



LJMU Research Online

Erskine, RM, Fletcher, G and Folland, JP

The contribution of muscle hypertrophy to strength changes following resistance training

<http://researchonline.ljmu.ac.uk/2992/>

Article

Citation (please note it is advisable to refer to the publisher's version if you intend to cite from this work)

Erskine, RM, Fletcher, G and Folland, JP (2014) The contribution of muscle hypertrophy to strength changes following resistance training. EUROPEAN JOURNAL OF APPLIED PHYSIOLOGY, 114 (6). pp. 1239-1249. ISSN 1439-6319

LJMU has developed **LJMU Research Online** for users to access the research output of the University more effectively. Copyright © and Moral Rights for the papers on this site are retained by the individual authors and/or other copyright owners. Users may download and/or print one copy of any article(s) in LJMU Research Online to facilitate their private study or for non-commercial research. You may not engage in further distribution of the material or use it for any profit-making activities or any commercial gain.

The version presented here may differ from the published version or from the version of the record. Please see the repository URL above for details on accessing the published version and note that access may require a subscription.

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

<http://researchonline.ljmu.ac.uk/>

The contribution of muscle hypertrophy to strength changes following resistance training

Robert M. Erskine^{1,2}, Gareth Fletcher¹ and Jonathan P. Folland¹

¹School of Sport, Exercise and Health Sciences, Loughborough University, Loughborough, United Kingdom; ²Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Liverpool, United Kingdom.

Running title: Muscle hypertrophy and strength gains

Address correspondence to:

R.M. Erskine, PhD; School of Sport and Exercise Sciences, Tom Reilly Building, Byrom Street Campus, Liverpool John Moores University, Liverpool L3 3AF, UK;
Tel: +44 (0)151 904 6256; Fax: +44 (0)151 904 6284; Email: R.M.Erskine@ljmu.ac.uk

Funding disclosure: Financial support for the conduct of this study was provided by GlaxoSmithKline Nutritional Healthcare UK.

Conflict of interest: The authors declare no conflict of interest.

Key words: 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

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 (θ_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 θ_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 (θ_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 θ_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 θ_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 θ_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 ± 3.0 yrs; height, 1.76 ± 0.06 m; body mass, 75.2 ± 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 ± 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 (Erschine 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 (θ_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 ≥ 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_{max}) 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 (θ_p)

201 BB short head (BBS) and BRACH θ_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 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 2nd 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

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. $sEMG_{\max}$, 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 θ_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 θ_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 ± 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 θ_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), ACSA_{max} (1-way ANOVA, $P =$
343 0.598 ; Table 2), or θ_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 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 $4.7 \pm 37.7\%$ 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 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 $\Sigma\text{ACSA}_{\text{max}}$ ($r = 0.406$, $P = 0.020$). 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 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 $\Sigma\text{ACSA}_{\text{max}}$. 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 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

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 (θ_p), which is thought to occur in response to muscle fibre hypertrophy

473 (Aagaard et al. 2001). Theoretically, an increase in θ_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 θ_p to be positively related to
477 hypertrophy (change in volume, $r = 0.43$; change in $\Sigma\text{ACSA}_{\text{max}}$, $r = 0.46$), but were
478 unrelated to any of the strength changes. The relative changes in θ_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 θ_p and normalized explosive force measured at 150
511 ms after force onset. All other factors remaining constant, an increase in θ_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 θ_p (Narici
514 1999). Thus, the greater the increase in θ_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

532 **ACKNOWLEDGEMENTS**

533 Financial support for the conduct of this study was provided by GlaxoSmithKline
534 Nutritional Healthcare UK.

535

536 **CONFLICTS OF INTERESTS**

537 The authors declare no conflicts of interest.

538

REFERENCES

- 539 Aagaard P, Andersen JL, Dyhre-Poulsen P, Leffers AM, Wagner A, Magnusson SP,
540 Halkjaer-Kristensen J, Simonsen EB (2001) A mechanism for increased
541 contractile strength of human pennate muscle in response to strength training:
542 changes in muscle architecture. *J Physiol* 534 (Pt. 2):613-623
- 543 Aagaard P, Simonsen EB, Andersen JL, Magnusson P, Dyhre-Poulsen P (2002)
544 Increased rate of force development and neural drive of human skeletal muscle
545 following resistance training. *J Appl Physiol* 93 (4):1318-1326
- 546 Alexander RM, Vernon A (1975) The dimensions of knee and ankle muscles and the
547 forces they exert. *J Hum Mov Studies* 1:115-123
- 548 Allen GM, McKenzie DK, Gandevia SC (1998) Twitch interpolation of the elbow
549 flexor muscles at high forces. *Muscle Nerve* 21 (3):318-328
- 550 Andersen LL, Andersen JL, Zebis MK, Aagaard P (2010) Early and late rate of force
551 development: differential adaptive responses to resistance training? *Scand J*
552 *Med Sci Sports* 20 (1):e162-169
- 553 Baecke JA, Burema J, Frijters JE (1982) A short questionnaire for the measurement of
554 habitual physical activity in epidemiological studies. *Am J Clin Nutr* 36
555 (5):936-942
- 556 Bamman MM, Newcomer BR, Larson-Meyer DE, Weinsier RL, Hunter GR (2000)
557 Evaluation of the strength-size relationship in vivo using various muscle size
558 indices. *Med Sci Sports Exerc* 32 (7):1307-1313
- 559 Blazeovich AJ, Cannavan D, Horne S, Coleman DR, Aagaard P (2009) Changes in
560 muscle force-length properties affect the early rise of force in vivo. *Muscle*
561 *Nerve* 39 (4):512-520
- 562 Blazeovich AJ, Horne S, Cannavan D, Coleman DR, Aagaard P (2008) Effect of
563 contraction mode of slow-speed resistance training on the maximum rate of
564 force development in the human quadriceps. *Muscle Nerve* 38 (3):1133-1146
- 565 Bottinelli R, Canepari M, Pellegrino MA, Reggiani C (1996) Force-velocity
566 properties of human skeletal muscle fibres: myosin heavy chain isoform and
567 temperature dependence. *J Physiol* 495 (Pt_2):573-586
- 568 Buckthorpe MW, Hannah R, Pain TG, Folland JP (2012) Reliability of neuromuscular
569 measurements during explosive isometric contractions, with special reference
570 to electromyography normalization techniques. *Muscle Nerve* 46 (4):566-576.
571 doi:10.1002/mus.23322
- 572 Burden A (2010) How should we normalize electromyograms obtained from healthy
573 participants? What we have learned from over 25 years of research. *J*
574 *Electromyogr Kinesiol* 20 (6):1023-1035
- 575 Cohen J (1992) A power primer. *Psychological bulletin* 112 (1):155-159

- 576 Cureton KJ, Collins MA, Hill DW, McElhannon FM, Jr. (1988) Muscle hypertrophy
577 in men and women. *Med Sci Sports Exerc* 20 (4):338-344
- 578 D'Antona G, Lanfranconi F, Pellegrino MA, Brocca L, Adami R, Rossi R, Moro G,
579 Miotti D, Canepari M, Bottinelli R (2006) Skeletal muscle hypertrophy and
580 structure and function of skeletal muscle fibres in male body builders. *J*
581 *Physiol* 570 (3):611-627. doi:10.1113/jphysiol.2005.101642
- 582 Davies J, Parker DF, Rutherford OM, Jones DA (1988) Changes in strength and cross
583 sectional area of the elbow flexors as a result of isometric strength training.
584 *Eur J Appl Physiol Occup Physiol* 57 (6):667-670
- 585 Erskine RM, Fletcher G, Hanson B, Folland JP (2012) Whey protein does not enhance
586 the adaptations to elbow flexor resistance training. *Med Sci Sports Exerc* 44
587 (9):1791-1800. doi:10.1249/MSS.0b013e318256c48d
- 588 Erskine RM, Jones DA, Maganaris CN, Degens H (2009) In vivo specific tension of
589 the human quadriceps femoris muscle. *Eur J Appl Physiol* 106 (6):827-838
- 590 Erskine RM, Jones DA, Williams AG, Stewart CE, Degens H (2010) Inter-individual
591 variability in the adaptation of human muscle specific tension to progressive
592 resistance training. *Eur J Appl Physiol* 110 (6):1117-1125
- 593 Folland JP, Irish CS, Roberts JC, Tarr JE, Jones DA (2002) Fatigue is not a necessary
594 stimulus for strength gains during resistance training. *Br J Sports Med* 36
595 (5):370-373; discussion 374
- 596 Fukunaga T, Miyatani M, Tachi M, Kouzaki M, Kawakami Y, Kanehisa H (2001)
597 Muscle volume is a major determinant of joint torque in humans. *Acta Physiol*
598 *Scand* 172 (4):249-255
- 599 Gandevia SC, Herbert RD, Leeper JB (1998) Voluntary activation of human elbow
600 flexor muscles during maximal concentric contractions. *J Physiol* 512 (Pt
601 2):595-602
- 602 Hakkinen K, Kallinen M, Izquierdo M, Jokelainen K, Lassila H, Malkia E, Kraemer
603 WJ, Newton RU, Alen M (1998) Changes in agonist-antagonist EMG, muscle
604 CSA, and force during strength training in middle-aged and older people. *J*
605 *Appl Physiol* 84 (4):1341-1349
- 606 Herbert RD, Dean C, Gandevia SC (1998) Effects of real and imagined training on
607 voluntary muscle activation during maximal isometric contractions. *Acta*
608 *Physiol Scand* 163 (4):361-368
- 609 Hubal MJ, Gordish-Dressman H, Thompson PD, Price TB, Hoffman EP,
610 Angelopoulos TJ, Gordon PM, Moyna NM, Pescatello LS, Visich PS, Zoeller
611 RF, Seip RL, Clarkson PM (2005) Variability in muscle size and strength gain
612 after unilateral resistance training. *Med Sci Sports Exerc* 37 (6):964-972
- 613 Jones DA, Rutherford OM (1987) Human muscle strength training: the effects of
614 three different regimens and the nature of the resultant changes. *J Physiol* 391
615 (1):1-11

- 616 Kanehisa H, Ikegawa S, Fukunaga T (1994) Comparison of muscle cross-sectional
617 area and strength between untrained women and men. *Eur J Appl Physiol*
618 *Occup Physiol* 68 (2):148-154
- 619 Kraemer WJ, Adams K, Cafarelli E, Dudley GA, Dooly C, Feigenbaum MS, Fleck SJ,
620 Franklin B, Fry AC, Hoffman JR, Newton RU, Potteiger J, Stone MH,
621 Ratamess NA, Triplett-McBride T (2002) American College of Sports
622 Medicine position stand. Progression models in resistance training for healthy
623 adults. *Med Sci Sports Exerc* 34 (2):364-380
- 624 Larsson L, Moss RL (1993) Maximum velocity of shortening in relation to myosin
625 isoform composition in single fibres from human skeletal muscles. *J Physiol*
626 472 (1):595-614
- 627 Lee JH, Kim HW, Im S, An X, Lee MS, Lee UY, Han SH (2010) Localization of
628 motor entry points and terminal intramuscular nerve endings of the
629 musculocutaneous nerve to biceps and brachialis muscles. *Surg Radiol Anat*
630 32 (3):213-220
- 631 Moritani T, deVries HA (1979) Neural factors versus hypertrophy in the time course
632 of muscle strength gain. *Am J Phys Med* 58 (3):115-130
- 633 Narici M (1999) Human skeletal muscle architecture studied in vivo by non-invasive
634 imaging techniques: functional significance and applications. *J Electromyogr*
635 *Kinesiol* 9 (2):97-103
- 636 Narici MV, Hoppeler H, Kayser B, Landoni L, Claassen H, Gavardi C, Conti M,
637 Cerretelli P (1996) Human quadriceps cross-sectional area, torque and neural
638 activation during 6 months strength training. *Acta Physiol Scand* 157 (2):175-
639 186
- 640 Reeves ND, Narici MV, Maganaris CN (2004) In vivo human muscle structure and
641 function: adaptations to resistance training in old age. *Exp Physiol* 89 (6):675-
642 689
- 643 Rutherford OM, Jones DA (1986) The role of learning and coordination in strength
644 training. *Eur J Appl Physiol Occup Physiol* 55 (1):100-105
- 645 Sahaly R, Vandewalle H, Driss T, Monod H (2001) Maximal voluntary force and rate
646 of force development in humans--importance of instruction. *Eur J Appl*
647 *Physiol* 85 (3-4):345-350
- 648 Seynnes OR, de Boer M, Narici MV (2007) Early skeletal muscle hypertrophy and
649 architectural changes in response to high-intensity resistance training. *J Appl*
650 *Physiol* 102 (1):368-373
- 651 Tillin NA, Folland JP (2013) Maximal and explosive strength training elicit distinct
652 neuromuscular adaptations, specific to the training stimulus. *Eur J Appl*
653 *Physiol*. doi:10.1007/s00421-013-2781-x
- 654 Tillin NA, Jimenez-Reyes P, Pain MT, Folland JP (2010) Neuromuscular
655 performance of explosive power athletes versus untrained individuals. *Med*

- 656 Sci Sports Exerc 42 (4):781-790
- 657 Tillin NA, Pain MT, Folland JP (2011) Short-term unilateral resistance training
658 affects the agonist-antagonist but not the force-agonist activation relationship.
659 Muscle Nerve 43 (3):375-384
- 660 Welle S, Totterman S, Thornton C (1996) Effect of age on muscle hypertrophy
661 induced by resistance training. J Gerontol A Biol Sci Med Sci 51 (6):M270-
662 275
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 ⁻¹)	3766 \pm 736	3800 \pm 798	+2.0 \pm 17.4	-33.3	+39.1
pRFD (iMVF \cdot s ⁻¹)	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⁻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 \pm SD (n = 33).

Muscle variable	Pre	Post	Change (%)	Min (%)	Max (%)
Muscle volume (cm ³)					
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 _{max} (cm ²)					
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
Σ ACSA _{max}	27.7 \pm 4.1	32.1 \pm 4.8*	15.9 \pm 5.8	+6.0	+33.6
θ_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
iMVF						
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
1-RM						
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
Explosive						
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.