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ORIGINAL ARTICLE

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Neuromuscular adaptations to detraining following resistance training in previously untrained subjects

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Abstract Resistance training has been shown to considerably increase strength and neural drive during maximal eccentric muscle contraction; however, less is known about the adaptive change induced by subsequent detraining. The purpose of the study was to examine the effect of dynamic resistance training followed by detraining on changes in maximal eccentric and concentric isokinetic muscle strength, as well as to examine the corresponding adaptations in muscle cross-sectional area (CSA) and EMG activity. Maximal concentric and eccentric isokinetic knee extensor moment of force was measured in 13 young sedentary males (age 23.5 ± 3.2 years), before and after 3 months of heavy resistance training and again after 3 months of detraining. Following training, moment of force increased during slow eccentric (50%, $P < 0.001$), fast eccentric (25%, $P < 0.01$), slow concentric (19%, $P < 0.001$) and fast concentric contraction (11%, $P < 0.05$). Corresponding increases in EMG were observed during eccentric and slow concentric contraction. Significant correlations were observed between the training-induced changes in moment of force and EMG ($R^2 = 0.33$–0.77). Muscle CSA (measured by MRI) increased by 10% ($P < 0.001$). After 3 months of detraining maximal muscle strength and EMG remained preserved during eccentric contraction but not concentric contraction. The present findings suggest that heavy resistance training induces long-lasting strength gains and neural adaptations during maximal eccentric muscle contraction in previously untrained subjects.

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Introduction

The expression of voluntary muscle force is influenced by both muscle cross-sectional area (Schantz et al. 1983), muscle architecture (Aagaard et al. 2001a) and neural drive to the muscle (Karlsson and Gerdle 2001). It has been suggested that a neural force-regulating mechanism exists, preventing utilization of maximum inherent muscle force, during conditions of very high muscle tension (Perrine and Edgerton 1978; Caiozzo et al. 1981; Wickiewicz et al. 1984). Superimposed electrical stimulation onto maximal eccentric muscle contraction yields marked gains in moment of force, which suggest the presence of substantial neural inhibition during this type of contraction (Dudley et al. 1990; Westing et al. 1990; Amiridis et al. 1996). Lowered EMG activity during eccentric relative to concentric or isometric contraction further supports this notion (Tesch et al. 1990; Westing et al. 1991; Seger and Thorstensson 1994, 2000; Higbie et al. 1996; Kellis and Baltzopoulos 1998; Komi et al. 2000; Aagaard et al. 2000). In response to resistance training intervention the most pronounced strength gains typically occur during maximal eccentric muscle contraction (Colliander and Tesch 1990; Aagaard et al. 1996, 2000). Furthermore, hypertrophy of the resistance-trained muscles (Narici et al. 1989, 1996; Aagaard et al. 2001a), changes in muscle architecture (Aagaard et al. 2001a; Reeves et al. 2004) and increased neural drive (Moritani and deVries 1979; Hakkinen and Komi 1983; Hakkinen et al. 1985; Narici et al. 1989; Higbie et al. 1996; Hortobagyi et al. 1996; Aagaard et al. 2000, 2002) have been reported. Thus, it is generally accepted that both neural and muscular morphological adaptations contribute to the strength gains observed in response to resistance training (Moritani and deVries 1979; Narici et al. 1989).

Cessation of resistance training, commonly known as detraining, is typically associated with a diminished physiological function. Reductions in maximal voluntary muscle strength (Hakkinen and Komi 1983; Houston et al. 1983; Narici et al. 1989; Colliander and Tesch 1992), muscle cross-sectional area (Narici et al. 1989) and neural drive to the muscle (Hakkinen and Komi 1983; Hakkinen et al. 1985; Narici et al. 1989) have been reported. In contrast, some studies have reported partially preserved gains in dynamic muscle strength when resistance training was followed by 6–12 weeks (LeMura et al. 2000; Kraemer et al. 2002) and even 30–32 weeks of detraining (Staron et al. 1991; Lemmer et al. 2000). Particularly eccentric muscle strength has been reported to be sensitive to detraining in trained subjects (Hortobagyi et al. 1993; Mujika and Padilla 2001; Kraemer et al. 2002) and in certain muscles of the shoulder (McCarrick and Kemp 2000). On the other hand, in previously untrained subjects a preservation of eccentric strength of the muscles of the lower limb for 8–12 weeks has been reported (Colliander and Tesch 1992; Housh et al. 1996). However, a learning effect (Rutherford and Jones 1986) may have influenced the latter results due to the use of identical training and testing devices. Thus some controversy appears to exist.

Therefore, the aim of the present study was to examine the effect of dynamic resistance training followed by detraining on changes in maximal eccentric and concentric isokinetic muscle strength; and furthermore, to evaluate the corresponding change in muscle cross-sectional area and EMG.

Methods

Subjects

Thirteen healthy sedentary male subjects (age 23.5 ± 3.2 years, stature 179 ± 8 cm, body mass 75.4 ± 9.6 kg, mean ± SD) volunteered to participate in the present training study. Additionally, ten subjects (age 23.9 ± 3.1 years, stature 181 ± 8 cm, body mass 76.0 ± 5.0 kg) were recruited as a control group that was tested but not trained. None of the subjects had previously participated in regular resistance training, and none of the subjects had participated in regular exercise within the last year. All subjects gave written informed consent to participate in the study, which was approved by the local Ethics Committee.

Training

Training was performed for 3 months with 38 sessions evenly dispersed, followed by 3 months of detraining (= no training). The resistance training program consisted of 4–5 sets of each of the following bilateral machine exercises: incline leg press, hack squat and isolated knee extension. In addition, 4–5 sets of hamstring curls were performed. All exercises were performed in a traditional manner using consecutive concentric and eccentric muscle contractions, i.e. raising and lowering the weight stack in a controlled manner without pause or breaks. In the early training phase (sessions 1–15), exercises consisted of 10–12 RM loads (4 sets), followed by 8–10 RM loads (4 sets) in the mid phase (sessions 16–25), and heavier loads of 6–8 RM (5 sets) in the later phase (sessions 26–38). During the final few weeks of the resistance training period very heavy loadings (4 RM) were used in the final set of each exercise. Absolute training loads were progressively increased to maintain relative loadings at the intended level. To ensure adequate training load and intensity, all training sessions were surveyed and supervised by the authors of the study.

EMG measurements

After careful preparation of the skin, bi-polar surface EMG electrodes (Medicotest M-00-S) were placed on the medial portion of the vastus lateralis (VL), vastus medialis (VM) and rectus femoris (RF) muscles. The EMG electrodes were connected directly to small preamplifiers located approximately 10 cm from the recording site. The signals were lead through shielded wires to custom-built differential instrumentation amplifiers, with a bandwidth of 10–10,000 Hz and a common mode rejection ratio > 100 dB (Aagaard et al. 2000). To quantify the degree of antagonist muscle involvement, EMG signals were obtained also in the biceps femoris caput longus and semitendinosus muscles (Aagaard et al. 2000).

Isokinetic strength testing

Unilateral isokinetic concentric and eccentric moment of force was measured for the knee extensors of the right leg using an isokinetic dynamometer (Kinetics Communicator, Chattecx Corp., Chattanooga, TN, USA). All strength testing was preceded by a 15-min warm-up and preconditioning to the isokinetic dynamometer (Aagaard et al. 1996). Isokinetic concentric and eccentric quadriceps moment of force was obtained at slow and fast angular velocities (30 and 240° s^{-1}, respectively). Range of motion was 10 to 90° anatomical knee joint angle (0° = knee fully extended). Trials were performed, separated by rest periods of at least 45 s until peak moment could not be visually improved any further, which was usually achieved within 6–8 maximal attempts.

Signal sampling

EMG and dynamometer strain gauge and lever arm position signals were sampled synchronously at

1,000 Hz using an external A/D-converter (dt2801-A, Data translation, Marlboro, USA) and stored on a personal computer for later analysis.

Data analysis

The dynamometer force and lever arm position signal was digitally lowpass-filtered at 15 and 8 Hz cut-off frequencies, respectively. Subsequently, the moment of force signal was corrected for the effect of gravity on the lower leg and foot (Aagaard et al. 1995). All raw EMG signals were filtered using linear EMG envelopes, which consisted of highpass filtering at a 5-Hz cut-off frequency, followed by full-wave rectification and subsequent lowpass filtering at a 10 Hz cut-off frequency (Winter 1990). All digital filtering routines were based on fourth-order zero phase lag Butterworth filters (Winter 1990). Mathematical integration of the filtered EMG signals with respect to time was performed, in the 60–80° knee joint range of motion, to yield integrated EMG (iEMG). The iEMG signal was then divided by the respective integration time to yield the average EMG in the 60–80° interval (Aagaard et al. 2000). Likewise, the average moment exerted in the 60–80° interval were calculated. The rationale for choosing this knee joint angle interval was that the highest moment of force occurred in this particular range of motion.

Normalized quadriceps EMG

According to previous procedures EMG obtained at slow and fast eccentric and slow concentric contraction were normalized relatively to that measured during fast concentric contraction (Seger and Thorstensson 1994; Aagaard et al. 2000). This was based on the assumption that full neuromuscular activation can be achieved during fast concentric contraction. Two lines of evidence exist to support this view. First, when electrical percutaneous muscle stimulation is superimposed onto fast concentric voluntary quadriceps contraction this does not result in an increased knee extension moment of force (Dudley et al. 1990; Westing et al. 1990; Amiridis et al. 1996). Second, the highest absolute EMG values consistently occur at fast concentric contraction (Westing et al. 1991; Seger and Thorstensson 1994; Aagaard et al. 2000). Uniform changes in VL, VM and RF EMG were observed with training. Therefore, data were collapsed for the three superficial muscles, i.e. normalized quadriceps EMG = (normalized VL + VM + RF EMG)/3.

Normalized hamstring EMG

Antagonist hamstring EMG was expressed relative to that measured in separate trials of maximal hamstring muscle contraction performed at identical contraction modes and angular velocities (Seger and Thorstensson 1994).

Muscle cross-sectional area

Quadriceps anatomical cross-sectional area (CSA) was determined at mid-femur (Narici et al. 1996; Aagaard et al. 2001b) from 2D T1-weighted fast field echo (TR/TE, 500/14 ms; FOV180; matrix 512×512; slice thickness 6 mm) magnetic resonance images (MRI) (Philips, Gyroscan ACS-NT 1.5 T, Best, Holland) at pre- and post-training and post-detraining. The length of the femur was determined in coronary scout scans as the distance from the greater trochanter to the lateral femur condyle. During analysis, CSAs of individual muscles of the thigh were determined using the computer software of the MRI scanner. CSA of the quadriceps muscle was defined as the sum of cross-sectional area of the vastus lateralis (VL), rectus femoris (RF), vastus medialis (VM) and vastus intermedius (VI) muscles. Due to the high cost of MRI these measurements were not performed for the non-trained control group.

Statistical analysis

Repeated measures ANOVA, and subsequent post-hoc analysis (Bonferroni corrected students t test) was used to answer the question if selected parameters changed over time. Values are reported as mean ± standard deviation unless otherwise stated. For correlation analysis Pearson's product moment correlation coefficient (R) were calculated.

Results

Muscle CSA

Anatomical quadriceps CSA increased by 10% in response to resistance training (77.4 ± 9.1–85.2 ± 10.1 cm^2, $P < 0.001$), and decreased to pre-training levels following the period of detraining (77.7 ± 7.6 cm^2, $P < 0.001$). Representative axial MRI scans are shown in Fig. 1.

Isokinetic muscle strength and EMG

Maximal muscle strength and normalized EMG at pre- and post-training and post-detraining are illustrated in Fig.2. Following the period of resistance training, slow eccentric strength increased by 50% ($P < 0.001$) with a parallel increase in normalized quadriceps EMG of 53% ($P < 0.001$), and these changes were closely correlated ($R^2 = 0.77$, $P < 0.001$) (Fig. 3). Fast eccentric strength increased by 25% ($P < 0.01$) while normalized EMG approached a significant increase of 21% ($P = 0.08$), with a correlation between these changes ($R^2 = 0.33$,

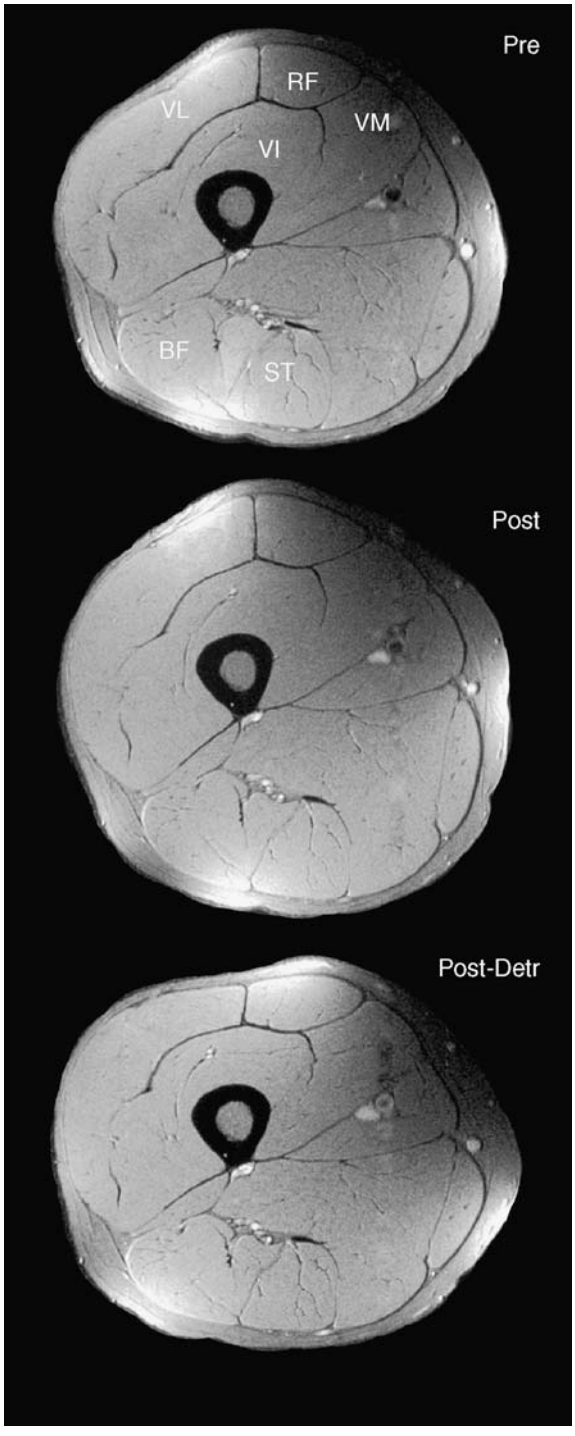

Fig. 1 Representative axial MRI scans of the right thigh obtained at 50% femur length pre-training (*top*), post-training (*middle*) and post-detraining (*bottom*). Hypertrophy (10%, $P < 0.001$) occurred in response to resistance training. Following detraining these hypertrophic gains were lost

$P < 0.05$). Slow concentric muscle strength and normalized EMG increased by 19% ($P < 0.001$) and 14% ($P < 0.05$), respectively, and these changes also showed a correlation ($R^2 = 0.35$, $P < 0.05$). Fast concentric strength increased by 11% ($P < 0.05$). Following 3 months of detraining, maximal muscle strength and normalized EMG during concentric contraction decreased to pre-training levels. In contrast, maximal moment of force and normalized EMG during slow and fast eccentric contraction were fully preserved compared to post-training (Fig. 2 *left*).

Antagonist EMG activity in the medial and lateral hamstring muscles did not change with resistance training or detraining. The levels of pre-training antagonist EMG activity in the biceps femoris were $14 \pm 1\%$, $13 \pm 2\%$, $17 \pm 3\%$ and $26 \pm 4\%$ during fast eccentric, slow eccentric, slow concentric and fast concentric contraction, respectively. Corresponding values in the semitendinosus were $12 \pm 2\%$, $13 \pm 1\%$, $16 \pm 1\%$ and $17 \pm 5\%$.

No significant changes occurred in the control group for any of the examined parameters (Fig. 2 *right*).

Discussion

The resistance training regime used in the present study resulted in considerable gains in isokinetic knee extensor strength and EMG, particularly during eccentric contraction. Likewise, quadriceps CSA was found to increase. Following 3 months of detraining, concentric strength as well as quadriceps CSA decreased towards pre-training levels; however, eccentric strength gains were fully preserved.

Eccentric strength gains evoked by heavy resistance training

It is well established that both muscular and neural factors are responsible for gains in voluntary muscle strength following a period of resistance training (e.g. Moritani and deVries 1979; Narici et al. 1989). The quadriceps CSA values measured prior to the present training intervention as well as the hypertrophic gains observed after the period of training are in agreement with previous reports (Narici et al. 1989; Aagaard et al. 2001a). In the present study gains in slow eccentric muscle strength markedly exceeded the magnitude of muscular hypertrophy. Further, strength gains in response to resistance training were matched by parallel increases in EMG. This relationship was most pronounced during slow eccentric contraction ($R^2 = 0.77$), which indicates a strong relation between increased neural drive and muscular strength during this particular type of contraction (Fig. 3). Increased EMG amplitude in response to training is commonly believed to reflect increased recruitment and firing rate of motor units (Suzuki et al. 2002), which in turn would increase force output of the muscle. However, increased EMG could also reflect increased synchronization of motor units (Yao et al. 2000; Day and Hulliger 2001), which may not per se increase muscle force. The present results indicate that neural factors contributed substantially to the gain in slow eccentric maximal muscle strength. It is possible

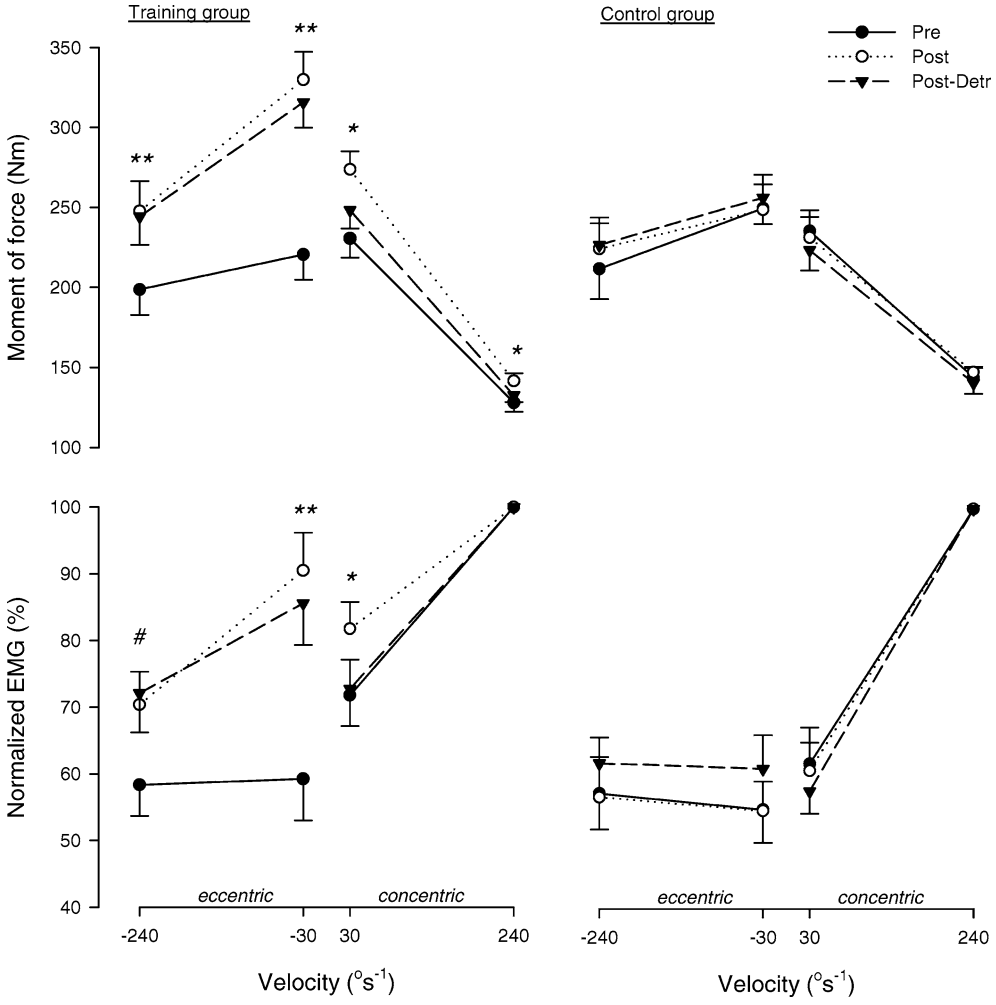

Fig. 2 Moment of force (*above*) and normalized quadriceps EMG (*below*) at slow and fast eccentric and slow and fast concentric contraction, at pre-, post-training and post-detraining in the training group (*left*) and the control group (*right*). Values are mean ± SE. Notice that the increase in strength and normalized EMG during maximal eccentric but not concentric contraction was preserved after 3 months of detraining. *: post-training > pre-training and post-detraining ($P < 0.001$–0.05), i.e. gains in response to resistance training that were lost after the period of detraining, **: post-training and post-detraining > pre-training ($P < 0.001$–0.05), i.e. gains in response to resistance training and full preservation following detraining. #: a tendency towards a significant increase, $P = 0.08$

however that changes in muscle architecture via an increased muscle fiber pennation angle could also have contributed to increase the maximal eccentric strength (Aagaard et al. 2001a; Reeves et al. 2004). The observed muscle hypertrophy must have contributed to some extent as well.

A twofold greater increase was observed in muscle strength and EMG during slow compared to fast eccentric contraction. The present resistance training exercises consisted of coupled concentric and eccentric contractions, i.e. raising and lowering the weight stack in a controlled manner. Training that included eccentric muscle contractions has previously been reported to induce similar fast and slow eccentric strength gains (Aagaard et al. 2000; Spurway et al. 2000) or greater slow eccentric strength gains (Colliander and Tesch 1990, 1991). One reason for these findings may reside in the velocity of training compared to that during testing. When employing free weight or machine training exercises it is difficult to control knee joint angular velocity. In the present study the subjects lowered the weight stack in a controlled manner (approximately 1–1.5 s ~ knee joint angular velocity of 50–100° s^{-1}). Eccentric resistance training has previously been shown to result in eccentric strength gains at and below the eccentric training velocity (Seger et al. 1998; Paddon-Jones et al. 2001). It can therefore be speculated that the relatively

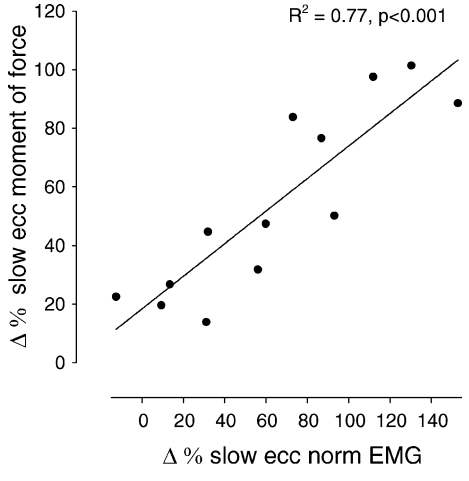

Fig. 3 The pre- to post-training change in moment of force and normalized EMG during maximal slow eccentric contraction were closely related ($R^2 = 0.77$, $P < 0.001$)

slow training speeds employed in the present study induced velocity-specific neural adaptations that were most pronounced during slow eccentric contraction, but less so during fast eccentric contraction.

Preservation of eccentric strength gains following detraining

A major finding of the present study was that muscle strength and EMG during maximal eccentric contraction remained preserved following 3 months of detraining. In contrast, quadriceps CSA decreased to pre-training levels. EMG and muscle strength during concentric contraction returned to pre-training levels following the period of detraining. This indicates that the present resistance training regime, using combined concentric and eccentric contractions and relatively heavy loadings induced long-lasting neural adaptations and parallel strength gains during maximal eccentric but not concentric contraction. Thus, although both concentric and eccentric muscle strength increased in response to resistance training, the underlying neural adaptation mechanisms may differ. In concert with the present findings, Colliander and Tesch (1992) observed that coupled concentric-eccentric quadriceps resistance training in an isokinetic dynamometer resulted in eccentric muscle strength gains that were partially preserved across 12 weeks of detraining. In another study dynamic eccentric resistance training resulted in increased dynamic eccentric muscle strength that was retained after 8 weeks of detraining (Housh et al. 1996). In contrast to the present study, these studies (Colliander and Tesch 1992; Housh et al. 1996) used identical training and testing devices, and the results may therefore have been influenced by the effect of learning (Rutherford and Jones 1986). Furthermore, these studies did not measure EMG.

The present results suggest that the neural circuitry involved in the control of force development during maximal eccentric muscle contraction exhibits considerable adaptive plasticity in response to strength training and that these adaptations are long-lasting. Possible mechanisms could involve modulated feedback from Golgi tendon organs and muscle spindles. In animal experiments Golgi tendon organs have been demonstrated to discharge proportionally to increasing force (Gregory and Proske 1979; Crago et al. 1982). In consequence, substantial autogenic motoneuron inhibition from Golgi tendon organs (Jami 1992), may be present during maximal eccentric contraction in untrained subjects as previously indicated (Dudley et al. 1990; Westing et al. 1990; Seger and Thorstensson 1994; Amiridis et al. 1996; Kellis and Baltzopoulos 1998; Aagaard et al. 2000). Furthermore, reduced H-reflex excitability has been observed during both active (Romano and Schieppati 1987; Abbruzzese et al. 1994) and passive muscle lengthening (Pinniger et al. 2001). Thus, muscle lengthening by itself appears to cause a reduced level of motoneuron excitability and/or an increased presynaptic inhibition of Ia afferents, which suggests influential neural feedback from spindle afferents. Importantly, interneurons at the spinal level are influenced by higher cortical centres via descending pathways (Nielsen and Petersen 1994; Bawa et al. 2000), which allow supraspinal pathways to modulate the magnitude of excitatory and inhibitory afferent input to the motoneurons. Speculatively, specific eccentric neural adaptation mechanisms could include disinhibition of inhibitory Ib-interneurons during maximal muscle contraction and/or reduced Ia-afferent presynaptic inhibition during muscle lengthening. However, it is likely that the resultant level of neuromuscular activity reflects the integration of a large number of neural pathways related to both central pre-programmed and peripheral feedback mechanisms, and that the neural adaptations induced by strength training can occur at several levels in the nervous system (Bawa 2002; Aagaard and Thorstensson 2002). Regardless of the exact nature of these neural adaptation mechanisms the present study is the first to suggest that the adaptations are long lasting.

In the present study, muscular atrophy as a result of detraining did not appear to significantly influence eccentric muscle strength. In contrast, Hortobagyi et al. (1993) reported that eccentric muscle strength and muscle fiber size decreased with 2 weeks of detraining in power athletes, whereas maximum EMG activity remained unchanged. In general it has been found that particularly eccentric muscle strength is susceptible to detraining in trained subjects (Hortobagyi et al. 1993; Mujika and Padilla 2001; Kraemer et al. 2002). Thus, the adaptive change in eccentric muscle strength with detraining is likely influenced by the initial training status of the subjects.

The control group did not display any change in isokinetic muscle strength and EMG during the three test sessions. Therefore, testing in the isokinetic dynamometer per se at pre- and post-training could not have influenced the test results at post-detraining.

In conclusion, the present findings suggest that heavy resistance training in previously untrained subjects induces long-lasting neural adaptations and parallel strength gains during maximal eccentric but not concentric muscle contraction.

References

Aagaard P, Thorstensson A (2002) Neuromuscular aspects of exercise—adaptive responses evoked by strength training. In: Kjær M (ed) Textbook of sport medicine. Blackwell, London, pp 70–106

Aagaard P, Simonsen EB, Trolle M, Bangsbo J, Klausen K (1995) Isokinetic hamstring/quadriceps strength ratio: influence from joint angular velocity, gravity correction and contraction mode. Acta Physiol Scand 154:421–427

Aagaard P, Simonsen EB, Trolle M, Bangsbo J, Klausen K (1996) Specificity of training velocity and training load on gains in isokinetic knee joint strength. Acta Physiol Scand 156:123–129

Aagaard P, Simonsen EB, Andersen JL, Magnusson SP, Halkjaer-Kristensen J, Dyhre-Poulsen P (2000) Neural inhibition during maximal eccentric and concentric quadriceps contraction: effects of resistance training. J Appl Physiol 89:2249–2257

Aagaard P, Andersen JL, Dyhre-Poulsen P, Leffers AM, Wagner A, Magnusson SP, Halkjaer-Kristensen J, Simonsen EB (2001a) A mechanism for increased contractile strength of human pennate muscle in response to strength training: changes in muscle architecture. J Physiol 534:613–623

Aagaard P, Simonsen EB, Andersen JL, Leffers AM, Wagner A, Magnusson SP, Halkjaer-Kristensen J, Dyhre-Poulsen P (2001b) MRI assessment of quadriceps muscle size before and after resistance training: determination of volume vs single-site CSA. Med Sci Sports Exerc 33: S147

Aagaard P, Simonsen EB, Andersen JL, Magnusson P, Dyhre-Poulsen P (2002) Neural adaptation to resistance training: changes in evoked V-wave and H-reflex responses. J Appl Physiol 92:2309–2318

Abbruzzese G, Morena M, Spadavecchia L, Schieppati M (1994) Response of arm flexor muscles to magnetic and electrical brain stimulation during shortening and lengthening tasks in man. J Physiol 481(Pt 2):499–507

Amiridis IG, Martin A, Morlon B, Martin L, Cometti G, Pousson M, Van Hoecke J (1996) Co-activation and tension-regulating phenomena during isokinetic knee extension in sedentary and highly skilled humans. Eur J Appl Physiol Occup Physiol 73:149–156

Bawa P (2002) Neural control of motor output: can training change it? Exerc Sport Sci Rev 30:59–63

Bawa P, Chalmers GR, Jones KE, Sogaard K, Walsh ML (2000) Control of the wrist joint in humans. Eur J Appl Physiol 83:116–127

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

Colliander EB, Tesch PA (1990) Effects of eccentric and concentric muscle actions in resistance training. Acta Physiol Scand 140:31–39

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

Colliander EB, Tesch PA (1992) Effects of detraining following short term resistance training on eccentric and concentric muscle strength. Acta Physiol Scand 144:23–29

Crago PE, Houk JC, Rymer WZ (1982) Sampling of total muscle force by tendon organs. J Neurophysiol 47:1069–1083

Day SJ, Hulliger M (2001) Experimental simulation of cat electromyogram: evidence for algebraic summation of motor-unit action-potential trains. J Neurophysiol 86:2144–2158

Dudley GA, Harris RT, Duvoisin MR, Hather BM, Buchanan P (1990) Effect of voluntary vs artificial activation on the relationship of muscle torque to speed. J Appl Physiol 69:2215–2221

Gregory JE, Proske U (1979) The responses of Golgi tendon organs to stimulation of different combinations of motor units. J Physiol 295:251–262

Hakkinen K, Komi PV (1983) Electromyographic changes during strength training and detraining. Med Sci Sports Exerc 15:455–460

Hakkinen K, Alen M, Komi PV (1985) Changes in isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of human skeletal muscle during strength training and detraining. Acta Physiol Scand 125:573–585

Higbie EJ, Cureton KJ, Warren GL, III, Prior BM (1996) Effects of concentric and eccentric training on muscle strength, cross-sectional area, and neural activation. J Appl Physiol 81:2173–2181

Hortobagyi T, Houmard JA, Stevenson JR, Fraser DD, Johns RA, Israel RG (1993) The effects of detraining on power athletes. Med Sci Sports Exerc 25:929–935

Hortobagyi T, Barrier J, Beard D, Braspennincx J, Koens P, Devita P, Dempsey L, Lambert J (1996) Greater initial adaptations to submaximal muscle lengthening than maximal shortening. J Appl Physiol 81:1677–1682

Housh TJ, Housh DJ, Weir JP, Weir LL (1996) Effects of eccentric-only resistance training and detraining. Int J Sports Med 17:145–148

Houston ME, Froese EA, Valeriote SP, Green HJ, Ranney DA (1983) Muscle performance, morphology and metabolic capacity during strength training and detraining: a one leg model. Eur J Appl Physiol Occup Physiol 51:25–35

Jami L (1992) Golgi tendon organs in mammalian skeletal muscle: functional properties and central actions. Physiol Rev 72:623–666

Karlsson S, Gerdle B (2001) Mean frequency and signal amplitude of the surface EMG of the quadriceps muscles increase with increasing torque—a study using the continuous wavelet transform. J Electromyogr Kinesiol 11:131–140

Kellis E, Baltzopoulos V (1998) Muscle activation differences between eccentric and concentric isokinetic exercise. Med Sci Sports Exerc 30:1616–1623

Komi PV, Linnamo V, Silventoinen P, Sillanpaa M (2000) Force and EMG power spectrum during eccentric and concentric actions. Med Sci Sports Exerc 32:1757–1762

Kraemer WJ, Koziris LP, Ratamess NA, Hakkinen K, Triplett-McBride NT, Fry AC, Gordon SE, Volek JS, French DN, Rubin MR, Gomez AL, Sharman MJ, Michael LJ, Izquierdo M, Newton RU, Fleck SJ (2002) Detraining produces minimal changes in physical performance and hormonal variables in recreationally strength-trained men. J Strength Cond Res 16:373–382

Lemmer JT, Hurlbut DE, Martel GF, Tracy BL, Ivey FM, Metter EJ, Fozard JL, Fleg JL, Hurley BF (2000) Age and gender responses to strength training and detraining. Med Sci Sports Exerc 32:1505–1512

LeMura LM, von Duvillard SP, Andreacci J, Klebez JM, Chelland SA, Russo J (2000) Lipid and lipoprotein profiles, cardiovascular fitness, body composition, and diet during and after resistance, aerobic and combination training in young women. Eur J Appl Physiol 82:451–458

McCarrick MJ, Kemp JG (2000) The effect of strength training and reduced training on rotator cuff musculature. Clin Biomech (Bristol, Avon) 15(Suppl 1): S42-S45

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

Mujika I, Padilla S (2001) Muscular characteristics of detraining in humans. Med Sci Sports Exerc 33:1297–1303

Narici MV, Roi GS, Landoni L, Minetti AE, Cerretelli P (1989) Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. Eur J Appl Physiol Occup Physiol 59:310–319

Narici MV, Hoppeler H, Kayser B, Landoni L, Claassen H, Gavardi C, Conti M, Cerretelli P (1996) Human quadriceps cross-sectional area, torque and neural activation during 6 months strength training. Acta Physiol Scand 157:175–186

Nielsen J, Petersen N (1994) Is presynaptic inhibition distributed to corticospinal fibres in man? J Physiol 477(Pt 1): 47–58

Paddon-Jones D, Leveritt M, Lonergan A, Abernethy P (2001) Adaptation to chronic eccentric exercise in humans: the influence of contraction velocity. Eur J Appl Physiol 85:466–471

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

Pinniger GJ, Nordlund M, Steele JR, Cresswell AG (2001) H-reflex modulation during passive lengthening and shortening of the human triceps surae. J Physiol 534:913–923

Reeves N D, Narici M V, Maganaris CN (2004) Effect of resistance training on skeletal muscle-specific force in elderly humans. J Appl Physiol 96:885–892

Romano C, Schieppati M (1987) Reflex excitability of human soleus motoneurones during voluntary shortening or lengthening contractions. J Physiol 390:271–284

Rutherford OM, Jones DA (1986) The role of learning and coordination in strength training. Eur J Appl Physiol Occup Physiol 55:100–105

Schantz P, Randall-Fox E, Hutchison W, Tyden A, Astrand PO (1983) Muscle fibre type distribution, muscle cross-sectional area and maximal voluntary strength in humans. Acta Physiol Scand 117:219–226

Seger JY, Thorstensson A (1994) Muscle strength and myoelectric activity in prepubertal and adult males and females. Eur J Appl Physiol Occup Physiol 69:81–87

Seger JY, Thorstensson A (2000) Electrically evoked eccentric and concentric torque-velocity relationships in human knee extensor muscles. Acta Physiol Scand 169:63–69

Seger JY, Arvidsson B, Thorstensson A (1998) Specific effects of eccentric and concentric training on muscle strength and morphology in humans. Eur J Appl Physiol Occup Physiol 79:49–57

Spurway NC, Watson H, McMillan K, Connolly G (2000) The effect of strength training on the apparent inhibition of eccentric force production in voluntarily activated human quadriceps. Eur J Appl Physiol 82:374–380

Staron RS, Leonardi MJ, Karapondo DL, Malicky ES, Falkel JE, Hagerman FC, Hikida RS (1991) Strength and skeletal muscle adaptations in heavy-resistance-trained women after detraining and retraining. J Appl Physiol 70:631–640

Suzuki H, Conwit RA, Stashuk D, Santarsiero L, Metter EJ (2002) Relationships between surface-detected EMG signals and motor unit activation. Med Sci Sports Exerc 34:1509–1517

Tesch PA, Dudley GA, Duvoisin MR, Hather BM, Harris RT (1990) Force and EMG signal patterns during repeated bouts of concentric or eccentric muscle actions. Acta Physiol Scand 138:263–271

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

Westing SH, Cresswell AG, Thorstensson A (1991) Muscle activation during maximal voluntary eccentric and concentric knee extension. Eur J Appl Physiol Occup Physiol 62:104–108

Wickiewicz TL, Roy RR, Powell PL, Perrine JJ, Edgerton VR (1984) Muscle architecture and force-velocity relationships in humans. J Appl Physiol 57:435–443

Winter DA (1990) Biomechanics and motor control of human movement. 2nd edn. Wiley, New York, pp 11–50

Yao W, Fuglevand RJ, Enoka RM (2000) Motor-unit synchronization increases EMG amplitude and decreases force steadiness of simulated contractions. J Neurophysiol 83:441–452