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The Adaptations to Strength Training

Morphological and Neurological Contributions to Increased Strength

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Contents

Abstract . . . 146
1. Morphological Adaptations . . . 146
  1.1 Changes in Whole-Muscle Size . . . 146
    1.1.1 Influence of Muscle Group . . . 148
    1.1.2 Influence of Sex . . . 148
    1.1.3 Influence of Age . . . 148
    1.1.4 Selective Growth (Hypertrophy) . . . 148
  1.2 Muscle Fibre Hypertrophy . . . 149
    1.2.1 Preferential Hypertrophy of Type 2 Fibres . . . 149
  1.3 Myofibrillar Growth and Proliferation . . . 150
    1.3.1 A Possible Mechanism of Myofibrillar Proliferation . . . 150
    1.3.2 Satellite Cells . . . 151
  1.4 Hyperplasia . . . 152
    1.4.1 Animal Studies . . . 152
    1.4.2 Human Studies . . . 152
  1.5 Other Morphological Adaptations . . . 153
    1.5.1 Changes in Fibre Type and Myosin Heavy-Chain Composition? . . . 153
    1.5.2 Density of Skeletal Muscle and Myofilaments . . . 153
    1.5.3 Tendon and Connective Tissue . . . 153
    1.5.4 Muscle Architecture . . . 154
2. Neurological Adaptations . . . 155
  2.1 Indirect Evidence of Neural Adaptations, Learning and Coordination . . . 155
    2.1.1 Specificity of Training Adaptations . . . 155
    2.1.2 Cross-over Training Effect . . . 156
    2.1.3 Imagined Contractions . . . 156
  2.2 A Change in Agonist Activation? . . . 156
    2.2.1 Electromyography . . . 156
    2.2.2 Tetanic Stimulation . . . 157
    2.2.3 Interpolated Twitch Technique . . . 158
    2.2.4 Dynamic Muscle Activity . . . 158
  2.3 Specific Mechanisms of Neurological Adaptation . . . 159
    2.3.1 Firing Frequency . . . 159
    2.3.2 Synchronisation . . . 159
    2.3.3 Cortical Adaptations . . . 160
    2.3.4 Spinal Reflexes . . . 160
    2.3.5 Antagonist Coactivation . . . 160
3. Conclusion . . . 161

Abstract

High-resistance strength training (HRST) is one of the most widely practiced forms of physical activity, which is used to enhance athletic performance, augment musculo-skeletal health and alter body aesthetics. Chronic exposure to this type of activity produces marked increases in muscular strength, which 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, which is due to an increase in myofibrillar size and number. Satellite cells are activated in the very early stages of training; 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 tendons.

Indirect evidence for neurological adaptations, which encompasses learning and coordination, 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; however, morphological factors (e.g. preferential hypertrophy of type 2 fibres, increased angle of fibre pennation, increase in radiological density) are also likely to contribute to this phenomenon. Changes in inter-muscular coordination appear critical. Adaptations in agonist muscle activation, as assessed by 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.

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 alter body aesthetics. The health benefits of HRST are primarily as a countermeasure to any circumstance where muscle weakness compromises function (e.g. sarcopenia, neuromusculo-skeletal disorders, or following immobilisation, 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 HRST, assessing the evidence for these adaptations, their interplay and contribution to enhanced strength and the methodologies employed.

1. Morphological Adaptations

1.1 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. This has been extensively documented in the scientific literature. Investigations employing a range of scanning techniques (e.g. magnetic resonance imaging [MRI]; computerised 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, because of 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 growth (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 ACSA 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 compared with ACSA, indicating an increase in whole-muscle specific tension. Whilst of interest, there are numerous methodological problems with the direct comparison of these parameters, mainly involving the methodology of muscle-size measurement. The vast majority of investigations have measured ACSA at just one level as the index of muscle size. A recent reliability study of muscle-size measurement concluded that cross-sectional area (CSA) measured at just one level was less reliable than measurement of multiple sections and should only be used if a relatively large change in 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.

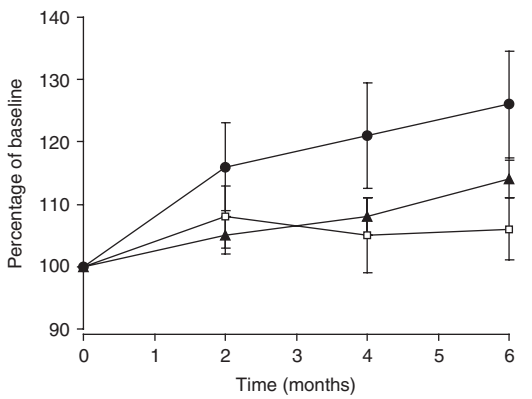

Fig. 1. Isometric maximal voluntary contraction (circles), integrated electromyography (squares) and quadriceps anatomical cross-sectional area (triangles) at mid-thigh during 6 months of strength training (data adapted from Narici et al.,[8] with permission).

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), as assessed by MRI, show significant changes after 8–12 weeks of regular training. 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.

1.1.1 Influence of Muscle Group

A greater hypertrophic response to resistance training has been observed in the upper body muscles compared with lower extremity muscles in previously untrained individuals.[21,22] When standard training was utilised, 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%, respectively. 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 with 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 the intermuscular differences in androgen receptor content, with some evidence for greater concentrations in the upper body muscles compared with lower limb muscles.[24]

1.1.2 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 hypothesised to cause less relative muscle hypertrophy in response to training when compared with 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 (+2.5%, with MRI), but greater increases in strength in women (+25%, 1-repetition maximum; +6% isometric) 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,[24] making them more responsive to higher blood androgen concentrations. The greater strength gains of females might reflect a greater capacity for neural adaptations,[41] 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.

1.1.3 Influence of Age

There is no doubt that older adults, including nonagenarians, undergo skeletal muscle hypertrophy in response to HRST (mid-thigh ACSA: +9% after 8 weeks;[42] +9.8% after 12 weeks[43]). The absolute increase in muscle size is smaller in old adults compared with 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,[34,44] whilst others seem to suggest a smaller hypertrophy response in older individuals.[14,23,45] 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]

1.1.4 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.[4,8,46,47] For example, Housh et al.[4] reported an 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) are each likely to have

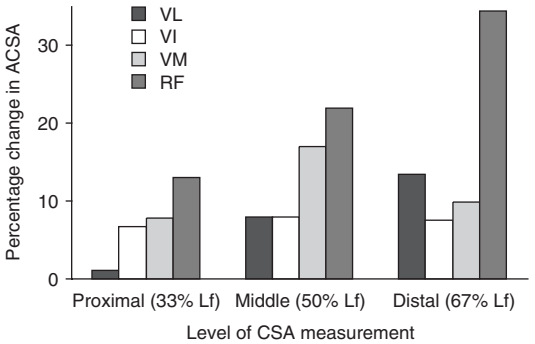

Fig. 2. Selective hypertrophy of the quadriceps femoris muscle after 8 weeks of isokinetic high-resistance strength training. The extent of hypertrophy varies according to the constituent muscle and level of cross-sectional area (CSA) assessment (adapted from the data of Housh et al.,[4] with permission). **ACSA** = anatomical cross-sectional area; **Lf** = length of the femur; **RF** = rectus femoris; **VI** = vastus intermedius; **VL** = vastus lateralis; **VM** = vastus medialis.

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).[5,13,48] However, others have found this to occur in proximal[46] or proximal and distal[8] regions of the muscle, possibly due to the 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 of 16.4 years), followed over 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.[49] 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.

1.2 Muscle Fibre Hypertrophy

An increase in the CSA of skeletal muscle fibres (fibre hypertrophy) is generally regarded as the primary adaptation to long-term strength training and has been widely documented (reviewed by McDonagh and Davies[50] and Jones et al.[51]). Fibre hypertrophy 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 an increase in 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 6 months resulted in type 1 and type 2 fibre hypertrophy of 27 and 33%, respectively.[52] Aagaard et al.[11] found a mean increase of 16% in fibre area after 14 weeks of resistance training, which 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 the mean fibre area despite muscle ACSA increasing by 19%. Such variability may be accounted for by a number of factors, including: (i) the poor reproducibility of the biopsy technique; (ii) the individual's responsiveness to training; and (iii) the precise nature of the training stimulus (e.g. 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 (coefficient of variation = 10–24%).[53-57] This appears to be largely due to heterogeneity of fibre size within skeletal muscle, which may be partially influenced by the depth of the biopsy site,[58] as well as variability in perpendicular slicing of muscle tissue and tracing of cell borders.[56] Thus, while the weight of evidence strongly supports fibre hypertrophy, data from single biopsy samples must be treated with caution.[59]

1.2.1 Preferential Hypertrophy of Type 2 Fibres

Preferential hypertrophy of type 2 fibres after strength training is another commonly reported finding.[60-63] The data presented by Hakkinen et al.[64] indicate a greater plasticity of type 2 fibres since they hypertrophy more rapidly during training and atrophy faster during detraining. Therefore, it is not surprising that many of the shorter studies (6–10 weeks) have only found significant hypertrophy of type 2 fibres,[11,63,65,66] whereas longer studies have more frequently found significant increases in the

fibre area of both type 1 and type 2 fibres.[52,64] The evidence from animal studies supports the greater hypertrophic response of type 2 fibres.[67] The proportion of type 2 fibres in human muscle has been significantly correlated with training-induced hypertrophy[45] and increases in strength.[65] However, strength gains have also been found to be unrelated to fibre composition[68] and positively related to the proportion of type 1 fibres.[63]

It has been suggested that type 2 fibres have a higher specific tension and their preferential hypertrophy contributes to the rise in the specific tension that is 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.[69] concluded that 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 the myosin heavy chain (MHC) IIX isoform, than in fibres expressing purely MHC I (+50%;[70] +20%;[71] +32%[72]). Studies that have related isometric specific tension to the fibre type composition of humans *in vivo* have found contradictory findings.[73-75] However, the proportion of type 2 fibres (or MHC II content) has been positively correlated with isokinetic strength at medium-to-high angular velocities[76] and relative force at high velocities.[73,77]

Recent evidence suggests that type 2 fibres have a significantly greater specific tension that, in combination with their greater hypertrophy response, likely contributes to increases in whole-muscle specific tension.

1.3 Myofibrillar Growth and Proliferation

MacDougall and colleagues[52] examined the myofibrillar structure of six subjects before and after 6 months of strength training. Despite wide variations in size, measurement of >3500 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.[78] 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, which may indicate a 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.[79] 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.[52] is interpreted as evidence for an increase in myofibril number (i.e proliferation) after training.

1.3.1 A Possible Mechanism of Myofibrillar Proliferation

The investigations by MacDougall and colleagues[52,78] 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.[80] In a series of investigations on the growth of post-natal mice, Goldspink[80,81] and Goldspink and Howells[82] proposed a mechanism for myofibrillar proliferation. Discrepancy in the arrays formed at the A and I bands causes 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[80,81] 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 the series may split in a similar manner until the entire myofibril has divided longitudinally. Evi-

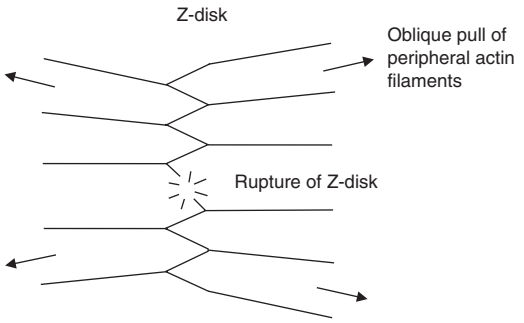

Fig. 3. Myofibrillar splitting due to the oblique pull of the peripheral actin filaments (redrawn from Goldspink,[83] with permission).

dence 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 also likely HRST, myofibrillar proliferation takes place as a result of Z-disk rupture and longitudinal division, which limits myofibrillar size and facilitates their effective control and regulation.

1.3.2 Satellite Cells

Many investigators have found that the ratio of nuclear to cytoplasmic material remains fairly constant throughout a wide range of growth conditions (in animals;[86,87] and 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 2 as for type 1 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 that 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. These authors 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 were higher in elite powerlifters than in untrained controls (total nuclei +35% in type 1 and +31% in type 2 fibres).[98] 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 and Thornell[100] reported myonuclear and satellite cell numbers in the trapezius muscle to increase substantially, and by proportionally more than fibre CSA (figure 4). They concluded 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]

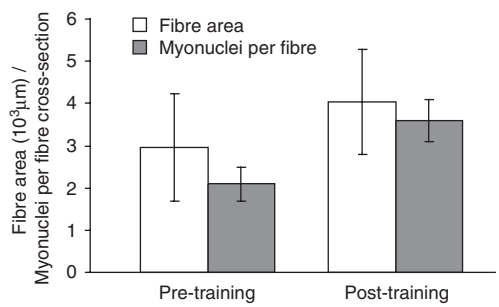

Fig. 4. The increase in fibre area during the early stages (10 weeks) of high-resistance strength training are matched by an increase in myonuclei number from proliferating satellite cells (data from Kadi and Thornell,[100] with kind permission of Springer and Business Media).

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, but thereafter, additional myonuclei derived from satellite cells are required.

In order for 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 regulation of protein synthesis is reviewed by Sartorelli and Fulco.[105]

1.4 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] 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.

1.4.1 Animal Studies

Work-induced splitting of muscle fibres has been observed and is 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. They found no evidence for hyperplasia and attributed muscle enlargement entirely to hypertrophy of existing fibres. In contrast, Gonyea and et al.[112] carried out fibre counts after an average of 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 with histological counting (21%). The degree of hyperplasia also seems to be dependent upon the experimental protocol that is used to induce the overload, with stretch causing more hyperplasia and small or no increase in fibre number with exercise or compensatory hypertrophy.[113,114]

1.4.2 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 et al.[117] found evidence of new myotube formation from satellite cell activity after 6 weeks of endurance training. In response to HRST, Kadi and 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 only have a small effect on muscle CSA and therefore strength improvements. A cadaver study by Sjostrom et al.[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.[54,118-121] 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 1.2, 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 the 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.

1.5 Other Morphological Adaptations

1.5.1 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,[67,125,126] although recent work indicates that more subtle changes can occur, specifically a transition of type 2B to type 2X.[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 2A fibres and a concomitant fall in type 2X fibres,[45,60,61,128] with one study 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 MHC isoforms. Schiaffino et al.[130] identified four 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 generally 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.

1.5.2 Density of Skeletal Muscle and Myofilaments

The gross muscle radiological density of skeletal muscle increases following strength training (+3%;[134] +5%[135,136]). Sipilä 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.[52] and includes all of the constituents of whole muscle (e.g. fat and connective tissue). In rats, the discrepancy in fibre and whole-muscle size increases with overload has been taken to suggest that fibres develop at the expense of the extracellular compartment.[138] It is also interesting to note that many of the human studies employing the muscle biopsy technique have found greater hypertrophy than those using the measurement of anatomical CSA.[11,45,68,139]

Studies of the packing density of myofilaments have found this to be very consistent pre- to post-training.[52,134] 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[72,140,141] and similar for sedentary and long-term (>6 years) 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.

1.5.3 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 hypertro-

phy 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 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 in theory, it could 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 is likely to change 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 with 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 tendons 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 that increased diameter and packing density of collagen fibrils and changes in collagen crimp structure (waviness of fibrils)[156,157] are likely to 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, probably due to a range of structural changes, and tendon hypertrophy also seems probable given a sufficient training period.

1.5.4 Muscle Architecture

The orientation of muscle fascicles (fibres), in relation to connective tissue/tendon and hence the relevant joint mechanics, influences 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 two 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 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 the resting fibre pennation angle of the VL to increase by 28–35%, according to the 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.

These recent studies provide strong 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.

2. 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 with 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 et al.[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 coordination and learning that facilitate improved recruitment and activation of the involved muscles during a specific strength task.

2.1 Indirect Evidence of Neural Adaptations, Learning and Coordination

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 in section 1, 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, notably Aagaard et al.,[11] 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, Gandevia[167] points out that it is difficult to estimate the cumulative effects of these necessary corrections.

2.1.1 Specificity of Training Adaptations

Other indirect, but more forceful, evidence for a substantial neurological adaptation comes from the observation in many strength-training investigations that the increase in dynamic lifting strength (1 repetition maximum) is disproportionately greater than the increase in isometric strength.[65,168] Undoubtedly, such findings point to a considerable facility for learning that is specific to the training task. Some proportion of this task specificity is 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, which may be different for apparently similar exercises.[168] Recent work by Nozaki et al.[171] has highlighted the variability, between and within subjects on a trial-to-trial basis, of inter-muscle coordination and adjacent joint activity, during even seemingly straight-forward single-joint actions (e.g. knee extension). This evidence reinforces the fact that apparently simple actions undoubtedly require a degree of skill in order for optimal expression of strength.

2.1.2 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] (a review is presented by Zhou[175]). 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 coordination (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 coordination/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 coordination 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.

2.1.3 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 be independent of morphological adaptations. Mechanistically, it supports the role of central-cortical adaptations in response to regular HRST.

2.2 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.

2.2.1 Electromyography

Surface electromyograph (SEMG) recordings have been used by many investigators in an attempt to measure the 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.[33,46,48,172,173,190,191] 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

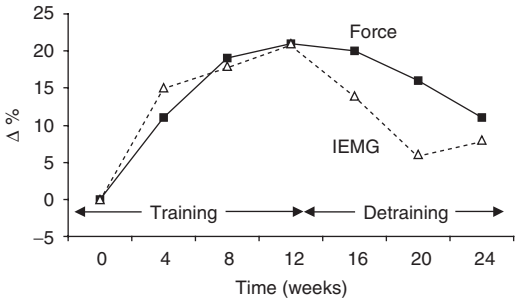

Fig. 5. Changes in the isometric force and surface electromyograph with 16 weeks of training and 8 weeks detraining (redrawn from Hakkinen & Komi,[190] with permission). **IEMG** = integrated electromyography.

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 reflecting an increased neural drive is also considered a simplification. Firstly, 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 that are likely to alter single-fibre action potential and SEMG, including: fibre type; fibre size; membrane potential;[195] intramuscular ionic concentrations; and sodium-potassium pump content.[196,197] Secondly, the large, fast motor units tend to be more abundant towards the periphery of the muscle, close to the skin,[58,198] and any change in their activity may have an exaggerated effect upon SEMG 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 the M-wave remained constant has been found,[199,200] whilst a parallel increase in EMG and M-wave has also 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 2.3.2).

2.2.2 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. As a result of 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, 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 voluntary 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) to that of 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.

2.2.3 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 Folland and Williams[216] and Shield and Zhou[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]

2.2.4 Dynamic Muscle Activity

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

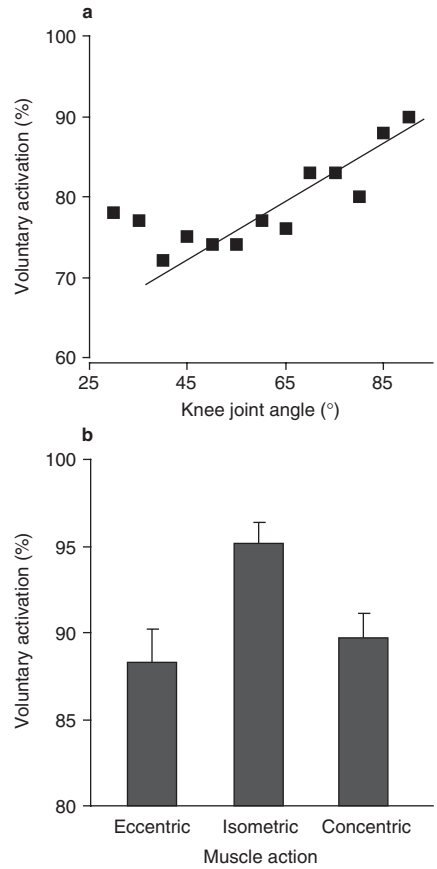

Fig. 6. Recent evidence using the interpolated twitch technique has suggested that the ability to maximally activate the agonist muscle varies with (**a**) joint position/muscle length (redrawn from Becker and Awiszus,[225] with permission of John Wiley & Sons, Inc.) and (**b**) type of muscle action (redrawn from Babault et al.,[227] with permission).

supraspinal activation or perhaps more likely spinal inhibition from a range of afferents (e.g. group Ib Golgi-organ afferents, group Ia, group II and 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 is likely to depend 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).

2.3 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.

2.3.1 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, which is 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 (100–200Hz[200]), with much lower rates at the instant of maximum force generation (20–30Hz[236,237,239,240]). It is curious that with involuntary stimulation the force-frequency relationship observed for motor units in human muscle suggests that discharge rates of at least 50Hz 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 frequencies than expected. An initial, brief, high-frequency burst of 2–4 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, may facilitate 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 (23.8Hz vs 19.1Hz, respectively).[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 are likely to 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 (5th finger abduction), low subject numbers or the short duration of the training.

2.3.2 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,[51,176] as at firing frequencies equivalent to MVC there is no effect of synchronisation upon force.[248,249]

2.3.3 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.

2.3.4 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 reflex (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 is 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 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, which 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 compared with 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]

2.3.5 Antagonist Coactivation

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 coactivation in the

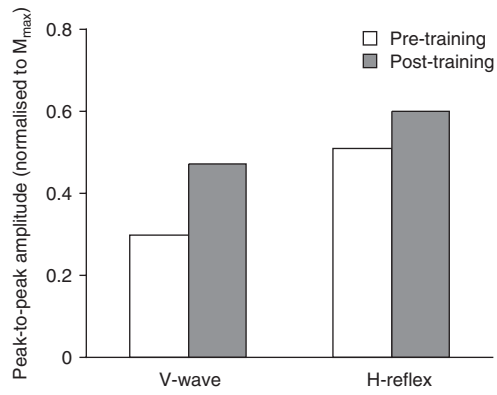

Fig. 7. V-wave and H-reflex amplitude (expressed relative to maximal compound muscle action potential [Mmax]) measured during isometric maximal voluntary contractions before and after 14 weeks of high-resistance strength training (data adapted from Aagaard et al.,[261] with permission).

strength/power of trained athletes than in 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 hamstring coactivation 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 coactivation with training.

3. Conclusion

A wide range of morphological and neurological factors are known to contribute to increased strength following HRST. 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 2 fibres and is accompanied by an increase in the angle of fibre pennation, which promotes a greater increase in PCSA and force production than is revealed by ACSA. These two factors are likely to 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 (e.g. 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 coordination of agonists, antagonists and synergists. The rapid rise in strength at the start of a training programme, within the first 2 weeks, which 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, which 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.

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