ResearchGate

See discussions, stats, and author profiles for this publication at: https://www.researchgate.net/publication/6562181

The adaptations to strength training: Morphological and neurological contributions to increased strength

Article *in* Sports Medicine · February 2007

Source: PubMed

CITATIONS
488

READS
7,083

2 authors:

Jonathan P. Folland
Loughborough University
116 PUBLICATIONS **2,910** CITATIONS
SEE PROFILE

Alun G Williams
Manchester Metropolitan University
104 PUBLICATIONS **3,421** CITATIONS
SEE PROFILE

Some of the authors of this publication are also working on these related projects:

Genetics of Sarcopenia View project

Osteoporosis View project

All content following this page was uploaded by Alun G Williams on 01 April 2015.

The user has requested enhancement of the downloaded file.

The Adaptations to Strength Training: Morphological and Neurological Contributions to increased Strength.

Folland, J.P. & Williams, A.G.

Sports Medicine (2007)

The final publication is available at Springer via *http://dx.doi.org/10.2165/00007256-200737020-00004*

The Adaptations to Strength Training: Morphological and Neurological Contributions to increased Strength.

Folland, J.P.[1] & Williams, A.G. [2]

[1] School of Sport and Exercise Sciences, Loughborough University, U.K.

[2]Institute for Biophysical and Clinical Research into Human Movement, Manchester Metropolitan University, UK.

Corresponding author:

Dr Jonathan P. Folland, School of Sport and Exercise Sciences, Loughborough University,

Ashby Road, Loughborough, Leicestershire, LE11 3TU, U.K.

Telephone: + 44 (0) 1509 226334

Fax: + 44 (0)1509 226301

E-mail: j.p.folland@lboro.ac.uk

Running title: Strength Training: Morphological and neurological adaptations.

Abstract

High resistance strength training (HRST) is one of the most widely practiced forms of physical activity, that is used to enhance athletic performance, augment musculo-skeletal health and to alter body aesthetics. Chronic exposure to this type of activity produces marked increases in muscular strength that are attributed to a range of neurological and morphological adaptations. This review assesses the evidence for these adaptations, their interplay and contribution to enhanced strength and the methodologies employed.

The primary morphological adaptations involve an increase in the cross-sectional area of the whole muscle and individual muscle fibres that is due to an increase in myofibrillar size and number. Satellite cells are activated in the very earliest stages of training, and their proliferation and later fusion with existing fibres appears to be intimately involved in the hypertrophy response. Other possible morphological adaptations include hyperplasia, changes in fibre type, muscle architecture, myofilament density, and the structure of connective tissue and tendon.

Indirect evidence for neurological adaptations that encompass learning and co-ordination comes from the specificity of the training adaptation, transfer of unilateral training to the contralateral limb and imagined contractions. The apparent rise in whole muscle specific tension has been primarily used as evidence for neurological adaptations, but morphological factors (preferential hypertrophy of type II fibres, increased angle of fibre pennation, increase in radiological density) likely also contribute to this phenomenon. Changes in inter-muscular co-ordination appear critical. Adaptations in agonist muscle activation assessed with electromyography, tetanic stimulation and the twitch interpolation technique suggest small but significant increases. Enhanced firing frequency and spinal reflexes most likely explain this improvement, although there is contrary evidence suggesting no change in cortical or corticospinal excitability.

The gains in strength with HRST are undoubtedly due to a wide combination of neurological and morphological factors. Whilst the neurological factors may make their greatest contribution during the early stages of a training programme hypertrophic processes also commence at the onset of training.

Contents

1. Introduction

High resistance strength training (HRST) is one of the most widely practiced forms of physical activity. In the early weeks of a resistance training programme voluntary muscle strength increases significantly and these gains continue for at least 12 months.[1] This type of exercise is used to enhance athletic performance, augment musculo-skeletal health and to alter body aesthetics. The health benefits of HRST are primarily as a countermeasure to any circumstance where muscle weakness compromises function (i.e. sarcopenia, neuromusculo-skeletal disorders, or following immobilization, injury or prolonged bed rest), but it also has a positive influence on metabolic and skeletal health. Whilst HRST is most readily associated with athletic events requiring strength and power, it has also been found to benefit endurance performance.[2] Thus the adaptations to this type of activity are of considerable interest. This review addresses the morphological and neurological adaptations to high resistance strength training, assessing the evidence for these adaptations, their interplay and contribution to enhanced strength and the methodologies employed.

2. Morphological Adaptations

2.1 Changes in Whole Muscle Size

It is a matter of common observation that regular high resistance activity causes a substantial increase in muscle size after a few months of training, and this has been extensively documented in the scientific literature. Investigations employing a range of scanning techniques (magnetic resonance imaging, MRI; computerized tomography, CT and Ultrasound) have typically found significant increases in muscle anatomical cross-sectional area (ACSA) over relatively short training periods (8-12 weeks[3-6]). MRI is regarded as the superior method of determining muscle ACSA, due to its greater resolution,[7] and has been used increasingly in the last decade. In a careful longer duration study Narici et al.[8] examined changes in muscle strength, ACSA (with MRI) and agonist muscle activation (with Electromyography, EMG) over 6 months of standard

heavy resistance training (Figure 1). They demonstrated that whole muscle hypertrophy evolved essentially in a linear manner from the onset of the training, with no indication of a plateau in this process after 6 months of training. Furthermore, after the first 2 months of training quadriceps strength and cross-sectional area (CSA) appeared to increase in parallel. It is intuitive that the growth of skeletal muscle must slow or plateau eventually. Quantitative evidence comes from a training study by Alway et al.[9] with experienced bodybuilders (>5 years training experience). They found no change in biceps brachii ACSA or fibre area with 24 weeks of strength training.

Another common observation with HRST is the disproportionate increase in muscle strength than ACSA, indicating an increase in specific tension. Whilst of interest there are numerous methodological problems with the direct comparison of these measurements, mainly involving the methodology of muscle size measurement. The vast majority of investigations have measured ACSA often at just one level as the index of muscle size. A recent reliability study of muscle size measurement concluded that CSA measured at just one level was less reliable than measurement of multiple sections and should only be used if a relatively large effect size is expected.[10] Theoretically, physiological CSA (PCSA) measured perpendicular to the line of pull of the fibres would seem a more valid index of the muscle's contractile capability. However, the precise measurement of PCSA is problematic,[11] requiring the measurement of muscle volume and the angle of fibre pennation as well as estimation of fibre length.[12] Alternatively, some studies have measured changes in whole muscle volume with MRI after resistance training (+14%, 12 weeks of elbow flexor training[13]; +9.1%, 12 weeks of first dorsal interosseous training[14]; +12%, 9 weeks of quadriceps training[5]; +10%, 14 weeks of quadriceps training[15]). The question of which of these measures of muscle size is the most valid indicator of muscular strength is disputed. Bamman et al.[16] concluded that ACSA and PCSA were more strongly correlated with strength performance, however, Fukanaga et al.[17] reported higher correlations for PCSA and muscle volume with peak joint torque, than for ACSA.

A further confounding factor is that muscle size measurements in relation to HRST have, to date, only been recorded in the passive state. Even during an isometric contraction, the contractile elements shorten and there can be considerable changes in

muscle morphology and the mechanics of the musculo-skeletal system.[18,19] For example, as the medial gastrocnemius changes from rest to a maximum voluntary contraction at a fixed position (isometric) the angle of muscle fibre pennation doubles and the PCSA increases by 35%.[20]

Various indices of muscle size (ACSA, PCSA or muscle volume) assessed by MRI show significant changes after 8-12 weeks of regular training, and this adaptation appears to proceed in a linear manner during the first 6 months of training. Unfortunately the most valid muscle size indicator of strength is unclear and the confounding issue of size measurements taken at rest has not been addressed.

2.1.1 Influence of Muscle Group

A greater hypertrophic response to resistance training has been observed in the upper body muscles compared to lower extremity muscles in previously untrained individuals.[21,22] When standard training was utilized, Welle et al.[23] found ACSA of the elbow flexors to increase by 22 and 9%, for young and old subjects respectively, whereas knee extensor ACSA increased by only 4 and 6%. A recent comparison of changes in muscle thickness (assessed by ultrasound) found a greater response to standard training for a range of upper body muscles compared to lower limb muscles.[6] A possible explanation for this is that lower limb muscles, particularly the anti-gravity quadriceps femoris and triceps surae, are habitually activated and loaded to a higher level during daily living activities than the upper body musculature,[22] and thus respond less to a given overload stimulus. An alternative explanation is intermuscular differences in androgen receptor content with some evidence for greater concentration in upper body than lower limb muscles.[24]

2.1.2 Influence of Sex

On average the skeletal muscle of women typically has 60-80% of the strength, muscle fibre CSA and whole muscle ACSA of men.[25-28] Therefore it is not surprising that the absolute changes in strength and muscle size after training are smaller in women[22] and in proportion to their smaller dimensions.[29] The lower blood androgen levels of women has also been hypothesized to cause less relative muscle hypertrophy in

response to training compared to men.[30-32] For lower body training a number of studies have failed to find any difference between males and females with similar relative improvements both in terms of hypertrophic and strength adaptations after HRST.[6,22,33-37] For example, Tracy et al.[5] compared the hypertrophic response of the quadriceps of older men and women, finding an identical 12% increase in muscle volume after 9 weeks of training. In contrast, results for upper body training indicate there may be sex-mediated differences in the response to HRST.[38-40] A recent large scale trial of 342 women and 243 men found greater increases in muscle ACSA in men (with MRI, +2.5%), but greater increases in strength in women (1-Repetition Maximum, +25%; Isometric, +6%) after 12 weeks of identical training.[39] Potentially, the greater hypertrophy of males following upper body training might be due to the greater androgen receptor content of these muscles[41] making them more responsive to higher blood androgen concentrations. The greater strength gains of females might reflect a greater capacity for neural adaptations,[42] perhaps due to less exposure and propensity towards upper body strength and power tasks that are not part of daily life in the untrained state.

2.1.3 Influence of Age

There is no doubt that older adults, including nonagenarians, do undergo skeletal muscle hypertrophy in response to HRST (mid thigh ACSA: +9% after 8 weeks[43]; +9.8% after 12 weeks[44]). The absolute increase in muscle size is smaller in old compared to young adults, likely due to the smaller size of a typical older adult's muscles.[23] Some comparative studies suggest that the relative change in muscle volume or ACSA in response to HRST is not affected by age,[34,45] whilst others seem to suggest a smaller hypertrophy response in older individuals.[14,23,46] The variability in findings is most likely accounted for by the low subject numbers of these studies and the large inter-individual variation in response to HRST. [39]

2.1.4 Selective Growth (Hypertrophy)

The extent of whole muscle growth has been found to vary within the constituent muscles of a muscle group as well as along the length of each constituent muscle.[4,8,47,48]

For example, Housh et al.[4] reported average hypertrophy of 23.2% for the rectus femoris as opposed to 7.5% for the vastus lateralis (Figure 2), and Narici et al.[8] found rectus femoris hypertrophy to vary from <10 to >50% at different lengths along the muscle. These authors went on to suggest that the hypertrophy of each component muscle may largely depend upon the extent of their loading and activation, which seems likely to be governed by the mechanics of each constituent muscle in relation to the training exercise(s). For example the four constituents of the knee extensors (quadriceps) each likely have different length-tension relationships and thus different contributions to torque production at any given joint angle.

Some studies have found the greatest hypertrophic response of the whole quadriceps or biceps brachii muscles to be in the region of maximum girth/CSA (e.g. mid thigh)[5,13,49]whilst others have found this to occur in proximal[47] or proximal and distal[8] regions of the muscle, possibly due to differences in the exercises prescribed. There is evidence that this phenomenon of selective growth can continue for an extended period of time. In experienced junior Weightlifters (average age 16.4 years) followed over a further 18 months of training quadriceps ACSA increased by 31% at 30% femur length from the knee (Lf) but with no change at 50 or 70% Lf.[50] From a measurement perspective selective growth suggests that multiple slice MRI scanning may be required to accurately quantify the growth of muscle tissue. Theoretically, muscle growth can be achieved either by an increase in the CSA of muscle fibres (fibre hypertrophy), an increase in the number of fibres (fibre hyperplasia) or an increase in the length of fibres that do not initially run the length of the muscle.

2.2 Muscle Fibre Adaptations

An increase in the cross-sectional area (CSA) of skeletal muscle fibres is generally regarded as the primary adaptation to long-term strength training and has been widely documented (Reviewed by: [51,52]). It is thought to account for the increase in muscle CSA, facilitating the increase in the contractile material (number of cross-bridges) arranged in parallel and thus force production. Changes in fibre CSA in humans can only be evaluated by taking biopsy samples of skeletal muscle. Widely varying changes in mean fibre area in response to HRST have been reported. Training the triceps brachii for

six months resulted in type I and II fibre hypertrophy of 27 and 33%, respectively.[53] Aagaard et al.[11]found a mean 16% increase in fibre area after 14 weeks of resistance training, and the change in fibre area with training correlated significantly with the increase in muscle volume. Whilst the vast majority of studies have found significant increases in fibre CSA, Narici et al.[8] found no change in mean fibre area despite muscle ACSA increasing by 19%. Such variability may be accounted for by a number of factors, including the poor reproducibility of the biopsy technique, the individual's responsiveness to training, and the precise nature of the training stimulus (muscle length, type and velocity of contraction, work intensity and duration). The poor repeatability of fibre area measurements with a single biopsy sample has been well documented (COV = 10-24%).[54-58] This appears to be largely due to heterogeneity of fibre size within skeletal muscle, which may be partially influenced by depth of the biopsy site,[59] as well as variability in perpendicular slicing of muscle tissue and tracing of cell borders.[57] Thus while the weight of evidence strongly supports fibre hypertrophy, data from single biopsy samples must be treated with caution.[60]

2.2.1 Preferential Hypertrophy of Type II Fibres

Preferential hypertrophy of type II fibres after strength training is another commonly reported finding.[61-64] The data presented by Hakkinen et al.[65] indicate a greater plasticity of type II fibres since they hypertrophy more rapidly during training and atrophy faster during detraining. It is not surprising therefore that many of the shorter studies (6-10 weeks) have only found significant hypertrophy of type II fibres,[11,64,66,67] whereas longer studies have more frequently found significant increases in the fibre area of both type I and II fibres.[53,65] The evidence from animal work supports the greater hypertrophic response of type II fibres.[68] The proportion of type II fibres in human muscle has been significantly correlated with training-induced hypertrophy[46] and increases in strength.[66] However, strength gains have also been found not to be related to fibre composition[69] and positively related to the proportion of type I fibres.[64]

It has been suggested that type II fibres have a higher specific tension and their preferential hypertrophy contributes to the rise in the specific tension often observed for

the whole muscle with training. However, there has been considerable debate about the specific tension of different fibre types. A review by Fitts et al.[70] concluded there were no significant differences in specific tension between fibre types in rat or human muscle. In contrast, more recent work suggests greater specific tension of human fibres expressing MHC IIX isoform than fibres expressing purely MHC I (+50%[71]; +20%[72]; +32%[73]). Studies that have related isometric specific tension to the fibre type composition of humans in-vivo have found contradictory findings.[74-76] However, the proportion of type II fibres (or MHC II content) has been positively correlated with isokinetic strength at medium to high angular velocities[77] and relative force at high velocities.[74,78]

Recent evidence suggests that type II fibres have a significantly greater specific tension that, in combination with their greater hypertrophy response likely contributes to increases in whole muscle specific tension.

2.3 Myofibrillar Growth and Proliferation

MacDougall and colleagues[53] examined the myofibrillar structure of 6 subjects before and after 6 months of strength training. Despite wide variations in size, measurement of over 3,500 myofibrils in each condition revealed a significant increase in myofibrillar CSA (16%, $p<0.01$) coincident with a 31% increase in mean fibre area. The methodology of this study was extremely thorough, and their findings reinforced some earlier work of this group.[79] The packing density of the myosin filaments within the myofibril was also investigated at the centre and periphery of ~500 myofibrils per subject. The packing density was extremely consistent within subjects, between conditions and within each myofibril, suggesting myofilament density was unchanged throughout myofibrils as well as being unresponsive to training. A three-fold increase in the number of myofibrils with 'splits' after training was also observed, perhaps indicating longitudinal division of myofibrils post-training.

The uniformity of myosin filament density throughout the myofibril indicated that myofibrillar growth was due to the addition of contractile proteins to the periphery of a myofibril. Furthermore, labelling studies have indicated that newly formed proteins tend

to be found around the periphery of existing myofibrils.[80] The increase in myofibrillar CSA clearly contributes to the increase in muscle fibre area, however, the disproportionately greater increase in fibre CSA (two fold more than myofibrillar area) suggests an additional adaptation. Given the consistency of the myosin filament packing and the increased number of myofibrils with 'splits' after training, the data of MacDougall et al.[53] is interpreted as evidence for an increase in myofibril number, proliferation, after training.

2.3.1 A Possible Mechanism of Myofibrillar Proliferation

The investigations by MacDougall and colleagues[53,79] indicate that myofibrillar growth and proliferation are the central morphological changes responsible for work-induced muscular growth in humans. During normal growth of mammalian muscle myofibrillar number has been found to increase by as much as 15-fold.[81] In a series of investigations on the growth of post-natal mice, Goldspink[81-83] proposed a mechanism for myofibrillar proliferation. Discrepancy in the arrays formed at the A and I bands causing the actin filaments to pull at a slightly oblique angle at the Z-disks. As myofibrillar size increases the peripheral filaments will be subjected to a greater lateral displacement between the A band and Z-disk, and will pull with increasing obliquity (Figure 3). Goldspink proposed that if this were developed sufficiently in two half sarcomeres it could cause the Z-disk to rupture.

Once one Z-disk has ruptured the next Z-disk in series may split in a similar manner until the entire myofibril has divided longitudinally. Evidence for myofibril splitting and z-disk rupture leading to myofibrillar proliferation has also been found in growing avian and fish muscle.[84,85] Thus in response to growth, and likely also HRST, myofibrillar proliferation takes place due to z-disk rupture and longitudinal division, that limits myofibrillar size and facilitates their effective control and regulation.

2.3.2 Satellite Cells

Many investigators have found that the ratio of nuclear: cytoplasmic material remains fairly constant throughout a wide range of growth conditions (in animals[86,87]; in

humans[88,89]). In human muscle, Landing and colleagues[90] found a direct correlation between the number of myonuclei and fibre diameter. Hence it seems that a single myonucleus may only be able to maintain a fixed volume of cytoplasmic material, and this ratio appears to be about twice as high for type II as type I fibres.[89]

Animal work has shown that, during normal growth and maturation, the increase in muscle fibre size is due to the addition of new nuclei originating from satellite cell populations.[86,87] Unlike the myonuclei inside the fibre, satellite cells, situated beneath the basal lamina that surround each fibre, can undergo mitosis and typically one of the daughter cells then becomes a true myonucleus.[91] New myonuclei derived from satellite cells, whilst no longer capable of dividing, begin to produce muscle specific proteins that increase fibre size.[92,93] In overloaded adult cat muscle, Allen et al.[94] found the increase in myonuclear number more than matched the increase in fibre volume. Rosenblatt and associates[95-97] studied changes in adult mammalian skeletal muscle in response to loading with an ablation model and reported significantly less hypertrophy following prior irradiation of the muscle, which prevents the division of satellite cells. They concluded that satellite cell proliferation is a prerequisite for hypertrophy following synergist ablation.

In humans, Kadi et al.[98,99] showed that both satellite cell numbers and myonuclei numbers (35% in type I and 31% in type II fibres) were higher in elite powerlifters than untrained controls. These authors concluded that the extreme hypertrophy of the muscle fibres of these athletes was dependent upon the enhanced myonuclear content. Longitudinal studies of HRST have demonstrated increases in the satellite cell population after 9-14 weeks of training,[100-102] and recent research suggests rapid proliferation of satellite cells within 4 days of a single bout of largely eccentric high load exercise.[103] However the influence of HRST on myonuclear number and the nuclear to cytoplasm ratio has been more controversial. In response to 10 weeks resistance training, Kadi & Thornell[100] reported myonuclear and satellite cell numbers in the trapezius muscle to increase substantially, and by proportionally more than fibre CSA (Figure 4), concluding that additional myonuclei appeared to be required to support the enlargement of skeletal muscle fibres following even short-term resistance training. Hikida et al.[104] also found the nuclei to cytoplasm ratio to remain unchanged after 16

weeks of strength training that elicited a 30% increase in the size of the same fibres. However, Kadi et al.[102] reported no change in myonuclei number and an increase in the fibre area controlled by each myonucleus after 90 days of HRST. Taken together these findings suggest that initial hypertrophy may involve a limited increase in the myonuclear domain and the quantity of cytosolic protein maintained by each nucleus, likely depending upon the muscle group involved and initial training status of the individual, but thereafter additional myonuclei derived from satellite cells are required.

In order for muscle growth (hypertrophy) to occur, additional contractile proteins must be manufactured and functionally integrated into the existing fibres and myofibrils. This net accretion of muscle proteins clearly requires a sustained excess of synthesis over degradation. Increased protein synthesis is reliant upon up-regulation of either transcription or translation and is beyond the scope of this review. The reader is referred to Sartorelli and Fulco[105].

2.4 Hyperplasia

Hyperplasia, an increase in the number of muscle fibres, could arise from fibre splitting/branching[106] with subsequent hypertrophy of daughter fibres and/or myogenesis,[107] and either of these processes could contribute to increased whole muscle CSA and strength gains in response to HRST. However, the phenomenon of hyperplasia remains controversial.

2.4.1 Animal Studies

Work-induced splitting of muscle fibres has been observed and thought to be responsible for hyperplasia in animal studies.[108-110] The methodology utilised of histologically counting the fibres in a cross-section at only one level in the muscle brings these results into question. Even in parallel fibred muscles, all the fibres may not run from origin to insertion. Consequently a number of studies have used nitric acid digestion to dissociate and count the total number of fibres. Using total fibre counting Gollnick and colleagues[111] studied the response to compensatory hypertrophy (ablation) and chronic stretch models in the rat, finding no evidence for hyperplasia and attributed muscle

enlargement entirely to hypertrophy of existing fibres. In contrast, Gonyea and associates[112] carried out fibre counts after an average 101 weeks of high resistance training in cats. A significant increase in fibre numbers (9%, $p<0.05$) was found and attributed to *de-novo* formation from satellite cells as no evidence for the longitudinal division of fibres was seen.

A review of 17 studies by Kelley[113] found less hyperplasia in mammalian muscle (8 vs 21% for avian muscle) and when the nitric acid digestion technique was used (11 %) compared to histological counting (21%). The degree of hyperplasia also seems to be dependant upon the experimental protocol used to induce overload with stretch causing more hyperplasia and small or no increase in fibre number with exercise or compensatory hypertrophy.[113,114]

2.4.2 Human Studies

The ethical and methodological problems of assessing the number of fibres in whole human muscles *in-vivo*, make the investigation of hyperplasia in humans extremely difficult. Even in cadaver studies there are large inter-individual differences that confound the observation of environmental adaptations.[115] The proliferative capacity of skeletal muscle tissue for regeneration is well documented.[116] Appell and associates[117] found evidence of new myotube formation from satellite cell activity after 6 weeks of endurance training. In response to HRST, Kadi & Thornell[100] discovered myotubes as well as small muscle fibres expressing embryonic and neonatal myosin heavy chain isoforms. However, Appell[107] suggested that because of the slow rate of new fibre formation, hyperplasia could have only a small effect on muscle CSA and therefore strength improvements. A cadaver study of Sjostrom and associates[115] supported the idea of hyperplasia in adult humans, but again at a very slow rate in terms of functional changes.

The comparison of mean fibre size of resistance trained subjects and controls has been used to infer or refute possible changes in muscle fibre number with HRST.[55,118-121] Given the previously discussed variability of fibre area measurements from biopsy specimens, often in combination with low subject numbers, this may produce erroneous conclusions. Somewhat more valid is the determination of fibre number by dividing the

CSA, established with CT/MRI scanning, by the average fibre area measured in biopsy specimens. However, this relies upon extrapolating a constant fibre area and angle of pennation throughout the muscle usually from a single biopsy sample, [111] which as discussed in section 2.2 may not be that reliable for fibre area measurement. Using this technique Alway et al. [122] reported a significant correlation between fibre number and anatomical CSA in elite bodybuilders that could be attributed to either an adaptive response or a process of self selection. In response to 3 months of HRST McCall et al.[123] found no change in estimated fibre number despite a 10% increase in CSA, and a comparison of muscle fibre number in bodybuilders and untrained subjects found no significant difference between the two. [124]

The quantitative contribution of hyperplasia to changes in human muscle CSA in response to exercise remains largely unknown. However, the study of human and mammalian muscle suggests hyperplasia accounts for at most a small proportion of the increase in muscle CSA in response to increased loading.

2.5 Other Morphological Adaptations

2.5.1 Changes in Fibre Type and Myosin Heavy Chain Composition?

Most of the research on muscular adaptations to strength training provides evidence against substantial fibre type changes. In animals a number of techniques used to manipulate muscle growth have revealed no change in gross fibre type with hypertrophy/atrophy,[68,125,126] although recent work indicates that more subtle changes can occur, specifically a transition of type IIB to IIX.[127] In humans resistance training also seems to produce subtle fibre type changes. Several studies have found a significant increase in the number of type IIA fibres and concomitant fall in type IIX fibres,[46,61,62,128] with one reporting this change to occur after only 18 training sessions.[129]

The most recent classification system for identifying muscle composition is based on the expression of myosin heavy chain (MHC) isoforms. Schiaffino et al.[130] identified

4 separate MHC isoforms (I, IIA, IIB, IIX) with the majority of fibres expressing just one MHC isoform that is indicative of functional and metabolic properties and in the main corresponds to other fibre type classification systems. In agreement with the findings on fibre type, measurements of muscle homogenate show the proportion of MHC IIX to fall by 5-11% with a similar rise in MHC IIA after 12-14 weeks of training.[131-133] Williamson et al.[132] examined single fibre MHC expression before and after 12 weeks of HRST. These authors found increases in the proportion of fibres expressing purely MHC IIa (+24% for young women, and +27% for young men) at the expense of a reduction of hybrid fibres (MHC I/IIA and IIA/IIX). In summary, subtle changes in fibre type and MHC composition appear to occur in the early phase (2-3 months) of training, but there is no evidence that this transformation continues over a prolonged period.

2.5.2 Density of Skeletal Muscle and Myofilaments

The gross muscle radiological density of skeletal muscle increases following strength training (+3%[134]; +5%[135,136]). Sipila and Suominen[137] found an 11% increase in radiological density of the triceps surae after 18 weeks of strength training in elderly women. This measure of density involves much larger sections of muscle tissue than the packing density of myosin filaments examined by MacDougall et al.[53] and includes all of the constituents of whole muscle e.g. fat and connective tissue. In rats, the discrepancy of fibre and whole muscle size increases with overload has been taken to suggest that fibres develop at the expense of the extra-cellular compartment.[138] It is also interesting in this context to note that many of the human studies employing the muscle biopsy technique have found greater hypertrophy than those using measurement of anatomical CSA.[11,46,69,139]

Studies of the packing density of myofilaments have found this to be very consistent pre to post training.[53,134] More contemporary research has revealed that the specific tension of muscle fibre types, divided according to myosin heavy chain expression, is unresponsive to 12 weeks of HRST[73,140,141] and similar for sedentary and long-term (>6 yrs) resistance trained individuals.[142] Therefore, there is no evidence for an adaptation of cross-bridge density or the intrinsic contractile properties of skeletal muscle (specific tension) after HRST.

2.5.3 Tendon and Connective Tissue

Skeletal muscle is enveloped in a connective tissue matrix that may play a role in transmitting force to the tendons[143] and work-induced hypertrophy is known to elevate collagen synthesis in animal muscle.[144] However, there is evidence for a fixed proportion of connective tissue in skeletal muscle throughout hypertrophy (~13% in bodybuilders and untrained controls[124]), although this does not rule out the possibility of some plasticity in the connective tissue matrix. The arrangement of connective tissue in relation to individual muscle fibres could also influence force production. For example, if connective tissue attachments were made between the tendons and intermediate parts of muscle fibres then the effective CSA of a fibre would increase.[145] Essentially a single longitudinal fibre with an extra tendinous attachment halfway along its length could in effect act with the force equivalent to two parallel fibres. Whether this occurs is unknown, but could in theory be tested, as it would cause substantial effects on the muscle mechanics.

Tendinous stiffness has been found to increase in animals in response to loading[146,147] and in humans after isometric[148] and isotonic HRST.[149,150] Reeves et al.[150] found 65% and 69% increases in patella tendon stiffness and Young's modulus respectively after 14 weeks of knee extensor training. Tendon stiffness affects the time required to stretch the series elastic component and will therefore affect both the electromechanical delay and the rate of force development,[151] thus enhancing the rapid application of force. Increased stiffness also reduces tendon elongation and likely changes the length-tension characteristics of a trained muscle, although this has not been formally investigated. A recent cross-sectional study found greater tendon thickness in athletes involved in high force activity compared to controls.[152] In animals, high intensity running has been found to cause tendon hypertrophy.[153,154] However, longitudinal studies in humans up to 14 weeks of HRST have failed to find any evidence for this,[149,150] perhaps because this is too short a period. Alternatively, a biphasic response with an initial atrophy followed by hypertrophy has been observed in pig tendon in response to endurance exercise.[147,155] Intra-tendon structural changes in response to HRST in humans have not been investigated, however animal studies suggest increased

diameter and packing density of collagen fibrils and changes in collagen crimp structure (waviness of fibrils)[156,157], which likely influence tendon stiffness.

Whilst the proportion of connective tissue in skeletal muscle does not change with HRST it is unknown if the arrangement of connective tissue changes. There is strong evidence for an increase in tendon stiffness likely due to a range of structural changes, and tendon hypertrophy also seems probable given a sufficient training period.

2.5.4 Muscle Architecture

The orientation of muscle fascicles (fibres) in relation to connective tissue/tendon and hence the relevant joint mechanics seems likely to influence muscular strength, and may exhibit a degree of plasticity with HRST. As the angle of fibre pennation (AoP) increases there is increased packing of muscle fibres within the same ACSA (essentially the effective PCSA increases), but less force from each fibre is resolved to the tendon due to their increasingly oblique angle of pull. Therefore the effect of AoP on strength is a trade-off of these 2 factors (packing vs mechanical disadvantage). Alexander and Vernon[158] calculated that the force produced by a muscle of fixed external dimensions was proportional to the sine of twice the angle of pennation. According to this relationship the optimum angle of pennation is 45°. Whilst most muscles have fibres that are pennate to the overall line of action, few muscles are pennate to this degree and therefore any increase in the angle of pennation would be expected to increase force, even if there were no increase in the anatomical CSA.

A number of studies have found a relationship between various muscle size indicies and the angle of pennation in a variety of strength trained and control groups.[159-161] This may suggest that hypertrophy involves an increase in the angle of fibre pennation. An early report[162] found no change in the angle of pennation in the vastus lateralis (VL) after 12 weeks of training, although these authors conceded that the sensitivity of their ultrasound measurement technique may have been insufficient to detect likely changes in the angle of fibre pennation. Aagaard et al.[11] reported an increase in VL pennation angle from 8.0° to 10.7° (+36%) after 14 weeks of quadriceps HRST. The increase in pennation angle facilitated PCSA and thus isometric strength to increase significantly more (+16%) than ACSA or muscle volume (+10%). HRST of the

triceps brachii has been found to increase the angle of fibre pennation after 10 weeks (17.0° - 19.2°, +16%,[163]) and 16 weeks (16.5° - 21.3°, +29%,[164]). Reeves et al.[165] found resting fibre pennation angle of the vastus lateralis to increase by 28-35%, according to knee joint angle, after 14 weeks of HRST. More uniquely these authors also measured pennation angle during maximal isometric contractions finding increases of 10-16% as a result of training.

There is strong recent evidence that the AoP increases with HRST and as most muscles have an AoP substantially below the optimum of 45° this is expected to make a substantial contribution to increased strength.

3. Neurological Adaptations

Neurological adaptations to high resistance training are of importance because of the specific nature of the adaptations in strength to the training task and also the apparent rise in specific tension after a period of strength training. In contrast to the morphological adaptations, considerable debate exists about the nature of the neurological changes that accompany strength training. Until recently much of the evidence on neurological adaptation came from somewhat indirect evidence that could be questioned methodologically or neurophysiologically, and there remain extensive methodological considerations with many of the techniques used to evaluate neural adaptations. Recent work has more precisely delineated the specific neural mechanisms contributing to the training induced increase in maximal muscle strength.

Sale[166] likened the expression of voluntary strength to a skilled act, where agonists must be maximally activated, while supported by appropriate synergist and stabiliser activation, and opposed by minimal antagonist activation. Neural adaptations are essentially changes in co-ordination and learning that facilitate better recruitment and activation of the involved muscles during a specific strength task.

3.1 Indirect Evidence of Neural Adaptations, Learning and Co-ordination

The disproportionately larger increase in muscle strength than size, particularly in the early stages of strength training, has been taken to indicate an increase in specific tension that is often largely ascribed to neurogenic factors. However, as discussed above numerous morphological changes could also account for this rise in specific tension (including changes in the architecture of muscle fibres as well as the parallel and series elastic components, fibre type and preferential hypertrophy). Whilst some investigators have attempted to include the contribution of some of these factors in order to calculate changes in muscle fibre specific tension *in-vivo* after training, notably Aagaard et al.[11], Gandevia[167] points out that it is difficult to estimate the cumulative effects of these necessary corrections.

3.1.1 Specificity of Training Adaptations

Other indirect, but more forceful evidence for a substantial neurological adaptation comes from the observation of many strength training investigations that the increase in dynamic lifting strength (1 RM) is disproportionately greater than compared to isometric strength.[66,168] Undoubtedly such findings point to a considerable facility for learning that is specific to the training task. Some proportion of this task specificity is likely attributable to postural activity associated with the task. As the human body is a linked mechanical system it is necessary to orientate the body segments and set the base of support prior to forceful muscle activity.[169] Strength and power improvements after training are specific to the postures employed[170] and the role of fixator muscles and their sequence of contraction may be different for apparently similar exercises.[168] Recent work by Nozaki et al.[171] has highlighted the variability between subjects, and within subjects on a trial to trial basis, of inter-muscle co-ordination and adjacent joint activity, during even seemingly straight forward single joint actions i.e. knee extension. This evidence reinforces the fact that apparently simple actions undoubtedly require a degree of skill in order for optimal expression of strength.

3.1.2 Cross-over Training Effect

There is considerable evidence of a cross-over effect with training of one limb causing strength increases in the contralateral untrained limb[172-174] (for review see [175]) and this supports the hypothesis of a central adaptation in the response to training.[176] However, some studies have observed no cross-over effect.[3,136,177] It has been suggested that the cross-over training effect may be partially due to stabilising or bracing activity of the 'untrained limb' during exercise,[178] although the EMG activity of the contralateral muscle has been found to be only 15% of that recorded during a maximal voluntary contraction (MVC).[179] Certainly the contribution of trained synergistic muscles, despite attempts to isolate a muscle group during strength measurements, might facilitate greater strength in the untrained limb.

The earliest phase of strength training may involve learning the right pattern of intermuscular co-ordination (i.e. stabilisers, synergists and antagonists),[168] and perhaps once learned this could be applied, for example, on the contralateral side.[167] Supporting evidence comes from the observation that cross-over training effects may also be muscle action and velocity specific.[180,181] The magnitude of this type of preliminary learning seems likely to depend upon the prior level of physical activity and co-ordination/skill of the participants at the training task, and is a likely explanation for the diverse findings on cross-over effects. There is recent evidence that cross-over effects may extend beyond general learning and co-ordination and include changes in agonist activation. Using the interpolated twitch technique (ITT) Shima et al.[182] found significant increases in agonist activation of the trained and contralateral limb after 6 weeks of training.

3.1.3 Imagined Contractions

In some muscles imagined contractions appear to increase strength by inducing purely central nervous system adaptations.[183,184] Similar experiments on the abductor digiti minimi,[183] an intrinsic hand muscle, and the dorsiflexors,[185] found equivalent strength increases for real and imagined training which were greater than a control group. More recently, Zijdewind et al.[184] contrasted the influence of 7 weeks of imagined contractions, low intensity training or a control group on plantar flexor torque. These authors found substantially greater strength gains with imagined contractions (+36%)

than for either controls (+14%) or low intensity training (+13%). In contrast, Herbert et al.[186] applied this idea to the elbow flexor muscles, finding imagined training produced strength gains only equivalent to a non-training control group and significantly less than real training. This could be because prior to training the elbow flexors are closer to maximum activation than other muscle groups[187] and therefore have less capacity for central neurological adaptations. Whilst further research is clearly required, overall this evidence suggests that substantial increases in the strength of major ambulatory muscle groups can be made without physical activity and independent of morphological adaptations. Mechanistically it supports the role of central cortical adaptations in response to regular HRST.

3.2 A Change in Agonist Activation?

The simple fact that even during maximum contractions recordings of force show substantial fluctuations has been taken to indicate that true maximum force is at best difficult to achieve.[167] Moreover, it has been widely suggested that healthy, but untrained individuals, cannot fully activate their muscles during maximum voluntary contractions even when fully motivated.[188,189] With HRST agonist muscle activation could increase through enhanced motor unit recruitment, or firing frequency, assuming these variables are sub-maximal prior to training.

3.2.1 Electromyography

Surface Electromyograph (SEMG) recordings have been used by many investigators in an attempt to measure changes in agonist muscle activation. Numerous studies have reported agonist muscle SEMG to increase significantly with strength training, particularly during the first 3-4 weeks, and this has been taken as evidence for a change in the neural drive to a muscle.[33,47,49,172,173,190,191] Hakkinen and Komi[190] found the changes in SEMG to closely follow the changes in force over 16 weeks of training and 8 weeks of detraining (Figure 5). In contrast some studies have found no change in EMG after training.[3,8,192,193] In order to examine the factors responsible for the rapid increase in strength at the onset of a training programme, Holtermann et al.[194] observed

changes in dorsiflexor strength and SEMG, of the tibialis anterior with a large grid electrode, over 9 training sessions in a 5 day period. Whilst strength increased by 16%, peak SEMG amplitude decreased by 11%. The controversy surrounding SEMG findings may be explained by a number of issues with SEMG measurement and interpretation. The technical difficulties of SEMG measurements are well recognised, and whilst electrode technology and signal processing of EMG recordings continues to improve, the reproducibility of EMG measurements remains questionable. Problems with relocating electrodes, variable impedance of the skin and subcutaneous tissue, as well as changes in muscle morphology tend to confound the ability to reliably detect longitudinal changes in SEMG.

The interpretation of increased SEMG as reflecting increased neural drive is also considered a simplification. First, SEMG is modified by changes in excitation-contraction coupling, specifically alteration of single fibre action potential.[167] A number of factors change during a period of resistance training, including: fibre type, fibre size, membrane potential,[195] intramuscular ionic concentrations and sodium-potassium pump content,[196,197] that likely alter single fibre action potential and SEMG. Second, large fast motor units tend to be more abundant towards the periphery of the muscle, close to the skin,[59,198] and any change in their activity may have an exaggerated effect upon surface EMG recording. The confounding influence of these factors, and the variability in electrical impedance, can be controlled/normalised by measurement of the compound muscle action potential (M-wave) produced by supramaximal nerve stimulation. Increased EMG whilst M-wave remained constant has been found,[199,200] whilst a parallel increase in EMG and M-wave has been reported.[201]

Finally, whilst increased SEMG may reflect an increase in fibre recruitment or firing frequency, the summation pattern of EMG is also sensitive to changes in synchronisation. Out-of-phase summation can lead to cancellation of motor unit action potentials that do not necessarily reflect any change in activation (possible changes in synchronisation are discussed in section 3.3.2 below).

3.2.2 Tetanic Stimulation

The maximality of the neural drive to the agonist has been measured by a variety of techniques, but typically only in relatively isolated circumstances i.e. unilateral, single joint isometric exercises. Supramaximal tetanic stimulation appears to be the most comprehensive method of evaluating the level of voluntary muscle activation, although a lack of activation of synergists and stabilisers does question the validity of this approach. Due to the associated difficulties and discomfort relatively few studies have been completed. The force from an isometric MVC has been found to match the force produced by tetanic stimulation in untrained subjects,[202-204] although the measurement sensitivity of these early investigations is dubious. After a period of training, the comparison of changes in voluntary and electrically evoked force have also been used to elucidate the importance of the voluntary drive to strength gain. However the evidence is equivocal, with reports that voluntarily training increases[199,205] and has no effect[206,207] on the force of electrically evoked tetanic contractions. A third strategy in this regard has been to compare the effect of training with electrical muscle stimulation (EMS) compared to voluntary efforts. A number of studies have employed EMS training reporting significant increases in strength,[208,209] similar strength increases as voluntary training[205,210,211] and greater strength and ACSA increases than voluntary training.[212] This evidence demonstrates that substantial improvements in strength are possible without central nervous system involvement.

3.2.3 Interpolated Twitch Technique

The interpolated twitch technique has been extensively employed to measure the level of muscle activation.[213-215] In numerous studies, insensitive forms of twitch interpolation have been used to conclude that untrained healthy subjects can achieve 'maximal' activation during isometric effort.[167] There is increasing acceptance of the importance of a number of technical and methodological issues in the use of this technique (see[216,217]). The maximality of neurological activation appears to be muscle specific[214] with for example the elbow flexors more completely activated than the quadriceps femoris.[187] Notably, more recent work provides evidence that activation of many muscle groups is rarely maximal, with for example, considerable evidence that

quadriceps femoris activation during isometric MVC is 85-95% in healthy untrained subjects.[182,218-221] Whilst a number of older studies have found no increase in voluntary activation after resistance training,[136,177,222] again more recent investigations have found increased activation following training.[165,182,223,224] Another development in this field is the suggestion that the maximality of muscle activation during isometric effort may well be angle specific. Becker & Awiszus[225] found quadriceps activation at 40° knee joint angle to be ~20% lower than at 90° (Figure 6a), and these findings have recently been replicated.[226]

3.2.4 Dynamic Muscle Activity

Numerous authors have hypothesised that during slow concentric contractions, typical of maximum lifting tasks, there is a reduced neural drive.[189,227,228] Using EMG, Aagaard et al.[15] found evidence for inhibition of neural drive during maximal slow concentric movements, that was partially abolished after 14 weeks HRST. Studies employing superimposed stimuli have tended to dismiss this suggestion.[229,230] However, using the ITT Babault et al.[231] found activation to be significantly lower for slow concentric than isometric contractions (89.7 vs 95.2% - Figure 6b).

During eccentric contractions there is considerable evidence of a sub-maximal neural drive in untrained subjects. The eccentric portion of the *in-vivo* force-velocity relationship for untrained individuals shows a marked difference in comparison to the *in-vitro* relationship – specifically force is no greater during lengthening (eccentric) activity than isometric actions.[232] Notably, this discrepancy does not exist for voluntary contraction of elite power-trained individuals,[232,233] and is removed with electrical stimulation of untrained subjects.[234] In addition, eccentric training of previously untrained individuals leads to considerably greater increases in eccentric-specific strength and EMG than concentric training upon concentric strength and EMG.[235] Taken together this evidence strongly indicates a failure in muscle activation during maximal eccentric efforts of untrained subjects either due to poor supraspinal activation or perhaps more likely spinal inhibition from a range of afferents (group IB Golgi organ afferents, group Ia and II muscle spindle afferents, group III muscle spindle afferents and Renshaw cells), although the precise mechanism remains unknown.[15]

There is increasing evidence that previously untrained yet healthy subjects have scope for increasing the neural drive to agonist muscles. The magnitude of this central reserve, and hence the capacity for improvement with training likely depends upon the muscle group(s) under consideration, the type of muscle contraction, the muscle lengths and joint positions involved as well as the complexity and familiarity of the movement task i.e. bilateral or multi-joint activity.

3.3 Specific Mechanisms of Neurological Adaptation

Enhanced agonist muscle activation after HRST could be due to increased motor unit recruitment or firing frequency. During a slow ramped contraction from rest the contribution of these two factors to increased activation is highly dependent upon the muscle under consideration, with large muscles appearing to rely more on recruitment to achieve high levels of voluntary force.[236,237] Definitive evidence of an increase in motor unit recruitment with training would require demonstration of a population of previously uninvolved motor units that can be recruited after training. Unfortunately this is beyond the capability of current techniques. Clearly, both increased recruitment and/or firing frequency would involve some form of increased neurological drive either at the spinal or supraspinal level.

3.3.1 Firing Frequency

Using a large grid electrode Holtermann and colleagues[194] evaluated changes in SEMG median frequency after 9 training session of the dorsiflexors. They found no change in median frequency, regarded as a measure of motor unit recruitment,[238] despite a 16% increase in strength. Intra-muscular EMG recording techniques offer the potential to accurately investigate motor unit firing frequency (MUFF) of humans *in-vivo*. The MUFF can be much higher for very brief periods (first three discharges) at the onset of a maximum voluntary effort (i.e. 100-200 Hz[200]) with much lower rates at the instant of maximum force generation (20-30 Hz[236,237,239,240]). It is curious that with involuntary stimulation the force-frequency relationship observed for motor units in human muscle suggests discharge rates of at least 50 Hz are required to achieve maximum tetanic

forces.[241,242] Taken in isolation this might suggest considerable capacity for increases, perhaps up to 2-fold, in MUFF during maximum voluntary contractions, contributing to increased strength after training. However, it is thought that phenomena such as the catch like properties of motor units[243] and twitch potentiation[244] may facilitate greater force production at lower than expected frequencies. An initial, brief, high-frequency burst of two to four pulses at the start of a contraction augments subsequent force production and is known as the catch like property of skeletal muscle.[243] Twitch potentiation refers to the greater contractile response to a single pulse following muscle activity, that likely leads to tetanic contractions at lower frequencies of innervation.

During maximum force generation MUFF has been found to be higher in trained elderly weight lifters than age-matched controls (23.8 vs 19.1 Hz,[245]). Two longitudinal studies have found increased MUFF after HRST.[174,200] Van Cutsem et al.[200] trained subjects for 12 weeks (60 training sessions) with fast ballistic contractions finding earlier motor unit activation, extra doublets and enhanced MUFF at the onset of ballistic contractions after training. Whilst these adaptations likely contribute to gains in the rate of force development and acceleration during fast dynamic contractions, their effect on the rate of MUFF and strength at the instant of maximum force generation during slower, high force contractions is unknown. Patten et al.[174] reported no effect of two weeks of strength training on maximal MUFF. In this study, the largest changes (in strength and MUFF) appeared to occur between the two baseline tests, perhaps due to the unfamiliar nature of the movement (5^{th} finger abduction), low subject numbers or the short duration of the training.

3.3.2 Synchronisation

Synchronisation quantifies the level of correlation between the timing of the action potentials discharged by concurrently active motor units. The motor units of strength athletes appear to exhibit greater synchronisation than untrained individuals and HRST appears to increase synchronisation.[246,247] However, it is not clear how an increase in synchronisation could promote strength[52,176] as at firing frequencies equivalent to MVC there is no effect of synchronisation upon force.[248,249]

3.3.3 Cortical Adaptations

In humans, motor skill training with low force muscle activity has been demonstrated, using neuroimaging techniques and transcranial magnetic stimulation, to induce changes in the primary motor cortex such as organisation of movement representations, and increased cortical or corticospinal excitability for specific muscles and movements.[250-257] These adaptations might also offer an explanation for how imaginary training/mental practice could increase strength. However, more specific studies employing transcranial stimulation techniques in response to strength training found an unexpected decrease in corticospinal excitability after training of the first dorsal interosseous[258] and biceps brachii[259] muscles that would question any significant cortical adaptation.

3.3.4 Spinal Reflexes

Afferent feedback in the form of spinal reflexes during contraction could enhance or dampen the supraspinal drive to the muscle. Evoked spinal reflexes have been investigated to examine any changes in spinal motoneurons after HRST, specifically their sensitivity to afferent feedback. The Hoffman (or H reflex) is an artificially elicited reflex that is used to test the efficacy of transmission of a stimulus as it passes from the afferent fibres through the motoneuron pool to the efferent fibres. It is thought to give an approximate measure of excitability of the motor neuron pool.[260] The V wave is an electrophysiological variant of the H-reflex, but delivered during an MVC, and may reflect efferent motor neuronal activity.[261] The H-reflex response has been measured at rest and found not to change after training,[223] although the relevance of this measurement has been questioned.[261] During maximum voluntary isometric contractions, Sale and colleagues measured the V1 and V2 wave responses after training, reporting both no potentiation[262] and a significant increase.[166] A thorough recent study by Aagaard and co-workers[261] carefully assessed and controlled M-wave amplitude even during maximal contractions. These authors found a 20% increase in isometric strength was accompanied by increased V-wave and H-reflex amplitudes (55% and 19% respectively, Figure 7) after 14 weeks of HRST. The increase in V-wave amplitude

indicates enhanced neural drive from the spinal motoneurons, that these investigators concluded was most likely due to increased motoneuron firing frequency. The enhanced H-reflex after training further suggests that the increase in motoneuron output was caused in part by a rise in motoneuron excitability, although the greater increase in V-wave than H-reflex indicates enhanced supraspinal activation. Whilst these changes seem certain to contribute to enhanced strength, the quantitative functional significance of these effects remains unknown,[263] and this evidence is clearly contrary to the surprising decrease in corticospinal excitability that has been observed after training.[258,259]

3.3.5 Antagonist Co-Activation

The extent of antagonist activation during any given exercise depends on a wide range of factors including the velocity and range of motion.[264] Any co-contraction of antagonists clearly reduces force output, but it also impairs, by reciprocal inhibition, the ability to fully activate the agonists. Cross-sectional studies have found lower co-activation in strength/power trained athletes than untrained controls.[265,266] Carolan and Cafarelli[267] found a significant decrease in antagonistic activation that mostly occurred in the first week of an isometric knee extensor training programme. Hakkinen and colleagues[268] found reduced hamstrings co-activation of older but not middle aged participants after 6 months of knee extensor HRST. However, other studies have found no change in antagonist activation after 9 dorsiflexor training sessions[194] or 14 weeks of knee extension training with older adults.[165] During more complex multi-joint or whole body movements the level of antagonist activation may be greater, perhaps providing more opportunity for a reduction in co-activation with training.

4. Conclusion

A wide range of morphological and neurological factors are known to contribute to increased strength following high resistance strength training. An increase in the size of the exercised muscles is typically regarded as the major long term adaptation, although this is highly variable between the muscles exposed to the training and along their length.

Whole muscle hypertrophy appears to proceed in a linear fashion during the first 6 months of training and is ascribed to hypertrophy of individual fibres by the processes of myofibrillar growth and proliferation, although hyperplasia may play a minor role. Whilst there may be an increase in the myonuclei to cytoplasm ratio by an upregulation of transcription or translation, satellite cells are activated in the very earliest stages of training. Their proliferation and fusion with existing myofibers enhances the number of myonuclei and appears to be intimately involved in the hypertrophy response. Muscle fibre hypertrophy is typically greater in type II fibres and is accompanied by an increase in the angle of fibre pennation, which promotes a greater increase in PCSA and force production than revealed by ACSA. These two factors likely contribute to increased strength and the apparent rise in whole muscle specific tension, despite the fact that individual fibre specific tension does not change.

The weight of indirect evidence (cross-over effects, task specificity, rapid gains in strength at the onset of a training programme) whilst not definitive suggests a substantial neurological adaptation that may well be predominantly due to learning and changes in intermuscular co-ordination of agonists, antagonists and synergists. The rapid rise in strength at the start of a training programme, within the first 2 weeks, that is primarily due to neurological adaptations, significantly increases the loading and training stimulus to which the muscle is then exposed. This helps to maximise further strength gains, particularly morphological adaptations, that occur as training continues.

More sensitive use of the interpolated twitch technique suggests that untrained individuals may not be able to fully activate agonist muscles, and this central reserve appears to depend upon a range of task specific factors. In addition, whilst controversial the weight of SEMG measurements indicates an increase in agonist activation after training. Studies employing transcranial stimulation have found no evidence for cortical or corticospinal adaptation and are at odds with investigations of spinal reflexes that indicate an increased supraspinal drive, motoneuron excitability, and a likely increase in MUFF after training.

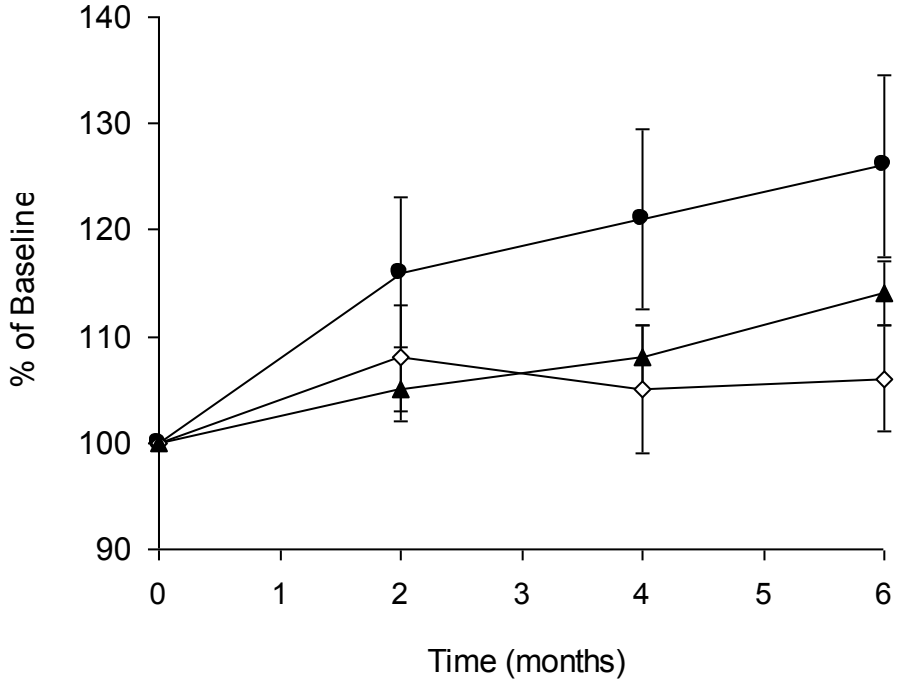

Figure 1. Isometric MVC (circles), Integrated EMG (diamonds) and quadriceps ACSA (triangles) at mid-thigh during 6 months strength training. Data adapted from Narici et al.[8]

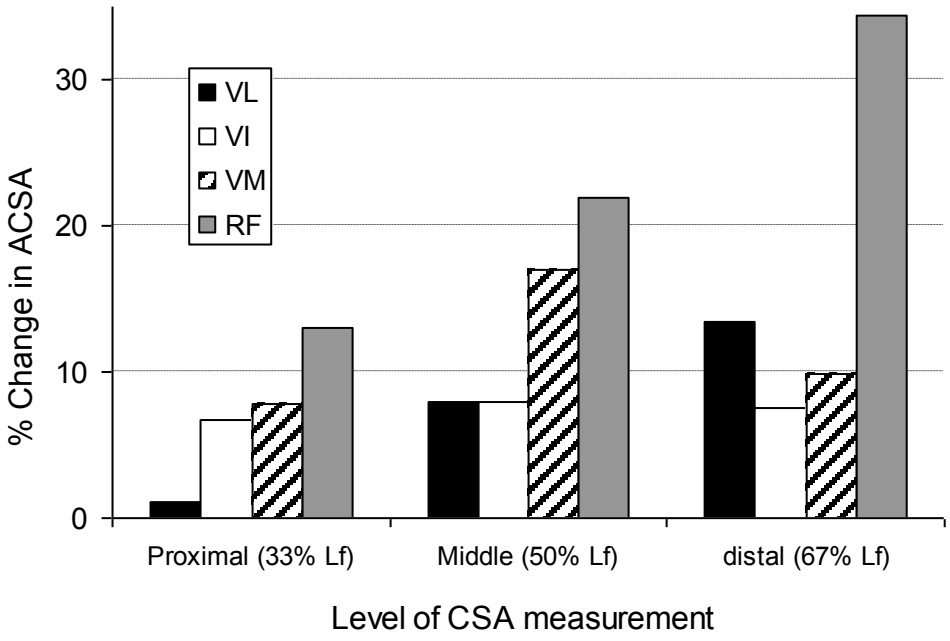

Figure 2. Selective hypertrophy of the quadriceps femoris muscle after 8 weeks of isokinetic HRST. The extent of hypertrophy varies according to the constituent muscle and level of CSA assessment (Lf, length of the femur). Adapted from the data of Housh et al.[4]

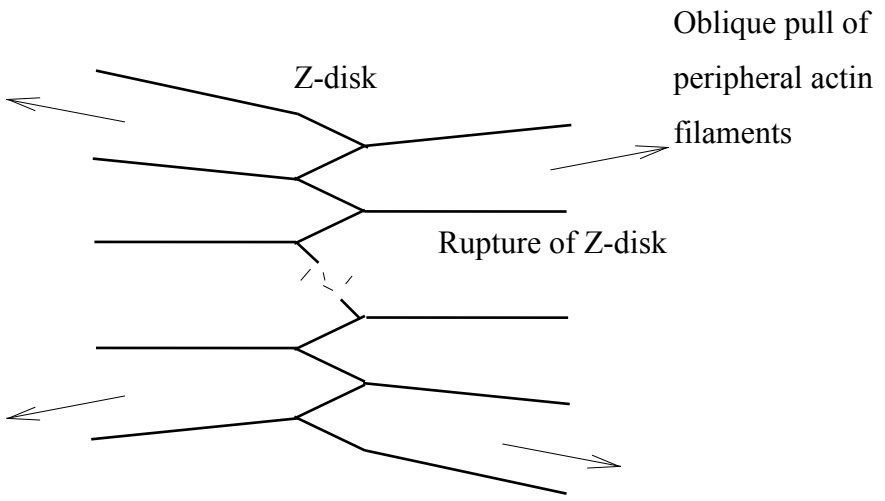

Figure 3 Myofibrillar splitting due to the oblique pull of the peripheral actin filaments. Redrawn from Goldspink.[82]

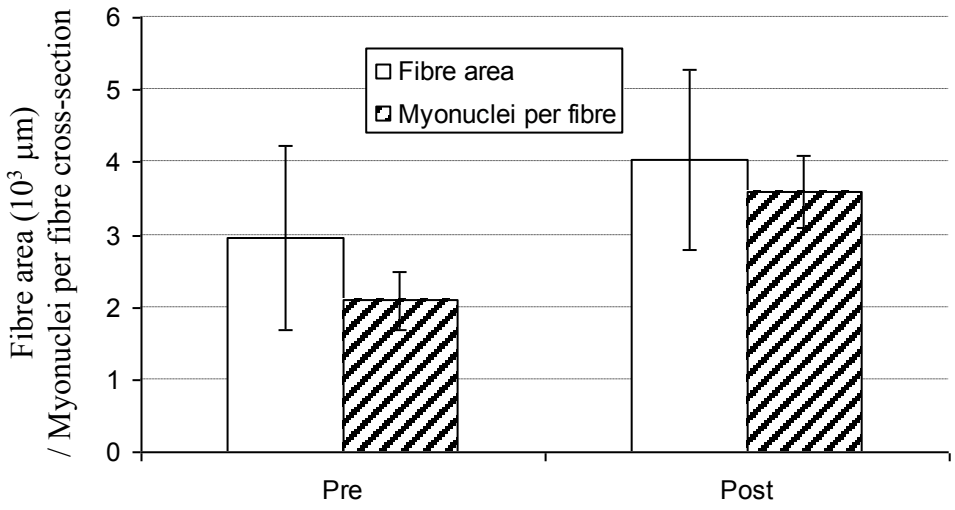

Figure 4. Whilst controversial there is strong evidence that increases in fibre area even during the early stages (10 weeks) of HRST, in this case of the trapezius muscle, involves an increase in myonuclei number from proliferating satellite cells. Data from Kadi and Thornell.[100]

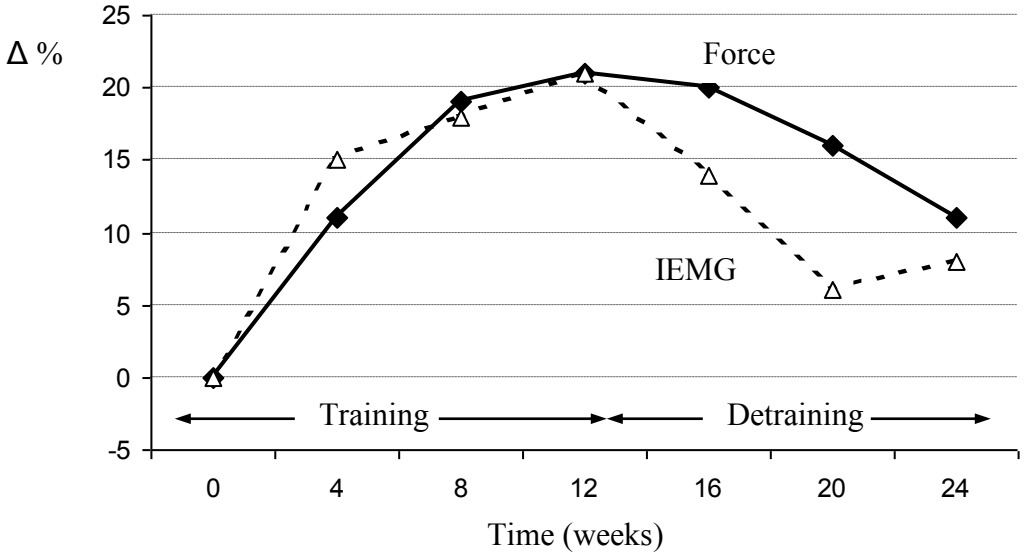

Figure 5. Changes in isometric force and SEMG with 16 weeks of training and 8 weeks detraining. Redrawn from Hakkinen & Komi.[190]

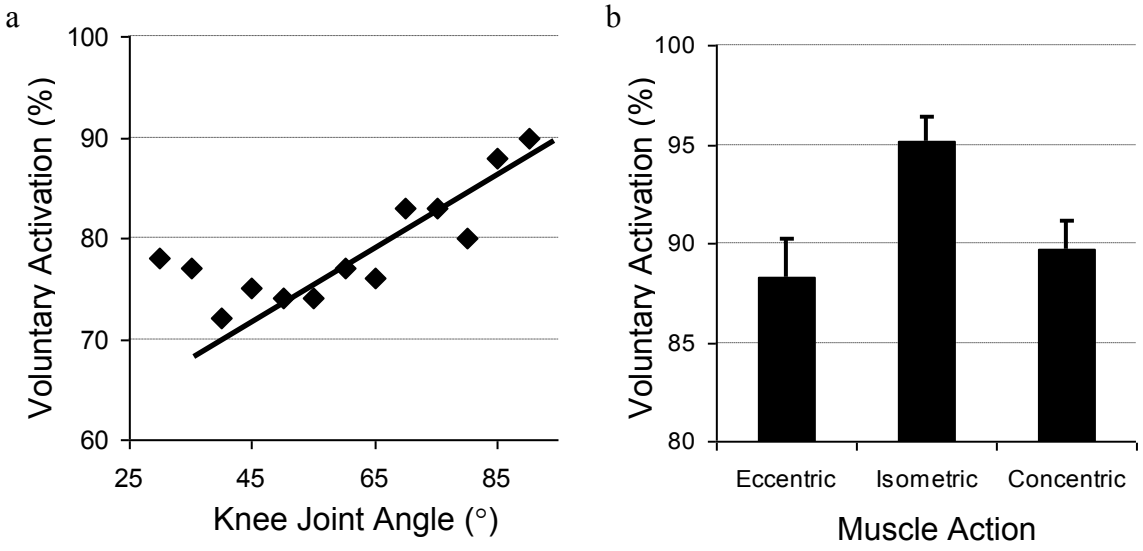

Figure 6. Recent evidence using the Interpolated Twitch Technique has suggested that the ability to maximally activate the agonist muscle varies with joint position / muscle length (a, Redrawn from Becker & Awiszus,[225]) and type of muscle contraction (b, Redrawn from Babault et al.[231]).

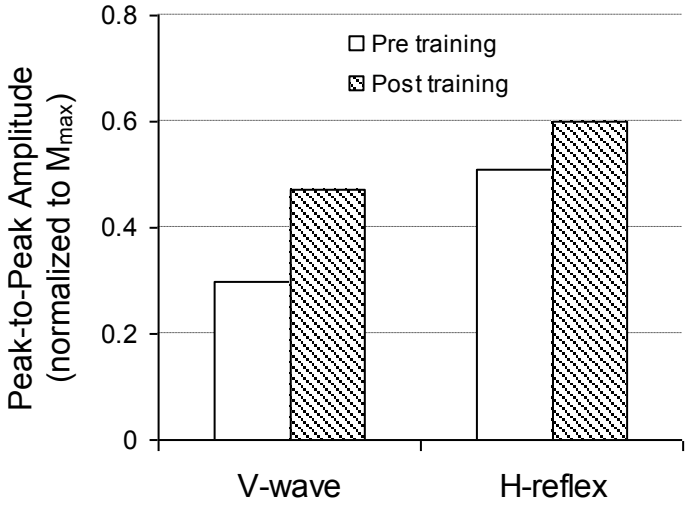

Figure 7. V-wave and H-reflex amplitude (expressed relative to Mmax) measured during isometric MVCs before and after 14 weeks of HRST. Data adapted from Aagaard et al.[261]

References

1. Morganti CM, Nelson ME, Fiatarone MA, et al. Strength improvements with 1 yr of progressive resistance training in older women. Med Sci Sports Exerc 1995; 27: 906-912

2. Paavolainen L, Paavolainen L, Hakkinen K, et al. Explosive-strength training improves 5-km running time by improving running economy and muscle power. J Appl Physiol 1999; 86: 1527-1533

3. Garfinkel S, Cafarelli E. Relative changes in maximal force, emg, and muscle cross- sectional area after isometric training. Med Sci Sports Exerc 1992; 24: 1220-1227

4. Housh DJ, Housh TJ, Johnson GO, et al. Hypertrophic response to unilateral concentric isokinetic resistance training. J Appl Physiol 1992; 73: 65-70

5. Tracy B, Ivey F, Hurlbut D, et al. Muscle quality. II. effects of strength training in 65- to 75-yr-old men and women. J Appl Physiol 1999; 86: 195-201

6. Abe T, DeHoyos D, Pollock M, et al. Time course for strength and muscle thickness changes following upper and lower body resistance training in men and women. Eur J Appl Physiol 2000; 81: 174-180

7. Engstrom CM, Loeb GE, Reid JG, et al. Morphometry of the human thigh muscles - a comparison between anatomical sections and computer tomographic and magnetic- resonance images. J Anat 1991; 176: 139-156

8. Narici M, Hoppeler H, Kayser B, et al. Human quadriceps cross-sectional area, torque and neural activation during 6 months strength training. Acta Physiol Scand 1996; 157: 175-186

9. Alway SE, Grumbt WH, Stray-Gundersen J, et al. Effects of resistance training on elbow flexors of highly competitive bodybuilders. J Appl Physiol 1992; 72: 1512-21

10. Tracy B, Ivey F, Jeffrey Metter E, et al. A more efficient magnetic resonance imaging-based strategy for measuring quadriceps muscle volume. Med Sci Sports Exerc 2003; 35: 425-433

11. Aagaard P, Andersen J, Dyhre-Poulsen P, et al. A mechanism for increased contractile strength of human pennate muscle in response to strength training: Changes in muscle architecture. J Physiol 2001; 534: 613-23

12. Fukunaga T, Roy RR, Shellock FG, et al. Specific tension of human plantar flexors and dorsiflexors. J Appl Physiol 1996; 80: 158-165

13. Roman WJ, Fleckenstein J, Straygundersen J, et al. Adaptations in the elbow flexors of elderly males after heavy- resistance training. J Appl Physiol 1993; 74: 750-754

14. Keen DA, Yue GH, Enoka RM. Training-related enhancement in the control of motor output in elderly humans. J Appl Physiol 1994; 77: 2648-2658

15. Aagaard P, Simonsen E, Andersen J, et al. Neural inhibition during maximal eccentric and concentric quadriceps contraction: Effects of resistance training. J Appl Physiol 2000; 89: 2249-2257

16. Bamman MM, Newcomer BR, Larson-Meyer DE, et al. Evaluation of the strength-size relationship in vivo using various muscle size indices. Med Sci Sports Exerc 2000; 32: 1307-1313

17. Fukunaga T, Miyatani M, Tachi M, et al. Muscle volume is a major determinant of joint torque in humans. Acta Physiol Scand 2001; 172: 249-255

18. Maganaris CN, Baltzopoulos V, Sargeant AJ. Changes in achilles tendon moment arm from rest to maximum isometric plantarflexion: In vivo observations in man. J Physiol 1998; 510: 977-985

19. Maganaris CN, Baltzopoulos V. Predictability of in vivo changes in pennation angle of human tibialis anterior muscle from rest to maximum isometric dorsiflexion. Eur J Appl Physiol Occup Physiol 1999; 79: 294-297

20. Narici MV, Binzoni T, Hiltbrand E, et al. In vivo human gastrocnemius architecture with changing joint angle at rest and during graded isometric contraction. J Physiol 1996; 496: 287-97

21. Wilmore JH. Alterations in strength, body composition and anthropometric measurements consequent to a 10-week weight training-program. Med Sci Sports Exerc 1974; 6: 133-138

22. Cureton KJ, Collins MA, Hill DW, et al. Muscle hypertrophy in men and women. Med Sci Sports Exerc 1988; 20: 338-344

23. Welle S, Totterman S, Thornton C. Effect of age on muscle hypertrophy induced by resistance training. J Gerontol Ser A-Biol Sci Med Sci 1996; 51: M270-M275

24. Kadi F, Bonnerud P, Eriksson A, et al. The expression of androgen receptors in human neck and limb muscles: Effects of training and self-administration of androgenic-anabolic steroids. Histochem Cell Biol 2000; 113: 9

25. Edwards RH, Young A, Hosking GP, et al. Human skeletal muscle function: Description of tests and normal values. Clin Sci Mol Med 1977; 52: 283-290

26. Schantz P, Randall-Fox E, Hutchison W, et al. Muscle fibre type distribution, muscle cross-sectional area and maximal voluntary strength in humans. Acta Physiol Scand 1983; 117: 219-226

27. Neder JA, Nery LE, Silva AC, et al. Maximal aerobic power and leg muscle mass and strength related to age in non-athlethic males and females. Eur J Appl Physiol Occup Physiol 1999; 79: 522-530

28. Toft I, Lindal S, Bønaa KH, et al. Quantitative measurement of muscle fiber composition in a normal population. Muscle Nerve 2003; 28: 101-108

29. Asmussen E. Development patterns in physical performance capacity. In: Larsson L, ed. Fitness, Health and Work capacity: International Standards for Assessment. NY: MacMillan, 1974; 435-438

30. Hettinger T. Physiology of Strength. Springfield, IL: CC Thomas, 1961

31. Brown CH, Wilmore JH. The effects of maximal resistance training on the strength and body composition of women athletes. Med Sci Sports 1974; 6: 174-177

32. Wells CL. Women, sport and Performance: A Physiological Perspective. Champaign, IL: Human Kinetics, 1985

33. Hakkinen K, Kallinen M, Linnamo V, et al. Neuromuscular adaptations during bilateral versus unilateral strength training in middle-aged and elderly men and women. Acta Physiol Scand 1996; 158: 77-88

34. Roth SM, Ivey FM, Martel GF, et al. Muscle size responses to strength training in young and older men and women. J Am Geriatr Soc 2001; 49: 1428-1433

35. Colliander EB, Tesch PA. Responses to eccentric and concentric resistance training in females and males. Acta Physiol Scand 1991; 141: 149-156

36. Lexell J, Downham DY, Larsson Y, et al. Heavy-resistance training in older scandinavian men and women: Short- and long-term effects on arm and leg muscles. Scand J Med Sci Sports 1995; 5: 329-341

37. Weiss LW, Clark FC, Howard DG. Effects of heavy-resistance triceps surae muscle training on strength and muscularity of men and women. Phys Ther 1988; 68: 208-213

38. O'Hagan FT, Sale DG, MacDougall JD, et al. Response to resistance training in young women and men. Int J Sports Med 1995; 16: 314-321

39. Hubal MJ, Gordish-Dressman H, Thompson PD, et al. Variability in muscle size and strength gain after unilateral resistance training. Med Sci Sports Exerc 2005; 37: 964-972

40. Knapik JJ, Wright JE, Kowal DM, et al. The influence of U.S. army basic initial entry training on the muscular strength of men and women. Aviat Space Environ Med 1980; 51: 1086-1090

41. Kadi F, Bonnerud P, Eriksson A, et al. The expression of androgen receptors in human neck and limb muscles: Effects of training and self-administartion of androgenic-anabolic steroids. Histochem Cell Biol 2000; 113: 25--29

42. Delmonico MJ, Kostek MC, Doldo NA, et al. Effects of moderate-velocity strength training on peak muscle power and movement velocity: Do women respond differently than men? J Appl Physiol 2005; 99: 1712-1718

43. Fiatarone MA, Marks EC, Ryan ND, et al. High-intensity strength training in nonagenarians - effects on skeletal-muscle. J Am Med Assoc 1990; 263: 3029-3034

44. Harridge SDR, Kryger A, Stensgaard A. Knee extensor strength, activation, and size in very elderly people following strength training. Muscle Nerve 1999; 22: 831-839

45. Ivey F, Tracy B, Lemmer J, et al. Effects of strength training and detraining on muscle quality: Age and gender comparisons. J Gerontol Ser A-Biol Sci Med Sci 2000; 55: B152--7

46. Hakkinen K, Newton RU, Gordon SE, et al. Changes in muscle morphology, electromyographic activity, and force production characteristics during progressive strength training in young and older men. J Gerontol Ser A-Biol Sci Med Sci 1998; 53: B415-B423

47. Narici MV, Roi GS, Landoni L, et al. Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. Eur J Appl Physiol Occup Physiol 1989; 59: 310-319

48. Hakkinen K, Pakarinen A, Kraemer WJ, et al. Selective muscle hypertrophy, changes in EMG and force, and serum hormones during strength training in older women. J Appl Physiol 2001; 91: 569-580

49. Häkkinen K, Alen M, Kraemer WJ, et al. Neuromuscular adaptations during concurrent strength and endurance training versus strength training. Eur J Appl Physiol 2003; 89: 42-52

50. Kanehisa H, Funato K, Kuno S, et al. Growth trend of the quadriceps femoris muscle in junior olympic weight lifters: An 18-month follow-up survey. Eur J Appl Physiol 2003; 89: 238-242

51. McDonagh MJN, Davies CTM. Adaptive response of mammalian skeletal-muscle to exercise with high loads. Eur J Appl Physiol Occup Physiol 1984; 52: 139-155

52. Jones DA, Rutherford OM, Parker DF. Physiological changes in skeletal muscle as a result of strength training. Q J Exp Physiol Cogn Med Sci 1989; 74: 233-256

53. MacDougall JD, Elder GCB, Sale DG, et al. Effects of strength training and immobilization on human-muscle fibers. Eur J Appl Physiol Occup Physiol 1980; 43: 25-34

54. Viitasalo JT, Saukkonen S, Komi PV. Reproducibility of measurements of selected neuromuscular performance variables in man. Electromyogr Clin Neurophysiol 1980; 20: 487-501

55. Schantz P, Fox ER, Norgren P, et al. The relationship between the mean muscle fibre area and the muscle cross-sectional area of the thigh in subjects with large differences in thigh girth. Acta Physiol Scand 1981; 113: 537-539

56. Halkjaer-Kristensen J, Ingemann-Hansen T. Variations in single fibre areas and fibre composition in needle biopsies from the human quadriceps muscle. Scand J Clin Lab Invest 1981; 41: 391-395

57. Blomstrand E, Celsing F, Fridén J, et al. How to calculate human muscle fibre areas in biopsy samples--methodological considerations. Acta Physiol Scand 1984; 122: 545-551

58. Mahon M, Toman A, Willan PL, et al. Variability of histochemical and morphometric data from needle biopsy specimens of human quadriceps femoris muscle. J Neurol Sci 1984; 63: 85-100

59. Lexell J, Taylor CC. Variability in muscle fibre areas in whole human quadriceps muscle. how much and why? Acta Physiol Scand 1989; 136: 561-568

60. Gollnick PD, Matoba H. The muscle fiber composition of skeletal muscle as a predictor of athletic success. an overview. Am J Sports Med 1984; 12: 212-217

61. Campos GER, Luecke TJ, Wendeln HK, et al. Muscular adaptations in response to three different resistance- training regimens: Specificity of repetition maximum training zones. Eur J Appl Physiol 2002; 88: 50-60

62. Staron RS, Malicky ES, Leonardi MJ, et al. Muscle hypertrophy and fast fiber type conversions in heavy resistance-trained women. Eur J Appl Physiol Occup Physiol 1990; 60: 71-79

63. Tesch PA. Skeletal-muscle adaptations consequent to long-term heavy resistance exercise. Med Sci Sports Exerc 1988; 20: S132-S134

64. Thorstensson A, Hulten B, Dobeln WV, et al. Effect of strength training on enzyme-activities and fiber characteristics in human skeletal-muscle. Acta Physiol Scand 1976; 96: 392-398

65. Hakkinen K, Komi P, Tesch P. Effect of combined concentric and eccentric strength training and detraining on force-time, muscle fiber and metabolic characteristics of leg extensor muscles. Scand J Sports Sci 1981; 3: 50-58

66. Dons B, Bollerup K, Bondepetersen F, et al. Effect of weight-lifting exercise related to muscle-fiber composition and muscle cross-sectional area in humans. Eur J Appl Physiol Occup Physiol 1979; 40: 95-106

67. Houston ME, Froese EA, Valeriote SP, et al. Muscle performance, morphology and metabolic capacity during strength training and detraining - a one leg model. Eur J Appl Physiol Occup Physiol 1983; 51: 25-35

68. Goldspink G, Ward PS. Changes in rodent muscle-fiber types during postnatal-growth, undernutrition and exercise. J Physiol 1979; 296: 453-469

69. Frontera WR, Meredith CN, Oreilly KP, et al. Strength conditioning in older men - skeletal-muscle hypertrophy and improved function. J Appl Physiol 1988; 64: 1038-1044

70. Fitts RH, McDonald KS, Schluter JM. The determinants of skeletal-muscle force and power - their adaptability with changes in activity pattern. J Biomech 1991; 24: 111-122

71. Stienen GJM, Kiers JL, Bottinelli R, et al. Myofibrillar ATPase activity in skinned human skeletal muscle fibres: Fibre type and temperature dependence. J Physiol 1996; 493: 299-307

72. Bottinelli R, Pellegrino MA, Canepari M, et al. Specific contributions of various muscle fibre types to human muscle performance: An in vitro study. J Electromyogr Kinesiol 1999; 9: 87-95

73. Widrick JJ, Stelzer JE, Shoepe TC, et al. Functional properties of human muscle fibers after short-term resistance exercise training. Am J Physiol Regul Integr Comp Physiol 2002; 283: R408-16

74. Nygaard E, Houston M, Suzuki Y, et al. Morphology of the brachial biceps muscle and elbow flexion in man. Acta Physiol Scand 1983; 117: 287-292

75. Maughan RJ, Nimmo MA. The influence of variations in muscle-fiber composition on muscle strength and cross-sectional area in untrained males. J Physiol 1984; 351: 299-311

76. Grindrod S, Round JM, Rutherford OM. Type-2 fiber composition and force per cross-sectional area in the human quadriceps. J Physiol 1987; 390: P154

77. Aagaard P, Andersen JL. Correlation between contractile strength and myosin heavy chain isoform composition in human skeletal muscle. Med Sci Sports Exerc 1998; 30: 1217-1222

78. Gur H, Gransberg L, vanDyke D, et al. Relationship between in vivo muscle force at different speeds of isokinetic movements and myosin isoform expression in men and women. Eur J Appl Physiol 2003; 88: 487-496

79. MacDougall JD, Sale DG, Moroz JR, et al. Mitochondrial volume density in human skeletal-muscle following heavy resistance training. Med Sci Sports Exerc 1979; 11: 164-166

80. Morkin E. Postnatal muscle fiber assembly: Localization of newly synthesized myofibrillar proteins. Science 1970; 167: 1499-1501

81. Goldspink G. The proliferation of myofibrils during muscle fibre growth. J Cell Sci 1970; 6: 593-603

82. Goldspink G. Changes in striated muscle fibres during contraction and growth with particular reference to myofibril splitting. J Cell Sci 1971; 9: 123-128

83. Goldspink G, Howells KF. Work-induced hypertrophy in exercised normal muscles of different ages and the reversibility of hypertrophy after cessation of exercise. J Physiol 1974; 239: 179-193

84. Patterson S, Goldspink G. Mechanism of myofibril growth and proliferation in fish muscle. J Cell Sci 1976; 22: 607-616

85. Ashmore CR, Summers PJ. Stretch-induced growth in chicken wing muscles: Myofibrillar proliferation. Am J Physiol 1981; 241: C93-7

86. Moss FP. The relationship between the dimensions of the fibres and the number of nuclei during restricted growth, degrowth and compensatory growth of skeletal muscle. J Anat 1968; 122: 555-563

87. Moss FP, Leblond CP. Satellite cells as the source of nuclei in muscles of growing rats. Anat Rec 1971; 170: 421-436

88. Burleigh IG. Observations on the number of nuclei within the fibres of some red and white muscles. J Cell Sci 1977; 23: 269-284

89. Eisenberg BR, Kennedy JM, Wenderoth MP, Dix DJ. Anonymous *Cellular and Molecular Biology of Muscle Development*. New York: A.R. Liss, 1989; 451-460

90. Landing BH, Dixon LG, Wells TR. Studies on isolated human skeletal muscle fibers, including a proposed pattern of nuclear distribution and a concept of nuclear territories. Hum Pathol 1974; 5: 441-461

91. Schmalbruch H. Muscle regeneration: Fetal myogenesis in a new setting. Bib Anat 1986; 29: 126-153

92. Allen RE, Merkel RA, Young RB. Cellular aspects of muscle growth: Myogenic cell proliferation. J Anim Sci 1979; 49: 115-127

93. Bourke DL, Wylie SR, Theon A, et al. Myosin heavy chain expression following transfer into regenerating chicken muscle. Basic Appl Myol 1995; 5: 43-56

94. Allen DL, Monke SR, Talmadge RJ, et al. Plasticity of myonuclear number in hypertrophied and atrophied mammalian skeletal muscle fibers. J Appl Physiol 1995; 78: 1969-1976

95. Rosenblatt JD, Parry DJ. Gamma irradiation prevents compensatory hypertrophy of overloaded mouse extensor digitorum longus muscle. J Appl Physiol 1992; 73: 2538-2543

96. Rosenblatt JD, Parry DJ. Adaptation of rat extensor digitorum longus muscle to gamma irradiation and overload. Pflugers Arch 1993; 423: 255-264

97. Rosenblatt JD, Yong D, Parry DJ. Satellite cell activity is required for hypertrophy of overloaded adult rat muscle. Muscle Nerve 1994; 17: 608-613

98. Kadi F, Eriksson A, Holmner S, et al. Cellular adaptation of the trapezius muscle in strength-trained athletes. Histochem Cell Biol 1999; 111: 189-195

99. Kadi F, Eriksson A, Holmner S, et al. Effects of anabolic steroids on the muscle cells of strength-trained athletes. Med Sci Sports Exerc 1999; 31: 1528-1534

100. Kadi F, Thornell LE. Concomitant increases in myonuclear and satellite cell content in female trapezius muscle following strength training. Histochem Cell Biol 2000; 113: 99-103

101. Roth S, Martel G, Ivey F, et al. Skeletal muscle satellite cell characteristics in young and older men and women after heavy resistance strength training. 2001;

102. Kadi F, Schjerling P, Andersen LL, et al. The effects of heavy resistance training and detraining on satellite cells in human skeletal muscles. J Physiol 2004; 558: 1005-1012

103. Crameri RM, Langberg H, Magnusson P, et al. Changes in satellite cells in human skeletal muscle after a single bout of high intensity exercise. J Physiol 2004; 558: 333-340

104. Hikida R, Staron R, Hagerman F, et al. Effects of high-intensity resistance training on untrained older men. II. muscle fiber characteristics and nucleo-cytoplasmic relationships. J Gerontol A Biol Sci Med Sci 2000; 55: B347-354

105. Sartorelli V, Fulco M. Molecular and cellular determinants of skeletal muscle atrophy and hypertrophy. Sci STKE 2004; 2004: re11

106. Reitsma W. Skeletal muscle hypertrophy after heavy exercise in rats with surgically reduced muscle function. Am J Phys Med 1969; 48: 237-258

107. Appell HJ. Muscular atrophy following immobilisation. A review. Sports Med 1990; 10: 42-58

108. Edgerton VR. Morphology and histochemistry of the soleus muscle from normal and exercised rats. Am J Anat 1970; 127: 81-88

109. Carrow RE, Heusener WW, Van Huss, W. D. Exercise and the incidence of muscle fibre splitting. Br Assoc Sports Med J 1973; 7: 39-41

110. Ho KW, Roy RR, Tweedle CD, et al. Skeletal muscle fiber splitting with weight-lifting exercise in rats. Am J Anat 1980; 157: 433-440

111. Gollnick PD, Timson BF, Moore RL, et al. Muscular enlargement and number of fibers in skeletal muscles of rats. J Appl Physiol 1981; 50: 936-943

112. Gonyea WJ, Sale DG, Gonyea FB, et al. Exercise induced increases in muscle fiber number. Eur J Appl Physiol 1986; 55: 137-141

113. Kelley G. Mechanical overload and skeletal muscle fiber hyperplasia: A meta-analysis. J Appl Physiol 1996; 81: 1584-1588

114. Antonio J, Gonyea WJ. Skeletal muscle fiber hyperplasia. Med Sci Sports Exerc 1993; 25: 1333-1345

115. Sjöström M, Lexell J, Eriksson A, et al. Evidence of fibre hyperplasia in human skeletal muscles from healthy young men? A left-right comparison of the fibre number in whole anterior tibialis muscles. Eur J Appl Physiol 1991; 62: 301-304

116. Mauro M. Muscle Regeneration. New York: Raven Press, 1979

117. Appell HJ, Forsberg S, Hollmann W. Satellite cell activation in human skeletal muscle after training: Evidence for muscle fiber neoformation. Int J Sports Med 1988; 9: 297-299

118. Tesch PA, Larsson L. Muscle hypertrophy in bodybuilders. Eur J Appl Physiol 1982; 49: 301-306

119. Larsson L, Tesch PA. Motor unit fibre density in extremely hypertrophied skeletal muscles in man. electrophysiological signs of muscle fibre hyperplasia. Eur J Appl Physiol 1986; 55: 130-136

120. MacDougall JD, Sale DG, Elder GC, et al. Muscle ultrastructural characteristics of elite powerlifters and bodybuilders. Eur J Appl Physiol 1982; 48: 117-126

121. Bell DG, Jacobs I. Muscle fibre area, fibre type & capillarization in male and female body builders. Can J Sport Sci 1990; 15: 115-119

122. Alway SE, Grumbt WH, Gonyea WJ, et al. Contrasts in muscle and myofibers of elite male and female bodybuilders. J Appl Physiol 1989; 67: 24-31

123. McCall GE, Byrnes WC, Dickinson A, et al. Muscle fiber hypertrophy, hyperplasia, and capillary density in college men after resistance training. J Appl Physiol 1996; 81: 2004-2012

124. MacDougall JD, Sale DG, Alway SE, et al. Muscle fiber number in biceps brachii in bodybuilders and control subjects. J Physiol 1984; 57: 1399-1403

125. Burke RE, Kanda K, Mayer RF. The effect of chronic immobilisation on defined types of motor units in cat medial gastrocnemius. Science 1975; 174: 709-712

126. Walsh JV Jr, Burke RE, Rymer WZ, et al. Effect of compensatory hypertrophy studied in individual motor units in medial gastrocnemius muscle of the cat. J Neurophysiol 1978; 41: 496-508

127. Haddad F, Qin AX, Zeng M, et al. Effects of isometric training on skeletal myosin heavy chain expression. J Appl Physiol 1998; 84: 2036-2041

128. Hather BM, Tesch PA, Buchanan P, et al. Influence of eccentric actions on skeletal muscle adaptations to resistance training. Acta Physiol Scand 1991; 143: 177-185

129. Carroll TJ, Abernethy PJ, Logan PA, et al. Resistance training frequency: Strength and myosin heavy chain responses to two and three bouts per week. Eur J Appl Physiol 1998; 78: 270-275

130. Schiaffino S, Gorza L, Sartore S, et al. Three myosin heavy chain isoforms in type 2 skeletal muscle fibres. J Muscle Res Cell Motil 1989; 10: 197-205

131. Andersen J, Aagaard P. Myosin heavy chain IIX overshoot in human skeletal muscle. Muscle Nerve 2000; 23: 1095-1104

132. Williamson DL, Gallagher PM, Carroll CC, et al. Reduction in hybrid single muscle fiber proportions with resistance training in humans. J Appl Physiol 2001; 91: 1955-1961

133. Andersen LL, Andersen JL, Magnusson SP, et al. Changes in the human muscle force-velocity relationship in response to resistance training and subsequent detraining. J Appl Physiol 2005; 99: 87-94

134. Claassen H, Gerber C, Hoppeler H, et al. Muscle filament spacing and short-term heavy-resistance exercise in humans. J Physiol 1989; 409: 491-495

135. Horber FF, Scheidegger JR, Grünig BE, et al. Thigh muscle mass and function in patients treated with glucocorticoids. Eur J Clin Invest 1985; 15: 302-307

136. Jones DA, Rutherford OM. Human muscle strength training: The effects of three different regimens and the nature of the resultant changes. J Physiol 1987; 391: 1-11

137. Sipilä S, Suominen H. Effects of strength and endurance training on thigh and leg muscle mass and composition in elderly women. J Appl Physiol 1995; 78: 334-340

138. Goldspink G. The combined effects of exercise and reduced food intake on skeletal muscle fibers. J Cell Comp Physiol 1964; 63: 209-219

139. Esmarck B, Andersen JL, Olsen S, et al. Timing of postexercise protein intake is important for muscle hypertrophy with resistance training in elderly humans. J Physiol 2001; 535: 301-311

140. Trappe S, Williamson D, Godard M, et al. Effect of resistance training on single muscle fiber contractile function in older men. J Appl Physiol 2000; 89: 143-152

141. Godard MP, Gallagher PM, Raue U, et al. Alterations in single muscle fiber calcium sensitivity with resistance training in older women. Pflugers Arch 2002; 444: 419-425

142. Shoepe TC, Stelzer JE, Garner DP, et al. Functional adaptability of muscle fibers to long-term resistance exercise. Med Sci Sports Exerc 2003; 35: 944-951

143. Street SF. Lateral transmission of tension in frog myofibers: A myofibrillar network and transverse cytoskeletal connections are possible transmitters. J Cell Physiol 1983; 114: 346-364

144. Goldberg AL, Etlinger JD, Goldspink DF, et al. Mechanism of work-induced hypertrophy of skeletal muscle. Med Sci Sports Exerc 1975; 7: 248-261

145. Huxley AF. Muscle structure and theories of contraction. Prog Biophysics Biophysical Chem 1957; 7: 255-318

146. Viidik A. Tensile strength properties of achilles tendon systems in trained and untrained rabbits. Acta Orthop Scand 1969; 40: 261-272

147. Woo SL, Gomez MA, Amiel D, et al. The effects of exercise on the biomechanical and biochemical properties of swine digital flexor tendons. Biomech Eng 1981; 103: 51-56

148. Kubo K, Kanehisa H, Fukunaga T. Effects of different duration isometric contractions on tendon elasticity in human quadriceps muscles. J Physiol 2001; 536: 649-55

149. Kubo K, Kanehisa H, Fukunaga T. Effects of resistance and stretching training programmes on the viscoelastic properties of human tendon structures in vivo. J Physiol 2002; 538: 219-226

150. Reeves ND, Maganaris CN, Narici MV. Effect of strength training on human patella tendon mechanical properties of older individuals. J Physiol 2003; 548: 971-981

151. Bojsen-Møller J, Magnusson SP, Rasmussen LR, et al. Muscle performance during maximal isometric and dynamic contractions is influenced by the stiffness of the tendinous structures. J Appl Physiol 2005; 99: 986-994

152. Kongsgaard M, Aagaard P, Kjaer M, et al. Structural achilles tendon properties in athletes subjected to different exercise modes and in achilles tendon rupture patients. J Appl Physiol 2005 in press;

153. Sommer HM. The biomechanical and metabolic effects of a running regime on the achilles tendon in the rat. Int Orthop 1987; 11: 71-75

154. Birch HL, McLaughlin L, Smith RK, et al. Treadmill exercise-induced tendon hypertrophy: Assessment of tendons with different mechanical functions. Equine Vet J Suppl 1999; 30: 222-226

155. Woo SL, Ritter MA, Amiel D, et al. The biomechanical and biochemical properties of swine tendons--long term effects of exercise on the digital extensors. Connect Tissue Res 1980; 7: 177-183

156. Wood TO, Cooke PH, Goodship AE. The effect of exercise and anabolic steroids on the mechanical properties and crimp morphology of the rat tendon. Am J Sports Med 1988; 16: 153-158

157. Michna H, Hartmann G. Adaptation of tendon collagen to exercise. Int Orthop 1989; 13: 161-165

158. Alexander RM, Vernon A. The dimensions of the knee and ankle muscles and the forces they exert. J Hum Movt Stud 1975; 1: 115-123

159. Kawakami Y, Abe T, Fukunaga T. Muscle-fiber pennation angles are greater in hypertrophied than in normal muscles. J Appl Physiol 1993; 74: 2740-2744

160. Ichinose Y, Kanehisa H, Ito M, et al. Relationship between muscle fiber pennation and force generation capability in olympic athletes. Int J Sports Med 1998; 19: 541-546

161. Abe T, Brechue WF, Fujita S, et al. Gender differences in FFM accumulation and architectural characteristics of muscle. Med Sci Sports Exerc 1998; 30: 1066-1070

162. Rutherford OM, Jones DA. Measurement of fibre pennation using ultrasound in the human quadriceps in vivo. Eur J Appl Physiol 1992; 65: 433-437

163. Kanehisa H, Nagareda H, Kawakami Y, et al. Effects of equivolume isometric training programs comprising medium or high resistance on muscle size and strength. Eur J Appl Physiol 2002; 87: 112-119

164. Kawakami Y, Abe T, Kuno SY, et al. Training-induced changes in muscle architecture and specific tension. Eur J Appl Physiol Occup Physiol 1995; 72: 37-43

165. Reeves ND, Narici MV, Maganaris CN. Effect of resistance training on skeletal muscle-specific force in elderly humans. J Appl Physiol 2004; 96: 885-892

166. Sale DG, MacDougall JD, Upton AR, et al. Effect of strength training upon motoneuron excitability in man. Med Sci Sports Exerc 1983; 15: 57-62

167. Gandevia SC. Spinal and supraspinal factors in human muscle fatigue. Physiol Rev 2001; 81: 1725-1789

168. Rutherford OM, Jones DA. The role of learning and coordination in strength training. Eur J Appl Physiol 1986; 55: 100-105

169. Horak FB, Macpherson JM. Postural orientation and equilibrium. In: Rowell LB, Shepherd JT, eds. Handbook of Physiology: Section 12 Exercise: Regulation and integration of multiple systems. New York: Oxford University Press, 1996; 255-292

170. Wilson GJ, Murphy AJ, Walshe A. The specificity of strength training: The effect of posture. Eur J Appl Physiol Occup Physiol 1996; 73: 346-352

171. Nozaki D, Nakazawa K, Akai M. Uncertainty of knee joint muscle activity during knee joint torque exertion: The significance of controlling adjacent joint torque. J Appl Physiol 2005; 99: 1093-1103

172. Komi PV, Viitasalo JT, Rauramaa R, et al. Effect of isometric strength training of mechanical, electrical, and metabolic aspects of muscle function. Eur J Appl Physiol Occup Physiol 1978; 40: 45-55

173. Moritani T, deVries HA. Neural factors versus hypertrophy in the time course of muscle strength gain. Am J Phys Med 1979; 58: 115-130

174. Patten C, Kamen G, Rowland D. Adaptations in maximal motor unit discharge rate to strength training in young and older adults. Muscle Nerve 2001; 24: 542-550

175. Zhou S. Chronic neural adaptations to unilateral exercise: Mechanisms of cross education. Exerc Sport Sci Rev 2000; 28: 177-184

176. Sale DG. Neural adaptation to resistance training. Med Sci Sports Exerc 1988; 20: S135-S145

177. Davies J, Parker DF, Rutherford OM, et al. Changes in strength and cross sectional area of the elbow flexors as a result of isometric strength training. Eur J Appl Physiol 1988; 57: 667-670

178. Young A, Stokes M, Round JM, et al. The effect of high-resistance training on the strength and cross-sectional area of the human quadriceps. Eur J Clin Invest 1983; 13: 411-417

179. Hortobágyi T, Scott K, Lambert J, et al. Cross-education of muscle strength is greater with stimulated than voluntary contractions. Motor Control 1999; 3: 205-219

180. Farthing JP, Chilibeck PD. The effect of eccentric training at different velocities on cross-education. Eur J Appl Physiol 2003; 89: 570-577

181. Seger JY, Thorstensson A. Effects of eccentric versus concentric training on thigh muscle strength and EMG. Int J Sports Med 2005; 26: 45-52

182. Shima N, Ishida, K.Katayama, K.Morotome, Y.Sato,Y.Miyamura, M. Cross education of muscular strength during unilateral resistance training and detraining. Eur J Appl Physiol 2002; 86: 287-294

183. Yue G, Cole KJ. Strength increases from the motor program: Comparison of training with maximal voluntary and imagined muscle contractions. J Neurophysiol 1992; 67: 1114-1123

184. Zijdewind I, Toering ST, Bessem B, et al. Effects of imagery motor training on torque production of ankle plantar flexor muscles. Muscle Nerve 2003; 28: 168-173

185. Sidaway B, Trzaska AR. Can mental practice increase ankle dorsiflexor torque? Phys Ther 2005; 85: 1053-1060

186. Herbert RD, Dean C, Gandevia SC. Effects of real and imagined training on voluntary muscle activation during maximal isometric contractions. Acta Physiol Scand 1998; 163: 361-368

187. Behm DG, Whittle J, Button D, et al. Intermuscle differences in activation. Muscle Nerve 2002; 25: 236-243

188. Westing SH, Seger JY, Karlson E, et al. Eccentric and concentric torque-velocity characteristics of the quadriceps femoris in man. Eur J Appl Physiol Occup Physiol 1988; 58: 100-104

189. Dudley GA, Harris RT, Duvoisin MR, et al. Effect of voluntary vs. artificial activation on the relationship of muscle torque to speed. J Appl Physiol 1990; 69: 2215-2221

190. Hakkinen K, Komi PV. Electromyographic changes during strength training and detraining. Med Sci Sports Exerc 1983; 15: 455-460

191. Reeves ND, Maganaris CN, Narici MV. Plasticity of dynamic muscle performance with strength training in elderly humans. Muscle Nerve 2005; 31: 355-364

192. Weir JP, Housh DJ, Housh TJ, et al. The effect of unilateral eccentric weight training and detraining on joint angle specificity, cross-training, and the bilateral deficit. J Orthop Sports Phys Ther 1995; 22: 207-215

193. Aagaard P, Simonsen E, Andersen J, et al. Increased rate of force development and neural drive of human skeletal muscle following resistance training. J Appl Physiol 2002; 93: 1318-1326

194. Holtermann A, Roeleveld K, Vereijken B, et al. Changes in agonist EMG activation level during MVC cannot explain early strength improvement. Eur J Appl Physiol 2005; 94: 593-601

195. Hicks AL, Cupido CM, Martin J, et al. Muscle excitation in elderly adults: The effects of training. Muscle Nerve 1992; 15: 87-93

196. Medbø JI, Jebens E, Vikne H, et al. Effect of strenuous strength training on the na-K pump concentration in skeletal muscle of well-trained men. Eur J Appl Physiol 2001; 84: 148-154

197. Dela F, Holten M, Juel C. Effect of resistance training on na,K pump and Na+/H+ exchange protein densities in muscle from control and patients with type 2 diabetes. Pflugers Arch 2004; 447: 928-933

198. Fuentes I, Cobos AR, Segade LA. Muscle fibre types and their distribution in the biceps and triceps brachii of the rat and rabbit. J Anat 1998; 192: 203-210

199. Duchateau J, Hainaut K. Isometric or dynamic training: Differential effects on mechanical properties of a human muscle. J Appl Physiol 1984; 56: 296-301

200. Van Cutsem M, Duchateau J, Hainaut K. Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. J Physiol 1998; 513: 295-305

201. Rich C, Cafarelli E. Submaximal motor unit firing rates after 8 wk of isometric resistance training. Med Sci Sports Exerc 2000; 32: 190-196

202. Bigland B, Lippold OC. Motor unit activity in the voluntary contraction of human muscle. J Physiol 1954; 125: 322-335

203. Bigland-Ritchie B, Johansson R, Lippold OC, et al. Contractile speed and EMG changes during fatigue of sustained maximal voluntary contractions. J Neurophysiol 1983; 50: 313-324

204. Grimby L, Hannerz J, Hedman B. The fatigue and voluntary discharge properties of single motor units in man. J Physiol 1981; 316: 545-554

205. Lyle N, Rutherford OM. A comparison of voluntary versus stimulated strength training of the human adductor pollicis muscle. J Sports Sci 1998; 16: 267-270

206. McDonagh MJ, Hayward CM, Davies CT. Isometric training in human elbow flexor muscles. the effects on voluntary and electrically evoked forces. J Bone Joint Surg Br 1983; 65: 355-358

207. Davies CT, Dooley P, McDonagh MJ, et al. Adaptation of mechanical properties of muscle to high force training in man. J Physiol 1985; 365: 277-284

208. Maffiuletti NA, Cometti G, Amiridis IG, et al. The effects of electromyostimulation training and basketball practice on muscle strength and jumping ability. Int J Sports Med 2000; 21: 437-443

209. Colson S, Martin A, Van Hoecke J. Re-examination of training effects by electrostimulation in the human elbow musculoskeletal system. Int J Sports Med 2000; 21: 281-288

210. Zhou S, Oakman A, Davie A. Effects of unilateral voluntary and electromyostimulation training on muscular strength of the contralateral limb. Hong Kong J Sports Med Sports Sci 2002; 14: 1-11

211. Lieber RL, Silva PD, Daniel DM. Equal effectiveness of electrical and volitional strength training for quadriceps femoris muscles after anterior cruciate ligament surgery. J Orthop Res 1996; 14: 131-138

212. Ruther CL, Golden CL, Harris RT, et al. Hypertrophy, resistance training, and the nature of skeletal muscle activation. J Strength Cond Res 1995; 9: 155-159

213. Merton PA. Voluntary strength and fatigue. J Physiol 1954; 123: 553-564

214. Belanger AY, McComas AJ. Extent of motor unit activation during effort. J Appl Physiol 1981; 51: 1131-1135

215. Rutherford OM, Jones DA, Newham DJ. Clinical and experimental application of the percutaneous twitch superimposition technique for the study of human muscle activation. J Neurol Neurosurg Psychiatry 1986; 49: 1248-1291

216. Folland J, Williams A. Methodological issues with the interpolated twitch technique. J Electromyog Kinesiol 2006; in press

217. Shield A, Zhou S. Assessing voluntary muscle activation with the twitch interpolation technique. Sports Med 2004; 34: 253-267

218. Nørregaard J, Bülow PM, Danneskiold-Samsøe B. Muscle strength, voluntary activation, twitch properties, and endurance in patients with fibromyalgia. J Neurol Neurosurg Psychiatry 1994; 57: 1106-1111

219. Jakobi JM, Cafarelli E. Neuromuscular drive and force production are not altered during bilateral contractions. J Appl Physiol 1998; 84: 200-206

220. Roos MR, Rice CL, Connelly DM, et al. Quadriceps muscle strength, contractile properties, and motor unit firing rates in young and old men. Muscle Nerve 1999; 22: 1094-1103

221. Kalmar JM, Cafarelli E. Effects of caffeine on neuromuscular function. J Appl Physiol 1999; 87: 801-808

222. Brown AB, McCartney N, Sale DG. Positive adaptations to weight-lifting training in the elderly. J Appl Physiol 1990; 69: 1725-1733

223. Scaglioni G, Ferri A, Minetti A, et al. Plantar flexor activation capacity and H reflex in older adults: Adaptations to strength training. J Appl Physiol 2002; 92: 2292-2302

224. Knight C, Kamen G. Adaptations in muscular activation of the knee extensor muscles with strength training in young and older adults. J Electromyogr Kinesiol 2001; 11: 405-412

225. Becker R, Awiszus F. Physiological alterations of maximal voluntary quadriceps activation by changes of knee joint angle. Muscle Nerve 2001; 24: 667-672

226. Kubo K, Tsunoda N, Kanehisa H, et al. Activation of agonist and antagonist muscles at different joint angles during maximal isometric efforts. Eur J Appl Physiol 2004; 91: 349-352

227. Perrine JJ, Edgerton VR. Muscle force-velocity and power-velocity relationships under isokinetic loading. Med Sci Sports 1978; 10: 159-166

228. Caiozzo VJ, Perrine JJ, Edgerton VR. Training-induced alterations of the in vivo force-velocity relationship of human muscle. J Appl Physiol 1981; 51: 750-754

229. Newham DJ, McCarthy T, Turner J. Voluntary activation of human quadriceps during and after isokinetic exercise. J Appl Physiol 1991; 71: 2122-2126

230. Gandevia SC, Herbert RD, Leeper JB. Voluntary activation of human elbow flexor muscles during maximal concentric contractions. J Physiol 1998; 512: 595-602

231. Babault N, Pousson M, Ballay Y, et al. Activation of human quadriceps femoris during isometric, concentric, and eccentric contractions. J Appl Physiol 2001; 91: 2628-2634

232. Amiridis IG, Martin A, Morlon B, et al. Co-activation and tension-regulating phenomena during isokinetic knee extension in sedentary and highly skilled humans. Eur J Appl Physiol Occup Physiol 1996; 73: 149-156

233. Hortobagyi T, Lambert NJ. Influence of electrical stimulation on dynamic forces of the arm flexors in strength-trained and untrained men. Scand J Med Sci Sports 1992; 2: 70-75

234. Westing SH, Seger JY, Thorstensson A. Effects of electrical stimulation on eccentric and concentric torque-velocity relationships during knee extension in man. Acta Physiol Scand 1990; 140: 17-22

235. Hortobágyi T, Hill JP, Houmard JA, et al. Adaptive responses to muscle lengthening and shortening in humans. J Appl Physiol 1996; 80: 765-772

236. Kukulka CG, Clamann HP. Comparison of the recruitment and discharge properties of motor units in human brachial biceps and adductor pollicis during isometric contractions. Brain Res 1981; 219: 45-55

237. De Luca CJ, LeFever RS, McCue MP, et al. Behaviour of human motor units in different muscles during linearly varying contractions. J Physiol 1982; 329: 113-128

238. Solomonow M, Baten C, Smit J, et al. Electromyogram power spectra frequencies associated with motor unit recruitment strategies. J Appl Physiol 1990; 68: 1177-1185

239. Monster AW, Chan H. Isometric force production by motor units of extensor digitorum communis muscle in man. J Neurophysiol 1977; 40: 1432-1443

240. Bellemare F, Woods JJ, Johansson R, et al. Motor-unit discharge rates in maximal voluntary contractions of three human muscles. J Neurophysiol 1983; 50: 1380-1392

241. Thomas CK, Bigland-Richie B, Johansson RS. Force-frequency relationships of human thenar motor units. J Neurophysiol 1991; 65: 1509-1516

242. Macefield VG, Fuglevand AJ, Bigland-Ritchie B. Contractile properties of single motor units in human toe extensors assessed by intraneural motor axon stimulation. J Neurophysiol 1996; 75: 2509-2519

243. Binder-Macleod SA, Barker CB,3rd. Use of a catchlike property of human skeletal muscle to reduce fatigue. Muscle Nerve 1991; 14: 850-857

244. Duchateau J, Hainaut K. Nonlinear summation of contractions in striated muscle. I. twitch potentiation in human muscle. J Muscle Res Cell Motil 1986; 7: 11-17

245. Leong B, Kamen G, Patten C, et al. Maximal motor unit discharge rates in the quadriceps muscles of older weight lifters. Med Sci Sports Exerc 1999; 31: 1638-1644

246. Milner-Brown HS, Stein RB, Lee RG. Synchronization of human motor units: Possible roles of exercise and supraspinal reflexes. Electroencephalogr Clin Neurophysiol 1975; 38: 245-254

247. Semmler JG, Nordstrom MA. Motor unit discharge and force tremor in skill- and strength-trained individuals. Exp Brain Res 1998; 119: 27-38

248. Rack PM, Westbury DR. The effects of length and stimulus rate on tension in the isometric cat soleus muscle. J Physiol 1969; 204: 443-460

249. Lind AR, Petrofsky JS. Isometric tension from rotary stimulation of fast and slow cat muscles. Muscle Nerve 1978; 1: 213-218

250. Perez MA, Lungholt BK, Nyborg K, et al. Motor skill training induces changes in the excitability of the leg cortical area in healthy humans. Exp Brain Res 2004; 159: 197-205

251. Pascual-Leone A, Nguyet D, Cohen LG, et al. Modulation of muscle responses evoked by transcranial magnetic stimulation during the acquisition of new fine motor skills. J Neurophysiol 1995; 74: 1037-1045

252. Lotze M, Braun C, Birbaumer N, et al. Motor learning elicited by voluntary drive. Brain 2003; 126:

253. Karni A, Meyer G, Jezzard P, et al. Functional MRI evidence for adult motor cortex plasticity during motor skill learning. Nature 1995; 377: 155-158

254. Hund-Georgiadis M, von Cramon DY. Motor-learning-related changes in piano players and non-musicians revealed by functional magnetic-resonance signals. Exp Brain Res 1999; 125: 417-425

255. Elbert T, Pantev C, Wienbruch C, et al. Increased cortical representation of the fingers of the left hand in string players. Science 1995; 270: 305-307

256. Classen J, Liepert J, Wise SP, et al. Rapid plasticity of human cortical movement representation induced by practice. J Neurophysiol 1998; 79: 1117-1123

257. Classen J, Liepert J, Hallett M, et al. Plasticity of movement representation in the human motor cortex. Electroencephalogr Clin Neurophysiol Suppl 1999; 51: 162-173

258. Carroll TJ, Riek S, Carson RG. The sites of neural adaptation induced by resistance training in humans. J Physiol 2002; 544: 641-652

259. Jensen JL, Marstrand PC, Nielsen JB. Motor skill training and strength training are associated with different plastic changes in the central nervous system. J Appl Physiol 2005; 99: 1558-1568

260. Hallett M, Berardelli A, Delwaide P, et al. Central EMG and tests of motor control. report of an IFCN committee. Electroencephalogr Clin Neurophysiol 1994; 90: 404-432

261. Aagaard P, Simonsen EB, Andersen JL, et al. Neural adaptation to resistance training: Changes in evoked V-wave and H-reflex responses. J Appl Physiol 2002; 92: 2309-2318

262. Sale D, McComas A, MacDougall J, et al. Neuromuscular adaptation in human thenar muscles following strength training and immobilization. J Appl Physiol 1982; 53: 419-424

263. Enoka RM. Neuromechanics of human movement. 3rd ed. Champaign, Ill.: Human Kinetics, 2002

264. Karst GM, Hasan Z. Antagonist muscle activity during human forearm movements under varying kinematic and loading conditions. Exp Brain Res 1987; 67: 391-401

265. Baratta R, Solomonow M, Zhou BH, et al. Muscular coactivation. the role of the antagonist musculature in maintaining knee stability. Am J Sports Med 1988; 16: 113-122

266. Osternig LR, Hamill J, Lander JE, et al. Co-activation of sprinter and distance runner muscles in isokinetic exercise. Med Sci Sports Exerc 1986; 18: 431-435

267. Carolan B, Cafarelli E. Adaptations in coactivation after isometric resistance training. J Appl Physiol 1992; 73: 911-917

268. Häkkinen K, Kallinen M, Izquierdo M, et al. Changes in agonist-antagonist EMG, muscle CSA, and force during strength training in middle-aged and older people. J Appl Physiol 1998; 84: 1341-1349

View publication stats