LJMU Research Online

Buckthorpe, M, Erskine, RM, Fletcher, G and Folland, JP

 Task-specific neural adaptations to isoinertial resistance training

http://researchonline.ljmu.ac.uk/id/eprint/2988/

Article

For more information please contact researchonline@ljmu.ac.uk

Task specific neural adaptations to isoinertial resistance training

Matthew Buckthorpe[1,2], Robert M. Erskine[3], Gareth Fletcher[1], Jonathan P. Folland[1]

[1] School of Sport, Exercise and Health Sciences, Loughborough University, UK.

[2] Isokinetic Medical Group, London, UK.

[3]Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Liverpool, UK

Corresponding Author:

Matthew Buckthorpe, Isokinetic London, 17a Welbeck Way, London, W1G 9YW, UK.
Tel: +44 (0)207.4865733
E-mail: M.Buckthorpe@isokinetic.com

Running Title: Neural adaptations to resistance training

ABSTRACT

This study aimed to delineate the contribution of adaptations in agonist, antagonist and stabiliser muscle activation to changes in isometric and isoinertial lifting strength after short-term isoinertial resistance training (RT). Following familiarisation, 45 men (23.2±2.8 yrs) performed maximal isometric and isoinertial strength tests of the elbow flexors of their dominant arm before and after three weeks of isoinertial RT. During these tasks surface EMG amplitude was recorded from the agonist (biceps brachii short and long heads), antagonist (triceps brachii lateral head) and stabiliser (anterior deltoid, pectoralis major) muscles and normalised to either M_{max} (agonists) or to maximum EMG during relevant reference tasks (antagonist, stabilisers). After training there was more than a two-fold greater increase in training task specific isoinertial than isometric strength (17 vs. 7%). There were also task specific adaptations in agonist EMG, with greater increases during the isoinertial than isometric strength task (ANOVA, training x task, P=0.005). A novel finding of this study was that training increased stabiliser muscle activation during all the elbow flexion strength tasks (P<0.001), although these were not task specific training effects. RT elicited specific neural adaptations to the training task that appeared to explain the greater increase in isoinertial than isometric strength.

Key Words: Neural activation, stabiliser muscles, elbow flexors, training specificity

INTRODUCTION

Marked increases in muscle strength during the early phase of a resistance training (RT) program have been observed (Abe et al., 2000; Pucci et al., 2006; Del Balso & Cafarelli, 2007) and these changes appear to be highly specific to the nature of the training task. For example, conventional dynamic isoinertial RT has repeatedly been found to produce disproportionately greater increases in isoinertial lifting strength (one repetition maximum [1RM]) than isometric strength (Thorstensson et al., 1976; Rutherford & Jones, 1986). This specificity of training phenomenon is often taken as evidence for neural adaptations to RT (Folland & Williams, 2007), however at present there is minimal direct evidence for either neural or morphological mechanisms that might explain this training task specificity. Furthermore, as functional tasks generally require isoinertial strength, establishing the mechanisms that lead to a greater improvement in isoinertial versus isometric strength is important from both a sports performance and injury rehabilitation perspective.

Early adaptations to RT are thought to be primarily explained by neural adaptations, e.g. adaptations in agonist, antagonist and stabiliser muscle activation (Folland & Williams, 2007), with a greater contribution from morphological adaptations, such as selective hypertrophy and/or architectural changes, as training duration progresses (Narici et al., 1996). Therefore, documenting training specific adaptations over a short term training period may distinguish between the neural and morphological explanations for the task specificity phenomenon. Although numerous studies have reported an increase in absolute agonist EMG amplitude during isometric tasks following RT (Hakkinen et al., 1983; 1998, Kubo et al., 2006; Tillin et al., 2011) only one investigation has found an increase in agonist EMG during an isoinertial task (Hakkinen et al., 1998). Moreover, the comparative changes in agonist activation during isometric and isoinertial tasks, or how these changes may explain the

strength gains that occur, have not been elucidated. Furthermore, the evidence for RT-induced changes in antagonist muscle co-activation is equivocal (Hakkinen et al., 1998; Pucci et al., 2006; de Boer et al., 2007; Tillin et al., 2011). Whilst an increase in antagonist co-activation might attenuate gains in strength, for some joints this may be a necessary adaptation to maintain joint integrity in response to increased loading post-training (Tillin et al., 2011).

In addition to agonist and antagonist activation, changes in stabiliser muscle activation could also influence strength gains and contribute to task specificity, but has not previously been investigated in any strength tasks. Learning to effectively stabilise the adjacent joints through increased stabiliser activation may facilitate an increase in strength during the early phase of RT. Additionally, isoinertial lifting tasks involve more degrees of freedom within the musculo-skeletal system than isometric strength tasks, and therefore may require a greater degree of stabilisation. Although the influence of RT on stabiliser EMG during submaximal contractions has been investigated (Cacchio et al., 2008), the contribution of adaptations in stabiliser activation to the changes in maximum isoinertial or isometric strength after RT has not been examined.

Explosive strength is also considered an important characteristic of muscle performance (de Ruiter et al., 2006; Tillin et al., 2013), as well as for the stabilisation of the musculoskeletal system following mechanical perturbation (Fleming et al., 1991; Izquierdo et al., 1999; Chang et al., 2005; Pijnappels et al., 2008), thus reducing the risk of falls and injury. The efficacy of RT for improving explosive isometric strength is controversial, with some reports finding an improvement (Hakkinen et al., 1998; Aagaard et al., 2002) and others no change (Andersen et al., 2010; Tillin et al., 2011). Moreover, it is not known whether just three weeks of conventional RT will influence explosive strength, and if so, whether this effect is due to specific neural adaptations.

The aim of the present study was to assess the task specific adaptations in isometric (maximum and explosive) and isoinertial strength following three weeks of isoinertial RT, and to document the concurrent neural changes in agonist, antagonist and stabiliser muscle activation. It was hypothesised that conventional isoinertial RT would induce greater increases in isoinertial, than isometric strength, and that this would be concomitant with greater changes in agonist and stabiliser muscle activation during the isoinertial task.

METHOD

Participants

Forty-five male participants (age, 23.2 ± 2.8 yr; height, 177.0 ± 7.7 cm; mass, 73.7 ± 9.9 kg) completed the study. The participants were physically active, healthy, injury free and had not taken part in any form of strenuous upper body exercise for the previous 12 months prior to the study. All participants provided written informed consent prior to their involvement in the study, which complied with the Declaration of Helsinki and was approved by the Ethical Advisory Committee of Loughborough University.

Overview

Participants were tested twice pre (pre-1, pre-2) and once post three weeks of isoinertial elbow flexor RT. Pre-1 and Pre-2 training measurements were separated by seven days, with pre-2 measurements recorded 3-7 days prior to the commencement of training. Post-training measurements were collected 3-5 days after the last training session. The measurement sessions were all performed at a consistent time of day for each participant. The training involved participants performing elbow flexion exercises (unilateral and bilateral preacher curls) three times per week (Monday, Wednesday, Friday) for three weeks. Although

participants trained both arms, measurements were only recorded from the dominant arm. During each measurement session participants performed a series of elbow flexion contractions to determine maximum isoinertial lifting strength (1RM), and isometric maximum and explosive strength. Surface EMG was recorded during pre-2 and post measurement sessions from the agonist, antagonist and stabiliser muscles throughout these volitional contractions as well as during reference tasks/measurements that were used for normalisation of EMG: a supramaximal evoked compound action potential (M_{max}) of the biceps brachii (agonists); maximum isometric elbow extension (antagonist); and maximum isometric bench press (stabilisers). Muscle thickness of the short-head of the biceps brachii was also measured at rest using ultrasound during both the pre-2 and post- measurement sessions.

Training

Each training session involved two similar elbow flexion exercises. Firstly, unilateral elbow flexion curls were performed with a dumbbell on a modified preacher bench (Body Solid, Forest Park, IL, USA; Figure 1A with participants performing alternate sets with the dominant and then the non-dominant arms. Secondly, participants performed bilateral elbow flexion exercises using a weights machine (Pro Club Line Bicep Curl; Body Solid, Forest Park, IL, USA). Both exercises were performed with a load of 8-10RM. Two sets of each exercise were performed, with two minutes rest between exercises involving the same muscle group. The training load was increased when participants could complete 10 repetitions on both sets. Participants were instructed to lower the weight in a controlled manner over 2-3 s during the eccentric phase, and then lift the weight as quickly as possible during the concentric phase, in order to maximise the rate of force development. They were also directed to move through a full range of motion (from ~15° to ~140°; 0° = full extension) throughout both exercises.

Measurement Procedures

Force

Participants sat upright (hip joint angle of 90°) in a custom built strength testing chair and were strapped at the hip and chest to the seat and back of the chair to prevent movement of the body (Figure 1A). The elbow and shoulder joints were flexed to 60 and 90°, respectively (0° being full elbow extension), with the upper arm placed on a horizontal board, and externally rotated with the elbow position maintained by blocks anterior and lateral to the joint. The forearm was supinated and the wrist was attached to an S-Beam tension-compression load cell (Applied Measurements Ltd, Berkshire, UK) positioned perpendicular to the forearm during elbow flexion/extension. The force signal was amplified ($\times$ 500) interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK) and sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK). During off-line analysis the force signal was low-pass filtered (500 Hz) with a fourth order zero-lag Butterworth digital filter. Real-time biofeedback of the force response was provided on a computer monitor.

[Figure 1]

Surface Electomyography (EMG)

Surface EMG was recorded using two Delsys Bangnoli-4 EMG systems (Delsys, Boston, USA) from two agonist muscles (short and long heads of the biceps brachii, BBS and BBL), the antagonist (triceps brachii lateral head, TB) and two stabilisers (anterior deltoid, AD; clavicular head of the pectoralis major, PM). Following preparation of the skin (shaving, abrading and cleansing with 70% ethanol), the same investigator attached double differential surface electrodes (1 cm inter-electrode distance, DE-3.1, Delsys) to the skin over each of the

muscles using adhesive interfaces. The electrodes were positioned at specific measured sites along the arm, in the centre of the muscle belly and parallel to the presumed orientation of the muscle fibers. BBS and BBL electrodes were positioned distally 75% of the distance between the coracoid process and medial epicondyle of the humerus (Lee et al., 2010). AD, PM and TB were positioned according to SENIAM guidelines. The EMG reference electrodes were placed on the contralateral clavicle. EMG signals were amplified ($\times$ 100, differential amplifier 20-450 Hz) and sampled at 2000 Hz with the same analogue to differential converter and PC as the force signal. During off-line analysis the EMG signals were band-pass filtered in both directions (6-500 Hz) using a 4th order zero-lag Butterworth digital filter.

Muscle Stimulation

The musculocutaneous nerve was electrically stimulated (via a constant current, variable voltage stimulator; DS7AH, Digitimer Ltd., UK) with square wave pulses (0.2 ms duration) to elicit twitch contractions, and facilitate measurement of compound muscle action potentials (M-waves) with EMG. The self-adhesive anode (5 x 5 cm; Verity Medical, Andover, UK) was attached to the skin over the triceps brachii. The cathode (1 cm diameter, Electro Medical Supplies, Wantage, UK) was held in place over the nerve between both biceps brachii heads at ~25% of the distance between the coracoid process and medial epicondyle of the humerus (Lee et al., 2010). However, the precise location was determined as the position that evoked the greatest response for a particular electrical current (10-30mA).

Protocol

Isoinertial Strength (1RM)

1RM was assessed with a series of incremental dumbbell lifts according to an adapted protocol (Baecke, 1982). Isoinertial strength was measured with the same modified preacher bench used during training (Figure 1A). Briefly, the preacher bench was modified with a horizontal rack at the end of the motion that provided a consistent starting point pre and post training for the isoinertial lifts. The height of the bench was adjusted according to the length of each participant's arm, so that it was underneath the axilla, with the participant leaning forward on to the bench such that the shoulder was flexed at ~75°. This position ensured the elbow was fully extended when each participant's hand gripped the dumbbell on the rack. Participants were instructed to lift the dumbbell through the full range of motion, from full elbow extension to full flexion. The non-lifting hand rested on the knee of the same side, both feet remained flat on the floor and the knees were flexed at 90°. Participants warmed up by performing 10 reps at 40% of their previous 1RM (or estimated 1RM for pre-1). After 1 min rest, participants performed 3 repetitions at 80% of their previous 1-RM. Thereafter they performed a series of single lifts interspersed with 1 min rest intervals, firstly at the previous 1-RM, and then at increments of +0.5 kg if the preceding lift was successful. The 1-RM was defined as the highest load lifted on that occasion, and was generally determined within 3-5 attempts, although more attempts were completed if necessary. EMG amplitude of all the muscles during the 1-RM lift was assessed for a 500 ms epoch that gave the highest agonist root-mean-square (RMS) EMG during the concentric phase of the lift. BBS and BBL RMS EMG were normalised to M_{max} peak-to-peak amplitude (see below) and then averaged to give a mean value for the biceps brachii (agonist EMG); AD and PM RMS EMG were normalised to the maximum value attained by each muscle during the isometric maximum bench press (see below) and then averaged to give a mean value for the stabiliser muscles (stabiliser EMG); and antagonist EMG was normalised to the TB EMG at elbow extension iMVF (antagonist EMG; see below).

Isometric Maximal Voluntary Contractions

Participants were transferred to the isometric elbow flexion/extension apparatus (Fig. 1B) and completed a warm up of submaximal isometric voluntary contractions. They then performed three elbow flexion isometric maximum voluntary contractions (iMVCs) (separated by 1 min) followed by three elbow extension iMVCs, in which participants were instructed to pull or push as hard as possible for ~3 seconds. Biofeedback and verbal encouragement were provided during and between each iMVC. Elbow flexor isometric maximal voluntary force (iMVF) was the greatest force achieved by the participant in any of the iMVCs. The amplitude of the EMG signal was assessed as the RMS of a 500 ms epoch around peak force (250 ms either side) for each muscle. EMG was normalised in the same manner as for the 1RM efforts. Maximal EMG of the TB (EMG_{max}) was assessed as a 500 ms epoch at elbow extension iMVF (250 ms either side) and used for normalisation of antagonist EMG during elbow flexion tasks.

Explosive Voluntary Contractions

Participants then performed 10 isometric explosive voluntary contractions (separated by 20 s). For each contraction participants were instructed to flex their arm as 'fast' and as hard as possible for ~1 s from a relaxed state (Sahaly et al., 2001) and achieve at least 80% iMVF. Participants were instructed to avoid any countermovement or pre-tension. To determine if countermovement had occurred, the resting force level was displayed on a sensitive scale. The slope of the force time curve (10 ms time constant) was displayed throughout and the peak slope was used to provide visual feedback to participants after each contraction. The three contractions with the highest peak slope, no discernible countermovement or pre-tension (change in force of < 0.5 N in the preceding 100 ms), and with a peak force of at least 80% iMVF were used for analysis, and all measurements were averaged across these three

contractions. Force was assessed at 50, 100, and 150 ms, from the onset of contraction and reported in absolute terms and normalised to iMVF. Peak rate of force development (pRFD) was measured as the maximum slope (10 ms time constant), and the time at which it occurred was also recorded. Impulse (the area beneath the force-time curve) was assessed in windows of 0-50, 0-100 and 0-150 ms from the onset of force. The RMS EMG was measured in windows of 0-50, 0-100 and 0-150 ms from the onset of EMG activity in the first agonist muscle to be activated, and normalised in the same manner as during the maximal strength tasks. The time between the first agonist muscle to be activated and onset of force was determined as the maximum electromechanical delay (EMD_{max}).

For the explosive voluntary contractions, identification of force and EMG onsets were made manually (visual identification) as this is considered the "gold standard" method (Allison, 2003; Moretti et al., 2003; Tillin et al., 2013; Folland et al., 2013). The same investigator identified signal onsets according to a standard method (see Tillin et al., 2010; Tillin et al., 2013) with a constant y-axis scale of ~ 2 N and 100 mV, for force and EMG respectively, and an x-axis scale of 500 ms. A vertical cursor was then placed on the onset and viewed at a higher resolution to determine its exact location (~ 0.5 N and 50 mV for force and EMG axes respectively using an x-axis of 25 ms).

M_{max}

Single twitch contractions were elicited by stimulation of the musculocutaneous nerve at incremental intensities until there was a plateau in the M-wave response of both heads of the bicep brachii. Thereafter, the current was increased by 20% and three supra-maximal twitches were elicited (separated by 12 s). The average M-wave peak to peak amplitude (M_{max}) of these three supramaximal M-waves was determined for each muscle and used for

normalisation of agonist EMG during elbow flexor strength tasks. Six participants were uncomfortable with the stimulation and thus EMG normalised to M_{max} is reported for N = 39.

Isometric Bench Press

Participants performed isometric bench press contractions to determine maximal EMG of the stabilising muscles (AD and PM). Participants lay supine on an inclined bench (head up ~15° to the horizontal) with their shoulders aligned vertically below a fixed immovable bar and their knees bent and feet positioned on the end of the bench, which was positioned on a portable force plate (Quattro Jump, Type 9290 AD, Kistler, Switzerland). The height of the bar was adjusted so that when participants grasped the bar their upper arm was horizontal, whilst their forearms were vertical (i.e. shoulders abducted at 90° and elbows flexed at 90°). Participants performed three iMVCs of 3 s duration with ≥ 30 s rest between efforts. Verbal encouragement was provided throughout the efforts and the recorded force during the effort was provided as feedback to the participants following each attempt. Maximal EMG of the AD and PM muscles was recorded over the highest 500 ms epoch during the series of bench press iMVCs (EMG_{max}) and used for normalisation of stabiliser EMG during elbow flexor strength tasks.

Muscle Thickness

In-vivo muscle thickness was examined prior to the start of each measurement session, using an ultrasound scanner (SSA-370A Power Vision 6000, Toshiba Corporation, Otawara-Shi, Japan) with a 6 cm (8 MHz) linear array transducer. Scans of the BBS were obtained from the dominant arm, whilst participants lay supine with the elbow fully extended and the shoulder abducted at 90°. Strips of surgical tape (50 mm long, 2 mm wide; 3M, Neuss, Germany) were placed at 50 mm intervals along the length of the upper arm from the cubital crease to the shoulder and acted as markers with which muscle length could be determined. Muscle

thickness was then assessed at 25, 50 and 75% BBS muscle length. The values at the three sites were averaged to give a mean BBS muscle thickness value. Reliability analysis was performed with N = 8 who performed measures in duplicate. For this, the surgical strips were removed, and the testing procedure was repeated. Due to technical reasons, nine of the participants were unable to complete both pre and post muscle thickness measurements. Therefore, muscle thickness data are reported for N = 36.

Statistical Analysis

Data are reported as mean ± standard error of the mean (SEM). Reliability analysis for strength data was performed using pre-1 and pre- 2 measurements in which 15 participants were chosen at random from the data sets of 1RM, iMVF and explosive force production. Significance testing to assess the consistency of the mean values was determined and the within-subject coefficient of variation (CV) and intra class coefficient (ICC) used to further determine reliability. Differences between pre-2 and post-training absolute measures (iMVF, 1RM and EMG during these maximal contractions, muscle thickness, and pRFD and time to pRFD during the explosive contractions) were determined using paired t-tests. As the absolute values of 1RM and iMVF are considerably different, we reported relative changes in 1RM and iMVF which were calculated as mean ± SEM of individual percentage changes, and compared these with a paired t-test. A Two-way repeated measures ANOVA was performed to contrast the changes in EMG variables for iMVF and 1RM with training (task: iMVF vs. 1RM; training, pre vs. post) and an interaction effect reported.

Time-series data during the explosive contractions (force and EMG) were assessed with two-way repeated measures ANOVA (training [pre vs. post] vs. epoch [0-50, 0-100, 0-150 ms]). Post-hoc pair-wise comparisons (Bonferroni corrected paired t-test) were used to determine if

there were pre- vs. post-training differences at specific time points. Statistical analysis was completed using SPSS version 17, and the significance level was set at P < 0.05.

RESULTS

Reliability

There were no significant changes in iMVF (paired t-test, P = 0.473), 1RM (paired t-test, P = 0.827) or explosive force production (ANOVA, P = 0.127) from pre-1 to pre-2 measurement sessions. Furthermore, these strength measures demonstrated moderate to excellent reliability (iMVF, CV 3.4%, ICC 0.97; 1RM, CV 3.5%, ICC 0.98; Explosive force, 50 ms, CV 14.6%, ICC 0.85; 100 ms, CV 4.1%, ICC 0.97; 150 ms, CV 3.5%, ICC 0.97). Additionally, muscle thickness measurements taken in duplicate before training were very consistent (paired t-test, P = 0.819) and showed high levels of reliability (CV 2.5%, ICC 0.94). There were no changes in EMG amplitude recorded during the reference tasks between the pre-2 and post-training measurement sessions: M_{max} amplitude (pre, 11.6 ± 1.3 vs. post, 12.2 ± 1.3 mV, paired t-test, P = 0.280); TB EMG_{max} during elbow extension (pre, 0.27 ± 0.03 vs. post 0.25 ± 0.03 mV; paired t-test, P = 0.130); AD and PM EMG_{max} during bench press (AD pre, 0.65 ± 0.04 vs. post, 0.62 ± 0.05; PM, pre, 0.50 ± 0.13 vs. post, 0.35 ± 0.04 mV; both, paired t-test, P ≥ 0.148).

Maximum Isometric Strength and 1RM

Elbow flexor iMVF increased from 240 ± 7 to 258 ± 8 N (paired t-test, P < 0.001) and 1RM lifting strength increased from 10.9 ± 0.4 to 12.6 ± 0.4 kg (paired t-test, P < 0.001) after three weeks of RT, with a greater percentage increase of 1RM than iMVF (17.0 ± 2.0 vs. 7.4 ± 1.4% , paired t-test, P < 0.001, Figure 2).

[Figure 2]

There was a 15.7% increase in agonist EMG amplitude during the 1RM (paired t-test, P <
0.001), with no change in agonist EMG at iMVF after the RT (paired t-test, P ≥ 0.541; Figure
3A). The increase in agonist EMG amplitude was greater for the 1RM than iMVF (ANOVA,
task x training P = 0.005, respectively). Agonist EMG amplitude was also 32.6% higher
during the 1RM than iMVF pre-training and 57.3% higher post-training (both, paired t-test, P
< 0.001).

Whilst, antagonist EMG during 1RM increased by 26.2% (paired t-test, P = 0.032, Figure 3B)
and co-activation at iMVF was unchanged (paired t-test, P = 0.701), there was no difference
between the changes in 1RM and iMVF (ANOVA, task x training, P = 0.143). Antagonist
EMG was 52.9% higher during the 1RM than at iMVF pre-training and 84.9% higher post-
training (both, paired t-test, P ≤ 0.001).

Stabiliser EMG also increased during the 1RM (43.2%, paired t-test, P < 0.001, Figure 3C)
and this reflected an increase for both the AD and PM (AD, pre, 56.0 ± 6.7 vs. post, 82.0 ±
6.5% EMG$_{max}$; PM, pre, 56.0 ± 5.3 vs. post, 80.7 ± 8.8% EMG$_{max}$, both, paired t-test, P ≤ 0.002).
Similarly, at iMVF overall stabiliser EMG (+53.1%, paired, t-test, P < 0.001) and that of each
of the stabiliser muscles was elevated post-training (AD, pre, 26.7 ± 2.2 vs. post, 44.7 ± 3.9;
PM, pre 41.5 ± 3.8 vs. post, 60.8 ± 4.6% EMG$_{max}$, both, paired t-test, P < 0.001). The
training-induced changes in stabiliser EMG were similar for 1RM and iMVF (ANOVA, task
x training, P ≥ 0.297). Stabiliser EMG was 65.4% higher during the 1RM than at iMVF pre-
training and 50.6% higher post-training (both, paired t-test, P≤ 0.001).

[Figure 3]

Explosive Isometric Contractions

During the explosive contractions there was no training effect on absolute force production at any time point (ANOVA, training, P = 0.595, Figure 4A), peak RFD (pre, 3224 ± 108.4 vs. post, 3210 ± 124.3 N.s^{-1}; paired t-test, P = 0.853) or impulse produced over any of the time periods (ANOVA, training, P = 0.495). However, there was a training effect for explosive force production normalised to iMVF (ANOVA, training, P = 0.008), with a significant decrease in % iMVF achieved at 50 ms (-16.5%, Bonferroni, P = 0.003), but no change at 100 (Bonferroni, P = 0.123), or 150 ms (Bonferroni, P = 0.735; Figure 4B). Similarly, there was a decrease in relative peak RFD (pre, 13.5 ± 0.3 vs. post, 12.6 ± 0.4 iMVF.s^{-1}, paired t-test, P = 0.005), and an increase in the time to reach peak RFD (pre, 57.2 ± 2.3 vs. post, 63.6 ± 2.3 ms, paired t-test, P = 0.004). Voluntary EMD$_{max}$ remained unchanged after training (pre, 25.1 ± 0.8 vs. post, 24.5 ± 0.9 ms, paired t-test, P = 0.417).

[Figure 4]

The training did not influence the EMG amplitude during the explosive contractions for either the agonist (ANOVA, training, P = 0.133, Figure 5A) or antagonist muscles (ANOVA, training, P = 0.682; Figure 5B), but there was an increase in stabiliser muscle activation (ANOVA, training, P < 0.001, Figure 5C) with significant increases over 0-50 (45.3%, Bonferroni, P = 0.003) and 0-150 ms (26.2%, Bonferroni, P < 0.001), but not 0-100 ms (Bonferroni, P = 0.201).

[Figure 5]

Muscle Thickness

Thickness of the biceps brachii short-head increased from 15.0 ± 0.5 to 15.7 ± 0.6 mm (paired t-test, P = 0.003) following training, representing a change of 5.3 ± 1.4 %.

DISCUSSION

This study investigated the changes in elbow flexion strength tasks (isoinertial, and isometric maximum and explosive strength), and evaluated the adaptations in agonist, antagonist and stabiliser neuromuscular activation that may contribute to improved strength following 3-wk RT. It was hypothesised that conventional isoinertial RT would induce greater increases in isoinertial, than isometric strength, and that this would be concomitant with greater changes in agonist and stabiliser muscle activation during the isoinertial task. After training there was more than a two-fold greater increase in training task specific isoinertial than isometric strength (17 vs. 7%) that appeared to be due to task specific neural adaptations during the 1RM. Specifically, task-specific adaptations in agonist EMG during the 1RM (+16%) with no change in agonist EMG at iMVF. A novel finding of this study was that training increased stabiliser muscle activation during all elbow flexion strength tasks, but with no task specific training effects. After training there was no change in absolute explosive force production, but there was a decrease in relative early phase explosive force production.

This study included a large cohort of participants (N = 45) and demonstrated good to excellent reliability of the strength and muscle thickness measurements. Furthermore, there was no change in any of the EMG reference measures following training. Normalisation of agonist, antagonist and stabiliser EMG during the elbow flexion contractions to these reference measurements would be expected to reduce measurement variability and enhance the statistical power of the experiment (Buckthorpe et al., 2012). These methodological strengths of the study provide confidence that the changes in performance and underlying physiological mechanisms are adaptations as a result of the short-term training.

We observed a 17% increase in the 1RM following RT which is equivalent to the changes observed in another upper body RT study [chest press 1RM 17% increase after 4 weeks, (Abe et al., 2000)]. This increase in 1RM was more than two-fold greater than the 7% increase in iMVF and this differential response is similar in magnitude to previous longer duration isoinertial RT studies [18 vs. 40% increase after 9 weeks, (Folland et al., 2002); 16 vs. 40% after 12 weeks (Hubal et al., 2005)]. This is the first study to provide strong evidence for neural mechanisms explaining this tasks specificity phenomenon. The task specific increase in agonist activation during the 1RM (no change in agonist EMG at iMVF) provides a ready explanation for the greater increase in isoinertial vs. isometric strength. Previous research has reported an increase in absolute agonist EMG during the 1RM after 12 weeks of RT [knee extensors, (Hakkinen et al., 1998)], but within the present study we also used the more robust method of normalised EMG (to M_{max} +16%) and found strong evidence that increased agonist activation contributes to isoinertial strength gains after RT. Considering isometric strength, evidence for changes in normalized agonist EMG (to M_{max}) at iMVF following RT is equivocal (Van Cutsem et al., 1998; Pucci et al., 2006; Tillin et al., 2011). The conflicting findings may relate to the muscle group investigated, as Behm and colleagues (2002) found agonist activation at iMVF to vary between muscle groups for untrained participants. Using the interpolated twitch technique (ITT), activation of the elbow flexors even in untrained participants has been reported to be very high (> 98%, (Allen et al., 1998)), which may explain why training did not increase agonist activation at iMVF in the current study.

Interestingly, agonist EMG during the 1RM was higher than at iMVF (pre, +33%; post +57%), which might indicate a higher level of muscle activation during the 1RM or simply reflect methodological differences between these measurements. As discussed above, elbow flexor activation at iMVF assessed with the ITT has been reported to be very high/maximal. Therefore, if the ITT and EMG are valid measures of voluntary activation, it does not seem

plausible for EMG during the 1RM to be over 50% higher than EMG at iMVF. On the other hand, the validity of the ITT for providing a quantifiable measurement of agonist activation at iMVF is controversial (e.g. de Hann et al., 2009). Likewise surface EMG has a number of technical limitations which may affect the recorded interference signal (see de Luca, 1997). Elbow flexor iMVF was recorded at 60° elbow flexion, whereas peak EMG during the 1RM typically occurred at a more extended joint angle, i.e. ~10-50°. There was also a subtle difference in shoulder flexion angle between the two tasks (1RM 75° vs. iMVF 90°). Joint angle has been found to influence the amplitude of volitional EMG (Kasprisin & Grabiner, 2000) and thus angle specific differences in both shoulder and elbow angles could explain the task specific discrepancy in EMG amplitude we have found. Despite the issues surrounding the quantification of voluntary muscle activation, the present results suggest that the greater gains in isoinertial lifting strength were explained by task specific adaptations in agonist neuromuscular activation.

The consistency of antagonist co-activation values pre and post training at iMVF is in accordance with two previous studies (Hakkinen et al., 1998; Pucci et al., 2006), but conflicts with studies that have reported both increased (Simoneau et al., 2006; de Boer et al., 2007) and decreased (Hakkinen et al., 1998; Tillin et al., 2011) co-activation following RT. Tillin et al. (2011) recently found increased co-activation at iMVF following four weeks of RT, but a reduction in antagonist activation expressed as a ratio of agonist activation, across a range of contraction intensities up to iMVF post-training. It may be that in the present study the increase in iMVF was not large enough to require an increase in co-activation to maintain joint integrity, or that the elbow joint is sufficiently stable at iMVF not to require increased co-activation as strength increases. There was however a large increase in co-activation during the 1RM following RT (26%), which may have attenuated the overall gains in isoinertial strength, and might reflect a greater need to stabilise the elbow joint during the

1RM (Cochrane et al., 2006; Tillin et al., 2011). There was no change in antagonist EMG during the explosive contractions, which may be due to the consistent force and agonist EMG of these contractions after training.

Stabiliser muscle activation increased by a similar extent during both the 1RM and at iMVF (39 vs. 53%, respectively) and is a novel finding, which likely contributed to changes in strength during both tasks. The effective stabilisation of joints is thought to be important for optimal force production (Folland & Williams, 2007), and the adaptation in stabiliser activation we have observed may help to explain the commonly observed discrepancy between muscle size and iMVF changes following RT (e.g. Narici et al., 1996). It was hypothesised that the greater increase in isoinertial, than isometric strength, would be concomitant with greater changes in stabiliser muscle activation during the isoinertial task. Whilst there was no task specific training effect on stabiliser activation, it is possible that increased stabiliser activation was of greater consequence during the 1RM that for iMVF. During isoinertial elbow flexion more movement is available at the shoulder than is the case during the isometric measurements where force production is constrained to the elbow joint by the apparatus and strapping. Therefore the elbow flexion 1RM may be more responsive to synergistic contributions from shoulder joint muscles or more effective stabilisation of the shoulder during this task. Thus the 1RM could be more responsive to enhanced stabiliser activation after training. Furthermore, increased stabiliser activation and shoulder joint stabilisation during the 1RM after RT may have facilitated an increase in agonist activation, particularly for the bi-articular bicep brachii. Thus the 1RM could be more responsive to enhanced stabiliser activation after training. It would be interesting for future work to consider the response of a wider range of stabiliser muscles after training. The 5% increase in muscle thickness we observed indicates the occurrence of hypertrophy after only three weeks of RT, and is in agreement with an earlier study that also found hypertrophy after only three

weeks of training [knee extensors, +3.5-5.2%, (Seynnes et al., 2007)]. Therefore, this study supports the notion that skeletal muscle hypertrophy can occur during the initial 3 weeks, or 9 sessions, of training, and might be expected to have made a greater relative contribution to the observed gains in isometric than isoinertial strength.

There was no change in absolute force and a decrease in early phase relative force production (50 ms) during the explosive voluntary contractions following RT, which further questions the efficacy of RT for enhancing explosive strength (Andersen et al., 2010, Tillin et al., 2011). There is some evidence that including an explosive strength component to RT (i.e., intending to lift the weight as quickly as possible) is sufficient to enhance early phase explosive strength and agonist neural drive (Behm & Sale, 1993; Van Cutsem et al., 1998; Barry et al., 2005; Del Balso & Cafarelli, 2007). However, during conventional isoinertial RT, the continuous, cyclic nature of the repetitions, with gradual controlled lowering (eccentric) immediately followed by lifting (concentric), may involve high levels of activation and force throughout each set. Therefore, even if attempting to perform the concentric phase of the lift as quickly as possible (as was the case in the present study) there may be no transition from low to high levels of activation/force that is required to enhance explosive force production. Alternatively, it is possible that adaptations in agonist neural drive and explosive force production with the training task may have been specific to the early concentric phase of the lift, i.e. at more extended joint angles than the angle used to measure isometric strength. Angle specific adaptations in maximal strength following RT have been known for some time (Knapik et al., 1983), but further investigation into angle-specific changes in explosive strength and associated mechanisms following isoinertial maximum and/or explosive strength training would have strong implications for athletic training.

Given the large increase in stabiliser activation during the explosive contractions in the absence of any changes in absolute force, it is likely that stabiliser muscle activation does not exert a strong independent influence on isometric explosive strength. However, future research should look to determine the role of stabiliser muscle activation on explosive muscle strength and address the inter-relationship of agonist and stabiliser muscle activation.

In summary, task specific neural adaptations, particularly increased agonist activation during the 1RM, appeared to explain the greater increase in isoinertial than isometric strength. Increased antagonist co-activation during the 1RM was the likely result of an increased load lifted and may be a protective mechanism to maintain joint integrity. Changes in iMVF were thought to be explained by increased muscle size and stabiliser muscle activation rather than changes in agonist or antagonist muscle activation. Despite participants attempting to lift the weight as quickly as possible, 3 weeks of RT resulted in no change in absolute explosive force production and a decrease in relative early phase explosive force production, which questions the efficacy of conventional isoinertial RT for enhancing explosive strength.

PERSPECTIVES

This is the first study to directly assess the neural mechanisms (agonist, antagonist, stabiliser activation) which contribute to training specific changes in isoinertial compared to isometric strength measures following isoinertial RT. The observation of short term training specific adaptations in strength following RT provides good indirect evidence that neural mechanisms are likely responsible for this phenomenon, and although the task-specific increase in agonist activation suggests the likely mechanism for increased strength, the role of stabiliser EMG changes to changes in strength are not fully understood. The changes in stabiliser activation are novel and despite only assessing two superficial global stabiliser muscles, the findings of

the study suggest marked changes in stabiliser activation elicited from RT. As RT with maximal intention to lift the weight as fast as possible did not elicit changes in absolute explosive force capabilities, it is suggested that specific training to enhance EMG rise at force onset is included to enhance RFD alongside training for maximal muscle strength.

ACKNOWEDGEMENTS

CONFLICTS OF INTEREST

REFERENCES

Aagaard P, Simonsen EB, Andersen JL, Magnusson P & Dyhre-Poulsen P. Increased rate of force development and neural drive of human skeletal muscle following resistance training. J Appl Physiol 2002: 93: 1318-26.

Abe T, Dehoyos DV, Pollock M & Garzella L. Time course for strength and muscle thickness changes following upper and lower body resistance training in men and women. Eur J Appl Physiol 2000: 81: 174-80.

Allen, GM, McKenzie DK & Gandevia SC. Twitch Interpolation of the elbow flexors at high muscle forces. Muscle Nerve 1998: 21: 318-28.

Allison GT. Trunk muscle onset detection technique for EMG signals with ECG artefact. J Electromyogr Kinesiol 2003: 13: 209-16.

Andersen LL, Andersen JL, Kebis MK & Aagaard P. Early and late rate of force development: differential adaptive responses to resistance training? Scand J Med Sci Sports 2010: 20 (1): e162-9.

Baecke JA, Burema J & Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. Am J Clin Nutr 1982: 36: 936-42.

Barry BK, Warman GE & Carson RG. Age-related differences in rapid muscle activation after rate of force development training of the elbow flexors. Exp Brain Res 2005: 162: 122-132.

Behm DG & Sale DG (1993). Intended rather than actual movement velocity determines velocity-specific training response. J Appl Physiol 1993: 74: 359-68.

Behm DG, Whittle J, Button D & Power K. Intermuscle differences in activation. Muscle Nerve 2002: 25: 236-43.

Buckthorpe MW, Hannah R, Pain MTG. & Folland JP. Reliability of neuromuscular measurements during explosive isometric contractions, with special reference to electromyography normalization techniques. Muscle Nerve 2002: 46: 566-76.

Cacchio A, Don R, Ranavolo A, Guerra E, McCaw ST, Procaccianti R, Camerota F & Santilli V. Effects of 8-week strength training with two models of chest press machines on muscular activity pattern and strength. J Electromyogr Kinesiol 2008: 18: 618-27.

Chang SH, Mercer VS, Giuliani CA & Sloane PD. Relationship between hip abductor rate of force development and mediolateral stability in older adults. Arch Phys Med Rehabil 2005: 86: 1843-50.

Cochrane J, Lloyd D, Ackland T, Besier T & Elliot B. The effect of lower limb training on muscular support of the knee and risk of anterior cruciate ligament injury. In: Proceedings of XXIV International Symposium on Biomechanics in Sport 2006 July 14-18: Salzburg, (Austria), p.1-4.

de Boer MD, Morse CI, Thom JM, de Haan A, & Narici MV. Changes in antagonist muscles' coactivation in response to strength training in older women. J Gerontol A Biol Sci Med Sci 2007: 62: 1022-7.

de Haan A, Gerrits K H &de Ruiter CJ. Counterpoint: the interpolated twitch does not provide a valid measure of the voluntary activation of muscle. J Appl Physiol 2009: 107: 355-77, Discussion 357-8.

Del Balso C & Cafarelli E. Adaptations in the activation of human skeletal muscle induced by short-term isometric resistance training. J Appl Physiol 2007: 103: 402-11.

de Ruiter CJ, Leeuwen DV, Heijblom A, Bobbert MF & de Haan A. Fast isometric knee extension torque development and bilateral jump height. Med Sci Sports Exerc 2006: 38: 1843-53.

Folland JP, Buckthorpe MW & Hannah R. Human capacity for explosive force production: Neural and contractile determinants. Scand J Med Sci Sports 2013: DOI: 10.1111/sms.12131.

Folland JP, Irish CS, Roberts JC, Tarr JE, Jones JA. Fatigue is not a necessary stimulus for strength gains during resistance training. Br J Sports Med 2002: 36: 370-4.

Folland JP & Williams A. The adaptations to strength training: Morphological and neurological contributions to increased strength. Sport Med 2007: 37 (2): 145-68.

Hakkinen K, Kallinen M, Izquierdo M, Jokelainen K, Lassila H, Malkia E, Kraemer WJ, Newton RU & Alen M. Changes in agonist-antagonist EMG, muscle CSA, and force during strength training in middle-aged and older people. J Appl Physiol 1998: 84: 134149.

Hubal MJ, Gordish-Dressman H, Thompson PD, Price TB, Hoffman EP, Angelopoulos TJ, Gordon PM, Moyna NM, Pescatello LS, Visich PS, Zoeller RF, Seip RL & Clarkson PM. Variability in muscle size and strength gain after unilateral resistance training. Med Sci Sports Exerc 2005: 37: 964-72.

Izquierdo M, Aguado X, Gonzalez R, Lopez JL & Hakkinen K. Maximal and explosive force production capacity and balance performance in men of different ages. Eur J Appl Physiol Occup Physiol 1999: 79: 260–7.

Kasprisin JE & Grabiner MD (2000). Joint angle dependence of elbow flexor activation during isometric and isokinetic maximum voluntary contractions. Clin Biomech 2000: 15: 743-9.

Knapik JJ, Mawdsley RH & Ramos MU. Angular specificity and test specificity of isometric and isokinetic strength training. J Orthop Sports Phys Therapy 1983: 5 (2): 58-65.

Kubo K, Ohgo K, Takeishi R, Yoshinaga K, Tsunoda N, Kaneshisa H et al. Effects of isometric training at different knee angles on the muscle tendon complex in vivo. Scand J Med Sci Sports 2006: 16: 159-67.

Lee JH, Kim HW, Im S, An X, Lee MS, Lee UY & Han SH. Localization of motor entry points and terminal intramuscular nerve endings of the musculocutaneous nerve to biceps and brachialis muscles. Surg Radiol Anat 2010: 32: 213-20.

Moretti DV, Babiloni F, Carducci F, Cincotti F, Remondini E, Rossine PM, Salinari S, Babiloni C. Computerized processing of EEG-EOG-EMG artifacts for multi-centric studies in EEG oscillations and event-related potentials. Int J Psychophysiol 2003: 47: 199-216.

Narici MV, Hoppeler H, Hayser B, Landoni L, Classen H, Gavardi C, Conti M, Cerretelli P. Human quadriceps cross-sectional area, torque and neural activation during 6 months strength training. Acta Physiol Scand.1996: 157: 175-86.

Pijnappels M, van der Burg PJ, Reeves ND & van Dieen JH. Identification ofelderly fallers by muscle strength measures. Eur J Appl Physiol: 102: 585–92.

Pucci AR, Griffin L & Cafarelli E. Maximal motor unit firing rates during isometric resistance training in men. Exp Physiol 2006: 91: 171-8.

Rutherford OM & Jones DA. The role of learning and coordination in strength training. Eur J Appl Physiol 1986: 55: 100-5.

Sahaly R, Vandewalle H, Driss T & Monod H. Maximal voluntary force and rate of force development in humans- importance of instruction. Eur J Appl Physiol 2001 : 85: 345-50.

Seynnes OR, de Boer M & Narici MV. Early skeletal muscle hypertrophy and architectural changes in response to high intensity resistance-training. J Appl Physiol 2007: 102: 368-73.

Simoneau E, Martin A, Porter M & Van Hoecke J. Strength training in old age: adaptations of antagonist muscles at the ankle joint. Muscle Nerve 2006: 33: 546-55.

Thorstensson A, Karlsson J, Viitasalo HT, Luhtanen P & Komi PV. Effect of strength training on EMG of human skeletal muscle. Acta Physiol Scand 1976: 98: 232-36.

Tillin NA, Jimenez-Reyes P, Pain MTG & Folland JP. Neuromuscular performance of explosive power athletes vs. untrained individuals. Med Sci Sports Exerc 2010: 42: 781-790.

Tillin NA, Pain MTG. & Folland JP. Short-term unilateral resistance training affects the agonist-antagonist but not the force-agonist activation relationship. Muscle Nerve 2011: 43: 375-84.

Tillin NA, Pain MTG & Folland JP. Explosive force production during isometric squats correlates with athletic performance in rugby union players. J Sports Sci 2013: 31: 66-76.

Van Cutsem M, Duchateau J & Hainaut K. Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. J Physiol 1998: 513 (Part 1): 295-305.

FIGURE LEGENDS

Figure 1. A, The modified preacher curl bench used for both training and testing of the elbow flexors and; B, the isometric strength testing apparatus used to measure elbow flexion/ extension force**.**

Figure 2. Percentage change in iMVF and 1RM following three weeks of resistance training. (Mean ± SEM, N = 45). ** indicates a significantly greater change in 1RM than isometric MVF (Paired t-test, $P < 0.001$).

Figure 3 Agonist (A), antagonist (B) and stabiliser (C) muscle EMG amplitude measured at isometric maximum voluntary force (open circles) and during the 1RM (filled squares). Data are reported as mean ± SEM (N= 45, [N = 39 for agonist EMG normalised to M_{max}]). A training effect is denoted by * ($P < 0.05$), ** ($P < 0.001$).

Figure 4. Absolute (A) and relative (B) force (relative to maximum voluntary force; MVF) during explosive isometric contractions of the elbow flexors pre (dashed line, open circles) and post (bold line, filled square) training. Data are mean ± SEM for the group (N = 45). A training effect is denoted by ** ($P < 0.01$).

Figure 5. Agonist (A), antagonist (B) and stabiliser (C) muscle EMG amplitude during explosive isometric contractions of the elbow flexors pre (black) and post (white) training. Date are reported as mean ± SEM for the group (N = 45). A training effect is denoted by * ($P \leq 0.05$), ** ($P \leq 0.001$).