

The contribution of muscle hypertrophy to strength changes following resistance training

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Abstract

Purpose Whilst skeletal muscle hypertrophy is considered an important adaptation to resistance training (RT), it has not previously been found to explain the inter-individual changes in strength after RT. This study investigated the contribution of hypertrophy to individual gains in isometric, isoinertial and explosive strength after 12 weeks of elbow flexor RT.

Methods Thirty-three previously untrained, healthy men (18–30 years) completed an initial 3-week period of elbow flexor RT (to facilitate neurological responses) followed by 6-week no training, and then 12-week elbow flexor RT. Unilateral elbow flexor muscle strength [isometric maximum voluntary force (iMVF), single repetition maximum (1-RM) and explosive force], muscle volume (V_m), muscle fascicle pennation angle (θ_p) and normalized agonist, antagonist and stabilizer sEMG were assessed pre and post 12-week RT.

Results Percentage gains in V_m correlated with percentage changes in iMVF ($r = 0.527$; $P = 0.002$) and 1-RM ($r = 0.482$; $P = 0.005$) but not in explosive force ($r \leq 0.243$; $P \geq 0.175$). Percentage changes in iMVF, 1-RM, and explosive force did not correlate with percentage changes in

agonist, antagonist or stabilizer sEMG (all $P > 0.05$). Percentage gains in θ_p inversely correlated with percentage changes in normalized explosive force at 150 ms after force onset ($r = 0.362$; $P = 0.038$).

Conclusions We have shown for the first time that muscle hypertrophy explains a significant proportion of the inter-individual variability in isometric and isoinertial strength gains following 12-week elbow flexor RT in healthy young men.

Keywords Strength training · Muscle volume · Muscle architecture · Inter-individual variability · Neuromuscular adaptations

Abbreviations

ACSA	Anatomical cross-sectional area
AD	Anterior deltoid
ANOVA	Analysis of variance
BBL	Biceps brachii long head
BBS	Biceps brachii short head
BR	Brachioradialis
BRACH	Brachialis
EMG	Electromyography
iMVF	Isometric maximal voluntary force
MVC	Maximum voluntary contraction
MRI	Magnetic resonance imaging
M_{max}	Evoked supramaximal compound muscle action potential
PM	Pectoralis major
RMS	Root mean square
RT	Resistance training
sEMG	Surface electromyography
V_m	Muscle volume
1-RM	Single repetition maximum
θ_p	Muscle fascicle pennation angle

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Introduction

The strength response to resistance training (RT) is known to vary considerably between previously untrained individuals (Erskine et al. 2010; Hubal et al. 2005). Considering that muscle size explains ~50 % of the inter-individual variability in maximum strength in the untrained state (Kanehisa et al. 1994; Bamman et al. 2000; Fukunaga et al. 2001), it is surprising that muscle hypertrophy does not appear to account for the variance in strength gains following RT (Jones and Rutherford 1987; Davies et al. 1988). However, it is possible that neural adaptations, also known to occur with RT, could confound the contribution of hypertrophy to strength gains. In fact, the first 2–3 weeks of a RT program have been shown to cause rapid increases in strength that have been largely attributed to neural adaptations, while the contribution of muscle hypertrophy to strength gains is considered to be increasingly more important after these initial weeks (Moritani and deVries 1979; Seynnes et al. 2007). Therefore, the role of hypertrophy in explaining strength gains may be elucidated by considering the RT responses after the first weeks of RT, i.e., once neural adaptations have largely taken place. An initial phase of RT may also serve as a standardized period of physical activity, thus reducing the variability in training status [which might also affect the individual training responses (Kraemer et al. 2002)] prior to a more prolonged experimental period of RT.

The contribution of muscle hypertrophy to strength gains may depend on the strength task assessed, e.g., isometric, isoinertial or explosive strength. Although it is well established that RT induces gains in both isometric and isoinertial strength (Rutherford and Jones 1986; Erskine et al. 2010; Folland et al. 2002), the effect of RT on explosive strength is controversial (Aagaard et al. 2002; Hakkinen et al. 1998; Andersen et al. 2010; Tillin et al. 2011; Blazevich et al. 2009, 2008). A better understanding of the how specific physiological adaptations contribute to the individual improvements in isometric, isoinertial and explosive strength after RT may help to optimize RT, to elicit specific adaptations and functional outcomes, such as improved physical performance in athletic groups and a reduced risk of falling in older populations.

In addition to neural and hypertrophic adaptations, RT is known to increase the muscle fascicle pennation angle (θ_p), i.e., the angle at which the muscle fascicles insert into the aponeurosis (Aagaard et al. 2001; Erskine et al. 2010). Although an increase in θ_p enables more contractile material to attach to the aponeurosis (leading to an increase in force output), there is a concomitant reduction in force resolved at the tendon due to the oblique line of pull of the fascicles (Alexander and Vernon 1975). Therefore, documenting inter-individual differences in θ_p

in response to RT may provide a more complete assessment of how morphological adaptations explain strength changes following RT.

The aim of this study was to determine the contribution of muscle hypertrophy to the inter-individual differences in isometric, isoinertial and explosive strength changes in response to RT. An upper-body elbow flexor RT model was used to maximize the hypertrophic response (Cureton et al. 1988; Welle et al. 1996), and changes in θ_p were also assessed. The unique design of this study incorporated an initial 3-week RT period to overcome neural adaptations and to standardize prior physical activity before participants completed a 12-week experimental RT period. Changes in neuromuscular activation of the agonist, antagonist and stabilizer muscles were assessed by normalizing surface EMG activity to appropriate reference measures to give context to the morphological adaptations.

Methods

Participants

Thirty-three healthy, recreationally active young men volunteered (mean \pm SD age 23.4 ± 3.0 years; height 1.76 ± 0.06 m; body mass 75.2 ± 10.7 kg) and provided written informed consent prior to their involvement in this 25-week study, which was approved by the Loughborough University Ethical Advisory Committee and conformed to the standards set by the 1964 Declaration of Helsinki. Health status and habitual physical activity were assessed using questionnaires and the physical activity rating was 2.6 ± 0.4 , where 1 = extremely inactive and 5 = exceptionally active (Baecke et al. 1982). Volunteers were excluded from the study if they reported use of purported anabolic supplements in the previous 6 months, had a history of upper-body exercise in the previous 12 months or were <18 or >30 years old.

Study overview

Some of the muscle response data reported here have been published in a previous report investigating the effects of protein supplementation on the gains in muscle size, strength and architecture with RT (Erskine et al. 2012). As no differences between protein and placebo supplementation groups were observed regarding any of the training adaptations, the data have been collapsed across groups for the purpose of answering the current (long-standing and previously unresolved) research question, i.e., what is the contribution of muscle hypertrophy to strength changes following RT? In addition to the previously reported data, stabilizer surface EMG (sEMG) and explosive force data

have been included here to provide a more comprehensive account of the neuromuscular adaptations to chronic RT.

The RT protocol and some of the pre and post-training measurements have been described in detail in the previously published study (Erskine et al. 2012). Therefore, they will be described briefly here. Thirty-three participants completed an initial 3-week period of elbow flexor RT, which was followed by 6 weeks of no training and then a 12-week period of experimental elbow flexor RT. The initial RT period provided extensive familiarization to the RT exercises and neuromuscular tests (data not reported here), whilst also standardizing participant training status and facilitating neural adaptations prior to the 12-week experimental RT period. All RT involved exercising both arms. Three to four days before and after the 12-week RT, strength [maximum isometric voluntary force (iMVf), single repetition maximum (1-RM) and explosive force], size [muscle volume and maximum anatomical cross-sectional area ($ACSA_{max}$)] and fascicle pennation angle (θ_p) of the elbow flexor muscles were measured in the dominant arm. To determine whether neural adaptations did occur during the 12-week RT (and to help differentiate neural from morphological contributions to strength gains), sEMG of the agonist, antagonist and stabilizer muscles was assessed during the three strength tasks and normalized to appropriate reference measures. All tests for each participant were performed at the same time of day before and after training.

Resistance training (RT)

Participants performed three training sessions per week (Monday, Wednesday and Friday) during both RT periods. Each session comprised unilateral seated elbow flexion ‘preacher curls’ using dumbbells, with alternating sets using the dominant and non-dominant arms, and then bilateral preacher curls on a resistance training machine (Body Solid, Forest Park, USA), with a 2-min rest between sets. The loading for both exercises was 8–10 RM and the load was increased when participants could lift 10 reps during the final set of an exercise. The 3-week RT involved two sets of each exercise, and this was the same for week 1–2 of the 12-week RT, but increased to three sets (unilateral) and two sets (bilateral) during week 3–4 and three sets of both exercises for week 5–12. Participant adherence was 100 %, i.e., all participants performed 9 and 36 training sessions during the 3- and 12-week RT periods, respectively.

Pre- and post-RT neuromuscular measurements

Unilateral single repetition maximum (1-RM)

A series of incremental unilateral elbow flexion preacher curl lifts of a dumbbell were performed whilst seated on

the same modified preacher bench that was used in training. After ten warm-up reps at 40 % 1-RM, three reps were performed at 80 % 1-RM. Thereafter, a series of single lifts were performed with 1-min rest intervals at increments of +0.5 kg if the preceding lift was successful. The last successful lift was defined as 1-RM.

Isometric maximum voluntary force (iMVf)

Elbow flexor iMVf was measured using a custom-built strength-testing chair with the elbow joint angle set to 60° (0° = full elbow extension). The wrist was strapped to an S-Beam tension–compression load cell (Applied Measurements Ltd, Aldermaston, UK), which was positioned perpendicular to the direction of forearm movement during isometric elbow flexion/extension. The force signal was interfaced with an analog-to-digital converter (CED micro 1401, CED, Cambridge, UK), sampled at 2 kHz with a PC using Spike 2 software (CED, Cambridge, UK) and low-pass filtered (500 Hz edge frequency) with a second-order Butterworth digital filter. Participants completed four isometric elbow flexion maximum voluntary contractions (MVCs), each lasting 3 s and separated by ≥ 30 s. Biofeedback and verbal encouragement were provided during and between each MVC. Participants then completed four isometric elbow extension MVCs with an identical protocol to determine the maximum sEMG ($sEMG_{max}$) amplitude of the TB (see below for details). Isometric MVf for elbow flexion and extension was the greatest instantaneous voluntary force achieved during that action.

Isometric explosive contractions

In addition to the MVCs detailed above, participants performed ten isometric explosive voluntary elbow flexion contractions (each separated by 20 s). During each contraction participants attempted to flex their elbow as ‘fast and hard’ as possible (Sahaly et al. 2001), with emphasis on fast, for 1 s from a relaxed state, while achieving at least 80 % iMVf. During each contraction, participants were instructed to avoid any countermovement (elbow extension prior to elbow flexion). A computer monitor displayed both force (on a sensitive scale around resting values) and the slope of the force–time curve. The latter was used to provide immediate biofeedback of performance, specifically peak rate of force development (RFD, 1 ms time constant) during each contraction, and the former highlighted any countermovement. The three contractions with the largest peak RFD and no discernible countermovement or pre tension (change of baseline force of <0.5 N during the 100 ms prior to contraction onset) were used for analysis of the force signal. Analysis consisted of measuring force at 50, 100 and 150 ms from force onset and peak RFD (which

typically occurred at 60–70 ms after force onset). Force at all three time points and peak RFD are reported both in absolute terms and relative to iMVf. Force onset was identified manually as previously described (Tillin et al. 2010), i.e., using constant y - and x -axis scales of ~ 1 N and 500 ms, respectively. After placing the vertical cursor on the onset, the resolution was increased (y -axis scale ~ 0.5 N; x -axis scale 25 ms) to confirm the exact location of force onset, i.e., the apex of the last trough before the signal deflected from the baseline noise.

Muscle hypertrophy

The dominant arm was scanned using a Magnetom Symphony 1.5-T MRI scanner (Siemens AG, Erlangen, Germany) with the participant supine. Three overlapping T1-weighted axial scans (time of repetition 420 ms; time to echo 1.2 s; matrix 284×448 pixels; field of view 181×200 mm; slice thickness 10 mm; interslice gap 0 mm) were performed perpendicular to the humerus/radius from the acromion process to below the wrist. Reference markers (lipid capsules) were placed on the skin midway along the humerus and radius to ensure accurate reconstruction of the scans offline using a dicom image viewer (Osirix Foundation, Geneva, Switzerland). Thus, the relevant slice from the first scan was matched with the identical slice in the second scan, and so on. The anatomical cross-sectional area (ACSA) of each muscle of interest (biceps brachii, BB; brachialis, BRACH; brachioradialis, BR) was then manually outlined (excluding visible fat and connective tissue) and plotted against bone length. A spline curve was fitted to the ACSA data points of each muscle and volume was calculated as the area under the curve (Erskine et al. 2009); the sum of the three volumes provided total elbow flexor muscle volume. The largest ACSA ($ACSA_{max}$) was recorded for BB, BRACH and BR, and the sum of the three $ACSA_{max}$ provided $\Sigma ACSA_{max}$.

Muscle fascicle pennation angle (θ_p)

BB short head (BBS) and BRACH θ_p were examined using B-mode ultrasonography (SSA-370A Power Vision 6000, Toshiba, Otawara-Shi, Japan) with a 6 cm, 8 MHz linear-array transducer. The participant lay supine with the dominant elbow fully extended and the shoulder abducted by 90° . Two millimeter-wide strips of ultrasound-absorbent tape (3 M, Neuss, Germany) were placed perpendicular to the long axis of the BBS at 50 mm intervals between the cubital crease and the shoulder, which formed markers on the sonographs and ensured that θ_p was analyzed at the same location pre- and post-RT. The probe was slowly glided in a straight line midway between the lateral and medial boundaries from the cubital crease to the

proximal end of BBS (in line with the direction of the muscle fascicles). Individual frames were analyzed offline (NIH ImageJ, Bethesda, USA). Fascicle θ_p was determined in 3 BBS fascicles within 50 mm of its distal end and in 3 BRACH fascicles within 50 mm of its proximal end. The mean of the three measurements determined θ_p for each muscle, and for each individual, the average of the θ_p for BBS and BRACH provided the mean elbow flexor θ_p .

Surface electromyography (sEMG) activity

Surface EMG activity was recorded from two agonists [the short and long heads of biceps brachii (BBS and BBL)], one antagonist [lateral head of triceps brachii (TB)] and two stabilizers [anterior deltoid (AD) and pectoralis major (PM)] on the dominant side using 2 Delsys Bagnoli-4 sEMG systems (Delsys, Boston, USA). Following preparation of the skin (shaving, lightly abrading and cleansing with 70 % ethanol), double-differential surface electrodes (1 cm inter-electrode distance, Model DE-3.1; Delsys) were attached over each muscle using adhesive interfaces. BBS and BBL electrodes were placed mid-belly at a location that corresponded to 75 % of the distance from the coracoid process to the medial epicondyle of the humerus, as this location is distal to the motor point region in each head (Lee et al. 2010). The TB electrode was placed over the distal third of the muscle, and the AD electrode was placed 5 cm distally from the acromion process over mid-sagittal plane. The PM electrode was placed at 50 % of the distance from the medial end of the clavicle to the axilla, and reference electrodes were placed on the clavicle. All electrode locations (with regard to distances from anatomical landmarks) were measured and recorded for relocation during subsequent tests. Surface EMG signals were amplified (100 \times , differential amplifier 20–450 Hz) and sampled at 2 kHz with the same analog-to-digital converter and PC as the force signal, prior to being band-pass filtered in both directions between 6 and 500 Hz using a second-order Butterworth digital filter.

The root mean square (RMS) of the sEMG signal over a 500 ms epoch around iMVf (± 250 ms) was used to assess activation of all muscles during elbow flexion iMVf. During the concentric phase of the 1-RM lift, the sEMG RMS of all muscles was assessed for the 200 ms period that gave the highest agonist sEMG RMS. During explosive contractions, the sEMG RMS from all muscles was determined in time periods of 0–50, 50–100 and 100–150 ms, from the onset of sEMG activity in the first agonist muscle to be activated. As with the onset of force, agonist sEMG onset was identified manually (Tillin et al. 2010), with the y - and x -axis scales set at 100 mV and 500 ms, respectively. The vertical cursor was placed on the onset and the scale was reduced to 50 mV and 25 ms for the y - and

x-axis, respectively, to confirm the exact location of sEMG onset, i.e., the apex of the last peak/trough before the signal deflected from the baseline noise.

To further minimize the variability in sEMG RMS amplitude (Burden 2010; Tillin et al. 2011), recordings during all three elbow flexion strength tasks were normalized to an appropriate reference measurement: BBL and BBS to the evoked supramaximal compound muscle action potential (M_{\max}) in each head (see below for details); TB to TB sEMG_{max} [recorded over a 500 ms epoch around elbow extension iMVF (± 250 ms)]; AD and PM to AD and PM sEMG_{max} (the highest sEMG RMS recorded over successive 500 ms periods) during a maximum isometric bench press (see below for details). The antagonist and stabilizer sEMG recordings during elbow flexion tasks were clearly submaximal and could therefore be normalized to the EMG_{max} of these muscles when acting as agonists (TB elbow extension; AD and PM bench press). Agonist (BBL and BBS) sEMG recordings during the elbow flexion tasks measured maximal volitional activation and thus for normalization purposes an independent non-volitional reference was used (evoked M_{\max}).

Evoked compound muscle action potential (*M*-wave)

To elicit *M*-waves from BBL and BBS, the musculocutaneous nerve was electrically stimulated (DS7AH, Digitimer Ltd., Welwyn Garden City, UK) with single square wave pulses (0.2 ms duration). A self-adhesive electrode (5 × 5 cm; Verity Medical, Andover, UK) served as an anode and was attached to the skin over the central portion of the TB muscle. The cathode (1 cm diameter, Electro Medical Supplies, Wantage, UK) was held to the skin over the musculocutaneous nerve, in between the BBS and BBL, at 50 % of the distance between the medial epicondyle of the humerus and the coracoid process [the motor entry point of the BB heads (Lee et al. 2010)]. The precise location of the cathode was determined (within 3–5 attempts) as the position that evoked the greatest *M*-wave response for a particular submaximal electrical current (typically 30–50 mA). *M*-waves were evoked at 10–20 mA incremental current intensities until a plateau was achieved (typically 80–140 mA). Thereafter, the electrical current was increased by 20 % and three supramaximal *M*-waves were evoked. M_{\max} was defined as the mean peak-to-peak sEMG response to these three stimuli.

Isometric bench press MVCs

PM and AD sEMG activity were recorded during isometric incline bench press MVCs. The participant lay supine on a bench, with the ‘head end’ raised and placed on a portable force plate (Kistler Quattro Jump 9290AD, Winterthur, Switzerland), thus producing a 15° incline. Shoulders

were abducted to 90° and the elbow angle was 90°, so that the forearms were perpendicular to a fixed horizontal bar positioned directly above the shoulders, while the feet were placed on the other end of the bench. Three isometric bench press MVCs were performed (30 s rest between each attempt) by pushing up against the immovable bar as hard as possible for 3 s. Verbal encouragement and biofeedback were provided during and after each MVC, and the highest sEMG, i.e., sEMG_{max}, for each stabilizer muscle was used for further analysis.

Statistical analysis

All data were analyzed by the same investigator. Pre- and post-RT differences in iMVF, 1-RM, muscle size, and θ_p were determined with paired *t* tests. Changes in force and sEMG during explosive contractions were identified with repeated measures ANOVAs [within factor: training (pre-/post-RT); between factor: time (Force: 50, 100 and 150 ms; sEMG: 0–50, 50–100 and 100–150 ms)]. Relative changes in all variables were calculated as percentage change from pre- to post-RT for each individual. Relative changes in the size of the three individual elbow flexor muscles were compared using a one-way ANOVA, while relative changes in BB and BRACH θ_p were compared with an independent *t* test. Pearson correlations were used to determine the relationships between relative changes in morphological and neural adaptations and the three indices of strength. Where two physiological adaptations, i.e., muscle hypertrophy and baseline 1-RM, were found to correlate with the % change in 1-RM, a partial correlation was used to determine the contribution of muscle hypertrophy while controlling for baseline 1-RM. Significance was defined as $P < 0.05$ and group data are expressed as mean \pm standard deviation (SD).

Results

Pre-training relationships between muscle strength and size

Pre-training iMVF was highly correlated with muscle volume ($r = 0.812$; $P < 0.001$) and ΣACSA_{\max} ($r = 0.806$; $P < 0.001$). Similarly, 1-RM pre-training was strongly correlated with muscle volume ($r = 0.768$, $P < 0.001$) and ΣACSA_{\max} ($r = 0.787$, $P < 0.001$). Prior to the 12-week RT period, explosive force production during the initial phase of contraction (50 ms) did not correlate with muscle volume ($r = 0.219$, $P = 0.21$) or ΣACSA_{\max} ($r = 0.176$, $P = 0.324$), but these muscle size indices were increasingly correlated with explosive force production as the contraction progressed (100 ms: muscle volume $r = 0.391$, $P = 0.024$; ΣACSA_{\max} $r = 0.428$, $P = 0.013$; 150 ms: muscle volume $r = 0.693$, $P < 0.001$; ΣACSA_{\max} $r = 0.725$, $P < 0.001$).

Muscle strength changes after RT

Relative increases in iMVf and 1-RM were 13.2 ± 9.1 and 41.6 ± 19.9 %, respectively (Table 1). Although absolute peak RFD did not change post 12-week RT, peak RFD normalized to iMVf decreased by 9.5 ± 16.3 % (Table 1). Absolute explosive force production at 50 ms after force onset was reduced after 12-week RT (ANOVA, training $P = 0.18$; training \times time $P = 0.029$; post hoc t test pre vs. post, $P = 0.001$), but there were no changes at 100 ms (t test, $P = 0.252$) or 150 ms (t test, $P = 0.695$; Fig. 1a). Explosive force normalized to iMVf was reduced at all three time points after force onset (ANOVA, training effect $P < 0.001$; group \times training $P = 0.449$; post hoc t test pre vs. post all $P < 0.001$; Fig. 1b).

Muscle size and architectural changes after RT

Total elbow flexor muscle volume ($+15.9 \pm 6.0$ %), Σ ACSA_{max} ($+15.9 \pm 5.8$ %) and θ_p ($+16.2 \pm 7.5$ %) all increased following the 12-week RT, and individual muscle responses are presented in Table 2. There were no

significant differences between the relative hypertrophic responses of the individual elbow flexor muscles regarding muscle volume (1-way ANOVA, $P = 0.189$; Table 2), ACSA_{max} (1-way ANOVA, $P = 0.598$; Table 2), or θ_p (t test, $P = 0.354$; Table 2). The individual relative increases in total elbow flexor muscle volume were unrelated to baseline muscle volume ($r = 0.055$, $P = 0.768$), habitual physical activity levels ($r = 0.134$, $P = 0.451$). However, the relative changes in elbow flexor muscle volume ($r = 0.429$, $P = 0.013$) and Σ ACSA_{max} ($r = 0.464$, $P = 0.007$) correlated with the individual gains in elbow flexor θ_p .

Neurological changes after RT

At elbow flexion iMVf post 12-week RT, normalized sEMG was unchanged after 12-week RT in the agonists (t test BBL, $P = 0.167$; BBS, $P = 0.537$; Table 3), antagonist (t test $P = 0.207$; Table 3) and stabilizers (PM, t test $P = 0.151$; AD, t test $P = 0.058$; Table 3). During the 1-RM, normalized sEMG did not change after 12-week RT in the agonists (t test, BBL, $P = 0.788$; BBS, $P = 0.182$; Table 3), or in the stabilizers (PM, t test $P = 0.074$; AD,

Table 1 Elbow flexor isometric, isoinertial and explosive strength before (Pre) and after (Post) 12-week RT

Strength variable	Pre	Post	Change (%)	Min (%)	Max (%)
iMVf (N)	262.3 \pm 42.3	296.4 \pm 50.5*	+13.2 \pm 9.1	-4.2	+36.4
1-RM (kg)	12.8 \pm 3.2	17.7 \pm 3.7*	+41.6 \pm 19.9	+14.3	+90.3
pRFD (N s ⁻¹)	3766 \pm 736	3800 \pm 798	+2.0 \pm 17.4	-33.3	+39.1
pRFD (iMVf s ⁻¹)	14.5 \pm 2.3	13.0 \pm 2.9	-9.5 \pm 16.3 %	-40.2	+36.3

Data are mean \pm SD ($n = 33$)

iMVf isometric maximum voluntary force, 1-RM single repetition maximum, pRFD peak rate of force development in absolute terms (N s⁻¹) and normalized (iMVf s⁻¹) to iMVf

* Significantly different to Pre-RT ($P < 0.0005$)

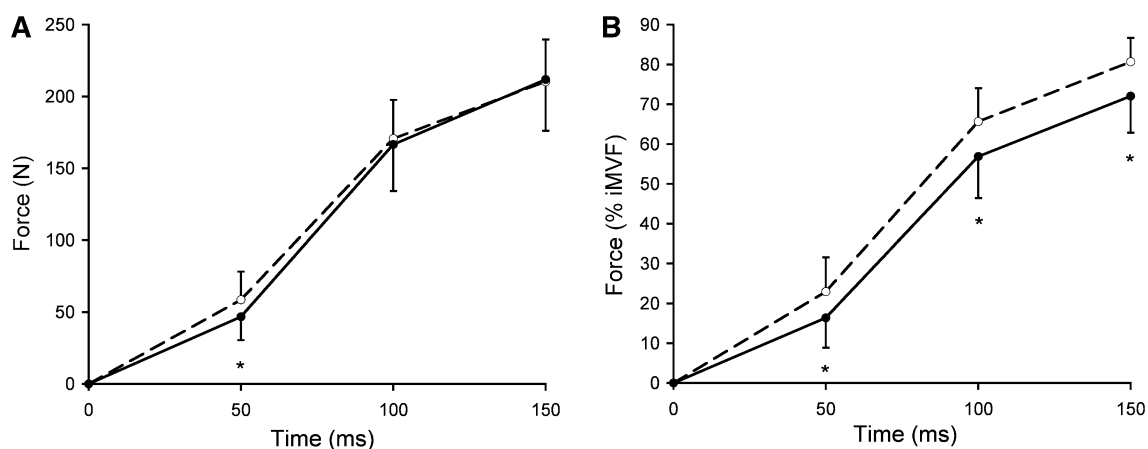


Fig. 1 Absolute (a) and normalized to iMVf (b) explosive force recorded at three time points (50, 100 and 150 ms) after the onset of force (0 ms) before (unfilled circle) and after (filled circle) 12-week RT; *Significantly different from pre-training values ($P < 0.05$)

Table 2 Elbow flexor muscle volume, maximum anatomical cross-sectional area (ACSA_{max}) and muscle fascicle pennation angle (θ_p) before (Pre) and after (Post) the 12-week RT period

Muscle variable	Pre	Post	Change (%)	Min (%)	Max (%)
Muscle volume (cm³)					
Biceps brachii	178.1 ± 31.9	208.7 ± 37.9*	17.3 ± 6.5	+5.9	+33.7
Brachialis	153.3 ± 27.9	175.3 ± 33.2*	14.3 ± 6.3	+1.6	+33.1
Brachioradialis	68.5 ± 14.7	79.5 ± 16.5*	16.5 ± 7.5	+3.7	+34.4
Total elbow flexor	400.0 ± 66.7	463.6 ± 79.2*	15.9 ± 6.0	+5.0	+33.4
ACSA_{max} (cm²)					
Biceps brachii	11.5 ± 2.1	13.5 ± 2.5*	16.9 ± 6.4	+6.6	+34.2
Brachialis	12.0 ± 1.8	13.8 ± 2.1*	15.1 ± 6.6	+1.3	+32.5
Brachioradialis	4.1 ± 0.8	4.8 ± 0.8*	16.0 ± 8.5	0.0	+35.1
∑ACSA _{max}	27.7 ± 4.1	32.1 ± 4.8*	15.9 ± 5.8	+6.0	+33.6
θ_p (°)					
Biceps brachii	14.4 ± 2.8	16.8 ± 3.4*	17.2 ± 8.3	+5.0	+35.6
Brachialis	10.8 ± 1.6	12.3 ± 1.6*	15.2 ± 9.0	+3.1	+35.6
Mean elbow flexor	12.6 ± 1.4	14.6 ± 1.7*	16.2 ± 7.5	+4.5	+35.1

Data are mean ± SD (*n* = 33)

* Significantly different to pre-training (*P* < 0.0005)

Table 3 Normalized sEMG RMS amplitude at isometric elbow flexion maximum voluntary force (iMVF), during single repetition maximum lifts (1-RM), and during 0–50, 50–100 and 100–150 ms time periods after agonist sEMG onset before (Pre) and after (Post) 12-week RT

Strength task	Normalized sEMG (%)				
	Agonists		Antagonist	Stabilizers	
	BBL	BBS	TB	PM	AD
iMVF					
Pre	9.3 ± 6.3	11.5 ± 9.3	14.3 ± 8.4	50.9 ± 20.2	44.9 ± 23.3
Post	8.0 ± 3.8	10.5 ± 7.1	12.8 ± 7.8	55.4 ± 26.7	37.5 ± 23.1
1-RM					
Pre	14.3 ± 8.2	14.7 ± 8.9	30.9 ± 21.0	55.1 ± 22.6	65.0 ± 27.3
Post	14.0 ± 4.7	16.2 ± 9.1	26.0 ± 14.8*	62.2 ± 26.0	65.7 ± 27.5
Explosive					
Pre 0–50 ms	5.0 ± 2.8	6.0 ± 3.4	7.3 ± 5.9	57.0 ± 43.0	43.5 ± 22.8
Post 0–50 ms	4.8 ± 2.8	5.3 ± 3.0	6.9 ± 5.1	47.7 ± 30.5	41.3 ± 25.5
Pre 50–100 ms	8.5 ± 5.9	9.8 ± 8.0	7.7 ± 6.0	51.2 ± 37.2	72.6 ± 40.2
Post 50–100 ms	7.3 ± 3.5	8.7 ± 4.0	6.2 ± 4.9	50.8 ± 31.1	70.1 ± 33.6
Pre 100–150 ms	8.2 ± 5.6	10.4 ± 7.2	7.3 ± 6.0	57.8 ± 40.5	66.9 ± 32.9
Post 100–150 ms	6.9 ± 2.8	9.6 ± 5.8	6.0 ± 8.9	52.8 ± 30.3	58.6 ± 24.0

Data are expressed relative to either M_{max} (agonists: BBL and BBS), sEMG_{max} during elbow extension (antagonist: TB), or sEMG_{max} during incline bench press (stabilizers: PM and AD). Data are mean ± SD

* Significantly different to Pre-training (*P* = 0.029)

t test *P* = 0.780; Table 3). However, normalized antagonist sEMG during 1-RM decreased by 4.7 ± 37.7 % after 12-week RT (*t* test *P* = 0.029; Table 3). During explosive force production, there were no changes in agonist (BBL, ANOVA, training *P* = 0.093, training × time *P* = 0.583; BBS, ANOVA, training *P* = 0.249, training × time

P = 0.965), antagonist (TB, ANOVA, training *P* = 0.117, training × time *P* = 0.803), or stabilizer (PM, ANOVA, training *P* = 0.164, training × time *P* = 0.582; AD, ANOVA, training *P* = 0.221, training × time *P* = 0.720) normalized sEMG in any of the three time windows (0–50, 50–100 and 100–150 ms) after agonist sEMG onset.

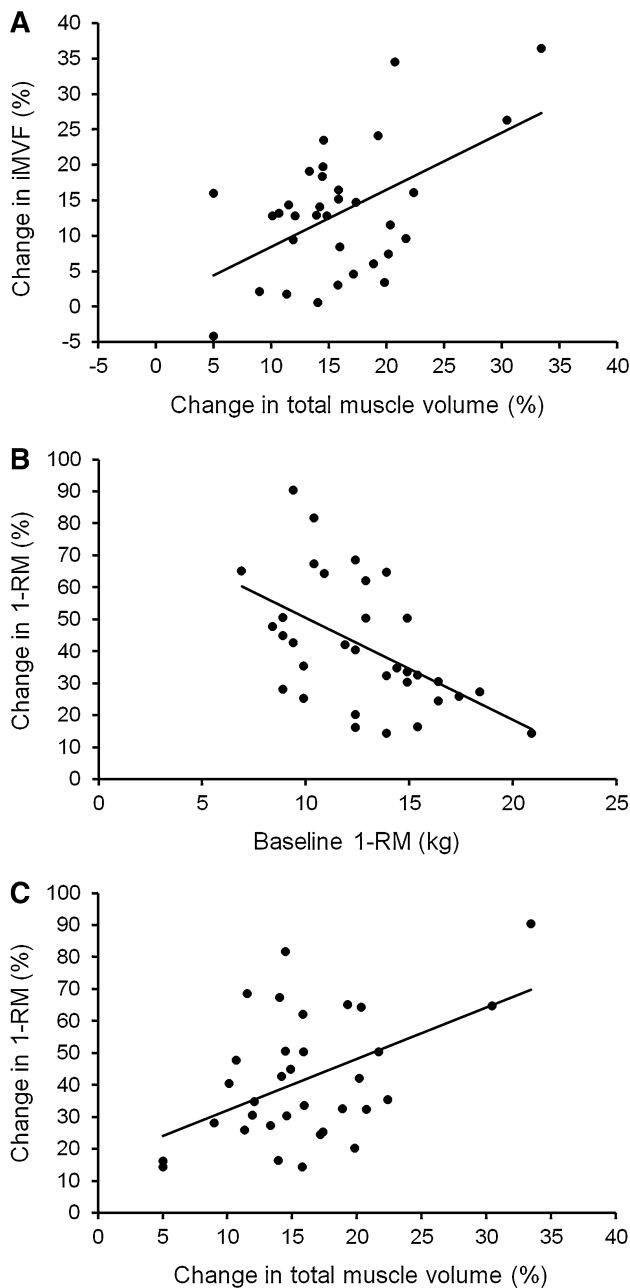


Fig. 2 The relationships between: the percentage changes in total elbow flexor muscle volume and iMVF (**a**; $r = 0.527$; $P = 0.002$); baseline 1-RM and percentage changes in 1-RM (**b**; $r = 0.519$; $P = 0.002$); the percentage changes in total elbow flexor muscle volume and 1-RM (**c**; $r = 0.482$; $P = 0.005$), after 12-week elbow flexor RT

Physiological contributors to the strength changes after RT

iMVF

Individual % changes in iMVF correlated with the relative changes in both total elbow flexor muscle volume ($r = 0.527$, $P = 0.002$; Fig. 2a) and elbow flexor

$\Sigma\text{ACSA}_{\text{max}}$ ($r = 0.493$, $P = 0.004$), but not with relative changes in θ_p ($r = 0.184$, $P = 0.304$). The relative changes in iMVF did not correlate with baseline iMVF ($r = 0.148$, $P = 0.416$), habitual physical activity levels ($r = 0.212$, $P = 0.239$), or relative changes in normalized agonist ($r = 0.187$, $P = 0.295$), antagonist ($r = 0.077$, $P = 0.656$), or stabilizer ($r = 0.184$, $P = 0.307$) sEMG at iMVF.

1-RM

The individual % gains in 1-RM were inversely correlated with baseline 1-RM values ($r = 0.519$, $P = 0.002$; Fig. 2b). Changes in 1-RM were also positively correlated with relative gains in total elbow flexor muscle volume ($r = 0.482$, $P = 0.005$; Fig. 2c) and elbow flexor $\Sigma\text{ACSA}_{\text{max}}$ ($r = 0.406$, $P = 0.020$). When controlling for baseline 1-RM, the correlations between changes in 1-RM and gains in total elbow flexor muscle volume ($r = 0.435$, $P = 0.013$) and changes in elbow flexor $\Sigma\text{ACSA}_{\text{max}}$ ($r = 0.383$, $P = 0.031$) were still significant. However, relative changes in 1-RM did not correlate with normalized agonist, antagonist, or stabilizer sEMG during 1-RM (All $r \leq 0.155$, $P \geq 0.389$). Further, the relative changes in 1-RM were not related to the percentage gains in elbow flexor θ_p ($r = 0.205$, $P = 0.254$).

Explosive strength

The individual relative changes in absolute and normalized explosive force at all three time points ($r \leq 0.243$, $P \geq 0.175$), and absolute and normalized peak RFD ($r \leq 0.190$, $P \geq 0.292$) were unrelated to the percentage changes in total elbow flexor muscle volume and $\Sigma\text{ACSA}_{\text{max}}$. Percentage changes in absolute (All $r \leq 0.285$, $P \geq 0.107$) and normalized (All $r \leq 0.281$, $P \geq 0.126$) explosive force (at any time point after force onset) did not correlate with % changes in normalized sEMG of any of the muscles investigated (at the appropriate time points). Percentage changes in θ_p were, however, inversely correlated with the % change in normalized force at 150 ms ($r = 0.362$, $P = 0.038$) but not at 50 ms ($r = 0.089$, $P = 0.615$) or 100 ms ($r = 0.192$, $P = 0.284$) after force onset.

Discussion

We aimed to determine the contribution of elbow flexor muscle hypertrophy to the changes in isometric, isoinertial and explosive strength following 12-week elbow flexor RT. By including an initial 3-week RT period, we attempted to overcome neural adaptations prior to the experimental 12-week RT intervention, and to highlight the role of

muscle hypertrophy in explaining the inter-individual variability in strength gains. Based on the correlations between the change in muscle volume and changes in isometric and isoinertial strength, we have shown for the first time that RT-induced muscle hypertrophy explains substantial proportions of the inter-individual changes in isometric and isoinertial, but not explosive, strength.

The individual percentage changes in muscle size and strength seen in our study were highly variable and comparable to previous studies that have investigated the variability in these training responses (Hubal et al. 2005; Erskine et al. 2010). In our study, the variable responses occurred after carefully controlling prior physical activity and RT status with a standardized 3-week period of RT and 6-week of no RT. The medium strength (Cohen 1992) correlations between the individual percentage changes in muscle volume and changes in maximum isometric and isoinertial strength suggest that muscle hypertrophy explained ~28 % ($R^2 = 0.28$) and ~23 % ($R^2 = 0.23$), respectively, of these strength gains. However, when baseline 1-RM values (another predictor of 1-RM changes) were taken into account, the contribution of muscle hypertrophy to isoinertial strength gains was reduced to ~19 % ($R^2 = 0.19$), i.e., still a moderate effect size (Cohen 1992). This is the first report to document the contribution of muscle hypertrophy to individual strength gains following RT. Two previous reports found no relationship, although their findings may have been confounded by limited elbow flexor [$+5.4 \pm 3.4$ % (Davies et al. 1988)] and quadriceps femoris [$+5.0 \pm 4.6$ % (Jones and Rutherford 1987)] muscle hypertrophy. Furthermore, relatively low sample sizes ($n = 12$) and no prior RT period to overcome neural adaptations are probable reasons for the discrepancy in the findings of these studies compared to ours.

Considering the strong relationships between muscle size (total volume and $\Sigma\text{ACSA}_{\text{max}}$) and isometric and isoinertial strength at baseline in this study (All, $r = 0.77$ – 0.81), which is in agreement with previous reports (Kanehisa et al. 1994; Bamman et al. 2000; Fukunaga et al. 2001), it is perhaps surprising that we did not find stronger relationships between the changes in muscle size and strength with RT. Despite strenuous efforts to minimize the test–retest variability of our measurements, resulting in high reproducibility (Erskine et al. 2012), any errors in the measurements of muscle strength and size, or discrepancies in the measurement of these variables, could confound their relationship. In addition, assessing the changes that occur with RT involves measurements at two time points, which is likely to lead to a greater accumulation of measurement errors than cross-sectional assessments that rely on a single measurement. Furthermore, RT-induced hypertrophy shows a steady increase for the first 6 months and after the first 2 months, hypertrophy and isometric strength gains appear

to increase in parallel (Narici et al. 1996). Therefore, it is possible that the relationship between hypertrophy and strength changes might have been even stronger had the current RT period been of a longer duration. Moreover, based on these issues, it seems likely that muscle hypertrophy exerts a stronger influence on the changes in isometric and isoinertial strength than we have documented in this study.

An alternative explanation for the weaker relationship between hypertrophy and strength changes (compared to the relationship at baseline) is that other physiological adaptations may be more important contributors to enhanced strength following RT. Regarding neural adaptations, we found only minor changes in neuromuscular activation: a small decrease in antagonist muscle co-activation during the 1-RM and no changes in agonist or stabilizer activation during any of the strength tasks. Thus, it would appear that the initial 3-week RT period served its purpose in eliciting neural adaptations prior to the experimental 12-week RT, and that neural changes played only a minor role in affecting strength changes following the 12-week RT. However, it should be noted that sEMG does not distinguish between motor unit recruitment, synchronization or firing rate. Therefore, it is possible that adaptations in one of these parameters may have been masked by the consistency, or even opposite changes, of the other parameters. Nevertheless, previous studies have reported high levels of elbow flexor muscle activation in the untrained state (Allen et al. 1998; Gandevia et al. 1998), with no increase in activation following RT (Herbert et al. 1998), thus suggesting a limited capacity for neural adaptation to RT in this muscle group.

Another physiological factor that could have explained the inter-individual differences in strength responses to RT was an increase in muscle fascicle pennation angle (θ_p), which is thought to occur in response to muscle fiber hypertrophy (Aagaard et al. 2001). Theoretically, an increase in θ_p leads to a trade-off between an increase in force from the hypertrophied muscle fibers, but a reduced transmission of force to the tendon due to the more oblique line of pull of the fascicles (Alexander and Vernon 1975). In fact, we found the changes in θ_p to be positively related to hypertrophy (change in volume, $r = 0.43$; change in $\Sigma\text{ACSA}_{\text{max}}$, $r = 0.46$), but were unrelated to any of the strength changes. The relative changes in θ_p varied considerably from +5 to +35 %, and might therefore have had a confounding effect on the relationship between hypertrophy and strength gains.

The inverse relationship observed between baseline 1-RM and RT-induced changes in 1-RM, although reported previously (Hubal et al. 2005), was surprising considering that we had standardized prior RT status and physical activity levels. Learning effects have been proposed to explain

the large increases in the 1-RM after RT (Rutherford and Jones 1986), and could conceivably explain this relationship. However, the lack of any substantive changes in agonist, antagonist and stabilizer activation during the 1-RM after RT in our study would argue against this possibility. Alternatively, inter-individual differences in RT-induced changes in muscle fascicle length (Erskine et al. 2010) could influence the length–tension relationship (Reeves et al. 2004), thus having a pronounced impact on the improvements in 1-RM.

Although we have been able to demonstrate that muscle hypertrophy explains a significant proportion of the inter-individual variability in strength gains, a substantial amount of the variability remains unexplained. We acknowledge that our measurement of muscle size did not account for possible changes in non-contractile material, myofibrillar packing or muscle fiber-type composition, all of which could have potentially influenced the muscle size–force relationship, and could therefore have confounded the relationship between hypertrophy and strength changes.

Despite substantial increases in muscle size and iMVF after 12-week RT, we found decreased absolute explosive force production at 50 ms and a reduced ability to express the available force generating capacity explosively, i.e., explosive force normalized to iMVF, during the first 150 ms of muscle contraction. This is in agreement with some previous work (Andersen et al. 2010; Tillin et al. 2011; Blazevich et al. 2009) but contrary to other reports (Aagaard et al. 2002; Hakkinen et al. 1998; Blazevich et al. 2008), and probably relates to the precise nature of the training stimulus (Tillin and Folland 2013). Although these changes were unrelated to muscle hypertrophy or neuromuscular activation, we did observe an inverse relationship between changes in θ_p and normalized explosive force measured at 150 ms after force onset. All other factors remaining constant, an increase in θ_p serves to decrease the shortening velocity of the whole muscle, as the amount of whole muscle shortening is the product of muscle fascicle shortening and the cosine of θ_p (Narici 1999). Thus, the greater the increase in θ_p , the lower the shortening velocity, leading to a reduction in RFD when normalized to iMVF. The fact that we saw this relationship only with changes in force measured at 150 ms after force onset could be due to the lower reliability of explosive force measured during the early phase of contraction (Buckthorpe et al. 2012). Alternatively, it may be that the early phase is more influenced by a reduction in the proportion of IIx muscle fibers (Andersen et al. 2010), which have faster contractile properties than IIa fibers (Bottinelli et al. 1996; D'Antona et al. 2006; Larsson and Moss 1993).

In conclusion, we have demonstrated for the first time that muscle hypertrophy explains a significant proportion of the inter-individual variability in isometric and isoinertial

strength changes in response to 12-week elbow flexor RT. However, a large amount of the variability remains unexplained and, although changes in intramuscular force transmission, myofibrillar packing and fiber-type composition cannot be discounted, due to limitations with measuring muscle size and strength in vivo, we suspect that muscle hypertrophy accounts for a greater proportion of the inter-individual variation in strength gains than reported here.

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Conflict of interest The authors declare no conflicts of interest.

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