

Adaptation of Skeletal Muscle to Resistance Training¹

WILLIAM D. BANDY, MA, PT, SCS, ATC,² VENITA LOVELACE-CHANDLER, PhD, PT, PCS,²
BETH MCKITRICK-BANDY, MA, PT, PCS³

Resistance training is frequently used in rehabilitation to improve musculoskeletal function. The increased ability of skeletal muscle to generate force following resistance training results from two important changes: 1) the adaptation of the muscle fiber, and 2) the extent to which the motor unit can activate the muscle (neural adaptation). The purpose of this article is to provide a review of research investigating the effects of resistance training on muscle fibers and on nervous system input. Muscle fiber adaptations caused by resistance training include increased cross-sectional area of the muscle (hypertrophy, hyperplasia, or both), selective hypertrophy of fast twitch fibers, decreased or maintained mitochondrial number and capillary density of muscle, and possible changes in energy sources. Changes in nervous system input resulting from resistance training include recruitment of an increased number and firing rate of motor units, increased reflex potentiation, and improved synchronization. An understanding of the adaptations occurring in muscle in response to resistance training provides a fundamental basis for which appropriate clinical exercise training programs can be developed for the rehabilitation of patients.

A review of physiologic mechanisms associated with resistance training is very important to physical therapists in the rehabilitation of musculoskeletal injuries. To provide a successful resistance training program for the patient, the physical therapist needs knowledge of neuromuscular adaptations to resistance training. An understanding of the adaptations occurring in skeletal muscle in response to resistance training provide a fundamental basis for which appropriate clinical exercise training programs can be developed for rehabilitation of patients.

The purpose of this paper is to review the literature related to adaptations of muscle fiber and nervous system response as a result of resistance training. The terms "resistance training" and "strength" will be operationally defined in order to establish baseline definitions.

OPERATIONAL DEFINITIONS

Rose and Rothstein (53) defined "strength training" as a muscle or muscle group lifting heavy loads for a relatively low number of repetitions resulting in increased muscle mass because of muscle hypertrophy. Fleck and Kraemer (10) used the terms "strength training," "weight training," and "resistance training" interchangeably to refer to forms of exercise involving the use of heavy loads with the primary goal to increase muscle strength, hypertrophy or both. Vogel (73) preferred the term "resistance training" to "strength training" or "weight training" because the term "strength" omits other targeted goals such as muscle hypertrophy. The term "weight" does not include the many devices used beyond free weights (73). In view of the concern expressed by Vogel, the term "resistance training" will be used in this literature review to refer to a muscle group exercising by lifting heavy loads for a relatively low number of repetitions for all types of contractions, including isometric, concentric, and eccentric.

Generalization of the results of research on resistance training are difficult because the term strength, as applied to exercise, has no single meaning (53). Kroemer (35) defined strength as the maximal force the muscle can exert iso-

¹ This paper was prepared in partial fulfillment for Doctoral Degree in Physical Therapy at Texas Woman's University in Houston, TX.

² On faculty for the Physical Therapy Department, University of Central Arkansas, Conway AR 72032.

³ Pediatric Physical Therapy Services, Little Rock, AR.

metrically in a single effort. Fleischman (11) defined three constructs of strength: static, dynamic, and explosive. Static strength referred to strength measured isometrically, dynamic strength to efforts repeated several times (until fatigue), and explosive strength to fast, maximal effort. Smidt and Rogers (61) operationally defined strength as the capacity of a muscle to provide the tension necessary for maintaining posture, initiating movement, or controlling movement during conditions of loading on the musculoskeletal system. Mayhew and Rothstein (43) have suggested avoiding the term "strength," stating that the word is inherently ambiguous while appearing to be specific. Instead, the authors recommend using the term "muscle performance" because the term is deliberately vague and forces the user to operationally define how to measure muscle performance.

For the purposes of this review, strength has been defined similarly to the definition used by Kroemer (35) as follows: simply the ability of the muscle to develop force against an unyielding resistance in a single maximal contraction of unrestricted duration. The definition appears to be supported in the literature (37, 50).

During a resistance training program, the muscle adapts physiologically in response to the challenge of increasing amounts of resistance (27). This increase in strength of the muscle is the result of both the adaptations at the muscle fiber level as well as the ability of the nervous system to recruit motor units involved in activation of the muscle (55, 65).

MUSCLE FIBER ADAPTATION

Muscle Cross-Sectional Area

Progressive overload of the muscle produced by resistance training has been shown to increase the cross-sectional area of the muscle (23, 27, 38–40, 75). The direct relation between increased cross-sectional area of the muscle and the production of increased force during a maximal muscle contraction has been demonstrated (23, 38–40, 75). Controversy exists as to whether the increase in cross-sectional area following resistance training results from an increase in cross-sectional area of the individual muscle fiber (hypertrophy) or an increase in the number of muscle fibers (hyperplasia).

Hypertrophy

Muscle hypertrophy represents a frequently reported response to resistance training and is characterized by an increase in the diameter of the individual muscle fiber (17, 23, 39, 40). Using computerized tomography (CT), Haggmark et al

(23) evaluated the cross-sectional area of the muscle of two groups of subjects, weight lifters and sedentary individuals. The weight lifters were found to have greater muscle cross-sectional area than the sedentary subjects, and the increase in cross-sectional area found in the weight lifters resulted from an increase in the size of individual muscle fibers.

More recently, MacDougall et al (39) used CT scanning in combination with muscle biopsy techniques to examine the cross-sectional area of the biceps brachii muscle of elite bodybuilders, intermediate bodybuilders, and sedentary subjects. The CT scan showed that average cross-sectional fiber area for both bodybuilder groups to be significantly greater than the sedentary group. No significant difference in fiber number existed between the three groups. The authors concluded that the increase in cross-sectional area resulting from heavy resistance training was because of increased cross-sectional area of the skeletal muscle fiber and not the result of increased fiber number.

Data obtained in dissections of rats by Goldspink (13) and Gollnick et al (17) indicated that the number of muscle fibers in skeletal muscle is fixed and that increases in muscle bulk produced by normal growth and functional overload were the result of fiber hypertrophy and not because of increased fiber number.

A longitudinal study by Luithi et al (38) examining six weeks of heavy resistance training reported an 8.4% increase in cross-sectional area of the vastus lateralis muscle in humans. MacDougall et al (40) also reported hypertrophy in human muscle following five months of resistance training of the muscle. These authors suggested that the increase in the cross-sectional area of the muscle was the result of the increase in myofibrillar volume of the individual muscle fiber (hypertrophy) and not because of increase in fiber number (38, 40).

Hyperplasia

The existence of hyperplasia, muscle fiber splitting in humans, remains controversial (10, 65). Gonyea and Erickson (20) theorized that fiber splitting of muscle was caused by heavy resistance training that created maximal hypertrophy of the muscle cells, making further hypertrophy impossible. These researchers suggested that after maximal hypertrophy was obtained, additional strength gains could only be made through the addition of new muscle fibers, which occurred when existing muscle fibers divided.

Edgerton (9) published early evidence to support the process of longitudinal fiber splitting in the muscle fibers of rats subjected to extensive resistance training. Since Edgerton's work, addi-

tional studies have reported the occurrence of hyperplasia following resistance training in chickens (63) and cats (18–22).

Recently, researchers have examined the occurrence of hyperplasia in human muscle through the use of cross-sectional studies comparing athletes who have the ability to display extreme muscle strength with sedentary subjects (36, 39). Larsson and Tesch (36) concluded that hyperplasia was more pronounced in successful body builders than nonweight lifters, while MacDougall et al (39) concluded that body builders did not possess more muscle fibers than untrained subjects. MacDougall et al (39) reported individual differences in muscle fiber number in humans and concluded that cross-sectional studies reporting enhanced hyperplasia in strength-trained athletes could not accommodate for the possibility that the athlete had an increased number of muscle fibers because of heredity.

Evidence supporting hyperplasia in human muscle following resistance training is scarce. In addition, research supporting hyperplasia in animals has been criticized because of methodological errors associated with estimation of fiber number from histological sections (17, 36). Gollnick et al (17) warn that the biopsy techniques used in the studies supporting hyperplasia in animals lack reliability, and conclusions based on such biopsy techniques provide no evidence of whether total number of fibers in a muscle increase, decrease, or remain constant as a result of resistance training. Most researchers agree that increased cross-sectional area of the muscle following resistance training is primarily the result of hypertrophy (10, 13, 17, 39, 40, 65).

Muscle Fiber Types

Human skeletal muscle is composed of varying percentages of fiber types. The percentage of composition of these fiber types varies widely between muscles and among individuals (16). Many classifications have been used to differentiate fiber types based on physiological, histochemical, and biochemical properties (49, 51, 58).

For many years, researchers used physiological techniques to examine the contractile properties of the muscle and the speed at which a fiber can produce peak tension. Two fiber types, fast twitch (FT) and slow twitch (ST), were identified (49). FT fibers develop high tension very quickly but maintain the high tension for only a short period of time. ST fibers develop less tension more slowly than FT fibers and are resistant to fatigue. FT fibers are recruited during short-term, high-intensity work (resistance training), while ST fibers are used for long-term, low-intensity work (endurance training) (49).

Using the two-fiber classification system,

Thorstensson et al (70) and Hakkinen et al (24) showed increased FT:ST fiber area ratio following two different types of resistance training programs. Thorstensson et al (70) achieved these results after eight weeks of heavy progressive resistance training using weights, while the study by Hakkinen et al (24) trained subjects for 24 weeks with explosive jumping activities. The authors of these two studies concluded that the data supported a selective hypertrophy of FT fibers following resistance training.

Researchers investigating fiber type composition have studied individuals with years of resistance training and individuals with years of endurance training (52, 67, 69). The researchers compared these two groups to each other and to an untrained sample. Examining muscle tissue samples in wrestlers, distance runners, weight lifters, and nonathletes, Tesch and Karlsson (67) reported significant increases in FT:ST fiber area ratio in the weight lifters compared to the three other groups. The authors concluded that weight training causes selective hypertrophy of FT fibers.

Selective hypertrophy of FT fibers in weight lifters was supported by additional research by Tesch et al (60) and Prince et al (52) who compared weight lifters, endurance athletes, and untrained subjects. The authors of both studies reported increased FT:ST fiber area ratio and concluded that weight lifting stimulated fiber growth in FT fibers more than ST fibers. In general, research indicates that muscles of resistance-trained athletes possess hypertrophied FT fibers, while the muscles of athletes trained for endurance activities possess hypertrophied ST fibers.

Recent research involving new staining techniques and electron microscopy have led to other classification systems (3, 5, 51). Peter et al (51) classified fibers according to biochemical, physiological, and histochemical properties. In contrast, Burke et al (5) combined histochemical and physiologic properties to classify fibers, while Brooke and Kaiser (3) used only histochemistry. Each of these studies identified a third fiber type in addition to fast and slow twitch fibers but the descriptions varied. A review of literature by Rose and Rothstein (53) combined the classification schemes of these three studies and described three muscle fiber types as follows:

"1) Type I (slow oxidative, slow twitch) fibers have large amounts of oxidative enzymes and small amounts of glycolytic enzymes. These fibers primarily use aerobic metabolism, are associated with extensive capillary density, and have a high number of mitochondria . . . These muscles generate a relatively small amount of tension, have a slow contraction time, and are resistant to fatigue.

2) Type IIB (fast glycolytic, fast twitch-fast fatiguable) fibers are well-supplied with glycolytic

enzymes and are poorly endowed with oxidative enzymes, which indicate a high capacity for anaerobic metabolism. These fibers are associated with relatively sparse capillary density, (and) have few mitochondria . . . These muscle units generate a large amount of tension in a short time, but they fatigue rapidly.

3) Type IIA (fast oxidative glycolytic, fast twitch-fatigue resistant) fibers possess intermediate amounts of oxidative and glycolytic enzymes, which indicates the use of both anaerobic and aerobic metabolism. These fibers have cytological properties that fall between the Type I and Type IIB fiber."

Two studies have investigated the effects of resistance training on muscle fibers using the classification system with I, IIA, and IIB categories (7, 64). A cross-sectional study by Staron et al (64) examined the differences in the proportion of type I, IIA, and IIB fibers in weight lifters, distance runners, and untrained individuals. The largest proportion of muscle fiber type in the weight lifters was type IIA fibers. In addition, the weight lifters presented a significantly larger type IIA fiber area than either the endurance-trained or the untrained groups. The authors concluded that weight lifting requires greater use of type IIA fibers than either type I or IIB.

Similar results were reported in a longitudinal study by Costill et al (7). These authors reported significant increase in the IIA:I and IIA:IIB fiber area ratios following seven weeks of isokinetic resistance training. The changes were theorized to result from hypertrophy in type IIA fibers following resistance training.

Cellular Changes in Muscle Cells

The number of mitochondria and the capillary density (number of capillaries per square millimeter of tissue) in muscle have been shown to correlate positively with both oxidative enzyme activities (6, 31, 33) and measurement of maximal oxygen uptake (2, 8, 28). Increase in number of mitochondria (15, 30) and increase in the capillary density (1, 2, 32) in muscle are well-documented changes following endurance training, but the effect of resistance training on the mitochondria and capillary supply in muscle is not as well-defined.

After performing a six-month resistance training program, MacDougall et al (41) concluded that resistance training resulted in decreased mitochondrial density (number of mitochondria per volume of muscle tissue) and mitochondrial number/myofibrillar volume ratio in the trained muscle. The authors claimed that the mitochondrial reduction was the result of the increase in total contractile protein because of hypertrophy, without a proportional increase in the number of mitochondria.

Luithi et al (38) also found decreased mitochondrial density in parallel with muscle hypertrophy following resistance training. These authors suggested that the anaerobic nature of the resistance training program failed to adequately stimulate the oxidative metabolic system and that the increased hypertrophy of the muscle fiber caused by resistance training occurred at a greater rate than the increase in the total number of mitochondria (oxidative metabolic system).

A cross-sectional study by Tesch et al (69) compared the number of capillaries in the muscle and the capillary density in weight lifters, endurance athletes, and nonathletes. The endurance athletes had a greater number of capillaries and greater capillary density than the weight lifters and nonathletes. No difference in number of capillaries was found between the weight lifters and nonathletes, and the weight lifters presented lower capillary density than the nonathletes. The authors concluded resistance training produced fiber hypertrophy without changing the number of capillaries and thus reduced capillary density.

In contrast, a second cross-sectional study by Schantz (60) compared resistance-trained athletes to endurance-trained athletes. The authors concluded that resistance training increased capillary growth as measured by increased number of capillaries but did not change capillary density.

The findings of these two studies on capillary density provide conflicting data on capillary proliferation occurring as a result of resistance training. However, the studies do suggest that the hypertrophic effect of the muscle fiber is greater than, or equal to, the increased number of capillaries, and the net effect is decreased or maintained capillary density. No evidence supports an increase in capillary density in resistance training as is seen with endurance training.

Effect on Energy Sources

Muscle contraction is accomplished by the hydrolysis of high energy phosphate, adenosine triphosphate (ATP), to adenosine diphosphate (ADP), inorganic phosphate, and utilization of energy stored in the hydrolyzed bond (44, 59). Only small amounts of ATP are stored in the muscle, and this energy supply is sufficient for only the first few seconds of exercise. In order to continue to contract, the muscle must maintain a supply of ATP. In the skeletal muscle, three primary sources of energy are available and are potentially affected by resistance training. During resistance training, stored phosphagen and anaerobic glycolysis are used as primary sources of energy. These two metabolic pathways supply ATP in the absence of oxygen. As oxygen becomes available in the cell, a third energy source called aerobic metab-

olism is directed towards oxidative utilization of carbohydrates and fatty acids (44, 59).

Stored Phosphagen

Since muscle ATP stores are depleted quickly, an immediate source of energy used for maximal muscle contraction is a high-energy stored phosphagen called creatine phosphate (CP). When CP is hydrolyzed, the energy is used to form new ATP molecules that can then be used as an energy source. Two enzymes, creatine phosphokinase (CPK) and myokinase (MK), catalyze the reaction between CP and ADP to produce more ATP and therefore produce more usable energy (44, 59).

MacDougall et al (42) reported an increase in intramuscular stores of ATP and CP after five months of resistance training. The authors emphasized that the increased ATP and CP stores would not be expected to affect the maximal strength of the muscle but would increase the total energy available from this source and thereby, prolong the time that a maximal contraction could be maintained. Following eight weeks of sprint training on a treadmill at high speed, Thorstensson et al (71) reported increased muscle enzyme activity of MK and CPK but no change in muscle concentrations of ATP and CP. Costill et al (7) reported no increase in activity of CPK or MK after seven weeks of six-sec bouts of isokinetic training, but an increase occurred in CPK and MK activity in subjects performing 30-sec bouts of isokinetic training. Conversely, the recent study by Tesch et al (68) reported decreased concentration of CPK and MK after six months of resistance training.

Anaerobic Glycolysis

When CP reaches a critical depletion level, ATP is produced by the breakdown of carbohydrates in the absence of oxygen, a process called anaerobic glycolysis. Two enzymes important to the process of anaerobic glycolysis are phosphofructokinase (PFK) and lactate dehydrogenase (LDH) (44, 59).

The research by Costill et al (7) showed increases in both PFK and LDH after resistance training with both six-sec and 30-sec bouts of isokinetic exercise. The authors suggested that the increased activity of PFK and LDH following resistance training assists the anaerobic glycolysis pathway by providing a rapid replenishment of ATP during maximal muscle contraction. Therefore, the combination of the anaerobic glycolysis pathway with the phosphagen energy pathway prolongs the time that a maximal contraction can be sustained. Conversely, the study by Tesch et al (68) reported a decreased concentration of PFK

and no change in the concentration of LDH. These authors concluded that resistance training was not associated with increased activity of enzymes reflecting the stored phosphagen or anaerobic glycolysis metabolic pathways.

Aerobic Metabolism

The energy for long-term exercise is derived from the final oxidative breakdown of carbohydrates and fat in the mitochondria of the muscle. Adaptation in muscle following endurance training is well-documented and demonstrates an enhanced capacity for aerobic metabolism (29, 30). Research shows significant increases in succinate dehydrogenase (SDH) and malate dehydrogenase (MDH), which are important enzymes used to measure activity of the Krebs cycle (indicative of aerobic metabolism) (14, 28, 29).

Research supporting change in the aerobic capacity after resistance training is scarce. Costill et al (7) reported increased SDH and MDH enzyme activity after seven weeks of isokinetic training using 30-sec bouts of maximal contractions at 180°/sec, but no change in these enzyme levels using six-sec bouts of maximal contractions. Tesch et al (68) reported no change in SDH or MDH levels after six months of heavy resistance training.

NEURAL ADAPTION

In addition to the adaptive changes that occur in muscle fibers, resistance training also affects the influence of the nervous system on the muscle. The changes in the nervous system following resistance training have been referred to in the literature as neural adaptation (55). Neural adaptation after resistance training has been inferred on the basis of several studies reporting increases in muscle strength with little or no change in cross-sectional area of the muscle (34, 48, 66, 70, 74). Research examining neural adaptations following resistance training has focused primarily on the changes occurring in the activation of the motor unit (55).

The final pathway by which the supraspinal centers of the nervous system can exert their control over motor activity is the motor unit (4, 12). The motor unit is considered the functional unit of skeletal muscle and consists of a single motor cell (with the body contained in the anterior horn of the spinal cord), the axon and terminal branches, and all the individual muscle fibers supplied by the axon (4, 12). The actual number of muscle fibers in a particular motor unit varies. Muscle involved in delicate movements of the eye have an innervation ratio (the total number of motor axons divided by the total number of muscle fibers in a muscle) of 1:4. Large postural

muscles not requiring a fine degree of control have an innervation ratio as large as 1:150 (4, 12).

Electromyography (EMG) has provided a method to directly assess the neural adaptation of the motor unit following resistance training (55). Electromyography is the study of muscle function through examining the electrical activity of the muscle (62). Surface or in-dwelling electrodes are connected to a recording apparatus and are used to gather information about the muscle. In general, EMG can be used to study pure function, the contraction/relaxation patterns of muscle during a particular movement, or to evaluate motor unit activity (62).

Motor Unit Activation during Maximal Contraction

One common method for evaluating neural adaptation of muscle is to use EMG to record the motor unit activity during a maximal contraction before and after resistance training. Using this method, motor unit activation during maximal contraction has been shown to increase after various types of resistance training (24–26, 48). In the study by Moritani and deVries (48), both isometric muscle strength and motor unit activation (as measured by EMG) increased following eight weeks of isotonic elbow flexion exercise. The authors concluded that increased strength following resistance training was due to the combination of increased neural input and hypertrophy of contractile tissue.

Three studies examining the concept of neural adaptation have been published by Hakkinen in corroboration with various co-investigators (24–26). Two of the studies were designed to evaluate adaptation of the knee extensor muscles following 24 weeks of a dynamic squat lift exercise program (24, 25), and in both studies, marked improvement in muscle strength and significant increases in neural activation of the motor units of knee extensor muscles as measured by EMG were reported.

A third study by Hakkinen et al (26) supported previous findings of increased strength and enhanced motor unit activation after 24 weeks of resistance training, but in this study, the method of resistance training was explosive jumping activities. The authors concluded that the increased EMG activity noted during maximal contraction following resistance training was the result of a combination of recruitment of an increased number of motor units and an increased firing rate of each unit.

Reflex Potentiation

Reflex potentiation attempts to measure the degree to which certain reflex responses are poten-

tiated during maximal contraction by employing surface EMG and indirect nerve stimulation (72). Response of the muscle to nerve stimulation at rest is compared to the response during maximal contraction, thereby calculating a reflex potentiation ratio (56). Sale et al (58) suggested that reflex potentiation is a measure of the ability of motor units to fully activate during maximal contraction.

A cross-sectional study by Sale et al (58) compared reflex potentiation in the triceps surae and thenar muscles of weight lifters to control subjects. Results indicated significantly greater reflex potentiation in the triceps surae muscle of the weight lifters than the control group but no differences between the two groups when comparing reflex potentiation of the thenar muscles. A study by Sale et al (57) described the effects of 18 weeks of training on the thenar muscles. Data from their study also indicated no change in reflex potentiation of the thenar muscles following training. The authors speculated that the thenar muscles played an important part in skilled movements of the thumb, and all subjects possessed a highly trained thenar muscle. Therefore, difficulty may arise when attempting to further enhance reflex potentiation of the thenar muscle through resistance training.

A third study by Sale et al (56) examined the change in reflex potentiation of four different muscles—the extensor digitorum brevis, soleus, brachioradialis, and hypothenar muscles—following resistance training ranging from nine to 21 weeks. Resistance training was found to increase reflex potentiation of all muscles tested except the soleus muscle. The authors theorized that the change in reflex potentiation after resistance training resulted from the enhanced ability of the subject to recruit additional motor units or discharge the motor units at a faster rate. In this way, more motoneurons were available to participate in the reflex response.

Synchronization

Relatively smooth performance is achieved in endurance activities because of the motor units firing asynchronously, allowing one group of muscle fibers within the muscle to rest while another group of muscle fibers contracts. Asynchronous firing of the motor units allows the muscle to contract for long periods of time at submaximal work loads. During the production of maximal muscle tension, the majority of the motor units of contracting muscle fire synchronously, so that the muscle fibers are activated at the same time. In this way, the muscle is able to generate maximal or near-maximal force, but the contraction can only be maintained for a short period of time (46, 47).

Milner-Brown et al (47) introduced a method

of quantifying motor unit synchronization by measuring motor unit activity via EMG. Using this method of quantifying motor unit synchronization, two different conditions were investigated in a second study by Milner-Brown et al (45). First, a six-week resistance training study indicated increased motor unit synchronization in the first dorsal interosseus muscle of the hand following training. Second, a cross-sectional study comparing weight lifters to a control group indicated that the weight lifters possessed a greater degree of motor unit synchronization during muscle contraction than the control subjects.

No further studies examining synchronization of motor units could be found in the literature, but research examining mechanisms of neural adaptation during resistance and endurance training have referenced the study by Milner-Brown et al (45) when attempting to explain the results (54, 56, 66).

SUMMARY

According to the literature, the increased ability of a muscle to generate force following resistance training was related to two important changes: the adaptation of the muscle fiber and the extent to which the motor units can activate the muscle. Increase in the cross-sectional area of skeletal muscle was the result of increased size (hypertrophy) of the FT type of muscle fiber. Hypertrophy of the FT muscle fiber was caused by the increased synthesis of the myofibrillar proteins, actin and myosin.

Some researchers reported the number of mitochondria and the capillary density to decrease in response to resistance training, while others reported no change. No support for an increase in mitochondrial number or capillary density secondary to resistance training existed in the literature. No definitive picture existed suggesting that the ability of the muscle to use anaerobic energy sources during exercise changes after resistance training, but the evidence indicated that no change occurred in the ability of the muscle to use aerobic metabolism.

Improvement in the ability of the motor unit to activate the muscle following resistance training has been inferred on the basis of several studies reporting increased strength without changes in the cross-sectional area of the muscle. Electromyography has been used to measure change in motor unit activity following maximal contraction, reflex potentiation, and synchronization. Data provided by EMG indicated that resistance-trained muscle exhibited recruitment of a greater number of motor units and a greater firing rate of each motor unit during a maximal contraction than untrained or less-trained muscle. The researchers of these studies concluded that the increased num-

ber of motor units firing at a high frequency facilitated increased activation of the muscle and, therefore, increased ability of the muscle to generate force.

CONCLUSION

Based on this review of research, documentation exists to support resistance training as causing increased size of the cross-sectional area of the muscle through hypertrophy of FT muscle fibers and as causing recruitment of an increased number and firing rate of motor units. More research using a resistance training program to examine the effects of training on changes in capillary density and mitochondrial number in muscle, on changes in anaerobic energy stores in muscle, and on the contribution of the neural components of reflex potentiation and synchronization is needed. Research in these areas will provide more information on the changes that occur in muscle as a result of resistance training. □

Acknowledgements: William Hanten, PhD, PT, Eve Sherwood, Pam Salkeld.

REFERENCES

1. Anderson P: Capillary density in skeletal muscle of man. *Acta Physiol Scand* 95:203-205, 1975
2. Anderson P, Henriksson J: Capillary supply of the quadriceps femoris muscle in man: adaptive response to exercise. *J Physiol* 270:677-690, 1977
3. Brooke MH, Kaiser KK: Muscle fiber types: how many and what kind? *Arch Neurol* 23:369-379, 1970
4. Buchtal F, Schmalbruch H: Motor unit of mammalian muscles. *Physiol Rev* 60:90-142, 1980
5. Burke RE, Levine DN, Tsairis P, Zajac III, FE: Physiological types and histochemical profiles in motor units of the cat gastrocnemius. *J Physiol (Lond)* 234:723-748, 1973
6. Bylund A, Bjuro T, Cederland G: Physical training in man. Skeletal muscle morphology and running ability. *Eur J Appl Physiol* 36:151-169, 1977
7. Costill DL, Coyle EF, Fink WF, Lesmes GR, Witzmann FA: Adaptation in skeletal muscle following strength training. *J Appl Physiol* 46:96-99, 1979
8. Costill DC, Daniels J, Evans W, Fink W, Krahenbuhl G, Saltin B: Skeletal muscle enzymes and fiber composition in male and female track athletes. *J Appl Physiol* 40:149-154, 1976
9. Edgerton VR: Morphology and histochemistry of the soleus muscle from normal and exercised rats. *Am J Anat* 127:81-86, 1970
10. Fleck SJ, Kraemer WJ: Resistance training: Physiological response and adaptations (Part 2 of 4). *Phys Sportsmed* 16(April):108-124, 1988
11. Fleischmann EA: Factor analysis of physical fitness tests. *Education Psych Measurement* 23:647-661, 1963
12. Freund HJ: Motor unit and muscle activity in voluntary motor control. *Physiol Rev* 63:387-436, 1983
13. Goldspink G: The proliferation of myofibrils during muscle fibre growth. *J Cell Sci* 6:593-604, 1970
14. Gollnick PD, Armstrong RB, Saltin B, Saubert IV, Sembrowich WL, Shepherd RE: Effect of training on enzyme activity and fiber composition of human skeletal muscle. *J Appl Physiol* 34:107-111, 1978
15. Gollnick PD, King D: Effect of exercise and training on mitochondria of rat skeletal muscle. *Am J Physiol* 216:1502-1506, 1969
16. Gollnick PD, Matoba H: The muscle fiber composition of skeletal muscle as a predictor of athletic success. *Am J Sports Med* 12:212-217, 1984
17. Gollnick PD, Timson BF, Moore RL, Reidy M: Muscular enlargement and number of fibers in skeletal muscle of rats. *J Appl Physiol* 50:936-943, 1981

18. Gonyea WJ: Role of exercise in inducing increases in skeletal muscle fiber number. *J Appl Physiol* 48:421-426, 1980
19. Gonyea WJ, Bonde-Peterson F: Alterations in muscle contractile properties and fiber composition after weight lifting exercise in cats. *Exp Neurol* 59:75-84, 1978
20. Gonyea WF, Erickson GC: Experimental model for study of exercise induced skeletal muscle hypertrophy. *J Appl Physiol* 40:630-633, 1976
21. Gonyea W, Erickson GC, Bonde-Peterson F: Skeletal muscle fiber splitting induced by weight lifting exercise in cats. *Acta Physiol Scand* 99:105-109, 1977
22. Gonyea WF, Sale DG, Gonyea FB, Erickson GC: Exercise induced increases in muscle fiber number. *Eur J Appl Physiol* 54:1292-1297, 1983
23. Haggmark T, Jansson E, Svane B: Cross-sectional area of the thigh muscle in man measured by computed tomography. *Scand J Clin Lab Invest* 38:355-360, 1978
24. Hakkinen K, Alen M, Komi PV: Changes in isometric force and relaxation time, electromyographic and muscle fibre characteristics of human skeletal muscle during strength training and detraining. *Acta Physiol Scand* 125:573-585, 1985
25. Hakkinen K, Komi PV: Electromyographic changes during strength training and detraining. *Med Sci Sports Exerc* 15:455-460, 1983
26. Hakkinen K, Komi PV, Alen M: Effect of explosive type strength training on isometric force- and relaxation time, electromyographic, and muscle fibre characteristics of leg extensor muscles. *Acta Physiol Scand* 125:587-600, 1985
27. Hellebrandt FA, Houtz SJ: Mechanisms of muscle training in man. Experimental demonstration of the overload principle. *Phys Ther Rev* 36:371-376, 1956
28. Hoeppler H, Lüthi P, Claassen H, Weibeler ER, Howald H: The ultrastructure of the normal human skeletal muscle: a morphometric analysis on untrained men, women, and well trained orienteers. *Pfluegers Arch* 344:217-232, 1973
29. Holloszy JO: Biochemical adaptations to exercise: aerobic metabolism. *Exerc Sport Sci Rev* 1:45-71, 1973
30. Holloszy JO, Booth FW: Biochemical adaptations to endurance exercise in muscle. *Annu Rev Physiol* 18:273-278, 1976
31. Howald H: Ultrastructure and biochemical function of skeletal muscle in twins. *Ann Hum Biol* 3:455-462, 1976
32. Inger F, Brodal P: Capillary supply of skeletal muscle fibers in untrained and endurance trained women. *Eur J Appl Physiol* 38:291-299, 1978
33. Kiessling K, Pilstrom L, Bylund A, Saltin B, Piehl, K: Enzyme activities and morphometry in skeletal muscle of middle aged men after training. *Scand J Clin Lab Invest* 33:63-69, 1974
34. Komi PV, Vitasalo J, Rauramaa R, Viikio V: Effect of isometric strength training on mechanical, electrical and metabolic aspects of muscle function. *Eur J Appl Physiol* 40:45-66, 1978
35. Kroemer KH: Human strength: terminology, measurement and interpretation of data. *Hum Factors* 12:515-522, 1972
36. Larsson L, Tesch PA: Motor unit fiber density in extremely hypertrophied skeletal muscles in man. *Eur J Appl Physiol* 55:130-136, 1986
37. Lehmkuhl D: Local factors in muscle performance. *Phys Ther* 46:473-484, 1966
38. Lüthi JM, Howald H, Claassen H, Rosler K, Vock P, Hoppeler H: Structural changes in skeletal muscle tissue with heavy resistance exercise. *Int J Sports Med* 7:123-127, 1986
39. MacDougall JD, Sale DG, Alway SE, Sutton JR: Muscle fiber number in biceps brachii in body builders and control subjects. *J Appl Physiol* 57:1399-1403, 1984
40. MacDougall JD, Sale DG, Elder G, Sutton JR: Ultrastructural properties of human skeletal muscle following heavy resistance training and immobilization. *Med Sci Sports Exerc* 8:72-73, 1976
41. MacDougall JD, Sale DG, Moroz JR, Elder G, Sutton JR, Howald H: Mitochondrial volume density in human skeletal muscle following heavy resistance training. *Med Sci Sports Exerc* 11:164-166, 1979
42. MacDougall JD, Ward GR, Sale DG, Sutton JR: Biochemical adaptation of human skeletal muscle in heavy resistance training and immobilization. *J Appl Physiol* 43:700-703, 1977
43. Mayhew TP, Rothstein JM: Measurements of muscle performance with instruments. In: Rothstein JM, Measurement in Physical Therapy, pp 57-102. New York: Churchill Livingstone, 1985
44. McCafferty WB, Horvath SM: Specificity of exercise and specificity of training: A subcellular review. *Res Q Exerc Sport* 48:358-371, 1977
45. Milner-Brown HS, Stein RB, Lee RG: Synchronization of human motor units: possible roles of exercise and supraspinal reflexes. *Electroencephalogr Clin Neurophysiol* 38:245-254, 1975
46. Milner-Brown HS, Stein RB, Lee RG: Synchronization of motor units. *Physiol Can* 4:193, 1973
47. Milner-Brown HS, Stein RB, Yemm R: The orderly recruitment of human motor units during voluntary isometric contractions. *J Physiol* 230:359-370, 1973
48. Moritani T, deVries HA: Neural factors vs hypertrophy in time course of muscle strength gain. *Am J Phys Med Rehabil* 58:115-130, 1979
49. Morris CJ: Human skeletal muscle fibre type grouping. *J Neurol Neurosurg Psychiatr* 32:440-444, 1968
50. Muller EA: Influence of training and of inactivity of muscle strength. *Arch Phys Med Rehabil* 51:449-462, 1970
51. Peter JB, Barnard RJ, Edgerton VR, et al: Metabolic profiles of three fiber types of skeletal muscle. *Biochemistry* 11:2627-2633, 1972
52. Prince FP, Hikida RS, Hagerman FC: Human muscle fiber types in power lifters, distance runners and untrained subjects. *Pfluegers Arch* 363:19-26, 1976
53. Rose SJ, Rothstein JM: Muscle mutability. Part 1. General concepts and adaptations to altered patterns of use. *Phys Ther* 62:1773-1787, 1982
54. Rosler K, Conley KE, Howald H, Gerber C, Hoppeler H: Specificity of leg power changes to velocities used in bicycle endurance training. *J Appl Physiol* 61:30-36, 1986
55. Sale DG: Neural adaptation to resistance training. *Med Sci Sports Exerc* 20:S135-S145, 1988
56. Sale DG, MacDougall JD, Upton A, McComas AJ: Effect of strength training upon motoneuron excitability in man. *Med Sci Sports Exerc* 15:57-62, 1983
57. Sale DG, McComas AJ, MacDougall JD, Upton ARM: Neuromuscular adaptation in human thenar muscles following strength training and immobilization. *J Appl Physiol* 53:419-424, 1982
58. Sale DG, Upton ARM, McComas AJ, MacDougall JD: Neuromuscular function in weight-trainers. *Exp Neurol* 82:521-531, 1983
59. Saltin B: Metabolic fundamentals in exercise. *Med Sci Sports Exerc* 5:137-46, 1973
60. Schantz P: Capillary supply in hypertrophied human skeletal muscle. *Acta Physiol Scand* 114:635-637, 1982
61. Smidt GL, Rogers MW: Factors contributing to the regulation and clinical assessment of muscular strength. *Phys Ther* 62:1283-1290, 1982
62. Soderberg GL, Cook TM: Electromyography in biomechanics. *Phys Ther* 64:1813-1820, 1984
63. Sole O, Christensen DL, Marten AW: Hypertrophy and hyperplasia of adult chicken anterior latissimus dorsi muscle following stretch with and without denervation. *Exp Neurol* 41:76-100, 1973
64. Staron R, Hikida RS, Hagerman FC, Dudley GA, Murray TF: Human muscle skeletal muscle fiber type adaptability to various workloads. *J Histochem Cytochem* 32:146-152, 1984
65. Tesch PA: Skeletal muscle adaptations consequent to longterm heavy resistance exercises. *Med Sci Sport Exerc* 20:S132-S134, 1988
66. Tesch PA, Hjort H, Balduin UI: Effects of strength training on G tolerance. *Aviat Space Environ Med* 54:691-695, 1983
67. Tesch PA, Karlsson J: Muscle fiber types and size in trained and untrained muscles of elite athletes. *J Appl Physiol* 59:1716-1720, 1985
68. Tesch PA, Komi PV, Hakkinen K: Enzymatic adaptations consequent to long term strength training. *Int J Sports Med (Suppl)* 8:66-69, 1987
69. Tesch PA, Thorsson A, Kaiser P: Muscle capillary supply and fiber type characteristics in weight and power lifters. *J Appl Physiol* 56:35-38, 1984
70. Thorstensson A, Hulten B, von Döbeln W, Karlsson J: Effect of strength training on enzyme activities and fibre characteristics in human skeletal muscle. *Acta Physiol Scand* 96:392-398, 1976
71. Thorstensson A, Spodin B, Karlsson J: Enzyme activities and muscle strength after "sprint training" in man. *Acta Physiol Scand* 94:313-316, 1975
72. Upton ARM, McComas AJ, Sica REP: Potentiation of 'late' responses evoked in muscles during effort. *J Neurol Neurosurg Psychiatry* 34:699-711, 1971
73. Vogel JA: Introduction to the symposium: Physiological responses and adaptations to resistance exercise. *Med Sci Sports Exerc* 20:S131-S134, 1988
74. Young K, McDonagh MJ, Davies CT: The effects of two forms of isometric training on the mechanical properties of the triceps surae in man. *Pfluegers Arch* 405:384-388, 1985
75. Young A, Stokes M, Round JM: The effect of high-resistance training on the strength and cross sectional area of the human quadriceps. *Eur J Clin Invest* 13:411-417, 1983