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Literature Review

Performance and Physiologic Adaptations to Resistance Training

ABSTRACT

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Weight lifting, or resistance training, is a potent stimulus to the neuromuscular system. Depending on the specific program design, resistance training can enhance strength, power, or local muscular endurance. These improvements in performance are directly related to the physiologic adaptations elicited through prolonged resistance training. Optimal resistance training programs are individualized to meet specific training goals. When trained properly (i.e., similar intensity and volume), these functional and physiologic adaptations are similarly impressive among women and the aged as they are among young men. Yet, in contrast to relative measurements, sex and age differences exist in the absolute magnitude of adaptation. Of equal importance, perhaps most notably among the elderly, are the important health benefits that may also be derived from resistance training. For example, bone density, insulin sensitivity, and co-morbidities associated with obesity can be effectively managed with resistance exercise when it is conducted on a regular basis. The extent of the functional and health benefits to be accrued from resistance training depend on factors such as initial performance and health status, along with the specification of program design variables such as frequency, duration, intensity, volume, and rest intervals.

Key Words: Exercise, Muscle, Weight Lifting, Strength

Resistance training is known to be an effective method of improving the functional capacity of the neuromuscular system. Several modes of resistance training equipment are now available (e.g., free weights, machines with stacked plates, and machines with pneumatic resistance), and they all serve as an effective stimulus to the neuromuscular system. Since the pioneering work of DeLorme[1] and DeLorme and Watkins[2] in the 1940s, when the basic principles of progressive resistance training were established, resistance exercise has been utilized as an effectual rehabilitative intervention. The focus of this article, however, is on the adaptations induced by resistance training among those who have no damage or injury to the neuromuscular system.

FUNCTIONAL ADAPTATIONS

Depending on program design, resistance exercise is capable of enhancing each of the functional constituents of the neuromuscular system, that is, strength, power, and local muscular endurance. For example, strength—the maximal amount of force exerted in a single attempt—is most effectively enhanced by a program featuring high resistance and few repetitions. But because strength is the product of both the ability of the nervous system to activate high-threshold motor units, and the amount of muscle mass available to contract, a periodized method of training that incorporates planned intervals of rest is indicated.[3,4]

Early within a periodized regimen, the stimulation of muscle hypertrophy is of paramount concern. Thus, in this phase of the cycle, "high-volume" training is emphasized. That is, a resistance that corresponds to 50–75% of the maximal resistance that the individual can overcome in a single repetition, or one repetition maximum (1 RM),

should be used to complete 8–12 repetitions. A total of three to five sets of these repetitions should be executed for each exercise.[5] Subsequent to this initial phase of the periodized cycle lasting several weeks, "high-intensity" exercise, for a similar number of weeks, is featured to elicit positive adaptations within the nervous system. The total volume—amount of pounds lifted—of training sessions is reduced, but a greater percentage of the individual's 1 RM, typically 80–90%, is used during the completion of only five to six repetitions. Again, three to five sets per exercise should be performed.[5] The aforementioned is an example of the original linear approach to periodization (Fig. 1). In the more recently developed nonlinear design, hypertrophy and strength sessions are included in a single week, and these different parameters of muscle fitness are developed concurrently, rather than sequentially, as in the linear design.

The superiority of periodized training has been confirmed in tightly controlled trials,[6,7] and al-though periodized training is typically employed with larger muscles, it may also be applied to smaller muscle groups. Because of the sophistication of the periodized model of resistance training, a full discussion of this topic is beyond the scope of this review. However, an in-depth description of the periodization technique has been presented by Fleck and Kraemer.[8]

Local muscular endurance is best described as the ability to resist muscular fatigue, particularly when using a submaximal resistance. In contrast to strength, muscular endurance is optimally developed by performing a high number of repetitions (no fewer than 20) per set. Accordingly, the use of a low resistance, about 50% of the 1 RM, is recommended. In addition, a low number of sets per exercise, no more than one or two, are also indicated when training to enhance muscular endurance.

Muscular power is defined as the force applied multiplied by the velocity of movement.[9] Because work produced is equal to the force multiplied by the distance moved, and velocity is
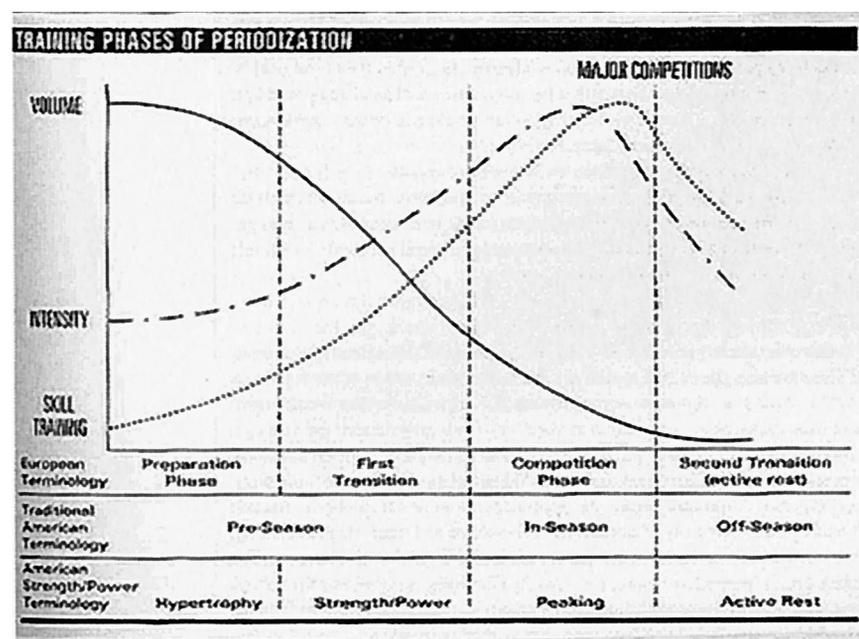

Figure 1: Linear periodization terminology. The classic interactions of the intensity and volume of exercise in a linear periodized training program. In sport, skill preparation plays an important role. Such classic models have been used around the world to develop optimal strength and power performances and have been adapted to strength fitness programs.

the distance traveled divided by the time taken, power can also be expressed as work completed per unit time (i.e., rate of performing work).[10] Thus, muscular power is a function of both strength and speed of movement. For most large muscle groups, it seems that maximal mechanical power is expressed at 30–45% of one's 1 RM.[11–13] A greater resistance than this slows the contractile speed, whereas a greater velocity of movement necessitates a considerably lower resistance. Maximal power output for a given load is the main determinant of performance in activities requiring a single movement sequence in which the goal is producing a high velocity on impact or release of an object.[14] Because many sport-specific skills, and even common everyday movements, take place in less than 0.2 msec, the ability to produce force quickly is vital not only to human performance, but also to daily activities. Indeed, it has been documented that a lack of muscle power is the main factor accounting for the high number of falls among the elderly.[15] Thus, power has many different applications, varying from everyday activities and job functions to elite athletic performance.

In attempting to optimize power, a periodized resistance training program that addresses each of the components of the power equation should be utilized. Strength training for high force development maximizes the force component, and ballistic resistance training with lighter loads (30–45% of maximal voluntary contraction) in appropriate exercises, movements, and modalities develops the time component. The key resides in proper exercise selection, which must include free-weight exercise movements that do not have a significant deceleration phase during the range of motion (e.g., power cleans, hang cleans).[16] Also effective are exercises allowing the mass to be released at the end of the range of movement, such as medicine ball ex-

ercise and computerized weight equipment that allows release of the mass, and the use of alternative modalities, including isokinetic, pneumatic, hydraulic, and plyometric training.[17] In a recent review of the principles of optimal resistance training progression, the most effective strategies of developing muscular power have been presented.[18]

For those who are interested in a moderate level of general muscle fitness as a part of an overall fitness program, without training specifically for strength, power, or local muscular endurance, the American College of Sports Medicine provides guidelines that are effective without being overly time consuming. The American College of Sports Medicine suggests as a starting point for any fitness program a total of eight to ten exercises featuring all major muscle groups be performed 2–3 days per week. For each exercise, only a single set of 8–12 repetitions is recommended (10–15 for the elderly or frail), and this program is most appropriate for previously untrained individuals during their initial 3–4 mo of training. However, it is recognized that performing multiple sets per exercise can derive additional benefits.[19] In fact, for optimal progression and maintenance of a resistance training program, more variation is needed to enhance the trainable characteristics of muscle, and if further improvements in muscle fitness are desired, multiple-set training is essential.[6,7] It is important to recognize, however, that even when using appropriate training methods, an individual's initial training status (i.e., novice or experienced lifter) influences the extent of strength gains realized (Fig. 2).

Finally, whether the goal of the resistance exercise program is to maximally develop strength or to improve general muscle fitness, both concentric (shortening) and eccentric (lengthening) muscle actions should be performed. Research has

shown that repetitions that include concentric and eccentric motions are most effective in eliciting strength gains and muscle hypertrophy.[20,21]

PHYSIOLOGIC ADAPTATIONS

Neural Adaptations. Several studies have demonstrated that early strength gains induced by resistance training are primarily due to modification of the nervous system rather than the contractile apparatus of skeletal muscle. In a commonly cited investigation, Moritani and deVries[22] found that "neural factors" accounted for the significant strength improvements observed during the first 4 wk of an 8-wk resistance-training program. After 4–6 wk of training, further strength gains were attributable mainly to muscle hypertrophy. Subsequent to this landmark study, numerous other investigators have reported similar conclusions, although specific timelines have differed. For example, Staron et al.[23] documented that in previously untrained subjects, significant muscle fiber hypertrophy was detected only after 6 wk of training, yet strength gains were evident with just 2 wk of training.[24] This suggests that adaptations of the nervous system were primarily responsible for
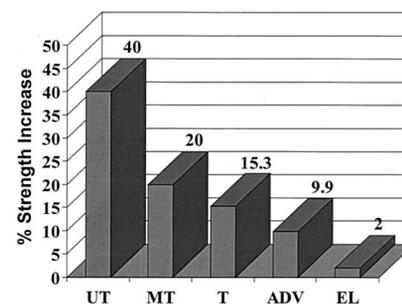

Figure 2: Effect of initial training status on resistance training–induced strength gains: data from approximately 150 studies. *UT*, untrained; *MT*, moderately trained; *T*, trained; *ADV*, advanced training; *EL*, elite training. Reproduced with permission from Kraemer et al.[18]

strength improvements during the initial 6 wk of a prolonged resistance-training protocol. Indeed, this research team demonstrated that even 8 wk of resistance training, sufficient to evoke significant strength increments, may fail to elicit myofiber hypertrophy.[24] This again suggests that initial training-induced strength increments are primarily mediated via the nervous system.

Views on the relative contributions of neural vs. muscle adaptation regarding the acquisition of strength have recently become more complex, however. More specifically, it is now believed that with prolonged resistance training, the degree of muscle hypertrophy is limited and that significant hypertrophic responses can occur only within a finite period of time lasting, perhaps, no more than 12 mo. Because strength gains continue beyond this interval, it seems that a secondary phase of neural adaptation takes place between the sixth and 12th month of training and accounts for the continued, albeit limited, strength gains exhibited by those with even extensive resistance training experience (Fig. 3).

To date, most of the available evidence regarding neural modifications consequent to strength training has been derived from surface electrode electromyography. This technique enables investigators to quantify changes in the electrical (i.e., neural) activation of skeletal muscle as a result of strength training. The technique is limited in that it does not directly distinguish between alterations in the capacity to recruit higher threshold motor units from an increased firing rate of neural impulses to the already activated motor units.[25] Either of these adaptations, or a combination of the two, would result in greater electromyographic activity within the muscle.

Numerous studies, as reviewed by Sale,[26] have confirmed that elevations in maximal force production are accompanied by increased elec-tromyographic activity of the muscle while maximally contracting. It has been postulated that the augmented electromyographic activity is the result of greater central drive from higher neural centers.[27]

Other neural adaptations elicited by resistance training include decreased co-contraction of antagonists[28,29] and an expansion in the dimensions of the neuromuscular junction, indicating greater content of presynaptic neurotransmitter and postsynaptic receptors.[30] Greater synchronicity in the discharge of motor units after strength training has also been detected.[31] Although this adaptation does not affect the maximal force developed by the muscle, it does increase the rate to peak force development and, thus, muscle power.[26,32]

Interestingly, unilateral resistance training has been found to enhance strength not only in the trained limb but also in the untrained contralateral limb, albeit to a lesser degree.[33–36] This phenomenon can be explained by modulations in the central nervous system. Since this greater central drive is not directed exclusively toward the trained limb, some of the activity is directed toward the untrained limb. Consequently, the strength improvements noted in the untrained contralateral limb are directly related to neural adaptations.[26]

Contractile Adaptations. Beyond the first few[4–8] weeks of resistance exercise among previously untrained individuals, an increased contractile capacity within muscle primarily accounts for strength improvements. The most likely explanation for this delay is the slow synthetic rate of contractile proteins (i.e., myosin and actin). In general, the assembly of muscle proteins is a deliberate process. Indeed, the turnover of muscle proteins is slower than that of the brain, liver, and even the whole body.[37] Perhaps of greater consequence is the fact that within skeletal muscle, the synthesis and accretion of contractile proteins lags behind that of other proteins, including those of the mitochondria and sarcoplasmic reticulum.[38,39]

It is the synthesis and accretion of contractile proteins that account for the hypertrophy that accompanies resistance training. This hypertrophy occurs both within the whole muscle (5–8%) and the myofibers themselves (25–35%) through the addition of intracellular myofibrils. Although there is evidence that resistance exercise may result in the formation of new myofibers, this hyperplasia contributes slightly (~5%), if at all, to the entire exercise-induced muscle enlargement.[40] In fact, a recent report indicated that the whole muscle hypertrophy demonstrated in resis-
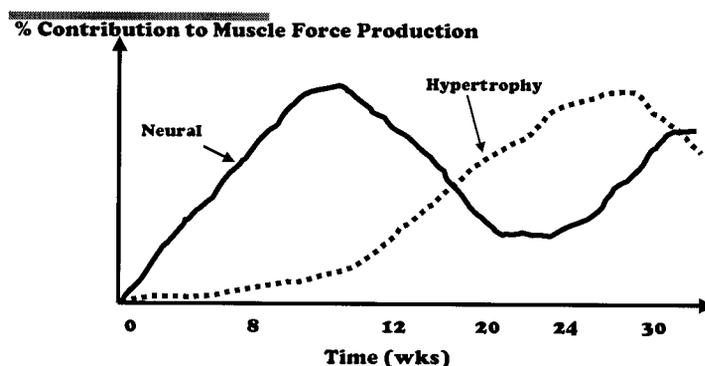

Figure 3: The interplay of neural and muscle hypertrophy factors have been hypothesized to be responsible for the changes in the dominant strategies of strength improvements over a training period.

tance-trained humans occurred in the absence of hyperplasia.[41]

Recent exciting experimental results have provided us with a greater understanding of the mechanisms involved in myofiber hypertrophy. Essential to this process is the activation of local satellite cells. These cells, first described by Mauro[42] in 1961, are myoblasts that are mitotically quiescent and located between the sarcolemma of the myofiber and its extracellular matrix. Found within this extracellular matrix is a potent mitotic cytokine, insulin-like growth factor-I (IGF-I).[43] On some sort of stress or physical disruption, IGF-I is able to interact with the nearby satellite cells, causing them to become mitotically active. The resultant sister cells are fused with the underlying myofiber adding nuclei to the existing fiber. It seems that it is the addition of these new nuclei that leads to the synthesis of additional contractile proteins, and thus hypertrophy, of the myofiber. Evidence suggests that the cytoplasmic/nuclear ratio is maintained in hypertrophied fibers,[44–46] supporting the "nuclear domain" theory as proposed by Hall and Ralston.[47] In short, in adult muscle, each nucleus maintains a small region within the myofiber, the size of which cannot vary. Consequently, to enlarge a fiber, it is necessary to first add nuclei, each of which can then regulate protein synthesis within a segment within that fiber.

Research has shown that irradiating satellite cells, thus destroying their mitotic potential, prevents the hypertrophy of overloaded myofibers.[48,49] The importance of IGF-I to the process is underscored by the fact that satellite cells isolated from skeletal muscle and its extracellular matrix resist mitotic activity until exposed to physiologic concentrations of IGF-I.[50–52]

Fiber Type–Specific Adaptations. It is well established that a prolonged program of resistance training brings about fiber type conversions within the trained muscle. The most common finding is an increase in the percentage of type IIA fibers concomitant with a decrease in the percentage of type IIB fibers,[53–55] which, in humans, have been found to predominantly express the type IIx myosin heavy chain.[56] It does not seem that resistance training significantly affects the relative contribution of type I fibers.

The changes in the fiber type profile described above were first identified with histochemical techniques used to stain myofibers according to the major isoform of myosin heavy chain expressed within. More recently, gel electrophoretic techniques, which fractionate individual myosin heavy chain isoforms within the whole muscle[24,57,58] and single myofibers,[59,60] have confirmed this pattern of fiber type conversion. These electrophoretic techniques are keenly sensitive to changes in the expression of myosin heavy chain isoforms. As evidence of this, Staron et al.[24] were able to detect remodeling of myosin heavy chain expression after only a few (<10) resistance-training sessions, when fiber type changes were not yet apparent using histochemical staining procedures. It has been suggested that these alterations in muscle protein "quality" may contribute to the strength gains made early in a training program, before hypertrophy occurs.[61]

Although resistance training promotes hypertrophy among each of the three major fiber types in humans—I, IIA, and IIB—the degree of this hypertrophy differs among those fiber types. In examining pretraining to posttraining muscle samples, it has been found that type IIA fibers display the greatest growth, followed by type IIB, with type I fibers typically exhibiting the least amount of hypertrophy.[23,24,53–55,62–64] This fiber type–specific pattern of hypertrophy is evident among both men and women. Among pretraining muscle samples, however, sex differences are apparent. In muscle cross-sections examined before resistance training, the type IIA fibers are the largest among men, whereas the type I fibers of women display the greatest size among the three categories of fibers.[65] With heavy resistance training the transition to type IIA is complete with few if any muscle fibers classified as type IIB. Thus, resistance training produces a shift in the myosin adenosine triphosphatase fiber type profile and the myosin heavy chain composition. Transitions seem to take place within the type II subtypes with training; no convincing evidence exists for detectable shifts between type I and II muscle fibers.

Neuroendocrine Adaptations. The stimulus of resistance exercise has been demonstrated to elicit acute, postexercise responses in blood-borne hormone levels and changes in basal, or resting, concentrations of hormones after prolonged, long-term training. Several studies have reported pronounced elevations in circulating testosterone levels subsequent to a resistance exercise workout.[41,66–70] This is true for both total testosterone and the unbound fraction, which is the biologically active form of this endogenous anabolic steroid. Moreover, it has been reported that long-term resistance training programs increase the blood-borne concentration of testosterone, even under resting conditions,[7,71] although a greater number of studies have failed to detect training-induced changes in basal concentrations of testosterone.[53,72–75]

Although testosterone is considered to be the principal muscle-building hormone among men, this is not the case in women. Circulating concentrations of testosterone among women are much less than those detected in men. In contrast, basal levels of growth hormone (GH) are higher in women than in men, and

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exhibit greater exercise-induced increments than those observed in men.[7,76–78] Accordingly, it has been suggested that GH, rather than testosterone, may be the most potent anabolic, or muscle building, hormone among women. But unlike testosterone, data indicate that long-term resistance-training programs do not alter basal blood-borne levels of GH.[78] In fact, small decreases in resting GH concentrations have been identified.[7] Research has determined that the extent of exercise-induced hormonal increments depends on the specific regimen of resistance exercise engaged. Regimens featuring a high volume of resistance coupled with brief rest intervals between sets—bodybuilding protocols—stimulate postexercise increases in circulating GH and testosterone that are greater in degree and duration than those observed after high-intensity lifting sessions incorporating long rest periods (i.e., power lifting protocols).[70,79,80] Moreover, new data suggest that GH responses to the exercise stress are linked to the characteristics of the muscle actions used (i.e., concentric/eccentric repetitions vs. concentric only repetitions), with GH increases specific to the type of muscle action used.[81] Other hormones, including testosterone and cortisol, do not exhibit this sensitivity to the type of muscle action performed.[81]

It may be a caveat that the majority of the exercise studies to date have made use of the immunoassay as a means of measuring circulating GH concentrations, despite the fact that often there is variability in hormone levels detected when different immunoassays are employed. In part, this can be explained by the epitope specificity of different antibodies used in various assays.[82] Furthermore, since the standard commercial immunoassay measures only the 22 kD GH monomer, this assay may neglect many of the GH isoforms that are cosecreted by the pituitary gland, in-

cluding GH fragments, dimers, and oligomers. As a result, the molecular nature of exercise-induced GH release is largely unknown. Baumann[83] offered the first insights into the size variants of GH found in the human circulation, and the manner in which these variants respond to an acute resistance exercise stimulus is only beginning to be revealed.[77]

In addition to its function as a mitogen, IGF-I directly acts as an anabolic agent on skeletal muscle. In this function, IGF-I seems to act primarily via autocrine and paracrine mechanisms rather than in a classical endocrine manner. Mainly it achieves this by decelerating the rate of proteolysis that naturally, and continuously, occurs within the tissue.[43,84] Several organs produce IGF-I, but the principal source for the IGF-I found in the bloodstream is probably the liver.[84] However, there have been reports that muscle tissue itself synthesizes and secretes IGF-I during contractile activity.[85,86] In fact, it has recently been reported that a whole family of IGF peptides exists in skeletal muscle, including mechano-growth factor, which demonstrates autocrine characteristics.[87]

Overall, the evidence regarding the acute effects of resistance exercise on blood-borne IGF-I are equivocal, with some studies reporting significant postexercise elevations[68,70,79] and others failing to note any alterations.[76,88,89] Because IGF-I affects anabolic responses through autocrine, paracrine, and endocrine mechanisms, circulating responses of IGF-I may not accurately reflect the overall influence it exerts on muscle metabolism after exercise.

Most investigations have concluded that prolonged resistance training programs do not affect basal concentrations of circulating IGF-I.[41,67,76,90,91] However, two recent studies reported significant increases in resting serum IGF-I levels after 12–13 wk of training.[7,92] Representative findings of the acute

responses and adaptations of basal levels of blood-borne anabolic hormones to resistance exercise or training are presented in Tables 1 and 2, respectively.

Cortisol, a glucocorticoid, is the major catabolic, or muscle wasting, agent among both men and women. This catabolic influence is due to the combined effect of muscle protein degradation and an inhibition in protein synthesis.[93] This anti-anabolic characteristic of cortisol is principally related to its attenuation of the impact of hormones such as testosterone, GH, and even insulin on skeletal muscle.[94–96] Interestingly, the overall catabolic processes of glucocorticoids, although evident in both fast- and slow-twitch muscle, are more pronounced among fast-twitch (type II) myofibers.[97]

Because cortisol is also considered a "stress" hormone,[98] blood-borne levels of cortisol are acutely amplified after a resistance exercise session.[66,78,99–101] However, several researchers have reported that participating in a long-term resistance-training program attenuates basal, or resting, levels of circulating cortisol.[7,67,74,78] The final outcome of this adaptation, of course, would be a hormonal environment more conducive to muscle hypertrophy. The findings of reduced basal concentrations of cortisol are not necessarily typical. Several authors have reported no change in resting cortisol after a prolonged program of resistance training.[41,72,75,76] It seems that variables such as age, gender, and, in particular, program design account for these disparate results.

A considerable body of literature has been accrued regarding exercise-induced alterations in blood-borne levels of hormones. We have previously presented more thorough reviews of these adaptations.[102,103] In contrast to changes in circulating levels of hormones, our understanding of the modifications made in the target tissue—skeletal muscle—in

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TABLE 1

Acute responses of circulating anabolic hormone concentrations to resistance exercise in young adults

Reference	Sex	Protocol (Sets × Reps)	Response, %
Testosterone			
Cumming et al.[99]	Females	18 × 10	↑ 20
Deschenes et al.[66]	Males	8 × 5/50	↑ 9
Guezennec et al.[100]	Males	15 × 8	↑ 3
Hakkinen et al.[145]	Males	4 × 10	↑ 27
Hakkinen & Pakarinen[157]	Females	5 × 10	No change
Jensen et al.[69]	Males	3 × 10	↑ 27
Kraemer et al.[158]	Males	4 × 10	↑ 38
Kraemer et al.[70]	Males	5 × 5	↑ 29
Kraemer et al.[70]	Males	3 × 10	↑ 49
Kraemer et al.[70]	Females	5 × 5	No change
Kraemer et al.[70]	Females	3 × 10	No change
Kraemer et al.[53]	Males	2–5 × 5–15	↑ 30
Kraemer et al.[67]	Males	4 × 10	↑ 19
Growth hormone			
Hakkinen et al.[145]	Males	4 × 10	↑ >1000
Hakkinen & Pakarinen[157]	Males	5 × 10	↑ >1000
Hakkinen & Pakarinen[157]	Females	5 × 10	↑ 225
Kraemer et al.[79]	Males	3 × 10	↑ 800
Kraemer et al.[79]	Males	5 × 5	↑ 300
Kraemer et al.[159]	Males	4 × 10	↑ 325
Kraemer et al.[78]	Males	3 × 6–8	↑ 600
Kraemer et al.[78]	Females	3 × 6–8	↑ 300
Kraemer et al.[158]	Males	4 × 10	↑ 700
Kraemer et al.[70]	Males	5 × 5	No change
Kraemer et al.[70]	Males	3 × 10	↑ >1000
Kraemer et al.[70]	Females	5 × 5	No change
Kraemer et al.[70]	Females	3 × 10	↑ 110
Kraemer et al.[67]	Males	4 × 10	↑ 425
IGF-I			
Kraemer et al.[89]	Males	3 × 10	No change
Kraemer et al.[67]	Males	4 × 10	No change
Kraemer et al.[70]	Males	5 × 5	↑ 26
Kraemer et al.[70]	Males	3 × 10	↑ 11
Kraemer et al.[70]	Females	5 × 5	No change
Kraemer et al.[70]	Females	3 × 10	↑ 14
Nindl et al.[89]	Males	5 × 5/10	No change

Reps, repetitions; IGF-I, insulin-like growth factor I.

response to resistance training is very limited. However, there is a report that resistance training alters the binding characteristics of skeletal muscle to anabolic steroids and that these modifications are fiber type specific. In that study,[104] it was determined that the soleus, which has primarily (~85%) type I myofibers, responded to an 11-wk program of resistance training by decreasing its maximal binding capacity for an anabolic steroidal hormone. On the other hand, the predominantly (~95%) type II extensor digitorum longus muscle demonstrated a significantly enhanced binding capacity for the anabolic steroid. Since in both muscles hormonal binding affinity was unaffected by resistance training, it was concluded that a down-regulation (soleus) or an up-regulation (extensor digitorum longus) in the number of receptors accounted for the observed alterations in maximal steroid binding capacity.

Bioenergetic Adaptations. Most of the available evidence suggests that the phosphagen (adenosine triphosphate and phosphocreatine) content of muscle remains unaffected by resistance training.[54,105] As would be expected, activities of the enzymes involved in phosphagen metabolism (i.e., myokinase and creatine kinase) also do not increase as a result of extended resistance-training programs.[106,107]

Muscles engaged in long-term resistance training may[108] or may

TABLE 2

Chronic adaptations in basal concentrations of circulating anabolic hormones after resistance training in young adults

Reference	Sex	Duration of training	Adaptation, %
Testosterone			
Alen et al.[74]	Males	24 wk	No change
Hakkinen et al.[72]	Males	1 yr	No change
Hakkinen et al.[75]	Females	16 wk	No change
Kraemer et al.[53]	Males	12 wk	No change
Kraemer et al.[78]	Males	8 wk	↑ 27
Kraemer et al.[78]	Females	8 wk	↑ 100
Kraemer et al.[158]	Males	10 wk	No change
Kraemer et al.[159]	Males	10 wk	No change
Marx et al.[7]	Females	12 wk (periodized)	↑ 27
Marx et al.[7]	Females	12 wk (single-set)	↑ 28
Marx et al.[7]	Females	24 wk (periodized)	↑ 45
Marx et al.[7]	Females	24 wk (single-set)	↑ 22
McCall et al.[41]	Males	12 wk	No change
Growth hormone			
Kraemer et al.[78]	Males	8 wk	No change
Kraemer et al.[78]	Females	8 wk	No change
Marx et al.[7]	Females	12 wk (periodized)	No change
Marx et al.[7]	Females	12 wk (single-set)	No change
Marx et al.[7]	Females	24 wk (periodized)	No change
Marx et al.[7]	Females	24 wk (single-set)	No change
McCall et al.[41]	Males	12 wk	No change
IGF-I			
Borst et al.[92]	Males/females	13 wk (multi-set)	↑ 18.5
Borst et al.[92]	Males/females	13 wk (single-set)	↑ 20.5
Kraemer et al.[158]	Males	10 wk	No change
Kraemer et al.[159]	Males	10 wk	No change
Marx et al.[7]	Females	12 wk (periodized)	↑ 33
Marx et al.[7]	Females	12 wk (single-set)	↑ 13
Marx et al.[7]	Females	24 wk (periodized)	↑ 43
Marx et al.[7]	Females	24 wk (single-set)	↑ 40
McCall et al.[41]	Males	12 wk	No change

IGF-I, insulin-like growth factor-I.

not[105] display greater glycogen content than they did in the pretraining state. However, phosphorylase and phosphofructokinase, enzymes essential to the energy-producing pathway of glycolysis, have consistently been found to remain unaffected by resistance training.[33,105,106,109]

Lipid depots, used during oxidative metabolism, do not seem to be increased, and may even be decreased, in skeletal muscle after resistance training.[110,111] Enzymes involved in oxidative metabolism, whether the substrate is of carbohydrate or lipid origin, generally show no adaptation in resistance-trained muscle. Specifically, citrate synthase[106] and succinate dehydrogenase[62] have failed to demonstrate increments and may, in fact, be diminished after prolonged resistance training.[112,113] The subcellular organelles housing these enzymes, the mitochondria, have been found to be reduced in density within resistance-trained muscle.[110] This result is probably due to the cellular hypertrophy associated with resistance exercise, thus causing a dilution effect on the resident mitochondria.[110,112]

These findings, which indicate a lack of improved oxidative capacity, substantiate the lack of enhanced myoglobin content in resistance-trained muscle. Indeed, the data suggest that myoglobin content may even be depressed as a result of resistance training.[107,113]

Cardiovascular Adaptations. Research indicates that a long-term regimen of resistance training may either improve or diminish the capillarity of muscle. Generally, when capillarity is quantified as capillary density, resistance training imparts a negative adaptation.[114] Capillary density is determined as the number of capillaries identified within a given area of tissue. Due to the myofiber

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hypertrophy evoked by resistance training, fewer fibers, and thus fewer capillaries, occupy the same area as before training. As with the decrease in mitochondrial density, the decrement in capillary density observed in resistance-trained muscle results from a dilution effect.

In contrast, when capillarity is assessed as the number of capillary contacts per myofiber, or capillary/fiber ratio, it seems that resistance training has a beneficial impact.[41,62,115] It has been postulated that this improved capillarity may be required to clear the lactate produced by muscle during resistance exercise.[61]

Consistent with the fact that resistance training fails to elicit adaptations in the capacity for oxidative metabolism within muscle, whole body maximal oxygen uptake does not respond to most resistance-training protocols.[53,116,117] The lone exception to this seems to be circuit weight training, in which the individual proceeds immediately from exercise to exercise with virtually no rest intervals between sets. Previously untrained subjects participating in this type of training display improvements in maximal oxygen uptake of up to 12%.[118–120] However, properly designed endurance-training programs featuring running or cycling exercise result in elevations of maximal oxygen uptake of up to 30%.[19]

Although resistance training, excepting circuit training, does not improve maximal oxygen uptake, perhaps this is not the most relevant measure of the cardiovascular benefits derived from this mode of training. In particular, it is quite conceivable that improving one's strength results in a muted cardiovascular strain when performing a given task requiring muscular exertion. If so, resistance training may convey a positive cardiovascular adaptation during the execution of normal daily activities. In fact, it has been demonstrated among older individuals that strength training lessens the cardiovascular stress—heart rate and blood pressure increases—during tasks such as walking, weight-loaded walking, and stair climbing, even in the absence of increases in maximal oxygen uptake.[121,122] This method of detecting the favorable adaptations of resistance training merits further investigation, especially among younger people with and without known cardiovascular conditions.

Blood Lipid Adaptations. Numerous studies have investigated the effects of resistance training on blood lipid profiles (e.g., triglycerides, total cholesterol, and high density lipoproteins). The findings sometimes conflict, and they are dependent on variables such as initial health status, age, and sex and the type and length of the training program. As a result, a recently published meta-analysis of the available literature on this topic summarized that no firm conclusions can be drawn regarding the impact of resistance training on blood lipid profiles.[123]

Body Composition Adaptations. Among the previously untrained, moderate, yet significant, positive adaptations in body composition have been derived from resistance training. This is particularly true when the program includes high-volume training with brief, between-set rest intervals (i.e., bodybuilding workouts). These changes include an increase in fat-free mass[108,124–126] and a reduced skinfold thickness, or subcutaneous fat.[23,55,127] These adaptations have been observed in men and women and among young and older individuals. A more thorough review of the body composition adaptations accompanying prolonged resistance training is presented in Fleck and Kraemer.[128]

DIFFERENCES BETWEEN SEXES

Early attempts to assess the efficacy of resistance training in women concluded that, relative to men, moderate strength gains were acquired, but in the absence of muscle hypertrophy.[129–131] The lack of hypertrophy was posited to be due to the low levels of testosterone produced by women. However, more recent studies that used training protocols equal in volume, intensity, and duration to those typically employed by men confirmed that relative strength gains—percentage increase from pretraining values—were the same in women as in men.[24,29,132,133] Research has also revealed that women undergo a similar degree of myofiber and whole-muscle hypertrophy as that observed in men.[23,55,134,135] Despite this cellular and whole-muscle hypertrophy, limb circumference, used to determine hypertrophy in early studies reporting an absence of increased muscle size,[129,130,131] was not increased in these recent studies because skinfold thickness had been reduced with training. In general, it seems that although women have less initial strength and smaller myofibers than men, relative increments in strength and fiber size induced by resistance training are similar in men and women if the same exercise stimulus is presented.

ADAPTATIONS IN THE AGED

Demographic data clearly illustrate that, overall, the United States population is growing older.[136] This "graying" of America has stimulated an increase in research examining the potential of older ($\geq$60 yr old) individuals to adapt to exercise, including resistance training. In a pioneering investigation, Frontera et al.[115] found that older men displayed impressive strength gains that were, relative to initial levels, similar to those quantified in younger subjects if the training stimulus—volume, intensity, and frequency—was the same as that presented to younger individuals. Subsequently, numerous inves-

tigations have confirmed the trainability of the older neuromuscular system exposed to resistance exercise.[28,137–139] This is evident among both men and women,[29,133,140,141] and even among people in their 90s.[142] It should be noted, however, that when directly comparing young and aged subjects, it seems that strength gains are more pronounced among the young.[133,143] However, these age-related differences regarding strength improvements were no longer evident when strength was expressed relative to whole-muscle size.[133,143]

Several studies have determined that strength improvements detected among the aged are coupled with cellular[115,137,139,144] and whole muscle-hypertrophy.[28,115,137,142,144] Also, relative to pretraining values, muscle hypertrophic responses to resistance training have been found to be indistinguishable between young and elderly people.[28,134] This is true despite the fact that the responses of anabolic and catabolic hormones to resistance exercise differ between young and aged subjects.[67,145]

The health benefits derived from resistance training may be even more impressive among the aged than in younger people. For example, resistance training, and the muscle mass that it builds, promotes greater insulin sensitivity and blood glucose tolerance among the elderly.[146–148] Resistance training has also been found to maintain bone density and health, and it thus serves as a useful prophylactic and treatment for osteoporosis.[149–151] Similar to the adaptations noted among the young, resistance training improves body composition among older individuals[28,121,127,152–154] and, by extension, manages co-morbidities associated with obesity such as hypertension, type II diabetes, and coronary artery disease. Indeed, the positive effects of resistance training on many of the health problems typically observed among the aged have recently been reviewed.[155,156]

CONCLUSIONS

The popularity of resistance training has grown immensely over the last 20–25 yr. This growth can be attributed to the increasing numbers of people other than highly trained athletes who are participating in this form of exercise. This change in the profile of those who regularly resistance train is related, at least in part, to the development of new types of equipment (i.e., machines) that are viewed as safer and less intimidating than traditional free weights. Moreover, both free weights and resistance exercise machines have become far more accessible to the general public.

Recent research has demonstrated that not only is resistance training an effective method to improve the function of the neuromuscular system, it can be equally effective in maintaining or improving one's health. These positive adaptations in performance and health can be realized among both men and women and among the young and the old. However, it must be appreciated that initial training and health status, along with the specific design of the training program—intensity, volume, frequency, duration—will affect the magnitude of the performance and physiologic adaptations derived from resistance exercise. Finally, it must be realized that all individuals bring different sets of backgrounds to any training program (e.g., genetic predispositions, training backgrounds, health status, sex, age) and, therefore, physiologic strategies available for adaptation to any resistance training program. Therefore, the use of individualized exercise prescriptions with very specific training targets and goals will represent the most optimal methods to achieve desired training results and physiologic adaptations.

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