The contribution of muscle hypertrophy to strength changes following resistance training

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Running title: Muscle hypertrophy and strength gains

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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

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 yr) completed an initial 3-wk period of elbow flexor RT (to facilitate neurological responses), followed by 6-wk no training, and then 12-wk 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-wk 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-wk elbow flexor RT in healthy young men.

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; Blazevich et al. 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, in order 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-wk RT period to overcome neural adaptations and to standardize prior physical activity before participants completed a 12-wk 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 in order to give context to the morphological adaptations.

METHODS

Participants

Thirty-three healthy, recreationally active young men volunteered (mean ± SD: age, 23.4 ± 3.0 yrs; 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 yrs 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-wk period of elbow flexor RT, which was followed by 6-wk of no training and then a 12-wk 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-wk experimental RT period. All RT involved exercising both arms. Three to 4 days before and after the 12-wk 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-wk 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 3 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-wk RT involved 2 sets of each exercise, and this was the same for wk 1-2 of the 12-wk RT, but increased to 3 sets (unilateral) and 2 sets (bilateral) during wk 3-4 and 3 sets of both exercises for wk 5-12. Participant adherence was 100%, i.e. all participants performed 9 and 36 training sessions during the 3 and 12-wk 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 10 warm-up reps at 40% 1-RM, 3 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° (0º = 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 4 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 4 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 10 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. by 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 x 448 pixels; field of view 181 x 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 mid-way 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

198 $\Sigma ACSA_{max}$.

199

200 *Muscle fascicle pennation angle (θ_p)*

201 BB short head (BBS) and BRACH θ_p was examined using B-mode ultrasonography
202 (SSA-37OA Power Vision 6000, Toshiba, Otawara-Shi, Japan) with an 8 MHz linear-
203 array transducer. The participant lay supine with the dominant elbow fully extended
204 and the shoulder abducted by 90°. Two millimeter-wide strips of ultrasound-absorbent
205 tape (3M, Neuss, Germany) were placed perpendicular to the long axis of the BBS at
206 50 mm intervals between the cubital crease and the shoulder, which formed markers
207 on the sonographs and ensured that θ_p was analyzed at the same location pre and post
208 RT. The probe was slowly glided in a straight line midway between the lateral and
209 medial boundaries from the cubital crease to the proximal end of BBS (in line with
210 the direction of the muscle fascicles). Individual frames were analyzed offline (NIH
211 ImageJ, Bethesda, USA). Fascicle θ_p was determined in 3 BBS fascicles within 50
212 mm of its distal end and in 3 BRACH fascicles within 50 mm of its proximal end. The
213 mean of the 3 measurements determined θ_p for each muscle, and for each individual,
214 the average of the θ_p for BBS and BRACH provided the mean elbow flexor θ_p.

215

216 *Surface electromyography (sEMG) activity*

217 Surface EMG activity was recorded from 2 agonists [the short and long heads of
218 biceps brachii (BBS and BBL)], 1 antagonist [lateral head of triceps brachii (TB)] and
219 2 stabilizers [anterior deltoid (AD) and pectoralis major (PM)] on the dominant side
220 using 2 Delsys Bagnoli-4 sEMG systems (Delsys, Boston, USA). Following
221 preparation of the skin (shaving, lightly abrading and cleansing with 70% ethanol),
222 double-differential surface electrodes (1 cm inter-electrode distance, Model DE-3.1;

223 Delsys) were attached over each muscle using adhesive interfaces. BBS and BBL
224 electrodes were placed mid-belly at a location that corresponded to 75% of the
225 distance from the coracoid process to the medial epicondyle of the humerus, as this
226 location is distal to the motor point region in each head (Lee et al. 2010). The TB
227 electrode was placed over the distal third of the muscle, and the AD electrode was
228 placed 5 cm distally from the acromion process over mid-sagittal plane. The PM
229 electrode was placed at 50% of the distance from the medial end of the clavicle to the
230 axilla, and reference electrodes were placed on the clavicle. All electrode locations
231 (with regard to distances from anatomical landmarks) were measured and recorded for
232 relocation during subsequent tests. Surface EMG signals were amplified (x100,
233 differential amplifier 20-450 Hz) and sampled at 2 kHz with the same analogue to
234 digital converter and PC as the force signal, prior to being band-pass filtered in both
235 directions between 6-500 Hz using a 2^{nd} order Butterworth digital filter.
236
237 The root mean square (RMS) of the sEMG signal over a 500 ms epoch around iMVF
238 (± 250 ms) was used to assess activation of all muscles during elbow flexion iMVF.
239 During the concentric phase of the 1-RM lift, the sEMG RMS of all muscles was
240 assessed for the 200 ms period that gave the highest agonist sEMG RMS. During
241 explosive contractions, the sEMG RMS from all muscles was determined in time
242 periods of 0-50, 50-100 and 100-150 ms, from the onset of sEMG activity in the first
243 agonist muscle to be activated. As with the onset of force, agonist sEMG onset was
244 identified manually (Tillin et al. 2010), with the y- and x-axis scales set at 100 mV
245 and 500 ms, respectively. The vertical cursor was placed on the onset and the scale
246 was reduced to 50 mV and 25 ms for the y- and x-axis, respectively, to confirm the
247 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 sub-maximal and could therefore be normalised 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 normalisation 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 x 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 3 supramaximal *M*-waves were evoked. M_{max} was defined as the mean peak-to-peak sEMG response to these 3 stimuli.

Isometric bench press MVCs

PM and AD sEMG activity was 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, Winterhur, Switzerland), thus producing a 15º incline. Shoulders were abducted to 90º and the elbow angle was 90º, 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 ± 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 $\Sigma ACSA_{max}$ ($r = 0.806$; $P < 0.001$). Similarly, 1-RM pre-training was strongly correlated with muscle volume ($r = 0.768$, $P < 0.001$) and $\Sigma ACSA_{max}$ ($r = 0.787$, $P < 0.001$). Prior to the 12-wk 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 $\Sigma ACSA_{max}$ ($r = 0.176$, $P = 0.324$), but these muscle size indices were increasingly correlated with explosive force production as the contraction progressed (100 ms: muscle volume $r = 0.391$, $P = 0.024$; $\Sigma ACSA_{max}$ $r = 0.428$, $P = 0.013$; 150 ms: muscle volume $r = 0.693$, $P < 0.001$; $\Sigma ACSA_{max}$ $r = 0.725$, $P < 0.001$).

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-wk 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-wk RT (ANOVA, training $P = 0.18$; training x 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 3 time points after force onset (ANOVA, training effect $P < 0.001$; group x training $P = 0.449$; post-hoc *t*-test pre vs. post all $P < 0.001$; Fig. 1B).

Insert Table 1 here.

Insert Fig. 1 here.

Muscle size and architectural changes after RT

Total elbow flexor muscle volume (+15.9 ± 6.0%), $\Sigma ACSA_{max}$ (+15.9 ± 5.8%) and θ_p (+16.2 ± 7.5%) all increased following the 12-wk 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 $\Sigma ACSA_{max}$ ($r = 0.464$, $P = 0.007$) were correlated with the individual gains in elbow

348 flexor θ_p.

349

350 *Insert Table 2 here.*

351

352 **Neurological changes after RT**

353 At elbow flexion iMVF post 12-wkRT, normalized sEMG was unchanged after 12-wk
354 RT in the agonists (*t*-test BBL, *P* = 0.167; BBS, *P* = 0.537; Table 3), antagonist (*t*-test
355 *P* = 0.207; Table 3) and stabilizers (PM, *t*-test *P* = 0.151; AD, *t*-test *P* = 0.058; Table
356 3). During the 1-RM, normalized sEMG did not change after 12-wk RT in the
357 agonists (*t*-test, BBL, *P* = 0.788; BBS, *P* = 0.182; Table 3), or in the stabilizers (PM,
358 *t*-test *P* = 0.074; AD, *t*-test *P* = 0.780; Table 3). However, normalized antagonist
359 sEMG during 1-RM decreased by 4.7 ± 37.7% after 12-wk RT (*t*-test *P* = 0.029;
360 Table 3). During explosive force production, there were no changes in agonist (BBL,
361 ANOVA, training *P* = 0.093, training x time *P* = 0.583; BBS, ANOVA, training *P* =
362 0.249, training x time *P* = 0.965), antagonist (TB, ANOVA, training *P* = 0.117,
363 training x time *P* = 0.803), or stabilizer (PM, ANOVA, training *P* = 0.164, training x
364 time *P* = 0.582; AD, ANOVA, training *P* = 0.221, training x time *P* = 0.720)
365 normalized sEMG in any of the three time windows (0-50 ms, 50-100 ms and 100-
366 150 ms) after agonist sEMG onset.

367

368 *Insert Table 3 here.*

369

370 **Physiological contributors to the strength changes after RT**

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

373 $\Sigma ACSA_{max}$ (r = 0.493, P = 0.004), but not with relative changes in θ_p (r = 0.184, P =
374 0.304). The relative changes in iMVF did not correlate with baseline iMVF (r =
375 0.148, P = 0.416), habitual physical activity levels (r = 0.212, P = 0.239), or relative
376 changes in normalized agonist (r = 0.187, P = 0.295), antagonist (r = 0.077, P =
377 0.656), or stabilizer (r = 0.184, P = 0.307) sEMG at iMVF.
378
379 *1-RM:* The individual % gains in 1-RM were inversely correlated with baseline 1-RM
380 values (r = 0.519, P = 0.002; Fig. 2B). Changes in 1-RM were also positively
381 correlated with relative gains in total elbow flexor muscle volume (r = 0.482, P =
382 0.005; Fig. 2C) and elbow flexor $\Sigma ACSA_{max}$ (r = 0.406, P = 0.020). When controlling
383 for baseline 1-RM, the correlations between changes in 1-RM and gains in total elbow
384 flexor muscle volume (r = 0.435, P = 0.013) and changes in elbow flexor $\Sigma ACSA_{max}$
385 (r = 0.383, P = 0.031) were slightly weaker but still significant. However, relative
386 changes in 1-RM did not correlate with normalized agonist, antagonist, or stabilizer
387 sEMG during 1-RM (All $r \leq 0.155$, $P \geq 0.389$). Further, the relative changes in 1-RM
388 were not related to the percentage gains in elbow flexor θ_p (r = 0.205, P = 0.254).
389
390 *Insert Fig. 2 near here.*
391
392 *Explosive strength:* The individual relative changes in absolute and normalized
393 explosive force at all three time points ($r \leq 0.243$, $P \geq 0.175$), and absolute and
394 normalized peak RFD ($r \leq 0.190$, $P \geq 0.292$), were unrelated to the percentage
395 changes in total elbow flexor muscle volume and $\Sigma ACSA_{max}$. Percentage changes in
396 absolute (All $r \leq 0.285$, $P \geq 0.107$) and normalized (All $r \leq 0.281$, $P \geq 0.126$)
397 explosive force (at any time point after force onset) did not correlate with % changes

398 in normalized sEMG of any of the muscles investigated (at the appropriate time
399 points). Percentage changes in θ_p were, however, inversely correlated with the %
400 change in normalized force at 150 ms (r = 0.362, P = 0.038) but not at 50 ms (r =
401 0.089, P = 0.615) or 100 ms (r = 0.192, P = 0.284) after force onset.
402
403 **DISCUSSION**
404 We aimed to determine the contribution of elbow flexor muscle hypertrophy to the
405 changes in isometric, isoinertial and explosive strength following 12-wk elbow flexor
406 RT. By including an initial 3-wk RT period, we attempted to overcome neural
407 adaptations prior to the experimental 12-wk RT intervention, and to highlight the role
408 of muscle hypertrophy in explaining the inter-individual variability in strength gains.
409 Based on the correlations between the change in muscle volume and changes in
410 isometric and isoinertial strength, we have shown for the first time that RT-induced
411 muscle hypertrophy explains substantial proportions of the inter-individual changes in
412 isometric and isoinertial, but not explosive, strength.
413
414 The individual percentage changes in muscle size and strength seen in our study were
415 highly variable and comparable to previous studies that have investigated the
416 variability in these training responses (Hubal et al. 2005; Erskine et al. 2010). In our
417 study, the variable responses occurred after carefully controlling prior physical
418 activity and RT status with a standardized 3-wk period of RT and 6-wk of no RT. The
419 medium strength (Cohen 1992) correlations between the individual percentage
420 changes in muscle volume and changes in maximum isometric and isoinertial strength
421 suggest that muscle hypertrophy explained ~28% (R^2 = 0.28) and ~23% (R^2 = 0.23),
422 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 ± 3.4% (Davies et al. 1988)] and quadriceps femoris [+5.0 ± 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 $\Sigma ACSA_{max}$) 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. Additionally, 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

448 the relationship between hypertrophy and strength changes might have been even
449 stronger had the current RT period been of a longer duration. Moreover, based on
450 these issues, it seems likely that muscle hypertrophy exerts a stronger influence on the
451 changes in isometric and isoinertial strength than we have documented in this study.
452
453 An alternative explanation for the weaker relationship between hypertrophy and
454 strength changes (compared to the relationship at baseline) is that other physiological
455 adaptations may be more important contributors to enhanced strength following RT.
456 Regarding neural adaptations, we found only minor changes in neuromuscular
457 activation: a small decrease in antagonist muscle co-activation during the 1-RM and
458 no changes in agonist or stabilizer activation during any of the strength tasks. Thus, it
459 would appear that the initial 3-wk RT period served its purpose in eliciting neural
460 adaptations prior to the experimental 12-wk RT, and that neural changes played only a
461 minor role in affecting strength changes following the 12-wk RT. However, it should
462 be noted that sEMG does not distinguish between motor unit recruitment,
463 synchronisation or firing rate. Therefore, it is possible that adaptations in one of these
464 parameters may have been masked by the consistency, or even opposite changes, of
465 the other parameters. Nevertheless, previous studies have reported high levels of
466 elbow flexor muscle activation in the untrained state (Allen et al. 1998; Gandevia et
467 al. 1998), with no increase in activation following RT (Herbert et al. 1998), thus
468 suggesting a limited capacity for neural adaptation to RT in this muscle group.
469
470 Another physiological factor that could have explained the inter-individual
471 differences in strength responses to RT was an increase in muscle fascicle pennation
472 angle (θ_p), which is thought to occur in response to muscle fibre 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 fibres, 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 $\Sigma ACSA_{max}$, 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 fibre-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-wk 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 fibres (Andersen et al. 2010), which have faster contractile properties than IIa fibres (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-wk elbow flexor RT. However, a large amount of the variability remains unexplained and, although changes in intramuscular force transmission, myofibrillar packing and fibre-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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CONFLICTS OF INTERESTS

The authors declare no conflicts of interest.

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TABLES

Table 1. Elbow flexor isometric, isoinertial and explosive strength before (Pre) and after (Post) 12-wk RT. Data are mean ± SD (*n* = 33).

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

iMVF, isometric maximum voluntary force; *1-RM*, single repetition maximum; *pRFD*, peak rate of force development in absolute terms ($N \cdot s^{-1}$) and normalized ($iMVF \cdot s^{-1}$) to iMVF; *significantly different to Pre-RT ($P < 0.0005$).

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-wk RT period. Data are mean ± SD (n = 33).

Muscle variable	Pre	Post	Change (%)	Min (%)	Max (%)
Muscle volume (cm^3)					
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
$ACSA_{max}$ (cm^2)					
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
$\sum ACSA_{max}$	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

*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-wk RT. 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.

	Normalized sEMG (%)				
Strength Task	Agonists		Antagonist	Stabilizers	
Pre/post RT	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

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

FIGURE LEGENDS

Figure 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 (○) and after (●) 12-wk RT; * significantly different from pre-training values ($P < 0.05$).

Figure 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-wks elbow flexor RT.