Journal of Strength and Conditioning Research, 2000, 14(1), 102–113

Review

Nonuniform Response of Skeletal Muscle to Heavy Resistance Training: Can Bodybuilders Induce Regional Muscle Hypertrophy?

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ABSTRACT

Skeletal muscle is a heterogeneous tissue that exhibits numerous inter- and intramuscular differences (i.e., architecture, fiber composition, and muscle function). An individual muscle cannot be simplistically described as a compilation of muscle fibers that span from origin to insertion. In fact, there are unique differences within a single muscle and within single muscle fibers with respect to fiber size and protein composition. Electromyographic data indicate that there is selective recruitment of different regions of a muscle that can be altered, depending on the type of exercise performed. Longitudinal resistance-training studies also demonstrate that individual muscles as well as groups of synergist muscles adapt in a regional-specific manner. The author speculates that no single exercise can maximize the hypertrophic response of all regions of a particular muscle. Thus, for maximal hypertrophy of an entire muscle, athletes (particularly bodybuilders) are justified in incorporating various exercises that purportedly stimulate growth in a regional-specific manner.

Key Words: muscle fiber, exercise, weight lifting

Reference Data: Antonio, J. Nonuniform response of skeletal muscle to heavy resistance training: can bodybuilders induce regional muscle hypertrophy? *J. Strength Cond. Res. 14(1):102–113. 2000.*

Introduction

Skeletal muscle has the unique ability to adapt in a very specific manner to different forms of stress. Specifically, high tension overload (i.e., resistance exercise, stretch, compensatory hypertrophy) results in profound changes in muscle mass, muscle fiber size, and to a lesser extent, muscle fiber number (2, 5, 6, 62, 96, 99). In humans, it is commonly accepted that heavy resistance training can produce significant skeletal muscle hypertrophy. Weight training programs for various sports are currently an integral component of the overall training scheme and are geared towards maximizing performance in those respective sports. However, bodybuilders engage in weight training not to enhance performance in any meaningful athletic sense, but to develop a high degree of muscle size, shape, symmetry, and definition. Minimizing subcutaneous fat is important part to a bodybuilder's physique. In a contest between bodybuilders, a panel of judges in essence determines who has the best physique on the basis of the various criteria listed previously.

In comparison with other athletes and with sedentary individuals, bodybuilders display higher body weights and limb circumferences (17, 100). However, muscle fiber size is not always greater in bodybuilders than in nonbodybuilders (63). In this case, greater muscle fiber number may account for the larger muscle mass seen in bodybuilders. In addition to emphasizing muscle hypertrophy in their training regimens, bodybuilders also engage in what some popular lay magazines refer to as isolation or shaping exercises. The practice of performing different exercises for the same muscle to alter the appearance or shape of that particular muscle is common among bodybuilders (79).

Although commonly accepted by bodybuilders, the notion that a particular muscle's overall shape can be altered via a combination of particular exercises has not been examined in the scientific literature. However, I would posit that there exists a plausible theoretical basis that supports the notion that bodybuilders can alter the shape of their muscles.

The primary aim of this review is not to list all the available publications related to the adaptive response of skeletal muscle to resistance training, but to provide a theoretical basis for the phenomenon of regional adaptation in skeletal muscle. As a general background, a brief account will be given of muscle fiber types and neural, metabolic, hormonal, and cellular adaptations to resistance training. Subsequently, this review will focus on evidence that supports the theory that regional adaptation of muscle and muscle fibers can be induced via resistance training.

Muscle Fiber Types

Skeletal muscle is a heterogeneous tissue that is composed of 2 basic fiber types: fast-twitch and slow-twitch. Fiber types have been categorized either on the basis of pH lability of myofibrillar (myosin) ATPase reaction (10), glycolytic/oxidative capacities (81), or myosin heavy chain (MHC) isoform content (9). According to the classification system of Peter et al. (81), there are 1 slow-twitch and 2 fast-twitch fiber types. Slow-twitch fibers are referred to as slow, oxidative fibers. These fibers have a slow contraction velocity, low tension capacity, and high fatigue resistance. Fast-twitch fibers can be divided into fast oxidative–glycolytic (FOG) or fast glycolytic (FG) fibers. FOG fibers have a high oxidative and glycolytic capacity. FG fibers have a low oxidative and high glycolytic capacity. These fibers generate the most tension and have the fastest contractile velocity, yet they fatigue the quickest. An alternative scheme by Brooke and Kaiser (10) uses the classifiers I, IIa, IIb, and IIc to type muscle fibers. Staron (92) further delineates fiber types (on the basis of pH lability of myofibrillar ATPase) into I, Ic, IIc, IIac, IIa, IIab, and IIb. The various C fibers (Ic, IIc) represent fibers that have intermediate staining properties of both the type I and II fibers and that are analogous to fibers that express both fast and slow MHC isoforms (92).

It is a common mistake to equate type IIa and IIb fibers with the FOG and FG fibers, respectively. There exists substantial overlapping in oxidative potentials of the IIa and IIb fiber types (84). Also, some fibers that may be type I fibers through histochemical criteria may have contractile properties representative of fast fibers. For example, the human adductor pollicis muscle is composed of 80% type I fibers, yet it exhibits a fast contractile time (24). Nonetheless, it is generally true that the type I fibers are the most oxidative, whereas the IIb fibers are the least.

Perhaps the best method of typing muscle fibers is via immunocytochemistry or electrophoresis. Interestingly, using these methods, the 3 major fiber types in humans (I, IIa, IIb) were found to correspond to the expression of 3 different MHC isoforms: MHCI, MHCIIa, MHCIIb (92). This is significant in that it is the MHC composition that determines the rate of cross-bridge interactions and hence the contractile velocity.

It should be noted that muscle fiber types are dynamic entities that can be altered through exercise (96). Rather than being static, it has been suggested that muscle fibers fall within a continuum of different types (i.e., I ↔ IIa ↔ IIab ↔ IIb); however, it has not been demonstrated that muscle fibers are obligated to follow this pattern (i.e., to transform a IIb fiber into a type I fiber, does the fiber need to first express the IIa MHC isoform?). For instance, rodents that were hind-limb-suspended and treated with thyroid hormone expressed combinations of fiber types (e.g., hybrid fibers that consisted of I/IIb) that were inconsistent with a fiber type continuum (12). For a complete review of skeletal muscle fiber types, see Staron (92).

Neural Adaptation

The motor unit serves as the functional unit of the neuromuscular system (72). In most mammalian skeletal muscle, motor units are comprised of a single motor neuron and the multiple muscle fibers that it innervates. In addition, motor unit populations differ between muscles (72). In general, small muscles such as the external rectus of the eye, lumbricals, and interossei muscles have few muscles per motor unit (~100 or less), whereas larger muscles such as the medial gastrocnemius can have up to nearly 2,000 muscle fibers per motor unit (25, 54).

According to the size principle of motor unit recruitment, the smallest motor units (i.e., slow-twitch or type I) are recruited before the larger motor units (i.e., fast-twitch—type IIa, type IIb) (72). Data derived via glycogen depletion methods show that both fast and slow units are scattered in a heterogeneous manner across a muscle. This would suggest that one could activate a muscle fiber (for instance in a slow motor unit) while an adjacent fast fiber remains inactive. Furthermore, using glycogen depletion techniques, one can determine the extent to which certain motor units are activated depending on the duration and intensity of exercise. Gollnick et al. (30) had subjects perform isometric contractions of the quadriceps femoris muscle group. On the basis of the amount of glycogen depleted from a particular fiber type, it was possible to determine which motor units were preferentially activated. They found that at 20% or less of maximal voluntary contraction (MVC), slow-twitch fibers demonstrated greater glycogen depletion than the fast-twitch fibers. As the percentage of MVC increased, the depletion of glycogen in the fast-twitch fibers increased as well. Recent work by Tesch et al. (101) examined the effects of different loads on glycogen depletion (5 sets of 10 concentric contractions at 3 loads). At loads as low as 30 to 45% of subjects' 1 repetition maximum (1RM) on the knee extension, 40% of type IIa fibers showed a significant loss of glycogen in the fibers of the vastus lateralis muscle. At loads of 60% of 1RM, 70% of these fibers showed a loss. Type IIab + IIb fibers showed no loss of glycogen at 30% or 45% of 1RM; however, approximately 30% of these fibers showed glycogen loss at 60% of 1RM. Moreover, the percentage of type I fibers demonstrating glycogen loss was similar (~35%) at all loads tested.

Thus, it is apparent that loads as low as 30–60% of MVC can recruit a significant number of fast motor units. It should be noted that attaining 100% MVC requires more than complete recruitment of motor units. In addition, firing rate must be sufficiently high to produce 100% of MVC. Small muscles such as the first dorsal interosseus muscle rely primarily on increased firing frequency to elicit a 100% MVC because all of its motor units are recruited at 50% of MVC, whereas a large muscle such as the biceps brachii recruits motor units up to 88% of MVC (71). There have been few reports suggesting a reversal of the size principle (i.e., fast units recruited before slow units). Apparently, high-force eccentric actions (74) preferentially recruit fast units while inhibiting the slow units. Thus, it may be possible to train oneself to selectively recruit specific fiber populations, depending on the type of exercise performed.

The initial increase in strength seen in beginning weightlifters is likely due to a neural adaptation because muscle hypertrophy usually does not occur until 4 weeks or later after the commencement of training (67, 76). Furthermore, the contribution of neural adaptation to strength development is affected by the complexity of the exercise task (16). Chilibeck et al. (16) measured gains in strength and lean body mass over a 20-week training period and found that hypertrophy of the upper extremity muscles occurred during the first 10 weeks of training, whereas the lower extremity and trunk muscles did not hypertrophy until the last 10 weeks. The authors believe that for simple motor tasks such as the biceps curl exercise, early gains in strength occur concurrently with muscle hypertrophy. On the other hand, the more complex tasks associated with trunk (e.g., bench press) and leg movements (e.g., leg press) require a longer period for neural adaptation, thus delaying muscle hypertrophy.

Metabolism

Heavy resistance training relies primarily on the phosphagen and glycolytic systems because of the high power outputs that are generated during this kind of exercise (20). The type of resistance training performed can have a profound effect on the energy systems that are used. The typical bodybuilding workout, which involves multiple sets using variable loads with limited rest periods (<1 minute), produces much higher levels of blood lactate than a typical powerlifting session, which uses lower volumes but higher loads and rest periods (20, 97). In addition, intramuscular triglycerides are used for energy during short-duration heavy-resistance exercise (23).

Furthermore, an examination of biochemical changes within skeletal muscle fibers reveals that resistance training can increase levels of intramuscular glycogen (65, 98). For instance, 5 months of resistance training increased muscle glycogen content of the triceps brachii by 35% (65); cross-sectional data show that bodybuilders have 50% higher glycogen concentration than their sedentary counterparts (98). Although longitudinal studies (20, 103) show that resistance training has no effect on the intramuscular concentration of ATP, phosphocreatine, creatine, and enzymes of aerobic or anaerobic metabolism, cross-sectional data comparing bodybuilders, Olympic weightlifters, powerlifters, and sedentary men show differences in muscle enzyme activities (103). Citrate synthase and 3-OH-acyl-CoA-dehydrogenase (markers of aerobic metabolism) are lower in bodybuilders, power lifters, and Olympic weightlifters than in sedentary men. On the other hand, lactate dehydrogenase activity was higher in the strength–power athletes than in their sedentary counterparts. In comparing different strength–power athletes, bodybuilders have a higher citrate synthase and myokinase activity within the fast-twitch fiber population in comparison with powerlifters and Olympic weightlifters (103).

With regard to metabolic characteristics, it is apparent that the concentration of various aerobic and anaerobic enzymes is usually sufficient to meet the energetic demands of heavy resistance training (102). It should be noted that changes in substrate levels can be enhanced via dietary supplementation (i.e., creatine), and thus, in addition to one's training protocol, the dietary habits of each athlete can influence the metabolic characteristics of skeletal muscle (32).

Hormonal Response

The pattern of acute hormonal changes during and after a resistance training session has been studied extensively (31, 39, 48, 50–53). It has been theorized that acute changes in serum hormones (e.g., testosterone, growth hormone) affect the adaptive response of skeletal muscle to resistance training. A training protocol that involves multiple sets of resistance exercises induces higher levels of growth hormone and testosterone in comparison with a single-set protocol (31). Kraemer et al. (50) demonstrated that Olympic-style weightlifting can produce increased concentrations of serum testosterone, cortisol, growth hormone, and beta-endorphin. In an attempt to parcel out which training variables most greatly affect acute hormonal status, Kraemer et al. (53) examined the effects of load (5 vs. 10RM), rest interval (1 vs. 3 minutes), and total work on hormonal response in 9 male subjects. These investigators found that serum testosterone increased regardless of the kind of training protocol. On the other hand, serum growth hormone levels were highest using the protocol that consisted of high total work, 1 minute of rest, and 10RM load. Kraemer et al. (51) found similar results using both male and female subjects with a protocol that consisted of a 5RM load, 3-minute rest interval, and lower total work vs. a protocol that consisted of a 10RM load, 1-minute rest interval, and higher total work. They found that serum growth hormone levels were significantly greater at all time points examined in the 10RM protocol. Thus, it is apparent that serum testosterone and growth hormone can increase dramatically in response to an acute bout of resistance training.

However, it is not entirely clear if acute changes in serum hormone levels are physiologically meaningful with regard to the hypertrophic response of resistance-trained skeletal muscle. Jensen et al. (43) demonstrated that serum testosterone concentration increased during both endurance and strength training sessions. In addition, maximal treadmill running, 2 hours of running at 70% of $\dot{V}O_2max$, 1 hour of running by middle-distance runners, and marathon running increased serum testosterone (34, 105, 106), whereas 14 hours of running (107-km race) and 19- and 42-km kayak races produced a decrease in serum testosterone (34, 59). It is not known why certain forms of exercise produce increases in serum testosterone and others do not.

With regard to a chronic adaptation, serum hormones (i.e., serum testosterone, free testosterone, and cortisol) in elite endurance athletes (swimmers) and elite weightlifters did not appreciably change over the course of 1 year (36). Although there is evidence that suggests that an acute bout of high-volume resistance training produces greater increases in serum growth hormone, it has yet to be demonstrated that this results in a greater skeletal muscle hypertrophy. Indeed, protocols that use high weight, low-repetition, low total work schemes are just as effective in producing gains in muscle mass as the higher-volume protocols (13, 18, 19, 73).

One could speculate that the transient increase in serum growth hormone and testosterone is not necessarily related to muscle protein accretion. For instance, aerobic exercise, such as distance running, may produce a rise in serum testosterone in response to the high levels of tissue damage. This increase in serum testosterone may be needed to blunt the degradation of skeletal muscle tissue. On the other hand, weight training may not produce the same absolute level of muscle tissue disruption, and therefore muscle protein accretion is observed. Or perhaps exercise, aerobic or resistance, can produce an increase in serum testosterone or growth hormone as long as a sufficient amount of one's total skeletal muscle mass is activated. At this point, the physiological significance of serum hormone changes in response to various exercise protocols is unclear.

Moreover, a confounding variable with regard to growth hormone measurements is the technique used to derive such measures. According to McCall et al. (70), there is an apparent discrepancy between immunoassay-derived growth hormone (IGH) and bioassay-derived growth hormone (BGH) measures. Work by Kraemer et al. (53) has shown that circulating levels of IGH can increase in response to high-volume resistance training; however, BGH was not determined. McCall et al. (70) demonstrated that exercise that consisted of unilateral plantar flexions had no effect on levels of IGH; however, exercise caused a significant increase in plasma concentrations of BGH. Future work may need to address the differences in BGH and IGH response to various resistance training programs.

Compartmentalization of Skeletal Muscle

An individual muscle is more than just a collection of muscle fibers spanning the entire muscle belly with a single muscle–nerve interaction. Instead, a muscle can be divided into neuromuscular compartments, which are distinct regions of a muscle, each of which is innervated by an individual nerve branch and therefore contains motor unit territories with a unique set of characteristics. In other words, different portions of a muscle may be called into play depending on the task demands of the situation.

An examination of various strap muscles of the human lower extremity reveals a compartmentalization within these muscles. The sartorius, gracilis, biceps femoris, and semitendinosus muscles are each subdivided into compartments by 1 or more fibrous bands or inscriptions (107). So rather than having a single muscle fiber spanning the entire muscle, it is apparent that each compartment must have its own distinct motor units. Thus, for these muscles to contract smoothly, there has to be cooperation between these different compartments. But also, because each compartment has its own innervation, it is possible that one can selectively recruit a particular region of that muscle.

Moreover, the gracilis and sartorius are composed of relatively short, in-series fibers (40). This contradicts a common assumption that muscle fibers span the entire origin to insertion of a muscle. Short in-series fibers can be found to terminate intrafascicularly. Thus, part of the tensile force from these short fibers is conveyed laterally onto surrounding fibers as well as proximodistally (40). The implications of such an anatomical arrangement are unknown with regard to muscle and muscle fiber hypertrophy. Could the presence of in-series short fibers contribute in part to regional differences in growth along the length of a muscle?

The distinction between anatomical and functional characteristics of skeletal muscles is more evident in other muscles. For instance, the human trapezius muscle is an example of how 1 muscle can basically be subdivided into 3 distinct functional regions on the basis of the origin and insertion of the muscle (56, 57). The upper portion elevates while the lower portion depresses the scapula. The midportion of the trapezius can adduct the scapula. Because each region of the trapezius has distinct attachments, it would make sense that specific movements could be performed that emphasize those regions. Moreover, there are other muscles (e.g., biceps brachii, extensor carpi radialis longus [ECRL]) that do not have an anatomical arrangement that would suggest that there are distinct functional abilities within that particular muscle, yet these muscles do in fact respond in a region-specific manner (11, 22).

Intermuscular Fiber Type Differences

Although many human muscles contain approximately 50% fast and 50% slow fibers (28), it is evident that there are certain muscles that may be primarily fast or slow. For instance, the paravertebral muscles in humans are predominantly small-diameter type I fibers (45, 104). The semitendinosus muscle contains a greater proportion (55–60%) of type II than type I fibers (28). Within the triceps surae, the soleus muscle demonstrates a range of slow fiber percentage from 70–100% in humans, whereas the gastrocnemius tends to have a higher percentage of fast fibers than other leg muscles (44). Thus, it is apparent that in addition to gross anatomical considerations, fiber type composition varies between muscles.

The significance of intermuscular fiber type variation with regard to training is at this point not fully understood. For instance, should a resistance training protocol be similar for the induction of hypertrophy in the slow soleus vs. the fast gastrocnemius? Heavy resistance training tends to produce hypertrophy of type I and II fibers; however, type II fibers enlarge proportionately more than type I fibers (49). Is there a training protocol that can consistently induce similar hypertrophy in type I fibers vs. type II? In essence, should all muscles be trained similarly if the goal is muscle mass accretion?

Intramuscular Fiber Type Differences

Fiber type differences are also evident within the same muscle. This regionalization of fiber types suggests that differing functional demands are placed on different regions of a muscle. According to Punkt et al. (83), muscle fibers of rat extensor digitorum longus (EDL) showed increased glycolytic activity near the insertion, whereas the soleus muscle had increased oxidative activity toward the midregion of the muscle. Along the superficial–deep axes, the oxidative capacity of all fibers from the soleus and EDL increased. Furthermore, the fiber cross-sectional areas of the soleus muscle fibers decreased from superficial to deep (83). Lexell et al. (55) have shown that type I fibers are predominant in the deeper regions of human vastus lateralis muscle, whereas type II fibers are predominant in the superficial regions. Sola et al. (91) have shown that there is a greater proportion of slow fibers in the deep regions of the human latissimus dorsi muscle, whereas fast fibers predominate superficially. Also, the fiber type distribution within the fascicles of the human vastus lateralis muscle show that a high proportion of type IIb fibers are located around the periphery, a prevalence of type I fibers are found within the fascicle, and type IIa fibers are somewhat uniformly distributed (80). Furthermore, type II fibers predominate around the periphery of the fascicles of the human biceps brachii, deltoid, and quadriceps femoris (66).

The semitendinosus muscle contains more type II fibers distally than proximally (28). The latissimus dorsi contains more fast fibers in the anterolateral and middle segments in comparison to the superior segment (90), and the short head of the biceps brachii contains a greater percentage of fast-twitch fibers vs. the long head (21). Furthermore, there is a greater percentage of fast-twitch fibers at the insertion of the biceps brachii (long and short head) and soleus muscles in comparison to the origin (21).

Intrafascicular fiber type differences suggest that functional properties of a muscle vary systematically within that muscle. Sjostrom et al. (89) hypothesize that muscle fibers on the boundaries of fascicles may be bound more firmly or at least differently than those located within the fascicle. Thus, muscle contraction or stretch impose different physical conditions on different fibers depending on their position. Is it possible that shear forces imposed during muscle contraction are greater on muscle fibers located at the periphery of fascicles and therefore cause more myofiber injury? Would an increase in injury lead to a different adaptive response with regard to muscle hypertrophy?

Further, the functional significance of regional differences in fiber composition within the same muscle may be similar to that posited with regard to intermuscular fiber type differences. On the basis of the preponderance of evidence that shows that heavy resistance training induces a preferential hypertrophy of type II or fast fibers, it would be plausible that a normal response of skeletal muscle would be to enlarge in a nonuniform manner. That is, those regions of a muscle that are predominantly fast-twitch would enlarge proportionately more than those that are slow-twitch. Thus, is it possible to emphasize particular kinds of exercise or training protocols (e.g., high vs. low volume) that could selectively hypertrophy parts of a muscle and in essence change the shape of that particular muscle?

Single Fiber Heterogeneity

Single fiber electrophoresis and immunocytochemical detection reveal that more than 1 MHC isoform can be expressed within 1 muscle fiber (4, 7, 47). Single fibers from the biceps brachii muscle coexpress MHC type I and IIa isoforms as well as a combination of type IIa and IIb isoforms (47). Interestingly, bodybuilders have a decreased coexpression of fibers with the IIa/IIb combination in comparison to sedentary individuals (47). Also, sprint training decreases the incidence of muscle fibers that coexpress the type IIa and IIb isoforms (4).

The physiological significance of having fibers with multiple isoforms is unclear. However, differential expression of MHC isoforms could arise along the length of a single fiber because of local control of myonuclei. The DNA unit (or myonuclear domain) represents a theoretical amount of cytoplasm that each myonucleus governs (15). Slow muscle fibers have a greater nucleus-to-cytoplasm ratio than fast fibers; thus, the DNA unit is more limited in slow than in fast muscle fibers (105). After bilateral surgical ablation of the plantaris and gastrocnemius muscles in the rat (i.e., functional overload), the soleus muscle demonstrated an increased fiber size and mean myonuclear number in comparison with intact rats (68). In addition, the administration of growth hormone plus insulin-like growth factor 1 further increased mean fiber size and myonuclear number above functional overload alone. Thus, the DNA unit is maintained as a muscle fiber enlarges (68).

It would seem plausible that different exercises would stress different parts of a muscle and its muscle fibers. Perhaps this could alter gene expression within that particular myonucleus, resulting in the expression of particular MHC isoforms. Nonetheless, it is evident that skeletal muscle is heterogeneous between muscles, within a muscle, and within single muscle fibers.

The notion that a particular resistance exercise will cause the entire muscle belly to grow in a uniform manner is at odds with the fact that muscles and muscle fibers are themselves heterogeneous. For instance, if one's goal was to induce maximal hypertrophy of the biceps brachii muscle, could any elbow flexion exercise achieve this? Would altering the angle of the shoulder joint affect the adaptive response of the biceps brachii muscle?

Electromyographic Evidence

There are several studies that have used electromyography (EMG) to determine the action of muscles surrounding a particular joint. The fact that a muscle does not have uniform EMG activity during a specific motion supports the notion that there is a regional-specific response during an acute bout of exercise. Whether a regional difference in EMG activity during an acute bout of exercise leads to regional differences in muscle hypertrophy is an unproven yet plausible notion. Nonetheless, it is clear that muscles do not act in a homogeneous fashion during an acute bout of exercise.

Barnett et al. (8) examined the effects of different bench angles and grip widths on muscle activity around the shoulder. The clavicular head of the pectoralis major as well as the long head of the triceps brachii were more active when using a narrow grip vs. a wide grip on the bench press. Also, the activity of the anterior deltoid increases as the degree of trunk inclination increases (8). Glass and Armstrong (29) found that the sternal portion of the pectoralis major is more active during a decline bench press in comparison with an incline bench press. There is evidence that suggests that a free weight bench press at a load equal to 60% of 1RM results in greater muscle activity than a machine bench press in the anterior and middle deltoid muscles (71). This is likely due to the need for these muscles in stabilization of the shoulder joint during a free weight bench press.

Brown et al. (11) showed that when the elbow was flexed to 120°, the short head was activated more than the long head of the biceps brachii muscle during supination. The greater the elbow was extended, the more the long head of the biceps came into play. English et al. (22) examined the ECRL muscle and found that when wrist extension was performed, the proximal ECRL was activated more than the distal ECRL. However, during radial deviation, only the proximal ECRL was activated. The distal ECRL is essentially inactive.

Sarti et al. (87) compared the average EMG activity of the upper and lower rectus abdominus muscle during a curl-up and posterior pelvic tilt exercise. The upper rectus abdominus muscle showed significantly greater activity than the lower rectus abdominus muscle during a curl-up. On the other hand, posterior pelvic tilt exercise produced greater activity than the curl-up in the lower rectus abdominus muscle. The notion that the lower and upper rectus abdominus muscles are activated differently in response to different exercises is supported by other investigators (58).

During the squat exercise, the rectus femoris, vastus lateralis, and vastus medialis were more active than the biceps femoris and semimembranosus muscles, indicating that the squat stresses the quadriceps muscle group more than the hamstrings (33). According to Signorile et al. (88), the vastus medialis produces the greatest electrical activity during a knee extension at the same angle as the vastus lateralis and rectus femoris, approximately 90°; however, Gryzlo et al. (33) found that during short-arc knee extension, the vastus medialis and lateralis were most active during the last 15° of extension.

Human Resistance Training

Heavy resistance training produces gains in muscle mass that are primarily due to muscle fiber hypertrophy; however, muscle fiber hyperplasia cannot be ruled out (5, 6, 35, 60–64, 69, 78, 82, 99). The increase in muscle cross-sectional area is primarily due to preferential hypertrophy of type II fibers (65, 82, 95, 96); however, type I fibers have been shown to experience hypertrophy similar to type II fibers under a low-repetition, high-load training protocol (18). One's training status can profoundly affect the adaptive response. Individuals who are already resistance-trained do not usually make the same gains in muscle mass observed in untrained individuals (2, 37, 38).

Staron et al. (96) examined 24 untrained women before and after a 20-week resistance training program that primarily stressed the muscles of the lower extremity. These investigators found that all fiber types increased in cross-sectional area: I (+15%), IIa (+45%), IIab + IIb (+57%). Furthermore, they found a significant decrease in the percentage of IIb fibers with an increase in IIa fibers. The decrease in the percentage of IIb fibers occurs after only 2 weeks of training in women and 4 weeks in men (94). This shift from type IIb to IIa muscle fibers is common with resistance training (49). In fact, the percentage of IIb fibers can decrease to <1% of the total fiber population (95). Moreover, there is no evidence that resistance training results in a type II to I shift or vice versa (93).

MacDougall et al. (61, 64) examined the response of untrained individuals to heavy resistance training over a 22–26-week period and found significant increases in type I (+27–31%) and type II (+33–39%) muscle fiber areas in the triceps brachii. However, a study of similar duration in elite male and female bodybuilders showed small ($p > 0.05$) gains in type I (+1.9–6.5%) and type II (1.2–11.4%) fiber areas (2). However, these competitive bodybuilders had a significant increase in whole-muscle cross-sectional area (+~8%) (2). Upon further inspection of the data from the study by Alway et al. (2), it is clear that making conclusions on the basis of grouped data can be highly misleading. Although most of the subjects had gains in muscle cross-sectional area of ~8%, 2 subjects experienced a decrease (−3.3%, −11.7%), whereas another had a tremendous increase (+19.2%). Thus, there are distinct differences in the training response that may be due to factors such as genetics, diet, or drug use.

A severe limitation of the majority of resistance training studies is the reliance on data from a single muscle biopsy. In fact, several studies have shown that changes in muscle cross-sectional area are incongruous with changes in muscle fiber size. This is due to region-specific adaptation that a muscle can undergo in response to resistance training.

Regional Adaptation—Human Resistance Training

Hisaeda et al. (41) examined college-age women who performed isotonic knee extension exercise 3 times a week for 8 weeks. These investigators found a preferential hypertrophy of the rectus femoris and vastus lateralis muscles in comparison with the vastus medialis and intermedius muscles. On the other hand, Narici et al. (77) showed that 8 weeks of isokinetic knee extensions resulted in preferential hypertrophy of the vastus medialis and intermedius muscles compared with the vastus lateralis and rectus femoris.

In a long-term study, Narici et al. (75) examined the effects of 6 months of weight training (unilateral isotonic knee extensions) on the quadriceps femoris. Quadriceps cross-sectional area increased ~19% in the proximal and distal regions but only 13% in the central portion of the muscle. There were intermuscular differences with regard to hypertrophy. The average increase in muscle cross-sectional area was as follows: rectus femoris (+27.9%), vastus lateralis (+19.5%), vastus medialis (18.7%), and vastus intermedius (17.4%). Moreover, there was nonuniform hypertrophy within each muscle. The vastus lateralis and rectus femoris showed the greatest hypertrophy in the distal region, whereas the vastus intermedius and medialis muscles demonstrated the greatest growth in the proximal portion. Interestingly, a biopsy taken from the midregion of the vastus lateralis showed little change (+1.9%, not significant) in mean fiber area.

Ten young adults (5 men and 5 women) performed leg-extension exercises concentrically with 1 leg and eccentrically on the other leg (14). They trained 3 times per week for 20 weeks, doing 4 sets of 10 reps with a 1-minute rest between sets. The cross-sectional area of the quadriceps muscles was measured at 2 levels: 25% and 75% of the femur's length measured from the knee joint. Both the concentrically and the eccentrically trained legs produced increases in muscle cross-sectional area (+5.1% concentric leg, +4.0% eccentric leg) but only in the proximal region of the quadriceps femoris muscle, with no change occurring distally. Although it is generally accepted that it is the eccentric part of a muscle contraction that is essential for growth, it is apparent that, at least in previously untrained persons, concentric contractions alone may provide a sufficient hypertrophic stimulus.

In a similar study done by Housh et al. (42), previously untrained male college students were trained to perform 6 sets of 10 reps of unilateral knee flexion/extension and elbow flexion/extension of the nondominant limbs concentrically on an isokinetic device 3 times a week for 8 weeks. The contralateral limb served as the control. The triceps brachii muscle experienced growth at the proximal and middle levels, but not distally, with the greatest changes occurring in the middle. For the quadriceps group, only the rectus femoris (at all 3 levels), the vastus lateralis (middle level), and vastus intermedius (middle level) increased in cross-sectional area. For the hamstring muscles, the biceps femoris (middle level) and the semitendinosus (distal level) increased in size with no change in the semimembranosus.

Other work further confirms that the upper-extremity muscles enlarge in a nonuniform manner similar to lower-extremity muscles (46). Sixteen weeks of unilateral triceps brachii exercises resulted in significant growth in the middle region of the triceps brachii muscle with no change in the proximal or distal end. Using male subjects with previous weightlifting experience, McCall et al. (69) found that 12 weeks of training (with emphasis on the elbow flexors) revealed greater increases in cross-sectional area in the biceps brachii (+12.6%) than the brachialis muscle (+7.7%). Interestingly, the triceps brachii muscle (+25.1%) increased more than either elbow flexor despite the lack of emphasis on this muscle.

Roman et al. (85) found that 12 weeks of training the elbow flexors in elderly men resulted in regional differences in muscle hypertrophy. The greatest increase in cross-sectional area occurred in the distal belly of the elbow flexor muscles with little if any change proximally. These investigators used magnetic resonance imaging technology to determine muscle cross-sectional area of the elbow flexors along the entire length of the arm. Interestingly, there was as much as a 73% difference in cross-sectional area between two segments 10 mm apart. This would suggest that the best measure of muscle growth should involve serial cross-sectional area measurements coupled with a muscle volume measurement. Relying solely on a single anatomical cross-sectional area may not accurately reflect muscle growth and may in fact be an overestimation.

In addition to the data on human subjects, various animal models of muscle enlargement have verified that skeletal muscle adapts in a nonuniform manner. Using the compensatory overload model, Sakuma et al. (86) demonstrated that type IIb fiber areas increased more in the middle and distal regions of the plantaris muscle vs. the proximal region. Gardiner et al. (27) found that in medial gastrocnemius undergoing compensatory growth, type I fibers were largest at the middle to rostral portion, whereas type II fibers were largest in the more caudal sections of the muscle. Stretch overload results in nonuniform increases in muscle fiber cross-sectional area and muscle fiber number in the anterior latissimus dorsi muscle (1, 3, 6, 7). Furthermore, the expression of various myosin isoforms varies between regions of the stretch-enlarged anterior latissimus dorsi muscle (3, 7).

Limitations of Current Research

It is evident that there are inter- and intramuscular variations in muscle size and fiber composition. These differences may be due to differing nerve supplies, genetic influences, or environmental stress (i.e., exercise). However, exercise (in the form of resistance training) is the paramount variable that is under the control of athletes. Although muscle enlargement is primarily due to increased fiber cross-sectional area, one cannot rule out de novo fiber formation. It is apparent that muscle growth is not merely a function of fiber hypertrophy or fiber hyperplasia. In fact, the few studies that have examined changes in muscle cross-sectional area and fiber cross-sectional area have produced odd results. Six months of unilateral isotonic knee extensions resulted in a 20% increase in the vastus lateralis muscle cross-sectional area, yet mean fiber area increased only 1.9% (75). Conversely, Frontera et al. (26) showed that 12 weeks of isotonic training of the lower extremity resulted in a 28% and 34% increase in type I and II fiber areas, respectively; however, quadriceps cross-sectional area increased only 11%. It is evident that relying solely on fiber area data derived from a single biopsy can grossly over- or underestimate the true hypertrophic response of a muscle.

Further, it is common to measure muscle cross-sectional area where the circumference is greatest. Yet even reliance on a single cross-section may not be an accurate indicator of the hypertrophic response. For instance, resistance training of the elbow flexors produced no change in type I fiber area of the biceps brachii, a 37% increase in type II fiber area of the biceps brachii, a 23% increase in the elbow flexor (biceps brachii + brachialis) cross-sectional area, and a 14% increase in elbow flexor muscle volume (85). It is apparent that measures of muscle fiber area and muscle cross-sectional area do not accurately reflect the true hypertrophy (i.e., changes in volume) of the muscle, though muscle cross-sectional area measurements are more accurate indicators of the hypertrophic response than muscle fiber area data derived from single biopsies. Future investigations should include measures of muscle volume or multiple cross-sectional area measures of the involved muscle and multiple biopsy sites for a more accurate picture of a muscle's adaptive response.

Certainly, the complexity of exercise-induced skeletal muscle hypertrophy is prodigious. Our current scientific understanding of the relations between the musculoskeletal, endocrine, and nervous systems with regard to the hypertrophic process is in its infancy. Numerous variables (i.e., age, training status, nutritional habits, drug use, sex, genetic predisposition) are known to affect the adaptive response of various biological systems to resistance training. Furthermore, the multiple variables that need to be accounted for in designing resistance training programs (i.e., repetitions, sets, exercise selection, exercise order, rest intervals, training frequency, recovery days) make it extremely difficult to make comparisons between different training programs. Thus, one should remain cognizant of the limitations that exist in the interpretation of the available data on the adaptive response to resistance training.

What Does This All Mean?

Skeletal muscle plasticity is amply demonstrated by the tremendous hypertrophy observed in resistance-trained individuals. It is nonetheless apparent that the adaptive response of skeletal muscle is quite heterogeneous. The particular muscle, muscle architecture (i.e., pennate vs. parallel), muscle fiber type, mode of contraction, exercise load and volume, training status, genes, etc. are all factors that affect the adaptive response of skeletal muscle.

Isotonic knee extension training produces preferential hypertrophy of the rectus femoris and vastus lateralis muscles in comparison with the vastus medialis and intermedius muscles (41, 75). On the other hand, isokinetic knee extensions resulted in preferential hypertrophy of the vastus medialis and intermedius compared with the rectus femoris and vastus lateralis. Are these differences due to the type of contraction used during training? The elbow flexors show greater hypertrophy in the distal vs. proximal region (85). Would altering the choice of exercise result in a different adaptive response? Or is the normal response of muscle to resistance training a nonuniform hypertrophy?

The majority of studies have examined the arm and thigh musculature. These studies have shown that significant gains in muscle cross-sectional area occur in the arm and thigh muscles as a result of a resistance training program. Anecdotal reports from bodybuilders suggest that certain muscles (e.g., wrist flexors, plantar flexors) do not respond as well as other muscles (e.g., elbow flexors, knee extensors) to resistance training. Is there an anatomical or physiological reason for this alleged difference in response between the distal and proximal regions of the limb muscles? Or should all muscles respond similarly regardless of location?

We know that the majority of resistance training studies demonstrate a preferential hypertrophy of type II fibers. Does that mean that type I fibers do not have the capacity to attain the same degree of hypertrophy as do type II fibers? Conversely, there may be resistance training programs that can induce similar or perhaps superior hypertrophy of type I fibers. Another factor that may affect muscle growth is the particular architecture of the muscle. Should all muscles respond equally regardless of their fiber arrangement (i.e., pennate vs. parallel)?

Conclusion

Skeletal muscle is a complex tissue that shows a prodigious capacity for growth. The notion that an individual muscle is just a compilation of muscle fibers that traverse from origin to insertion is simplistic and egregiously flawed. There are obvious differences between muscles with regard to size, architecture, and fiber composition. Moreover, within the same muscle, one can find regional differences in fiber size and fiber composition. Within a single fiber, one can find differences in MHC isoform expression and diameter.

Thus, it would make sense that the response of skeletal muscle to resistance training would be a nonuniform hypertrophy. In fact, the idea that a muscle would respond in a uniform fashion would seem implausible in light of the fact that there are distinct physiological/anatomical differences within a single muscle.

The existing studies (both acute and chronic training) show that within a given muscle, there is not a homogeneous response with regard to electrical activity (as measured by EMG), changes in muscle area, muscle fiber area, or even fiber number. To further our understanding of skeletal muscle plasticity, future studies should describe the regional differences in skeletal muscle with regard to muscle growth and perhaps elucidate the mechanism(s) behind such differences.

Practical Applications

I would posit that there is no single best exercise for inducing hypertrophy of specific skeletal muscles. Nor has it been shown that there is a single best training protocol for inducing skeletal muscle hypertrophy. The designation of a ''hypertrophy phase'' using the classical periodization scheme is misleading because low-repetition, high-load protocols can also induce significant hypertrophy. The fact that muscle responds in a nonuniform manner indicates that there may be different recruitment patterns with different exercises and as such, it would be advisable for bodybuilders to engage in different exercises to induce hypertrophy of different regions of a muscle.

On the basis of the limited available data, it would seem that with regard to the pectoralis major, biceps brachii, triceps brachii, rectus abdominus, and quadriceps femoris muscles, varying exercise selection should be an integral feature of the bodybuilders' training program. For other strength–power athletes (e.g., Olympic-style weightlifters), this may not be as important because the goals are quite different (i.e., performance vs. appearance).

References

1. Alway, S.E. Stretch induces non-uniform isomyosin expression in the quail anterior latissimus dorsi muscle. *Anat. Rec.* 237:1–7. 1993.
2. Alway, S.E., W.H. Grumbt, J. Stray-Gundersen, and W.J. Gonyea. Effects of resistance training on elbow flexors of highly competitive bodybuilders. *J. Appl. Physiol.* 72:1512–1521. 1992.

3. Alway, S.E., P.K. Winchester, M.E. Davis, and W.J. Gonyea. Regionalized adaptations and muscle fiber proliferation in stretch-induced enlargement. *J. Appl. Physiol.* 66:771–781. 1989.
4. Andersen, J.L., H. Klitgaard, and B. Saltin. Myosin heavy chain isoforms in single fibres from m. vastus lateralis of sprinters: Influence of training. *Acta Physiol. Scand.* 151:135–142. 1994.
5. Antonio, J., and W.J. Gonyea. Skeletal muscle fiber hyperplasia. *Med. Sci. Sports Exerc.* 25:1333–1345. 1993.
6. Antonio, J., and W.J. Gonyea. Muscle fiber splitting in stretch-enlarged avian muscle. *Med. Sci. Sports Exerc.* 26:973–977. 1994.
7. Antonio, J., and W.J. Gonyea. Ring fibres express ventricular myosin in stretch overloaded quail muscle. *Acta Physiol. Scand.* 152:429–430. 1994.
8. Barnett, C., V. Kippers, and P. Turner. Effects of variations of the bench press exercise on the EMG activity of five shoulder muscles. *J. Strength Cond. Res.* 9:222–227. 1995.
9. Billeter, R., H. Weber, H. Lutz, H. Howald, H.M. Eppenberger, and E. Jenny. Myosin types in human skeletal muscle fibers. *Histochemistry* 65:249–259. 1980.
10. Brooke, M.H., and K.K. Kaiser. Three myosin ATPase systems: The nature of their pH lability and sulfhydryl dependence. *J. Histochem. Cytochem.* 18:670–672. 1970.
11. Brown, J.M.M., C. Solomon, and M. Paton. Further evidence of functional differentiation within biceps brachii. *Electromyogr. Clin. Neurophysiol.* 33:301–309. 1993.
12. Caiozzo, V.J., M.J. Baker, and K.M. Baldwin. Are skeletal muscle fibers obligated to follow the I ↔ IIA ↔ IIX ↔ IIB scheme of MyHC isoform plasticity? [Abstract]. *Med. Sci. Sports Exerc.* 30(Suppl.):S38. 1998.
13. Campos, G.R., T. Luecke, K. Toma, F.C. Hagerman, R.S Hikida, K. Ragg, W.J. Kraemer, and R.S. Staron. The effects of three different resistance training regimens on muscle fiber type composition [Abstract]. *Med. Sci. Sports. Exerc.* 30(Suppl.): S207. 1998.
14. Carey Smith, R., and O.M. Rutherford. The role of metabolites in strength training. *Eur. J. Appl. Physiol.* 71:332–336. 1995.
15. Cheek, D.B. The control of cell mass and replication. The DNA unit—A person 20-year study. *Early Hum. Dev.* 12:211–239. 1985.
16. Chilibeck, P.D., A.W. Calder, D.G. Sale, and C.E. Webber. A comparison of strength and muscle mass increases during resistance training in young women. *Eur. J. Appl. Physiol.* 77:170–175. 1998.
17. Colliander, E.B., and P.A. Tesch. Blood pressure in resistance-trained athletes. *Can. J. Sport Sci.* 13:31–34. 1988.
18. Crill, M., G. Campos, R.S. Staron, F.C. Hagerman, T. Luecke, T. Bailey, W.J. Kraemer, and R.S. Hickida. Different resistance training protocols and skeletal muscle fiber hypertrophy [Abtract]. *Med. Sci. Sports Exerc.* 30(Suppl.):S207. 1998.
19. Davies, J., D.F. Parker, O.M. Rutherford, and D.A. Jones. Changes in strength and cross-sectional area of the elbow flexors as a result of isometric strength training. *Eur. J. Appl. Physiol.* 57:667–670. 1988.
20. Dudley, G.A. Metabolic consequences of resistive-type exercise [Abstract]. *Med. Sci. Sports Exerc.* 20(Suppl.):S158–S161. 1988.
21. Elder, G.C.B., K. Bradbury, and R. Roberts. Variability of fiber type distributions within human muscles. *J. Appl. Physiol.* 53:1473–1480. 1982.
22. English, A.W., S.L. Wolf, and R.L. Segal. Compartmentalization of muscles and their motor nuclei: The partitioning hypothesis. *Phys. Ther.* 73:857–867. 1993.
23. Essen-Gustavsson, B., and P.A. Tesch. Glycogen and triglyceride utilization in relation to muscle metabolic characteristics in men performing heavy-resistance exercise. *Eur. J. Appl. Physiol.* 61:5–10. 1990.
24. Faulkner, J.A., D.R. Claflin, and K.K. McCully. Power output of fast and slow fibers from human skeletal muscles. In: *Human Muscle Power.* N.L. Jones, N. McCartney, and A.J. McComas, eds. Champaign, IL: Human Kinetics Publishers, 1986. pp. 81–90.
25. Feinstein, B., B. Lindegard, E. Nyman, and G. Wohlfart. Morphologic studies of motor units in normal human muscles. *Acta Anat.* 23:127–142. 1955.
26. Frontera, W.R., C.N. Meredith, K.P. O'Reilly, H.G. Knuttgen, and W.J. Evans. Strength conditioning in older men: Skeletal muscle hypertrophy and improved function. *J. Appl. Physiol.* 64:1038–1044. 1988.
27. Gardiner, P.F., B.J. Jasmin, and P. Corriveau. Rostrocaudal pattern of fiber-type changes in an overloaded rat ankle extensor. *J. Appl. Physiol.* 71:558–564. 1991.
28. Garrett, W.E., J.C. Califf, and F.H. Bassett. Histochemical correlates of hamstring injuries. *Am. J. Sports Med.* 12:98–103. 1984.
29. Glass, S.C., and T. Armstrong. Electromyographical activity of the pectoralis muscle during incline and decline bench presses. *J. Strength Cond. Res.* 11:163–167. 1997.
30. Gollnick, P.D., J. Karlsson, K. Piehl, and B. Saltin. Selective glycogen depletion in skeletal muscle fibers of man following sustained contraction. *J. Physiol.* 241:59–67. 1974.
31. Gotshalk, L.A., C.C. Loebel, B.C. Nindl, M. Putukian, W.J. Sebastianelli, R.U. Newton, K. Hakkinen, and W.J. Kraemer. Hormonal responses of multiset versus single set heavy resistance exercise protocols. *Can. J. Appl. Physiol.* 22:244–255. 1997.
32. Greenhaff, P.L., K. Bodin, K. Soderlund, and E. Hultman. Effect of oral creatine supplementation on skeletal muscle phosphocreatine resynthesis. *Am. J. Physiol.* 266:E725–E730. 1994.
33. Gryzlo, S.M., R.M. Patek, M. Pink, and J. Perry. Electromyographic analysis of knee rehabilitation exercises. *J. Orthop. Sports Phys. Ther.* 20:36–43. 1994.
34. Guglielmini, C., A.R. Paolini, and F. Conconi. Variations of serum testosterone concentrations after physical exercises of different duration. *Int. J. Sports Med.* 5:246–249. 1984.
35. Haggmark, T., E. Jansson, and B. Svane. Cross-sectional area of the thigh muscle in man measured by computed tomography. *Scand. J. Clin. Lab. Invest.* 38:355–360. 1978.
36. Hakkinen, K., K.L. Keskinen, M. Alen, P.V. Komi, and H. Kauhanen. Serum hormone concentrations during prolonged training in elite endurance-trained and strength-trained athletes. *Eur. J. Appl. Physiol.* 59:233–238. 1989.
37. Hakkinen, K., P.V. Komi, M. Alen, and H. Kauhanen. EMG, muscle fibre and force production during a 1 year training period in elite weight-lifters. *Eur. J. Appl. Physiol.* 56:419–427. 1987.
38. Hakkinen, K., A. Pakarinen, M. Alen, H. Kauhanen, and P.V. Komi. Neuromuscular and hormonal adaptations in athletes to strength training in two years. *J. Appl. Physiol.* 65:2406–2412. 1988.
39. Hakkinen, K., A. Pakarinen, R.U. Newton, and W.J. Kraemer. Acute hormone responses to heavy resistance lower and upper extremity exercise in young versus old men. *Eur. J. Appl. Physiol.* 77:312–319. 1998.
40. Heron, M.I., and F.J.R. Richmond. In-series fiber architecture in long human muscles. *J. Morphol.* 216:35–45. 1993.
41. Hisaeda, H., K. Miyagawa, S.-Y. Kuno, T. Fukunaga, and I. Muraoka. Influence of two different modes of resistance training in female subjects. *Ergonomics* 39:842–852. 1996.
42. Housh, D.J., T.J. Housh, G.O. Johnson, and W.-K. Chu. Hypertrophic response to unilateral concentric isokinetic resistance training. *J. Appl. Physiol.* 73:65–70. 1992.
43. Jensen, J., H. Oftebro, B. Breigan, A. Johnsson, K. Ohlin, H.D. Meen, S.B. Stromme, and H.A. Dahl. Comparison of changes in testosterone concentrations after strength and en-

durance exercise in well trained men. *Eur. J. Appl. Physiol.* 63: 467–471. 1991.
44. JOHNSON, M.A., J. POLGAR, D. WEIGHTMAN, AND D. APPLETON. Data on the distribution of fibre types in thirty-six human muscles: An autopsy study. *J. Neurol. Sci.* 18:111–129. 1973.
45. JORGENSEN, K., T. NICHOLAISEN, AND M. KATO. Muscle fiber distribution, capillary density, and enzymatic activities in the lumbar paravertebral muscles of young men. *Spine* 18:1439–1450. 1993.
46. KAWAKAMI, Y., T. ABE, S.-Y. KUNO, AND T. FUKUNAGA. Training-induced changes in muscle architecture and specific tension. *Eur. J. Appl. Physiol.* 72:37–43. 1995.
47. KLITGAARD, H., M. ZHOU, AND E.A. RICHTER. Myosin heavy chain composition of single fibers from m. biceps brachii of male body builders. *Acta Physiol. Scand.* 140:175–180. 1990.
48. KRAEMER, W.J., S.J. FLECK, J.E. DZIADOS, E.A. HARMAN, L.J. MARCHITELLI, S.E. GORDON, R. MELLO, P.N. FRYKMAN, L.P. KOZIRIS, AND N.T. TRIPLETT. Changes in hormonal concentrations following different heavy resistance exercise protocols in women. *J. Appl. Physiol.* 75:594–604. 1993.
49. KRAEMER, W.J., S.J. FLECK, AND W.J. EVANS. Strength and power training: Physiological mechanisms of adaptation. *Exerc. Sport Sci. Rev.* 24:363–398. 1996.
50. KRAEMER, W.J., A.C. FRY, B.J. WARREN, M.H. STONE, S.J. FLECK, J.T. KEARNEY, B.P. CONROY, C.M. MARESH, C.A. WESEMAN, N.T. TRIPLETT, AND S.E. GORDON. Acute hormonal responses in elite junior weightlifters. *Int. J. Sports Med.* 13:103–109. 1992.
51. KRAEMER, W.J., S.E. GORDON, S.J. FLECK, L.J. MARCHITELLI, R. MELLO, J.E. DZIADOS, K. FRIEDL, E. HARMAN, C. MARESH, AND A.C. FRY. Endogenous anabolic hormonal and growth factor responses to heavy resistance exercises in males and females. *Int. J. Sports Med.* 12:228–235. 1991.
52. KRAEMER, W.J., K. HAKKINEN, R.U. NEWTON, M. MCCORMICK, B.C. NINDL, J.S. VOLEK, L.A. GOTSHALK, S.J. FLECK, W.W. CAMBELL, S.E. GORDON, P.A. FARRELL, AND W.J. EVANS. Acute hormonal responses to heavy resistance exercise in younger and older men. *Eur. J. Appl. Physiol.* 77:206–211. 1998.
53. KRAEMER, W.J., L. MARCHITELLI, S.E. GORDON, E. HARMAN, J.E. DZIADOS, R. MELLO, P. FRYKMAN, D. MCCURRY, AND S.J. FLECK. Hormonal and growth factor responses to heavy resistance exercise protocols. *J. Appl. Physiol.* 69:1442–1450. 1990.
54. LEE, R.G., P. ASHBY, D.G. WHITE, AND A.J. AGUAYO. Analysis of motor conduction velocity in the human median nerve by computer simulation of compound muscle action potentials. *Electroenceph. Clin. Neurophysiol.* 39:225–237. 1975.
55. LEXELL, J., K. HENRIKSSON-LARSEN, AND M. SJOSTROM. Distribution of different fiber types in human skeletal muscles. 2. A study of cross-sections of whole m. vastus lateralis. *Acta Physiol. Scand.* 117:115–122. 1983.
56. LINDMAN, R., A. ERIKSSON, AND L.-E. THORNELL. Fiber type composition of the human male trapezius muscle: Enzyme–histochemical characteristics. *Am. J. Anat.* 189:236–244. 1990.
57. LINDMAN, R., A. ERIKSSON, AND L.-E. THORNELL. Fiber type composition of the human female trapezius muscle: Enzyme–histochemical characteristics. *Am. J. Anat.* 190:385–392. 1991.
58. LIPETZ, S., AND B. GUTIN. An electromyographic study of four abdominal exercises. *Med. Sci. Sports* 2:35–38. 1970.
59. LUTOSLAWSKA, G., Z. OBMINSKI, A. KROGULSKI, AND W. SENDECKI. Plasma cortisol and testosterone following 19-km and 42-km kayak races. *J. Sports Med. Phys. Fit.* 31:538–542. 1991.
60. MACDOUGALL, J.D. Morphological changes in human skeletal muscle following strength training and immobilization. In: *Human Muscle Power.* N.L. Jones, N. McCartney, and A.J. McComas, eds. Champaign, IL: Human Kinetics, 1986. pp. 269–288.
61. MACDOUGALL, J.D., G.C.B. ELDER, D.G. SALE, J.R. MOROZ, AND J.R. SUTTON. Effects of strength training and immobilization on human muscle fibers. *Eur. J. Appl. Physiol.* 43:25–34. 1980.
62. MACDOUGALL, J.D., D.G. SALE, S.E. ALWAY, AND J.R. SUTTON. Muscle ultrastructural characteristics of elite powerlifters and bodybuilders. *Eur. J. Appl. Physiol.* 48:117–126. 1982.
63. MACDOUGALL, J.D., D.G. SALE, S.E. ALWAY, AND J.R. SUTTON. Muscle fiber number in biceps brachii in bodybuilders and control subjects. *J. Appl. Physiol.* 57:1399–1403. 1984.
64. MACDOUGALL, J.D., D.G. SALE, J.R. MOROZ, E.C.B. ELDER, AND J.R. SUTTON. Mitochondrial volume density in human skeletal muscle following heavy resistance training. *Med. Sci. Sports Exerc.* 11:164–166. 1979.
65. MACDOUGALL, J.D., G.R. WARD, D.G. SALE, AND J.R. SUTTON. Biochemical adaptation of human skeletal muscle to heavy resistance training and immobilization. *J. Appl. Physiol.* 43:700–703. 1977.
66. MANTA, P., N. KALFAK, E. KARARIZOU, D. VASSILOPOULOS, AND C. PAPAGEORGIOU. Size and proportion of fiber types in human muscle fascicles. *Clin. Neuropathol.* 15:116–118. 1996.
67. MAYHEW, T.P., J.M. ROTHSTEIN, S.D. FINUCANE, AND R.L. LAMB. Muscular adaptation to concentric and eccentric exercise at equal power levels. *Med. Sci. Sports Exerc.* 27:868–873. 1995.
68. MCCALL, G.E., D.L. ALLEN, J.K. LINDERMAN, R.E. GRINDELAND, R.R. ROY, V.R. MUKKU, AND V.R. EDGERTON. Maintenance of myonuclear domain size in rat soleus after overload and growth hormone/IGF-1 treatment. *J. Appl. Physiol.* 84:1407–1412. 1998.
69. MCCALL, G.E., W.C. BYRNES, A. DICKINSON, P.M. PATTANY, AND S.J. FLECK. Muscle fiber hypertrophy, hyperplasia, and capillary density in college men after resistance training. *J. Appl. Physiol.* 81:2004–2012. 1996.
70. MCCALL, G.E., C. GOULET, R.E. GRINDELAND, J.A. HODGSON, A.J. BIGBEE, AND V.R. EDGERTON. Bed rest suppresses bioassayable growth hormone release in response to muscle activity. *J. Appl. Physiol.* 83:2086–2090. 1997.
71. MCCAW, S.T., AND J.J. FRIDAY. A comparison of muscle activity between a free weight and machine bench press. *J. Strength Cond. Res.* 8:259–264. 1994.
72. MCCOMAS, A.J. Motor units. In: *Skeletal Muscle—Form and Function.* Champaign, IL: Human Kinetcs, 1996. pp.183–203.
73. MOSS, B.M., P.E. RENES, A. ABILDGAARD, K. NICOLAYSEN, AND J. JENSEN. 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. 1997.
74. NARDONE, A., C. ROMANO, AND M. SCHIEPPATI. Selective recruitments of high-threshold human motor units during voluntary isotonic-lengthening of active muscles. *J. Physiol.* 409: 451–471. 1989.
75. NARICI, M.V., H. HOPPELER, B. KAYSER, L. LANDONI, H. CLAASSEN, C. GAVARDI, M. CONTI, AND P. CERRETELLI. Human quadriceps cross-sectional area, torque, and neural activation during 6 months of strength training. *Acta Physiol. Scand.* 157:175–186. 1996.
76. NARICI, M.V., AND B. KAYSER. Hypertrophic response of human skeletal muscle to strength training in hypoxia and normoxia. *Eur. J. Appl. Physiol.* 70:213–219. 1995.
77. NARICI, M.V., G.S. ROI, L. LANDONI, A.E. MINETTI, AND P. CERRETELLI. Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. *Eur. J. Appl. Physiol.* 59:310–319. 1989.
78. NYGAARD, E., H. NIELSEN, AND B. SALTIN. Number of muscle fibers in human muscle determined in vivo. *Acta Physiol. Scand. Suppl.* 473:37. 1979.
79. O'CONNELL, J. Volcanic peaks. *Muscle Fit.* 59:58–65. 1998.
80. PERNUS, F., AND I. ERZEN. Arrangement of fiber types within fascicles of human vastus lateralis muscle. *Muscle Nerve* 14:304–309. 1991.
81. PETER, J.B., R.J. BARNARD, V.R. EDGERTON, C.A. GILLESPIE, AND K.E. STEMPEL. Metabolic profiles of three fiber types of skeletal

muscle in guinea pigs and rabbits. *Biochemistry* 11:2627–2633. 1972.

82. Prince, F.P., R.S. Hikida, and F.C. Hagerman. Human muscle fiber types in powerlifters, distance runners, and untrained subjects. *Pflugers Arch.* 363:19–26. 1976.
83. Punkt, K., H. Mehlhorn, and H. Hilbig. Region- and age-dependent variations of muscle fiber properties. *Acta Histochem.* 100:37–58. 1998.
84. Reichmann, H., and D. Pette. A comparative microphotometric study of succinic dehydrogenase activity levels in type I, IIA, and IIB fibers of mammalian and human muscles. *Histochemistry* 74:27–41. 1982.
85. Roman, W.J., J. Fleckenstein, J. Stray-Gundersen, S.E. Alway, R. Peshock, and W.J. Gonyea. Adaptations in the elbow flexors of elderly males after heavy resistance training. *J. Appl. Physiol.* 74:750–754. 1993.
86. Sakuma, K., A. Yamaguchi, and S. Katsuta. Are region-specific changes in fibre types attributable to nonuniform muscle hypertrophy by overloading? *Eur. J. Appl. Physiol.* 71:499–504. 1995.
87. Sarti, M.A., M. Monfort, M.A. Fuster, and L.A. Villaplana. Muscle activity in upper and lower rectus abdominus during abdominal exercises. *Arch. Phys. Med. Rehabil.* 77:1293–1297. 1996.
88. Signorile, J.F., D. Kacsik, A. Perry, B. Robertson, R. Williams, I. Lowensteyn, S. Digel, J. Caruso, and W.G. LeBlanc. Effect of foot position on the electromyographical activity of the superficial quadriceps muscles during the parallel squat and knee extension. *J. Strength Cond. Res.* 9:182–187. 1995.
89. Sjostrom, M., D.Y. Downham, and J. Lexell. Distribution of different fiber types in human skeletal muscles: Why is there a difference within a fascicle? *Muscle Nerve* 9:30–36. 1986.
90. Sola, O.M., L.C. Haines, B.A. Kakulas, T. Ivey, D.H. Dillard, R. Thomas, Y. Shoji, Y. Fujimura, and L. Dahm. Comparative anatomy and histochemistry of human and canine latissimus dorsi muscle. *J. Heart Transplant.* 9:151–159. 1990.
91. Sola, O.M., S. Herring, G. Zhang, X. Huang, N. Hayashida, L.C. Haines, R. Thomas, B.A. Kakulas, and L.R. Sauvage. Significance of the biopsy site of the latissimus dorsi muscle for fiber typing. *J. Heart Transplant.* 11:S315–S319. 1992.
92. Staron, R.S. Human skeletal muscle fiber types: Delineation, development, and distribution. *Can. J. Appl. Physiol.* 22:307–327. 1997.
93. Staron, R.S., and P. Johnson. Myosin polymorphism and differential expression in adult human skeletal muscle. *Comp. Biochem. Physiol.* 106B:463–475. 1993.
94. Staron, R.S., D.L. Karapondo, W.J. Kraemer, A.C. Fry, S.E. Gordon, J.E. Falkel, F.C. Hagerman, and R.S. Hikida. *J. Appl. Physiol.* 76:1247–1255. 1994.
95. Staron, R.S., M.J. Leonardi, D.L. Karapondo, E.S. Malicky, J.E. Falkel, F.C. Hagerman, and R.S. Hikida. Strength and skeletal muscle adaptations in heavy-resistance–trained women after detraining and retraining. *J. Appl. Physiol.* 70:631–640. 1991.
96. Staron, R.S., E.S. Malicky, M.J. Leonardi, J.E. Falkel, F.C. Hagerman, and G.A. Dudley. Muscle hypertrophy and fast fiber type conversions in heavy resistance trained women. *Eur. J. Appl. Physiol.* 60:71–79. 1990.
97. Tesch, P.A. Short- and long-term histochemical and biochemical adaptations in muscle. In: *Strength and Power in Sports.* P. Komi, ed. Oxford: Blackwell, 1992. pp. 239–248.
98. Tesch, P.A., E.B. Colliander, and P. Kaiser. Muscle metabolism during intense, heavy-resistance exercise. *Eur. J. Appl. Physiol.* 55:362–366. 1986.
99. Tesch, P.A., and L. Larsson. Muscle hypertrophy in bodybuilders. *Eur. J. Appl. Physiol.* 49:301–306. 1982.
100. Tesch, P.A., and S. Lindeberg. Blood lactate accumulation during arm exercise in world class paddlers and strength trained athletes. *Eur. J. Appl. Physiol.* 52:441–445. 1984.
101. Tesch, P.A., L.L. Ploutz-Snyder, L. Ystrom, M.J. Castro, and G.A. Dudley. Skeletal muscle glycogen loss evoked by resistance exercise. *J. Strength Cond. Res.* 12:67–73. 1998.
102. Tesch, P.A., A. Thorsson, and E.B. Colliander. Effects of eccentric and concentric resistance training on skeletal muscle substrates, enzyme activities and capillary supply. *Acta. Physiol. Scand.* 140:575–580. 1990.
103. Tesch, P.A., A. Thorsson, and B. Essen-Gustavsson. Enzyme activities of FT and ST muscle fibers in heavy-resistance trained athletes. *J. Appl. Physiol.* 67:83–87. 1989.
104. Thorstensson, A., and H. Carlson. Fibre types in human lumbar back muscles. *Acta Physiol. Scand.* 131:195–202. 1987.
105. Tseng, B.S., C.E. Kasper, and V.R. Edgerton. Cytoplasm-to-myonucleus ratios and succinate dehydrogenase activities in adult rat slow and fast muscle fibers. *Cell Tissue Res.* 275:39–49. 1994.
106. Webb, M.L., J.P. Wallace, C. Hamill, J.L. Hodgson, and M.M. Mashaly. Serum testosterone concentration during two hours of moderate intensity treadmill running in trained men and women. *Endocr. Res.* 10:27–38. 1984.
107. Wickiewicz, T.L., R.R. Roy, P.L. Powell, and V.R. Edgerton. Muscle architecture of the human lower limb. *Clin. Orthop. Related Res.* 179:275–283. 1983.