

## **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

$\theta_p$  Muscle fascicle pennation angle

**ABSTRACT**

1 **Purpose:** Whilst skeletal muscle hypertrophy is considered an important adaptation to  
2 resistance training (RT) it has not previously been found to explain the inter-  
3 individual changes in strength after RT. This study investigated the contribution of  
4 hypertrophy to individual gains in isometric, isoinertial and explosive strength after  
5 12 weeks of elbow flexor RT. **Methods:** Thirty-three previously untrained, healthy  
6 men (18-30 yr) completed an initial 3-wk period of elbow flexor RT (to facilitate  
7 neurological responses), followed by 6-wk no training, and then 12-wk elbow flexor  
8 RT. Unilateral elbow flexor muscle strength [isometric maximum voluntary force  
9 (iMVF), single repetition maximum (1-RM) and explosive force], muscle volume  
10 ( $V_m$ ), muscle fascicle pennation angle ( $\theta_p$ ) and normalized agonist, antagonist and  
11 stabilizer sEMG were assessed pre and post 12-wk RT. **Results:** Percentage gains in  
12  $V_m$  correlated with percentage changes in iMVF ( $r = 0.527$ ;  $P = 0.002$ ) and 1-RM ( $r =$   
13  $0.482$ ;  $P = 0.005$ ) but not in explosive force ( $r \leq 0.243$ ;  $P \geq 0.175$ ). Percentage  
14 changes in iMVF, 1-RM, and explosive force did not correlate with percentage  
15 changes in agonist, antagonist or stabilizer sEMG (all  $P > 0.05$ ). Percentage gains in  
16  $\theta_p$  inversely correlated with percentage changes in normalized explosive force at 150  
17 ms after force onset ( $r = 0.362$ ;  $P = 0.038$ ). **Conclusions:** We have shown for the first  
18 time that muscle hypertrophy explains a significant proportion of the inter-individual  
19 variability in isometric and isoinertial strength gains following 12-wk elbow flexor  
20 RT in healthy young men.

21

22

## 23 INTRODUCTION

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

42

43 The contribution of muscle hypertrophy to strength gains may depend on the strength  
44 task assessed, e.g. isometric, isoinertial or explosive strength. Although it is well  
45 established that RT induces gains in both isometric and isoinertial strength  
46 (Rutherford and Jones 1986; Erskine et al. 2010; Folland et al. 2002), the effect of RT  
47 on explosive strength is controversial (Aagaard et al. 2002; Hakkinen et al. 1998;

48 Andersen et al. 2010; Tillin et al. 2011; Blazevich et al. 2009; Blazevich et al. 2008).  
49 A better understanding of the how specific physiological adaptations contribute to the  
50 individual improvements in isometric, isoinertial and explosive strength after RT may  
51 help to optimize RT, in order to elicit specific adaptations and functional outcomes,  
52 such as improved physical performance in athletic groups and a reduced risk of falling  
53 in older populations.

54

55 In addition to neural and hypertrophic adaptations, RT is known to increase the  
56 muscle fascicle pennation angle ( $\theta_p$ ), i.e. the angle at which the muscle fascicles insert  
57 into the aponeurosis (Aagaard et al. 2001; Erskine et al. 2010). Although an increase  
58 in  $\theta_p$  enables more contractile material to attach to the aponeurosis (leading to an  
59 increase in force output), there is a concomitant reduction in force resolved at the  
60 tendon due to the oblique line of pull of the fascicles (Alexander and Vernon 1975).  
61 Therefore, documenting inter-individual differences in  $\theta_p$  in response to RT may  
62 provide a more complete assessment of how morphological adaptations explain  
63 strength changes following RT.

64

65 The aim of this study was to determine the contribution of muscle hypertrophy to the  
66 inter-individual differences in isometric, isoinertial and explosive strength changes in  
67 response to RT. An upper body elbow flexor RT model was used to maximize the  
68 hypertrophic response (Cureton et al. 1988; Welle et al. 1996), and changes in  $\theta_p$  were  
69 also assessed. The unique design of this study incorporated an initial 3-wk RT period  
70 to overcome neural adaptations and to standardize prior physical activity before  
71 participants completed a 12-wk experimental RT period. Changes in neuromuscular  
72 activation of the agonist, antagonist and stabilizer muscles were assessed by

73 normalizing surface EMG activity to appropriate reference measures in order to give  
74 context to the morphological adaptations.

75

## 76 **METHODS**

### 77 **Participants**

78 Thirty-three healthy, recreationally active young men volunteered (mean  $\pm$  SD: age,  
79  $23.4 \pm 3.0$  yrs; height,  $1.76 \pm 0.06$  m; body mass,  $75.2 \pm 10.7$  kg) and provided  
80 written informed consent prior to their involvement in this 25-week study, which was  
81 approved by the Loughborough University Ethical Advisory Committee and  
82 conformed to the standards set by the 1964 Declaration of Helsinki. Health status and  
83 habitual physical activity were assessed using questionnaires and the physical activity  
84 rating was  $2.6 \pm 0.4$ , where 1 = extremely inactive and 5 = exceptionally active  
85 (Baecke et al. 1982). Volunteers were excluded from the study if they reported use of  
86 purported anabolic supplements in the previous 6 months, had a history of upper body  
87 exercise in the previous 12 months or were  $<18$  or  $>30$  yrs old.

88

### 89 **Study Overview**

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

98 explosive force data have been included here to provide a more comprehensive  
99 account of the neuromuscular adaptations to chronic RT.

100

101 The RT protocol and some of the pre and post-training measurements have been  
102 described in detail in the previously published study (Erskine et al. 2012). Therefore,  
103 they will be described briefly here. Thirty-three participants completed an initial 3-wk  
104 period of elbow flexor RT, which was followed by 6-wk of no training and then a 12-  
105 wk period of experimental elbow flexor RT. The initial RT period provided extensive  
106 familiarization to the RT exercises and neuromuscular tests (data not reported here),  
107 whilst also standardizing participant training status and facilitating neural adaptations  
108 prior to the 12-wk experimental RT period. All RT involved exercising both arms.  
109 Three to 4 days before and after the 12-wk RT, strength [maximum isometric  
110 voluntary force (iMVF), single repetition maximum (1-RM) and explosive force], size  
111 [muscle volume and maximum anatomical cross-sectional area ( $ACSA_{max}$ )] and  
112 fascicle pennation angle ( $\theta_p$ ) of the elbow flexor muscles were measured in the  
113 dominant arm. To determine whether neural adaptations did occur during the 12-wk  
114 RT (and to help differentiate neural from morphological contributions to strength  
115 gains), sEMG of the agonist, antagonist and stabilizer muscles was assessed during  
116 the three strength tasks and normalized to appropriate reference measures. All tests  
117 for each participant were performed at the same time of day before and after training.

118

### 119 **Resistance Training (RT)**

120 Participants performed 3 training sessions per week (Monday, Wednesday and  
121 Friday) during both RT periods. Each session comprised unilateral seated elbow  
122 flexion ‘preacher curls’ using dumbbells, with alternating sets using the dominant and

123 non-dominant arms, and then bilateral preacher curls on a resistance training machine  
124 (Body Solid, Forest Park, USA), with a 2 min rest between sets. The loading for both  
125 exercises was 8-10 RM and the load was increased when participants could lift 10  
126 reps during the final set of an exercise. The 3-wk RT involved 2 sets of each exercise,  
127 and this was the same for wk 1-2 of the 12-wk RT, but increased to 3 sets (unilateral)  
128 and 2 sets (bilateral) during wk 3-4 and 3 sets of both exercises for wk 5-12.  
129 Participant adherence was 100%, i.e. all participants performed 9 and 36 training  
130 sessions during the 3 and 12-wk RT periods, respectively.

131

### 132 **Pre and post RT neuromuscular measurements**

#### 133 *Unilateral single repetition maximum (1-RM)*

134 A series of incremental unilateral elbow flexion preacher curl lifts of a dumbbell were  
135 performed whilst seated on the same modified preacher bench that was used in  
136 training. After 10 warm-up reps at 40% 1-RM, 3 reps were performed at 80% 1-RM.  
137 Thereafter, a series of single lifts were performed with 1 min rest intervals at  
138 increments of +0.5 kg if the preceding lift was successful. The last successful lift was  
139 defined as 1-RM.

140

#### 141 *Isometric maximum voluntary force (iMVF)*

142 Elbow flexor iMVF was measured using a custom-built strength-testing chair with the  
143 elbow joint angle set to 60° (0° = full elbow extension). The wrist was strapped to an  
144 S-Beam tension-compression load cell (Applied Measurements Ltd, Aldermaston,  
145 UK), which was positioned perpendicular to the direction of forearm movement  
146 during isometric elbow flexion/extension. The force signal was interfaced with an  
147 analog-to-digital converter (CED micro 1401, CED, Cambridge, UK), sampled at 2



148 kHz with a PC using Spike 2 software (CED, Cambridge, UK) and low-pass filtered  
149 (500 Hz edge frequency) with a second order Butterworth digital filter. Participants  
150 completed 4 isometric elbow flexion maximum voluntary contractions (MVCs), each  
151 lasting 3 s and separated by  $\geq 30$  s. Biofeedback and verbal encouragement were  
152 provided during and between each MVC. Participants then completed 4 isometric  
153 elbow extension MVCs with an identical protocol to determine the maximum sEMG  
154 (sEMG<sub>max</sub>) amplitude of the TB (see below for details). Isometric MVF for elbow  
155 flexion and extension was the greatest instantaneous voluntary force achieved during  
156 that action.

157

#### 158 *Isometric explosive contractions*

159 In addition to the MVCs detailed above, participants performed 10 isometric  
160 explosive voluntary elbow flexion contractions (each separated by 20 s). During each  
161 contraction participants attempted to flex their elbow as 'fast and hard' as possible  
162 (Sahaly et al. 2001), with emphasis on fast, for 1 s from a relaxed state, while  
163 achieving at least 80% iMVF. During each contraction, participants were instructed to  
164 avoid any countermovement (elbow extension prior to elbow flexion). A computer  
165 monitor displayed both force (on a sensitive scale around resting values) and the slope  
166 of the force-time curve. The latter was used to provide immediate biofeedback of  
167 performance, specifically peak rate of force development (RFD, 1 ms time constant)  
168 during each contraction, and the former highlighted any countermovement. The three  
169 contractions with the largest peak RFD and no discernible countermovement or pre  
170 tension (change of baseline force of  $< 0.5$  N during the 100 ms prior to contraction  
171 onset) were used for analysis of the force signal. Analysis consisted of measuring  
172 force at 50, 100 and 150 ms from force onset and peak RFD (which typically occurred

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

180

### 181 *Muscle hypertrophy*

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

201 BB short head (BBS) and BRACH  $\theta_p$  was examined using B-mode ultrasonography  
202 (SSA-370A 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  $\theta_p$  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  $\theta_p$  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  $\theta_p$  for each muscle, and for each individual,  
214 the average of the  $\theta_p$  for BBS and BRACH provided the mean elbow flexor  $\theta_p$ .

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<sup>nd</sup> order Butterworth digital filter.

236

237 The root mean square (RMS) of the sEMG signal over a 500 ms epoch around iMVF  
238 ( $\pm 250$  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

248 deflected from the baseline noise.

249

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

263

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

265 To elicit *M*-waves from BBL and BBS, the musculocutaneous nerve was electrically  
266 stimulated (DS7AH, Digitimer Ltd., Welwyn Garden City, UK) with single square  
267 wave pulses (0.2 ms duration). A self-adhesive electrode (5 x 5 cm; Verity Medical,  
268 Andover, UK) served as an anode and was attached to the skin over the central  
269 portion of the TB muscle. The cathode (1 cm diameter, Electro Medical Supplies,  
270 Wantage, UK) was held to the skin over the musculocutaneous nerve, in between the  
271 BBS and BBL, at 50% of the distance between the medial epicondyle of the humerus  
272 and the coracoid process [the motor entry point of the BB heads (Lee et al. 2010)].

273 The precise location of the cathode was determined (within 3-5 attempts) as the  
274 position that evoked the greatest  $M$ -wave response for a particular submaximal  
275 electrical current (typically 30-50 mA).  $M$ -waves were evoked at 10-20 mA  
276 incremental current intensities until a plateau was achieved (typically 80-140 mA).  
277 Thereafter, the electrical current was increased by 20% and 3 supramaximal  $M$ -waves  
278 were evoked.  $M_{\max}$  was defined as the mean peak-to-peak sEMG response to these 3  
279 stimuli.

280

### 281 *Isometric bench press MVCs*

282 PM and AD sEMG activity was recorded during isometric incline bench press MVCs.  
283 The participant lay supine on a bench, with the 'head end' raised and placed on a  
284 portable force plate (Kistler Quattro Jump 9290AD, Winterhur, Switzerland), thus  
285 producing a 15° incline. Shoulders were abducted to 90° and the elbow angle was 90°,  
286 so that the forearms were perpendicular to a fixed horizontal bar positioned directly  
287 above the shoulders, while the feet were placed on the other end of the bench. Three  
288 isometric bench press MVCs were performed (30 s rest between each attempt) by  
289 pushing up against the immovable bar as hard as possible for 3 s. Verbal  
290 encouragement and biofeedback were provided during and after each MVC, and the  
291 highest sEMG, i.e. sEMG<sub>max</sub>, for each stabilizer muscle was used for further analysis.

292

### 293 **Statistical analysis**

294 All data were analyzed by the same investigator. Pre and post-RT differences in  
295 iMVF, 1-RM, muscle size, and  $\theta_p$  were determined with paired  $t$ -tests. Changes in  
296 force and sEMG during explosive contractions were identified with repeated measures  
297 ANOVAs [within factor: training (pre/post RT); between factor: time (Force: 50, 100

298 and 150 ms; sEMG: 0-50, 50-100 and 100-150 ms)]. Relative changes in all variables  
299 were calculated as percentage change from pre- to post-RT for each individual.  
300 Relative changes in the size of the three individual elbow flexor muscles were  
301 compared using a one-way ANOVA, while relative changes in BB and BRACH  $\theta_p$   
302 were compared with an independent *t*-test. Pearson correlations were used to  
303 determine the relationships between relative changes in morphological and neural  
304 adaptations and the three indices of strength. Where two physiological adaptations,  
305 i.e. muscle hypertrophy and baseline 1-RM, were found to correlate with the %  
306 change in 1-RM, a partial correlation was used to determine the contribution of  
307 muscle hypertrophy while controlling for baseline 1-RM. Significance was defined as  
308  $P < 0.05$  and group data are expressed as mean  $\pm$  standard deviation (SD).

309

## 310 **RESULTS**

### 311 **Pre-training relationships between muscle strength and size**

312 Pre-training iMVF was highly correlated with muscle volume ( $r = 0.812$ ;  $P < 0.001$ )  
313 and  $\Sigma\text{ACSA}_{\text{max}}$  ( $r = 0.806$ ;  $P < 0.001$ ). Similarly, 1-RM pre-training was strongly  
314 correlated with muscle volume ( $r = 0.768$ ,  $P < 0.001$ ) and  $\Sigma\text{ACSA}_{\text{max}}$  ( $r = 0.787$ ,  $P <$   
315  $0.001$ ). Prior to the 12-wk RT period, explosive force production during the initial  
316 phase of contraction (50 ms) did not correlate with muscle volume ( $r = 0.219$ ,  $P =$   
317  $0.21$ ) or  $\Sigma\text{ACSA}_{\text{max}}$  ( $r = 0.176$ ,  $P = 0.324$ ), but these muscle size indices were  
318 increasingly correlated with explosive force production as the contraction progressed  
319 (100 ms: muscle volume  $r = 0.391$ ,  $P = 0.024$ ;  $\Sigma\text{ACSA}_{\text{max}}$   $r = 0.428$ ,  $P = 0.013$ ; 150  
320 ms: muscle volume  $r = 0.693$ ,  $P < 0.001$ ;  $\Sigma\text{ACSA}_{\text{max}}$   $r = 0.725$ ,  $P < 0.001$ ).

321

### 322 **Muscle strength changes after RT**

323 Relative increases in iMVF and 1-RM were  $13.2 \pm 9.1$  and  $41.6 \pm 19.9\%$ , respectively  
324 (Table 1). Although absolute peak RFD did not change post 12-wk RT, peak RFD  
325 normalized to iMVF decreased by  $9.5 \pm 16.3\%$  (Table 1). Absolute explosive force  
326 production at 50 ms after force onset was reduced after 12-wk RT (ANOVA, training  
327  $P = 0.18$ ; training x time  $P = 0.029$ ; post-hoc  $t$ -test pre vs. post,  $P = 0.001$ ), but there  
328 were no changes at 100 ms ( $t$ -test,  $P = 0.252$ ) or 150 ms ( $t$ -test,  $P = 0.695$ ; Fig. 1A).  
329 Explosive force normalized to iMVF was reduced at all 3 time points after force onset  
330 (ANOVA, training effect  $P < 0.001$ ; group x training  $P = 0.449$ ; post-hoc  $t$ -test pre vs.  
331 post all  $P < 0.001$ ; Fig. 1B).

332

333 *Insert Table 1 here.*

334

335 *Insert Fig. 1 here.*

336

337 **Muscle size and architectural changes after RT**

338 Total elbow flexor muscle volume ( $+15.9 \pm 6.0\%$ ),  $\Sigma\text{ACSA}_{\text{max}}$  ( $+15.9 \pm 5.8\%$ ) and  $\theta_p$   
339 ( $+16.2 \pm 7.5\%$ ) all increased following the 12-wk RT, and individual muscle  
340 responses are presented in Table 2. There were no significant differences between the  
341 relative hypertrophic responses of the individual elbow flexor muscles regarding  
342 muscle volume (1-way ANOVA,  $P = 0.189$ ; Table 2),  $\text{ACSA}_{\text{max}}$  (1-way ANOVA,  $P =$   
343  $0.598$ ; Table 2), or  $\theta_p$  ( $t$ -test,  $P = 0.354$ ; Table 2). The individual relative increases in  
344 total elbow flexor muscle volume were unrelated to baseline muscle volume ( $r =$   
345  $0.055$ ,  $P = 0.768$ ), habitual physical activity levels ( $r = 0.134$ ,  $P = 0.451$ ). However,  
346 the relative changes in elbow flexor muscle volume ( $r = 0.429$ ,  $P = 0.013$ ) and  
347  $\Sigma\text{ACSA}_{\text{max}}$  ( $r = 0.464$ ,  $P = 0.007$ ) were correlated with the individual gains in elbow



348 flexor  $\theta_p$ .

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  $4.7 \pm 37.7\%$  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  $\Sigma\text{ACSA}_{\text{max}}$  ( $r = 0.493$ ,  $P = 0.004$ ), but not with relative changes in  $\theta_p$  ( $r = 0.184$ ,  $P =$   
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  $\Sigma\text{ACSA}_{\text{max}}$  ( $r = 0.406$ ,  $P = 0.020$ ). 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  $\Sigma\text{ACSA}_{\text{max}}$   
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  $\theta_p$  ( $r = 0.205$ ,  $P = 0.254$ ).

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  $\Sigma\text{ACSA}_{\text{max}}$ . 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  $\theta_p$  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

423 predictor of 1-RM changes) were taken into account, the contribution of muscle  
424 hypertrophy to isoinertial strength gains was reduced to ~19% ( $R^2 = 0.19$ ), i.e. still a  
425 moderate effect size (Cohen 1992). This is the first report to document the  
426 contribution of muscle hypertrophy to individual strength gains following RT. Two  
427 previous reports found no relationship, although their findings may have been  
428 confounded by limited elbow flexor [ $+5.4 \pm 3.4\%$  (Davies et al. 1988)] and  
429 quadriceps femoris [ $+5.0 \pm 4.6\%$  (Jones and Rutherford 1987)] muscle hypertrophy.  
430 Furthermore, relatively low sample sizes ( $n = 12$ ) and no prior RT period to overcome  
431 neural adaptations are probable reasons for the discrepancy in the findings of these  
432 studies compared to ours.

433

434 Considering the strong relationships between muscle size (total volume and  
435  $\Sigma\text{ACSA}_{\text{max}}$ ) and isometric and isoinertial strength at baseline in this study (All,  $r =$   
436  $0.77-0.81$ ), which is in agreement with previous reports (Kanehisa et al. 1994;  
437 Bamman et al. 2000; Fukunaga et al. 2001), it is perhaps surprising that we did not  
438 find stronger relationships between the changes in muscle size and strength with RT.  
439 Despite strenuous efforts to minimize the test-retest variability of our measurements,  
440 resulting in high reproducibility (Erskine et al. 2012), any errors in the measurements  
441 of muscle strength and size, or discrepancies in the measurement of these variables,  
442 could confound their relationship. Additionally, assessing the changes that occur with  
443 RT involves measurements at two time points, which is likely to lead to a greater  
444 accumulation of measurement errors than cross-sectional assessments that rely on a  
445 single measurement. Furthermore, RT-induced hypertrophy shows a steady increase  
446 for the first 6 months and after the first 2 months, hypertrophy and isometric strength  
447 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 ( $\theta_p$ ), which is thought to occur in response to muscle fibre hypertrophy

473 (Aagaard et al. 2001). Theoretically, an increase in  $\theta_p$  leads to a trade-off between an  
474 increase in force from the hypertrophied muscle fibres, but a reduced transmission of  
475 force to the tendon due to the more oblique line of pull of the fascicles (Alexander and  
476 Vernon 1975). In fact, we found the changes in  $\theta_p$  to be positively related to  
477 hypertrophy (change in volume,  $r = 0.43$ ; change in  $\Sigma ACSA_{max}$ ,  $r = 0.46$ ), but were  
478 unrelated to any of the strength changes. The relative changes in  $\theta_p$  varied  
479 considerably from +5% to +35%, and might therefore have had a confounding effect  
480 on the relationship between hypertrophy and strength gains.

481

482 The inverse relationship observed between baseline 1-RM and RT-induced changes in  
483 1-RM, although reported previously (Hubal et al. 2005), was surprising considering  
484 that we had standardized prior RT status and physical activity levels. Learning effects  
485 have been proposed to explain the large increases in the 1-RM after RT (Rutherford  
486 and Jones 1986), and could conceivably explain this relationship. However, the lack  
487 of any substantive changes in agonist, antagonist and stabilizer activation during the  
488 1-RM after RT in our study would argue against this possibility. Alternatively, inter-  
489 individual differences in RT-induced changes in muscle fascicle length (Erskine et al.  
490 2010) could influence the length-tension relationship (Reeves et al. 2004), thus having  
491 a pronounced impact on the improvements in 1-RM.

492

493 Although we have been able to demonstrate that muscle hypertrophy explains a  
494 significant proportion of the inter-individual variability in strength gains, a substantial  
495 amount of the variability remains unexplained. We acknowledge that our  
496 measurement of muscle size did not account for possible changes in non-contractile  
497 material, myofibrillar packing or muscle fibre-type composition, all of which could

498 have potentially influenced the muscle size-force relationship, and could therefore  
499 have confounded the relationship between hypertrophy and strength changes.

500

501 Despite substantial increases in muscle size and iMVF after 12-wk RT, we found  
502 decreased absolute explosive force production at 50 ms and a reduced ability to  
503 express the available force generating capacity explosively, i.e. explosive force  
504 normalized to iMVF, during the first 150 ms of muscle contraction. This is in  
505 agreement with some previous work (Andersen et al. 2010; Tillin et al. 2011;  
506 Blazevich et al. 2009) but contrary to other reports (Aagaard et al. 2002; Hakkinen et  
507 al. 1998; Blazevich et al. 2008), and probably relates to the precise nature of the  
508 training stimulus (Tillin and Folland 2013). Although these changes were unrelated to  
509 muscle hypertrophy or neuromuscular activation, we did observe an inverse  
510 relationship between changes in  $\theta_p$  and normalized explosive force measured at 150  
511 ms after force onset. All other factors remaining constant, an increase in  $\theta_p$  serves to  
512 decrease the shortening velocity of the whole muscle, as the amount of whole muscle  
513 shortening is the product of muscle fascicle shortening and the cosine of  $\theta_p$  (Narici  
514 1999). Thus, the greater the increase in  $\theta_p$ , the lower the shortening velocity, leading  
515 to a reduction in RFD when normalized to iMVF. The fact that we saw this  
516 relationship only with changes in force measured at 150 ms after force onset could be  
517 due to the lower reliability of explosive force measured during the early phase of  
518 contraction (Buckthorpe et al. 2012). Alternatively, it may be that the early phase is  
519 more influenced by a reduction in the proportion of IIX muscle fibres (Andersen et al.  
520 2010), which have faster contractile properties than IIA fibres (Bottinelli et al. 1996;  
521 D'Antona et al. 2006; Larsson and Moss 1993).

522

523 In conclusion, we have demonstrated for the first time that muscle hypertrophy  
524 explains a significant proportion of the inter-individual variability in isometric and  
525 isoinertial strength changes in response to 12-wk elbow flexor RT. However, a large  
526 amount of the variability remains unexplained and, although changes in intramuscular  
527 force transmission, myofibrillar packing and fibre-type composition cannot be  
528 discounted, due to limitations with measuring muscle size and strength *in vivo*, we  
529 suspect that muscle hypertrophy accounts for a greater proportion of the inter-  
530 individual variation in strength gains than reported here.

531

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535

#### 536 **CONFLICTS OF INTERESTS**

537 The authors declare no conflicts of interest.

538



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663  
664

## TABLES

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

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 $\cdot$ s <sup>-1</sup> )	3766 $\pm$ 736	3800 $\pm$ 798	+2.0 $\pm$ 17.4	-33.3	+39.1
pRFD (iMVf $\cdot$ s <sup>-1</sup> )	14.5 $\pm$ 2.3	13.0 $\pm$ 2.9	-9.5 $\pm$ 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<sup>-1</sup>) and normalized (iMVf $\cdot$ s<sup>-1</sup>) to iMVf; \*significantly different to Pre-RT ( $P < 0.0005$ ).

**Table 2.** Elbow flexor muscle volume, maximum anatomical cross-sectional area (ACSA<sub>max</sub>) and muscle fascicle pennation angle ( $\theta_p$ ) before (Pre) and after (Post) the 12-wk RT period. Data are mean  $\pm$  SD ( $n = 33$ ).

Muscle variable	Pre	Post	Change (%)	Min (%)	Max (%)
Muscle volume (cm <sup>3</sup> )					
Biceps brachii	178.1 $\pm$ 31.9	208.7 $\pm$ 37.9*	17.3 $\pm$ 6.5	+5.9	+33.7
Brachialis	153.3 $\pm$ 27.9	175.3 $\pm$ 33.2*	14.3 $\pm$ 6.3	+1.6	+33.1
Brachioradialis	68.5 $\pm$ 14.7	79.5 $\pm$ 16.5*	16.5 $\pm$ 7.5	+3.7	+34.4
Total elbow flexor	400.0 $\pm$ 66.7	463.6 $\pm$ 79.2*	15.9 $\pm$ 6.0	+5.0	+33.4
ACSA <sub>max</sub> (cm <sup>2</sup> )					
Biceps brachii	11.5 $\pm$ 2.1	13.5 $\pm$ 2.5*	16.9 $\pm$ 6.4	+6.6	+34.2
Brachialis	12.0 $\pm$ 1.8	13.8 $\pm$ 2.1*	15.1 $\pm$ 6.6	+1.3	+32.5
Brachioradialis	4.1 $\pm$ 0.8	4.8 $\pm$ 0.8*	16.0 $\pm$ 8.5	0.0	+35.1
$\Sigma$ ACSA <sub>max</sub>	27.7 $\pm$ 4.1	32.1 $\pm$ 4.8*	15.9 $\pm$ 5.8	+6.0	+33.6
$\theta_p$ (°)					
Biceps brachii	14.4 $\pm$ 2.8	16.8 $\pm$ 3.4*	17.2 $\pm$ 8.3	+5.0	+35.6
Brachialis	10.8 $\pm$ 1.6	12.3 $\pm$ 1.6*	15.2 $\pm$ 9.0	+3.1	+35.6
Mean elbow flexor	12.6 $\pm$ 1.4	14.6 $\pm$ 1.7*	16.2 $\pm$ 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  $\pm$  SD.

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