hypertrophy (to a greater extent than the ST fibers), one will see a greater level of fatiguability (increased inroad/time under tension). Effectively, the level of intensity (inroad/time) tends to increase as one gets stronger, since the FT contribution to total muscular force becomes greater. Thus, if you want to continue maximizing hypertrophic adaptations of this newly hypertrophied pool of fibers, you may also need to decrease TUL to match the new intensity levels. Thus, for long-term strength and muscle growth, one may need to continue adjusting training parameters such as recovery, volume, frequency, TUL, etc. to allow for the best growth stimulus of one's largest and most abundant fiber pool. Failure to do so, may result in less than optimal results.

3. Regardless of loads employed (at least, within reasonable limits), a set of any exercise movement carried to momentary muscular failure (or close to failure) will normally recruit the maximal number of MUs available for that exercise. This includes the entire spectrum of muscle fibers, from the most fatigue-resistant, slow-twitch (ST)-oxidative or type I muscle fibers, to the moderately fatigue-resistant, fast-twitch (FT)-oxidative or type IIA fibers to the most powerful, fatigable, FT-glycolytic or type IIB fibers. In fact, in a set taken to or close to momentary muscular failure, as the % of a 1-RM used decreases (at least, within reasonable limits), one is effectively fatiguing a greater percentage of available fibers (including the larger units). This explains why one is momentarily weaker when training to failure with a set involving a lower % of a 1-RM. It also explains the greater level of strength decrement that occurs in successive sets carried to failure, when the initial set employs higher repetitions or TULs. Effectively, you have exhausted a greater % of total fibers (including the larger fibers) when employing a higher set duration or TUL in the first set. Furthermore, if a set using a lower % of a 1-RM (taken to failure) recruits the same number of fibers (and fatigues a greater number of them) than a set with a higher % of a 1-RM taken to failure (as has been suggested in 15, 18) and yet, one finds a lower level of strength and/or hypertrophic adaptations with the lower % of a 1-RM protocol (as has been suggested in 13, 21, 43, 48), then, the concurrent maximal hypertrophy of all fibers seems unlikely. Incompatibility of different modes of training is a strong possibility (as has been proposed in 14, 60), so that attempts to maximally stimulate all the different components of a muscle (as has been suggested by some authors) is very likely not possible. One cycle or protocol that may be optimal for stimulating certain fiber sub-types and/or enzymes may compromise the development of other fiber sub-types and/or enzymes. In fact, one may speculate that under conditions of competing exercise stimuli (where there is fatigue of both glycolytic and oxidative fibers), one's physiological adaptive processes may shift optimal hypertrophic adaptation towards the fatigue-resistant fiber spectrum. Fiber adaptation/transformation studies already suggest this. If this scenario is accurate, then, if one is primarily interested in maximizing muscle strength and hypertrophic adaptations, it may be necessary to seek out a training protocol (through experimentation) that optimizes adaptation of one's most abundant pool of fibers and adjust training parameters accordingly as has been suggested in point 2. above.

REFERENCES

1. Abernethy, P., Thayer, R., Taylor, A. (1990). Acute and chronic responses of skeletal muscle to endurance and sprint exercise. *Sports Medicine* 10(6):365-389.
2. Abernethy, P.J., Jurimae, J., Logan, P.A., Taylor, A.W., Thayer, R.E. (1994). Acute and chronic response of skeletal muscle to resistance training. *Sports Med* 17(1):22-38.
3. Bacou, F., Rouanet, P., Barjot, C., Janmot, C., Vigneron, P., d'Albis, A. (1996). Expression of myosin isoforms in denervated, cross-reinnervated and electrically stimulated rabbit muscles. *Eur J Biochem* 236:539-547.
4. Barjot, C., Rouanet, P., Vigneron, P., Janmot, C., d'Albis, A., Bacou, F. (1998). Transformation of slow-or fast-twitch rabbit muscles after cross-reinnervation or low frequency stimulation does not alter the in vitro properties of their satellite cells. *J of Muscle Research and Cell Motility* 19:25-32.
5. Bellanger, A.Y., McComas, J. (1981). Extent of motor unit activation during effort. *J Appl Physiol* 51(5):1131-1135.
6. Bellemare, F., Woods, J.J., Johansson, R., Bigland-Ritchie, B. (1983). Motor-unit discharge rates in maximal voluntary contractions of three human muscles. *J. of Neurophysiol* 50(6):1380-1392.
7. Bigland-Ritchie, B.R., Dawson, N.J., Johansson, R.S., Lippold, O.C.J. (1986). Reflex origin for the slowing of motorneuron firing rates in fatigue of human voluntary contractions. *J Physiol* 379:451-459.
8. Buchthal, F., Schmalbruch, H. (1980). Motor unit of mammalian muscle. *Physiol Reviews* 60 (1):90-140.
9. Carpinelli, R.N. (1996). A big question mark for "Periodization of strength". *Hard training* 6 (1): 6-10.
10. Carvalho, A.J., McKee, N.H. (1996). Simultaneous assessment of isometric forces in fast-and slow-twitch muscles of single rat hindlimbs in situ. *Can J Appl Physiol* 21(1):23-34.
11. DeLuca, C.J., LeFever, R.S., McCue, M.P., Xenakis, A.P. (1982). Behaviour of human motor units in different muscles during linearly varying contractions. *J Physiol* 329:113-128.
12. Dietz, V. (1978). Analysis of the electrical muscle activity during maximal contraction and the influence of ischaemia. *J of Neurol Sc* 37:187-197.
13. Dons, B., Bollerup, K., Bonde-Peterson, Hancke, S. (1979). The effect of weight lifting exercise related to muscle fiber composition and muscle cross-sectional area in humans. *Eur J Appl Physiol* 40:95-106.
14. Dudley, G.A., Djamil, R. (1985). Incompatibility of endurance and strength-training modes of exercise. *J Appl Physiol* 59:1446-1451.
15. Edstrom, L., Grimby, L. (1986). Effect of exercise on the motor unit. *Muscle and Nerve* 9:104-126.

16. Enoka, R.M., Trayanova, N., Laouris, Y., Bevan, L., Reinking, R.M., Stuart, D.G. (1992). Fatigue-related changes in motor unit action potentials of adult cats. *Muscle and Nerve* 14:138-150.
17. Enoka, R. M. (1996). Commentary-Neural and neuromuscular aspects of muscle fatigue. *Muscle and Nerve Suppl* 4:S31-S32.
18. Fallentin, N., Jorgensen, K., Simonsen, E.B. (1993). Motor unit recruitment during prolonged isometric contractions. *Eur J Appl Physiol* 67:335-341.
19. Feigenbaum, M.S., Pollock, M.L. (1997). Strength training. Rationale for current guidelines for adult fitness programs. *The physician and sports Medicine* 25(2):44-64.
20. Fitts, R.H. (1994). Cellular mechanisms of muscle fatigue. *Physiol Reviews* 74(1):49-94.
21. Fleck, S.J., Kraemer, W.J. (1987). Designing resistance training programs. Champaign, Illinois:Human Kinetics Books.
22. Freund, H. (1983). Motor unit and muscle activity in voluntary motor control. *Physiol Reviews* 63(2):387-436.
23. Gerdle, B., Karlsson, A.G., Crenshaw, A.G., Friden, J. (1997). The relationships between EMG and muscle morphology throughout sustained static knee extensions at two submaximal force levels. *Acta Physiol Scand* 160:341-351.
24. Granier, P., Dubouchaud, H., Mercier, B., Ahmaidi, S., Prefaut, U. (1996). Lactate uptake by forearm skeletal muscles during repeated periods of short-term intense leg exercise in humans. *Eur J Appl Physiol* 72:209-214.
25. Grillner, S., Udo, M. (1971). Recruitment in the tonic stretch reflex. *Acta Physiol Scand* 81:571-573.
26. Grotmol, S., Totland, G., Kryvi, H. (1988). A general, computer-based method for the study of the spatial distribution of muscle fiber types in skeletal muscle. *Anat Embryol* 177:421-426.
27. Hakkinen, K., Alien, M., Komi, P.V. (1985). Changes in isometric force and relaxation time, electromyographic and muscle fiber characteristics of human skeletal muscle during strength training and detraining. *Acta Physiol Scand* 125:573-585.
28. Housh, T.J., deVries, H.A., Johnson, G.O., Housh, D.J., Evans, S.A., Stout, J.R., Evetovich, T.K., Bradway, R.M. (1995). Electromyographic fatigue thresholds of the superficial muscles of the quadriceps femoris. *Eur J Appl Physiol* 71:131-136.
29. Inbar, O., Kaiser, P., Tesch, P. (1981). Relationships between leg muscle fiber type distribution and leg exercise performance. *Int J Sports Medicine* 2(3):154-159.
30. Jones, A. (1993). The lumbar spine, the cervical spine and the knee: testing and rehabilitation. Ocala, FL:MedX Corporation.
31. Jones, A. (1986). Exercise 1986: The present state of the art. *Athletic Journal* (April) 66:53-79.
32. Juel, C. (1997). Lactate-proton cotransport in skeletal muscle. *Physiol Reviews* 72(2):321-358.
33. Jurimae, J., Abernethy, P., Quigley, B., Blake, K., McEniery, M. (1997). Differences in muscle contractile characteristics among bodybuilders, endurance trainers and control subjects. *Eur J Appl Physiol* 75:357-362.
34. Kanehisa, H., Ikegawa, S., Fukunaya, T. (1997). Force-velocity relationships and fatiguability of strength and endurance-trained subjects. *Int J Sports Med* 18(2):106-112.
35. Katsuta, S., Takamatsu, K. (1987). "Estimation of muscle fiber composition using performance tests" in *Biomechanics XB*. Champaign, IL:Human Kinetics, p. 989-993.
36. Kernell, D. (1998). Muscle Regionalization. *Can J Appl Physiol* 23(1):1-22.
37. Krogh-Lund, C. (1993). Myo-electric fatigue and force failure from submaximal static elbow flexion sustained to exhaustion. *Eur J Appl Physiol* 67:389-401.
38. Kukulka, C.G., Clamann, P.H. (1981). Comparison of the recruitment and discharge properties of motor units brachial biceps and adductor pollicis during isometric contractions. *Brain Research* 219:45-55.
39. Liljedahl, M. E., Holm, I., Sylven, C., Jansson, E. (1996). Different responses of skeletal muscle following sprint training in men and women. *Eur J Appl Physiol* 74:375-383.
40. Linossier, M., Dormois, Geyssant, A., Denis, C. (1997). Performance and fiber characteristics of human skeletal muscle during short sprint training and detraining on a cycle ergometer. *Eur J Appl Physiol* 75:491-498.
41. Linossier, M.T., Dormois, D., Perier, C., Frey, J., Geyssant, A., Denis, C. (1997). Enzyme adaptations of human skeletal muscle during bicycle short-sprint training and detraining. *Acta Physiol Scand* 161:439-445.
42. Linssen, H. J. P. et al. (1991). Fatigue in type I fiber predominance: a muscle force and surface EMG study on the relative role of type I and type II muscle fibers. *Muscle and Nerve* 14:829-837.
43. McDonagh, M.J.N., Davies, C.T.M. (1984). Adaptive response of mammalian skeletal muscle to exercise with high loads. *Eur J Appl Physiol* 52:139-155.
44. McGuff, D.M., Carter, T. (1998). Time under load, a new standard of measurement. *Ultimate Exercise article*.
45. Miller, E.J., MacDougall, J.D., Tarnopolsky, M.A., Sale, D.G. (1993). Gender differences in strength and muscle characteristics. *Eur J Appl Physiol* 66:254-262.
46. Millner-Brown, H.S., Stein, R.B., Yemm, R. (1993). Changes in firing rate of human motor units during linearly changing voluntary contractions. *J Physiol* 230:371-390.
47. Moritani, T., Gaffney, F.D., Carmichael, T., Hargis, J. (1985). "Interrelationships among muscle fiber types, electromyogram and blood pressure during fatiguing isometric contraction" in *Biomechanics IXA*. Champaign, IL:Human Kinetics, p. 287-292.

48. Moss, B.M., Refsnes, P.E., Abildgaard, A., Nicolaysen, K., Jensen, J. (1997). Effects of maximal effort strength training with different loads on dynamic strength, cross-sectional area, load-power and load-velocity relationships. *Eur J Appl Physiol* 75:193-199
49. Nagata, A., Christianson, J.C. (1995). M-Wave modulation at relative levels of maximal voluntary contraction. *Eur J Appl Physiol* 71:77-86.
50. Ostrowski, K.J., Wilson, G.J., Weatherby, R., Murphy, P.W., Lyttle, A.D. (1997). The effect of weight training volume on hormonal output and muscular size and function. *J. of Strength and Conditioning Research* 11(1): 148-154.
51. Pette, D. (1998). Training effects on the contractile apparatus. *Acta Physiol Scand* 162:367-376.
52. Pipes, T.V. (1994). Strength training and fiber types. *Scholastic Coach* (March)63:67-70.
53. Sale, D.G. (1988). Neural adaptation to resistance training. *Med Sci Sports Exerc* 20(5) Suppl. S135-S145.
54. Sahlin, K., Tonkonogi, M., Soderlund, K. (1998). Energy supply and muscle fatigue in humans. *Acta Physiol Scand* 162:261-266.
55. Schmidtbleicher, D., Haralombie, G. (1981). Changes in contractile properties after strength training in man. *Eur J Appl Physiol* 46:221-228.
56. Sejersted, O.M., Vollestad, N., Hallen, J., Bahr, R. (1998). Introduction-Muscle performance-fatigue, recovery and trainability. *Acta Physiol Scand* 162:181-182.
57. Starkey, D.B., Pollock, M.L., Ishida, Y., Welsh, M.A., Brechue, W.F., Graves, J.E., Feigenbaum, M.S. (1996). Effect of resistance training volume on strength and muscle thickness. *Med Sci Sports Exerc* 28(10): 1311-1320.
58. Staron, R. (1997). Human skeletal muscle fiber types:delineation, development, and distribution. *Can J Appl Physiol* 22(4):307-327.
59. Stephens, J.A., Usherwood, T.P. (1977). The mechanical properties of human motorunits with special reference to their fatigability and recruitment threshold. *Brain Research* 125:91-97.
60. Stone, J., Brannon, T., Haddad, F., Qin, A., Baldwin, K.M. (1996). Adaptive responses of hypertrophying skeletal muscle to endurance training. *J Appl Physiol* 81(2):665-672.
61. Tesch, P., Sjodin, B., Thorstenson, A., Karlsson, J. (1978). Muscle fatigue and its relation to lactate accumulation and LDH activity in man. *Acta Physiol Scand* 103:413-420.
62. Tesch, P. A., Komi, P.V., Jacobs, I., Karlsson, J. (1983). Influence of lactate accumulation of EMG frequency spectrum during repeated concentric contractions. *Acta Physiol Scand* 119:61-67.
63. Tesch, P. A. (1988). Skeletal muscle adaptation consequent to long-term heavy resistance exercise. *Med Sci Sports Exerc* 20(5) Suppl. S132-S134.
64. Theriault, R., Boulay, M., Theriault, G., Simoneau, J.A. (1996). Electrical stimulation-induced changes in performance and fiber type proportion of human knee extensor muscles. *Eur J Appl Physiol* 74:311-317.
65. Thorstenson, A., Karlsson, J. (1976). Fatiguability and fibre composition of human skeletal muscle. *Acta Physiol Scand* 98:318-322.
66. Van Cutsem, M., Feiereisen, P., Duchateau, J., Hainaut (1997). Mechanical properties and behaviour of motor units in the tibialis during voluntary contractions. *Can J Appl Physiol* 22(6):585-597.
67. West, W., Hicks, A., Clements, L., Dowling, J. (1995). The relationship between voluntary electromyogram, endurance time and intensity of effort in isometric handgrip exercise. *Eur J Appl Physiol* 71:301-305.
68. Westcott, W.L. (1989). Strength training research: sets and repetitions. *Scholastic Coach* (May/June)58:98-100.
69. Westerblad, H., Allen, D.G., Bruton, J.D., Andrade, F.H., Lannergren, J. (1998). Mechanisms underlying the reduction of isometric force in skeletal muscle fatigue. *Acta Physiol Scand* 162:253-260.
70. Woods, J.J., Furbush, F., Bigland-Ritchie, B. (1987). Evidence for a fatigue-induced reflex inhibition of motorneuron firing rates. *J of Neurophysiol* 58(1):125-137.