The Role of Fiber Types in Muscle Hypertrophy: Implications for Loading Strategies

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Many individuals who resistance train do so with the primary goal of increasing muscular size. Current theory suggests that exercise-induced muscle hypertrophy is regulated by a phenomenon called mechanotransduction whereby mechanical forces are converted into chemical signals that mediate intracellular anabolic and catabolic pathways, ultimately leading to a shift in muscle protein balance that favors synthesis over degradation (42). The net effect is an accretion of myofibrillar contractile proteins that augments the diameter of individual fibers and thereby results in an increase in whole muscle cross sectional area (CSA) (28).

Both endurance-oriented type I (slow twitch) fibers and strength-oriented type II (fast twitch) fibers have the ability to hypertrophy. However, research shows that the growth capacity of fast twitch fibers is approximately 50% greater than that of slow twitch fibers (3), although a high degree of inter-individual variability is seen with respect the extent of hypertrophic adaptation (20). Based on the aforementioned information, it may assumed that recruitment of type II fibers should be the main focus of exercise program design for the accretion of muscle mass, given the enhanced rate of hypertrophy in these fibers as compared to type I fibers.

Despite a logical rationale, emerging evidence suggests that such an approach may be simplistic and perhaps misguided, at least if the goal is to maximize muscle size. Therefore, the purpose of this paper will be to review the research regarding fiber type-specific hypertrophy and draw evidence-based conclusions as to their implications for program design.

Hypertrophy of Type II Fibers with High Intensity Strength Training

Type II muscle fibers have long displayed superior growth following high-intensity strength training (1, 10, 11, 13, 14, 20, 31, 32). These experimental results are often extrapolated to represent a growth capacity exceeding that of type I fibers; however, it is important to note that these findings are specific to the training intensities at which the study is performed, and may not apply universally across the repetition continuum. The superior capacity for growth of this particular fiber type may be more a consequence of the models in which we study them than an intrinsic property of the fiber itself. In support of this hypothesis, bodybuilders display greater Type I fiber hypertrophy than powerlifters, presumably as a result of routinely training with higher repetition ranges (13).

Our previous understanding of the relationship between training intensity and the resultant fiber type specific hypertrophy is best summarized by Fry's (13) comprehensive review of resistance training program variables. Using a regression analysis to assess the intensity-related percent change in fiber growth across various studies, it was determined that for the majority of training intensities above 50% 1RM, type II fiber growth exceeded that of the type I fibers. In addition, the peak growth rate of type I fibers was lower than for type IIs, regardless of training intensity. This work is limited by the fact that research to date has been biased towards training intensities greater than 60% 1RM, with a lack of lower-intensity strength training studies in the literature (13, 38). Thus, the paucity of low-intensity resistance training research precludes equal weighting in analysis, predisposing results toward higher-intensity training. Furthermore, regression merely shows the correlation between two variables, in this case fiber type-specific hypertrophy and training intensity; it does not imply causation.

Direct comparisons of training intensities and fiber-type specific hypertrophy

Strength training interventions using intensities above 50% 1RM abound (38), however direct comparisons including low-intensity training that address fiber-type specific hypertrophy are currently lacking and those that have been conducted are disparate and contradictory in their findings. Campos et al. (10) compared three different repetition ranges against a sedentary control group and found that groups who performed sets with either a 3-5 RM or 9-11 RM load to failure produced significant growth in both fiber types as compared to training with 20-28 RM, where the extent of hypertrophy did not reach statistical significance. In agreement, Schuenke et al. (31) found high intensity training (3x6-10 RM; ~80-85% 1RM) to failure resulted in significant growth of all fiber types as compared to a lack of growth following lower intensity training (3x20-30 RM; ~40-60% 1RM). Interesting, training with low-intensity at a slow speed (10 seconds concentric and 4 seconds eccentric) induced a greater hypertrophic response compared to training with a comparable resistance at traditional speed, but adaptations were less than that seen with high-intensity loads.

Employing a quasi within-subject design, Mitchell et al. (24) compared training with three sets at 80%- 1RM against one set at 80% 1RM and three sets with 30% 1RM over 10 weeks, with all groups taking every set to concentric failure. Whole muscle hypertrophy was equivalent between all training groups at completion of the study, and fiber-type specific hypertrophy indicated substantial hypertrophy in all fiber types, with no significant differences in fiber-type specific growth indicated. It should be noted, however, that the low-intensity condition resulted in a ~23% increase in type I fiber area compared to ~16% in the high-intensity condition. Conversely, high-intensity training

resulted in a ~15% increase in type II fiber area compared to a ~12% in the low-intensity condition. The study was likely underpowered to detect significance, suggesting that an increase type I fiber area substantially contributed to the hypertrophic-effects of low-intensity training.

The influence of training intensity on whole muscle hypertrophy

A number of studies have compared whole muscle hypertrophy in low-intensity versus high-intensity training protocols. Holm et al. (17) employed a within-subject design whereby subjects performed 10 sets of unilateral leg extensions, training one leg at 70% 1RM and the contralateral leg at 15.5% 1RM in a randomized, counterbalanced fashion. After 12 weeks, magnetic resonance imaging (MRI) showed an approximately threefold greater increase in quadriceps muscle CSA with higher intensity exercise compared to low-intensity exercise. A limitation of the study was that training at 15.5% 1RM was not carried out to concentric muscular failure, which has been shown to be necessary to maximize a hypertrophic response in low-intensity exercise (24).

Leger et al. (23) and Lamon et al. (22) employed a similar protocol to that of Campos et al. (10); neither study found significant differences in muscle CSA between low-intensity and high-intensity groups. The discrepancy in results between studies may be attributable to the fact that both Leger et al. (23) and Lamon et al. (22) evaluated middle-aged males, who conceivably were detrained relative to the younger males studied by Campos et al. (10). Another important difference between studies was the techniques used to measure hypertrophy. Campos et al. (10) employed muscle biopsies while Leger et al. (23) and Lamon et al. (22) used computerized tomography (CT). Since CT does not allow evaluation of differential effects on fiber typing, it is not clear whether

differences in slow twitch versus fast twitch fibers played a role in explaining the discrepancies in these findings. Moreover, it is possible that non-contractile increases in sarcoplasmic proteins (i.e. mitochondria, fluid, etc) may have contributed to results. This is an important consideration as mitochondrial protein synthesis is particularly sensitive to extended time under tension during strength training, and may increase disproportionately as time under tension and total work are higher with low-load training (8, 9). As such, it is important to consider the possibility that even if whole muscle hypertrophy is equivalent in response to various training intensities, the increase in cross-sectional area may be mediated by differing cellular adaptations (28) and muscle strength and function may vary accordingly. This is partially supported by the observation that strength is increased to a greater extent with high intensity training (10, 31), even when whole muscle hypertrophy is comparable (24), although adaptations independent to muscle could also explain such a response.

Recently, Ogasawara et al. (26) found similar increases in CSA of the triceps brachii and pectoralis major as determined by MRI in subjects performing training at 75% 1RM versus 30% 1RM. The study employed a within-subject design whereby participants performed the higher intensity exercise for 12 weeks initially and, after a 12-month washout period of detraining, performed 12 weeks of the low-intensity exercise in non-randomized fashion. Although intriguing, these findings must be viewed with caution as "muscle memory" via both neural mechanisms and satellite accretion may have influenced results (7, 32).

Interactions of load and motor unit recruitment

The premise behind high-intensity training is rooted firmly in Henneman's Size principle that states the orderly recruitment of motor units during a given task (15, 16). This principle dictates that motor units are recruited based on their size: smaller, low-threshold slow motor units are recruited initially, followed by progressively larger, higher-threshold motor units as the force demands of the task increases. Training with heavy loads requires substantial force production, and therefore calls upon both low and high threshold motor units to maximize force. As load (training intensity) decreases, the required force production from the muscle is reduced, and fewer motor units are required to complete the lift. Given that fibers must be recruited in order to respond and adapt to resistance exercise (21), it seems logical to conclude that training at very high levels of intensity is required to maximize recruitment and therefore muscular development.

However, this argument discounts the role of fatigue in the stimulation of hypertrophy, and its ability to influence motor unit recruitment. During low-load contractions, initial motor unit recruitment is lower than under high load conditions. However, as fatigue increases, the recruitment threshold of higher threshold motor units is progressively reduced (2). Conceivably, this results in the gradual recruitment of higher threshold, presumably fast motor units as fatigue increases. This recruitment process provides a mechanism whereby low-intensity strength training can activate fast-twitch motor units and ultimately, stimulate the growth of these fibers.

The exact mechanisms whereby fatigue enhances high-threshold motor unit recruitment are not well understood. It has been speculated that results are mediated by metabolic stress associated with exercise relying primarily on the fast glycolytic energy system (29). The accumulation of hydrogen ions, increased muscle hypoxia, and free

radical generation all have been implicated as playing a role in the process (29). Regardless of the mechanism, the fatigue-induced activation of FT fibers is believed to be an attempt to maintain necessary levels of force generation so that work output is maintained (2).

Although intuitively it would seem that a given threshold of intensity is required to achieve recruitment of the full spectrum of fibers, studies to date have failed to establish a specific percentage of RM at which this occurs. Surface electromyography (EMG), which provides a global measure of motor unit activity of a given muscle, has been used to provide an indirect measure of motor unit recruitment during resistance exercise. Cook et al. (12) recently demonstrated that EMG amplitude of the quadriceps femoris during knee extension exercise to failure was significantly greater at a high intensity (70% 1RM) than at low intensity (20% 1RM). This suggests that the threshold for optimal motor unit recruitment exceeds 20% of 1RM. In contrast, Wernbom et al. (39) reported that peak EMG activity was similar between 3 sets of low intensity (30% 1RM) unilateral knee extensions. In attempting to justify the discrepancies between the studies of Cook et al. (12) and Wernbom et al. (39), one could infer that 30% 1RM represents a lower threshold for type II recruitment. However, Suga et al. (33) found that fiber recruitment during blood flow restricted training at 30% 1RM did not reach levels achieved with traditional high-intensity resistance exercise, as determined by inorganic phosphate splitting via ^{31}P-magnetic resonance spectroscopy. Only when blood flow restricted exercise was carried out at an intensity of 40% 1RM did P_i peaks equate to those associated with the high-intensity training. Given the conflicting results, it is difficult to draw any firm conclusions as to a minimum intensity threshold for recruiting

the full spectrum of muscle fibers, although evidence indicates that intensity below 30% 1RM is suboptimal for inducing substantial recruitment of high-threshold motor units.

Understanding the relationship between load and time-under-tension

The hypothesis that we have underestimated both the growth potential of type I fibers and the ability of low-intensity training to stimulate hypertrophy relies on two basic premises: 1) that hypertrophy requires a minimum time-under-tension (TUT) that varies with training intensity, and; 2) that this TUT is greater for type I than type II fibers. To address this relationship Burd et al. (9) compared the protein synthetic response following four sets at three training intensity conditions: 90% 1RM lifted to failure, 30% 1RM to failure, and 30%-1RM lifted such that the total work was matched to the 90% 1RM condition. The protein synthetic response, while of differing time-course, was similar when either 90% 1RM or 30% 1RM were lifted to concentric failure, however the low-load work-matched condition did not stimulate mixed, myofibrillar and sarcoplasmic protein synthesis to a similar extent as the other conditions. Although the protein synthetic response to an acute bout of resistance exercise is not necessarily indicative of long-term hypertrophic adaptations (3) there is some evidence, albeit conflicting, that low intensity exercise can produce comparable hypertrophy with higher intensity exercise provided sets are taken to failure (24, 26).

Methodology may at least partially explain the divergent findings regarding the effects of training intensity on fiber-type specific and whole muscle hypertrophy. The studies that do not equate total intra-session work tend to show similar growth between high- and low-intensity exercise (24, 26) with the exception of Schuenke et al. (31) who displayed greater hypertrophy when training at a high-intensity. Conversely, studies that

do equate work show a hypertrophic advantage for high-intensity exercise (10, 17). It is also important to consider the contribution of muscular failure to these experimental findings. While the majority of studies identify concentric failure as the end point for the set (10, 24, 31), Holm et al. (17) did not and only the low-intensity condition trained to failure in Ogasawara et al. (26). While fatigue may contribute to the hypertrophic response (29, 30), it is currently unknown how the relationship between training intensity and hypertrophy compare when exercise is fatiguing, but terminated prior to the point of concentric failure.

Nevertheless, given that low-intensity resistance exercise appears to preferentially increase hypertrophy of type I fibers (24), it is logical to conclude that an increased time-under-tension is necessary to fully stimulate these fibers. This would be consistent with the endurance-oriented nature of slow-twitch fibers that renders them resistant to fatigue.

Do Type I Fibers Have a Limited Growth Capacity?

It remains questionable as to whether type I fibers have the ultimate hypertrophic potential of type II fibers. There is evidence that they do not. Studies evaluating the relationship between cross-sectional area and mitochondrial density suggest that the growth of a given muscle fiber is achieved at the expense of its endurance capacity (36). This implies that the smaller size of highly oxidative slow twitch fibers is an evolutionary design constraint, limiting their inherent ability to hypertrophy in comparison to fast-twitch fibers with a low oxidative capacity (36).

One way that type I fiber hypertrophy may be self-limiting is via competition between anabolic and catabolic intra-cellular signaling pathways. Expression of MuRF (muscle ring finger) and MAFbx (muscle atrophy F-box)--ubiquitin ligases that mediate

protein degradation--is approximately twofold higher in oxidative versus non-oxidative fibers (36). Moreover, there is evidence that type I fibers have a reduced ability to increase phosphorylation of ribosomal protein kinase S6 (p70S6K) after resistance exercise compared to type II fibers, which in turn blunts their protein translational capacity (19). p70S6K is a downstream effector in the mTOR pathway, and its activation via mechanical stimuli has been deemed critical to the hypertrophic response (41). These events are consistent with the "AMPK-PKB switch" hypothesis, whereby signaling is switched to either a catabolic AMPK/PGC-1α- or anabolic PKB-TSC2-mTOR-dominated state depending on whether the imposed demand is endurance- or resistance-based (5). While signaling interactions are unquestionably more complex than a simple switch mechanism, especially given the stimulation of AMPK with traditional resistance exercise (19), research does suggest that the endurance-oriented nature of type I fibers provide a greater inclination to turnover rates that favor proteins involved in metabolism (i.e., mitochondrial proteins) over structural muscle protein (i.e., myofibrillar proteins) (36).

Another potential limiting factor in type I growth is their ability to increase satellite cell proliferation. Satellite cells are muscle stem cells that reside between the basal lamina and sarcolemma, which remain dormant in the resting state. When stimulated by mechanical stress, however, satellite cells generate precursor cells (myoblasts) that proliferate and ultimately fuse to existing cells, providing the necessary agents for remodeling of muscle tissue (35, 40). In addition, satellite cells are able to donate their nuclei to existing muscle fibers, enhancing their capacity for protein synthesis (6, 25). In young, untrained subjects, satellite cell number is approximately

equal between slow- and fast-twitch fiber types (18). However, there is evidence that mixed modes exercise training, even of an endurance-oriented nature, results in a greater increase of the satellite cell pool in type II fibers (37), indicating a greater long-term growth potential for these fiber types. The heightened exercise-induced expression of the protein MyoD in type II fibers (4), which is predominantly responsible for upregulating satellite proliferation and thus facilitates the formation of new myoblasts, could be a potential mechanisms to explain their enhanced satellite cell response. This suggests that over time, fast-twitch fibers may have a favorable pre-disposition to growth as a result of an enhanced capacity to synthesize new contractile proteins.

Considering the aforementioned factors, evidence suggests that while type I fibers possess significant growth potential, they may have a lower ceiling for hypertrophy in comparison to type II fibers. Unfortunately, no study to date has directly examined this hypothesis. Furthermore, as the study documenting enhanced phosphorylation of p70s6k only relied on higher training intensities, it remains to be determined whether low intensity strength training can augment protein synthetic signaling pathways (24) in type I fibers specifically to a greater extent than high intensity training in type II fibers (19). This topic should be explored in future research.

Practical Applications

Based on the current body of research, there is emerging evidence indicating that type I fibers can substantially contribute to overall muscle cross sectional area. Research also suggests that low-load resistance exercise may help to maximize type I fiber hypertrophy, provided that training is carried out to concentric muscular failure (24). From a practical perspective, the following recommendations can be made.

If one's goal is simply to maximize overall muscle mass, exercise prescriptions should include training across a wide spectrum of repetition ranges. Higher intensity exercise appears necessary to fully stimulate fast-twitch fiber growth (13), while lower intensity exercise preferentially enhances hypertrophy in slow-twitch fibers (24). A periodized approach combining high- and low-intensity training may help to ensure an optimal hypertrophic response in the full continuum of fiber types. Both linear and non-linear models are viable approaches here, as neither has been shown to be superior to the other in this regard. Thus, intensities can be varied within and/or across multiple training sessions, or by alternating target repetition zones every few microcycles. Training loads can easily be manipulated based on the characteristics of the exercises within a training session, favoring high-load training on multi-joint exercises like the squat and deadlift, saving higher repetition ranges for single-joint, isolation type exercises that may be better suited to lighter training loads.

While training percentages or repetition ranges can be varied in a periodized manner, advanced training techniques may provide additional hypertrophic benefit. Drop sets, where training load is progressively decreased on subsequent sets with little to no rest once the point of fatigue or technical failure is achieved at each training load may provide the best of both worlds (27). Training load can be maximized initially to capitalize on type II fiber activation, however as fatigue sets in training loads can be progressively decreased to increase the time-under-tension to maximally stimulate the Type I fibers. In addition, rest-pause training, where a set with a given training load is extended beyond the point of fatigue by taking small rests within the set may provide a similar benefit by increasing the duration of loading. Care must be taken when utilizing

these techniques to balance the need for increased muscle recruitment and time under tension to promote optimal hypertrophy against the potential for elevated levels of fatigue and overuse (27).

On the other hand, if hypertrophy is a means to maximize strength, then higher intensity loads should be favored over lighter loads, as gains in strength are greater with high as compared to low load training (10) even when a comparable hypertrophic response occurs (24). If strength is to be maximized in specific exercises, as in sports such as powerlifting, higher intensity training is essential due to the specificity of strength (13) and the fact that exercise technique can differ across the training intensity continuum (34). Fiber-type specificity plays a role in this process. Fast-twitch fibers are innervated by larger motor neurons compared to their slow-twitch counterparts, allowing for enhanced high force production. Because of the smaller size of the neurons associated with type I fibers, they simply cannot cycle fast enough to carry out tasks involving high levels of force. Hence, the major focus here should be on high-load training (greater than ~75% 1RM).

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