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Changes in agonist neural drive, hypertrophy and pre-training strength all contribute to the individual strength gains after resistance training

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Abstract

Purpose Whilst neural and morphological adaptations following resistance training (RT) have been investigated extensively at a group level, relatively little is known about the contribution of specific physiological mechanisms, or pre-training strength, to the individual changes in strength following training. This study investigated the contribution of multiple underpinning neural [agonist EMG (QEMG_{MVT}), antagonist EMG (HEMG_{ANTAG})] and morphological variables [total quadriceps volume (QUADS_{VOL}), and muscle fascicle pennation angle (QUADS_{θ_p})], as well as pre-training strength, to the individual changes in strength after 12 weeks of knee extensor RT.

Methods Twenty-eight healthy young men completed 12 weeks of isometric knee extensor RT (3/week). Isometric maximum voluntary torque (MVT) was assessed pre- and post-RT, as were simultaneous neural drive to the agonist (QEMG_{MVT}) and antagonist (HEMG_{ANTAG}). In addition QUADS_{VOL} was determined with MRI and QUADS_{θ_p} with B-mode ultrasound.

Results Percentage changes (Δ) in MVT were correlated to Δ QEMG_{MVT} ($r = 0.576$, $P = 0.001$), Δ QUADS_{VOL}

($r = 0.461$, $P = 0.014$), and pre-training MVT ($r = -0.429$, $P = 0.023$), but not Δ HEMG_{ANTAG} ($r = 0.298$, $P = 0.123$) or Δ QUADS_{θ_p} ($r = -0.207$, $P = 0.291$). Multiple regression analysis revealed 59.9% of the total variance in Δ MVT after RT to be explained by Δ QEMG_{MVT} (30.6%), Δ QUADS_{VOL} (18.7%), and pre-training MVT (10.6%).

Conclusions Changes in agonist neural drive, quadriceps muscle volume and pre-training strength combined to explain the majority of the variance in strength changes after knee extensor RT (~60%) and adaptations in agonist neural drive were the most important single predictor during this short-term intervention.

Keywords Strength training · Neural drive · Muscle volume · Muscle architecture · Between-individual variability

Abbreviations

Δ	Percentage change
BF	Biceps femoris
CON	Control
EMG	Electromyography
HEMG _{ANTAG}	Hamstrings electromyography recorded during knee extension maximum voluntary torque production
HEMG _{MAX}	Hamstrings electromyography recorded during knee flexion maximum voluntary torque production
ITT	Interpolated twitch technique
M _{MAX}	Maximal M-wave
MRI	Magnetic resonance imaging
MVC	Maximum voluntary contraction
MVT	Maximum voluntary torque
PCSA	Physiological cross-sectional area

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QEMG _{MVT}	Quadriceps electromyography recorded during knee extension maximum voluntary torque production
QUADS _{VOL}	Total quadriceps femoris muscle volume
QUADS _{θ_p}	Quadriceps femoris muscle fascicle pennation angle
RF	Rectus femoris
RMS	Root mean square
RT	Resistance training
ST	Semitendinosus
VI	Vastus intermedius
VIF	Variance inflation factor
VL	Vastus lateralis
VM	Vastus medialis

Introduction

Muscular strength, the maximum force or torque a muscle group can produce, is a major contributor to function in both athletic (Wisloff et al. 2004) and day-to-day tasks (Samuel et al. 2012), and is a risk factor for muscle injury (Opar et al. 2015) as well as the development (Slemenda et al. 1998) and progression (Amin et al. 2009) of joint degeneration in osteoarthritis. Consequently, resistance training (RT) is frequently employed when aiming to: improve athletic performance (Wilson et al. 1996; Comfort et al. 2012); enhance mobility of middle-aged and older adults (Brochu et al. 2002; Brandon et al. 2003); reduce injury risk (Brooks et al. 2006; Noyes and Barber Westin 2012); prevent or slow the progression of joint degeneration (Zhang and Jordan 2010). On a cohort level, neural (agonist activation, Komi et al. 1978; Narici et al. 1996; Tillin et al. 2011, 2011; antagonist co-activation, Carolan and Cafarelli 1992; Häkkinen et al. 1998) and morphological (hypertrophy, O'Hagan et al. 1995; Tracy et al. 1999; Erskine et al. 2010a; muscle architecture, Aagaard et al. 2001; Seynnes et al. 2006; Blazevich et al. 2007) adaptations have been widely documented to occur after RT and are presumed to explain the observed improvements in strength. However, on an individual basis there is great variability in the changes in strength after RT (Folland et al. 2000; Hubal et al. 2005), and relatively little is known about the contribution of specific physiological mechanisms to these individual changes, including which is the most important adaptation for determining strength gains. Knowledge of the contribution of specific adaptations may facilitate more effective RT prescription.

The relationship between the individual changes in strength following training and the putative underpinning neural and morphological adaptations has previously been investigated primarily using bivariate correlations. These studies have reported changes in strength to

be both significantly related and unrelated to the changes in neural [*significant*: agonist electromyography (EMG): $r = 0.66$ – 0.74 (Häkkinen et al. 1985; Häkkinen and Komi 1986; Shima et al. 2002); *non-significant*: agonist activation assessed with the interpolated twitch technique (ITT): $r = -0.124$ – 0.47 (Erskine et al. 2010b), antagonist EMG: $r = 0.09$ (Erskine et al. 2010b) or morphological variables [*significant*: muscle volume assessed via magnetic resonance imaging (MRI): $r = 0.48$ – 0.53 (Erskine et al. 2014); *non-significant*: muscle volume assessed via MRI: $r = 0.15$ (Erskine et al. 2010b); muscle fascicle pennation angle (θ_p) assessed via ultrasonography: $r = -0.33$ – 0.26 (Nimphius et al. 2012)]. Therefore, there is considerable confusion about the contribution of specific physiological variables to the individual changes in strength.

Only a few studies have examined two or more predictor variables to assess neural and morphological factors, typically finding only one variable to make a significant contribution to the explained variance (Erskine et al. 2010b, 2014), negating the need for a more comprehensive multi-factorial analysis. The exception is Higbie et al. (1996) who found a remarkable 65% of the variability in concentric strength gains to be due to the combination of neural (agonist activation assessed with EMG) and hypertrophic (muscle cross-sectional area) adaptations. However, this study has not been corroborated, and was based on absolute changes in strength and hypertrophy that may predispose towards positive correlations [for the same proportional change (e.g. 10%) individuals with larger and stronger muscles at baseline will tend to have bigger absolute changes in both variables]. It is also possible that inclusion of the changes in antagonist co-activation and θ_p may help to explain an even greater proportion of the variance in strength gains than those reported by Higbie et al. (1996). Finally, in addition to neural and morphological adaptations, pre-training strength has also been demonstrated to be related to strength changes (Hubal et al. 2005; Erskine et al. 2014). Therefore, a detailed investigation of the contribution of a range of putative variables, including pre-training strength, neural and morphological factors, to the changes in strength after training is currently lacking.

The purpose of this study was to assess the individual and combined contribution of the adaptations in neural (agonist quadriceps EMG, antagonist hamstring EMG) and morphological (quadriceps muscle volume and θ_p) variables, whilst also accounting for the influence of pre-training strength, to the individual changes in strength after RT. Existing literature and logical deduction were used to select these five discrete hypothesis-driven predictor variables.

Materials and methods

Participants

Forty-eight young, healthy, asymptomatic, males who had not completed lower body RT for >18 months and were not involved in systematic physical training were recruited and provided written informed consent prior to their participation in study, which was approved by the Loughborough University Ethical Advisory Committee. Following familiarization participants were randomly allocated to RT or control (CON) groups that were matched for maximum voluntary torque (MVT) and body mass. Six participants withdrew from the study (four due to personal reasons and two were excluded due to non-compliance). Forty-two participants completed the study (RT group, $n = 28$: age 25 ± 2 years; height 1.75 ± 0.07 m; body mass 70 ± 9 kg; baseline physical activity 2067 ± 1157 MET min week⁻¹. CON group, $n = 14$: age 25 ± 3 years; height 1.76 ± 0.06 m; body mass 72 ± 7 kg; baseline physical activity 2321 ± 1614 MET min week⁻¹). Baseline recreational physical activity was assessed with the International Physical Activity Questionnaire (short format; Craig et al. 2003).

Overview

Participants visited the laboratory for a familiarization session involving maximum voluntary and evoked twitch knee extension contractions. Two duplicate laboratory measurement sessions were conducted both pre (sessions 7–10 days apart prior to the first training session) and post (2–3 days after the last training session and 2–3 days later) 12 weeks of unilateral knee extensor RT. Axial T1-weighted MRI scans of the thigh and quadriceps ultrasonography recordings were also conducted pre (5 days prior to the first training session) and post (2–3 days after the final training session). Training and testing were completed with the same isometric apparatus. The laboratory testing sessions involved recordings of the dominant leg isometric knee extension torque and surface EMG of the superficial quadriceps muscles during voluntary maximum contractions and evoked maximum twitch contractions (via electrical stimulation of the femoral nerve). Measurement sessions were at a consistent time of day and started between 12:00 and 19:00. After pre-training measurements RT participants were randomized to one of two RT interventions: explosive-contraction ($n = 13$) or sustained-contraction training ($n = 15$) (matched for body mass and pre-training strength). Training involved unilateral isometric contractions (4×10 repetitions) of both legs three times a week (36 sessions in total). CON group participants attended only the measurement sessions. All participants were

instructed to maintain their habitual physical activity and diet throughout the study. Cohort level comparisons of the two RT interventions have previously been reported elsewhere (Balshaw et al. 2016). Both interventions resulted in increased MVT from pre- to post-training and, therefore, data were pooled to form the RT group in the current study.

Training

After a brief warm-up of submaximum contractions of both legs, participants completed four sets of ten unilateral isometric knee extensor contractions of each leg; with sets alternating between dominant and non-dominant legs. Each set took 60 s with 2 min between successive sets on the same leg. The explosive-contraction group completed short, explosive contractions with participants instructed to perform each contraction “as fast and hard as possible” up to $\geq 80\%$ MVT for ~ 1 s, and then relax for 5 s between repetitions. A computer monitor displayed rate of torque development (10-ms time epoch) to provide biofeedback of explosive performance. The torque–time curve was also shown: with a horizontal cursor at 80% MVT to ensure sufficiently forceful contractions; on a sensitive scale highlighting baseline torque to observe and correct any pre-tension or countermovement. The sustained-contraction group completed prolonged contractions at 75% MVT, with 2-s rest between contractions. These participants were presented with a target torque trace 2 s before every contraction and instructed to match this target, which increased torque linearly from rest to 75% MVT over 1 s before a plateau at 75% MVT for a further 3 s. All participants performed three maximum voluntary isometric contractions (MVCs, see below) at the start of each training week to re-establish MVT and prescribe training torques.

Force and EMG recording

Measurement and training sessions were completed in a rigid custom-made isometric dynamometer with knee and hip angles of 115° and 126° ($180^\circ =$ full extension), respectively. Adjustable straps were tightly fastened across the pelvis and shoulders to prevent extraneous movement. An ankle strap (35 mm width reinforced canvas webbing) was placed $\sim 15\%$ of tibial length (distance from lateral malleolus to knee joint space), above the medial malleolus, and positioned perpendicular to the tibia and in series with a calibrated S-beam strain gauge (Force Logic, Swallowfield, UK). The analogue force signal from the strain gauge was amplified ($\times 370$) and sampled at 2000 Hz using an external A/D converter (Micro 1401; CED Ltd., Cambridge, UK) and recorded with Spike 2 computer software (CED Ltd., Cambridge, UK). In offline analysis, force data were low-pass filtered at 500 Hz using a fourth-order zero-lag

Butterworth filter, gravity corrected by subtracting baseline force, and multiplied by lever length, the distance from the knee joint space to the centre of the ankle strap, to calculate torque values.

Surface EMG was recorded from the superficial quadriceps (rectus femoris, RF; vastus lateralis, VL; vastus medialis, VM) and hamstring (biceps femoris, BF; and semitendinosus, ST) muscles using a wireless EMG system (Trigno; Delsys, Boston, MA, USA). Following skin preparation (shaving, abrading, and cleansing with 70% ethanol), single differential Trigno Standard EMG sensors (Delsys, Boston, MA, USA), each with a fixed 1 cm interelectrode distance, were attached at six separate sites over the superficial quadriceps muscles at set percentages of thigh length above the superior border of the patella (RF 65 and 55%; VL 60 and 55%; VM 35 and 30%) and at two sites over the BF and ST (both 45% of thigh length above the popliteal fossa). Sensors were positioned parallel to the presumed orientation of the underlying fibres. EMG signals were amplified at source ($\times 300$; 20- to 450-Hz bandwidth) before further amplification (overall effective gain, $\times 909$), and sampled at 2000 Hz via the same A/D converter and computer software as the force signal, to enable data synchronization. In offline analysis, EMG signals were corrected for the 48-ms delay inherent to the Trigno EMG system and band-pass filtered (6–500 Hz) using a fourth-order zero-lag Butterworth filter.

Pre- and post-measurement sessions

Following a brief warm-up of the dominant leg [3 s contractions at 50% ($\times 3$), 75% ($\times 3$), and 90% ($1\times$) of perceived maximum] measurements were completed in the following order.

Knee extension maximum voluntary contractions

Participants performed 3–4 MVCs and were instructed to “push as hard as possible” for 3–5 s and rest for ≥ 30 s between efforts. A torque–time curve with a horizontal cursor indicating the greatest torque obtained within that session was displayed for biofeedback and verbal encouragement was provided during all MVCs. Knee extensor MVT was the greatest instantaneous torque achieved during MVC in that measurement session. Quadriceps root mean square (RMS) EMG for a 500 ms epoch at MVT (250 ms either side) was normalized to maximal M-wave (M_{MAX}) area (see below) from the corresponding EMG site and then averaged across all quadriceps EMG sites to provide a whole quadriceps measurement ($QEMG_{MVT}$). Similarly, Hamstrings RMS EMG for the same 500 ms epoch at knee extension MVT was normalized to $HEMG_{MAX}$ (a 500 ms EMG epoch measured at knee flexion MVT; see below)

from the corresponding EMG site before being averaged across the two hamstrings sites to provide an overall antagonist hamstrings measurement during knee extension MVT ($HEMG_{ANTAG}$).

Evoked twitch contractions

A constant current variable voltage stimulator (DS7AH; Digitimer Ltd., Welwyn Garden City, UK), cathode probe (1 cm diameter, Electro-Medical Supplies Ltd., Wantage, UK), and anode electrode (7×10 cm carbon rubber electrode; Electro-Medical Supplies Ltd., Wantage, UK) were used to electrically stimulate the femoral nerve. The cathode and anode were coated with electrode gel and securely taped to the skin over the femoral nerve in the femoral triangle and over the greater trochanter, respectively. Cathode location was determined by delivering single electrical impulses (square wave-pulses of 0.2 ms duration, ≥ 12 s apart) to identify the position that elicited the greatest sub-maximum twitch response. The current intensity was progressively increased until plateaus in peak twitch force and peak-to-peak M-wave amplitude were reached. Then three supra-maximal twitch and M_{MAX} responses were evoked (15 s apart) at a higher current ($\geq 50\%$) to ensure maximal stimulation. Cumulative M_{MAX} area from EMG onset (after stimulation artefact) to the point where the signal returned to baseline for each of the six EMG sites was averaged across the three supra-maximal twitch contractions.

Knee flexion maximum voluntary contractions

Knee flexion MVCs were performed in the same manner as knee extension, except participants performed a series of submaximum knee flexion efforts to warm-up and were instructed to “pull as hard as possible” for 3–5 s, rather than “push”. Knee flexor MVT was the greatest instantaneous torque achieved during any flexor MVC during that measurement session. RMS EMG for a 500 ms epoch at knee flexor MVT (250 ms either side) was measured from each hamstring EMG site.

Total quadriceps volume ($QUADS_{VOL}$)

A 1.5 T MRI scan of the dominant leg was made in the supine position at a knee joint angle of $\sim 163^\circ$ using a receiver 8-channel whole body coil (Signa HDxt, GE). T1-weighted axial slices (5 mm thick, 0 mm gap) were acquired from the anterior superior iliac spine to the knee joint space in two overlapping blocks. Oil-filled capsules placed on the lateral side of the participants’ thigh allowed alignment of the blocks during analysis. MR images were analysed by two investigators using Osiris software (version 6.0, Pixmeo, Geneva, Switzerland).

Pre- and post-scans of each participant were analysed by the same investigator. The quadriceps (RF, VL, VM, and vastus intermedius; VI) muscles were manually outlined in every third image (i.e. every 15 mm) starting from the most proximal image in which the muscle appeared. The volume of each muscle was calculated using cubic spline interpolation (GraphPad Prism 6, GraphPad Software, Inc.). Total quadriceps volume ($QUADS_{VOL}$) was the sum of the individual muscle volumes. Inter- and intra-rater reliability for $QUADS_{VOL}$ calculated from the repeated analysis of MRI scans of five participants was 1.2 and 0.4%, respectively.

Quadriceps muscle fascicle pennation angle ($QUADS\theta_p$)

RF, VL, VM, and VI θ_p were examined using B-mode ultrasonography (EUB-8500, Hitachi Medical Systems UK Ltd, Northamptonshire, UK) with a 9.2 cm, 5–10 MHz linear-array transducer (EUP-L53L). The participant sat in the same isometric apparatus used for testing and training whilst images were captured at rest from the following percentage of thigh length proximal to the knee joint space: RF 55%, VL 50%, VM 40%, and VI 50%. The transducer (coated with water-soluble transmission gel) was positioned along the median longitudinal line (50% of superficial medio-lateral width) of the muscle at the imaging locations, with minimal pressure applied on the dermal surface. The transducer was orientated perpendicular to the skin and parallel to the fascicular path and appropriate orientation was defined as the orientation resulting in an image with the aponeuroses and the perimysium trajectory of several fascicles being clearly identifiable with no visible fascicle distortion at the edge of the image. Video output from the ultrasound machine was transferred to a computer (via an S-video to USB converter) and images were recorded using ezcip video capture software. Images were later imported into public domain software (Image J, v1.48, National Institutes of Health, Bethesda, USA) for analysis.

For each constituent muscle of the quadriceps, θ_p was measured as the angle of insertion of the fascicles onto the deep aponeurosis. To calculate an overall measure of $QUADS\theta_p$ the θ_p of each constituent muscle was multiplied by the ratio of its respective muscle volume to $QUADS_{VOL}$ (see previous section). The sum of these values produced $QUADS\theta_p$ expressed as the weighted mean of the constituent muscles based on their relative contribution to total $QUADS_{VOL}$ (Massey et al. 2015). All ultrasound images were collected and analysed by the same investigator. Intra-rater reliability calculated from the repeated analysis of RF, VL, VM, and VI ultrasound images of five participants was 1.6% for θ_p .

Data analysis and statistics

All data were anonymised prior to analysis. MVT, $QEMG_{MVT}$, and $HEMG_{ANTAG}$ measurements from the duplicate test sessions were averaged to produce criterion pre- and post-values for statistical analysis; unless within-participant coefficient of variation [(SD/mean) \times 100] for the MVT was $\geq 10\%$ (calculated from duplicate test sessions), in which case the lowest MVT value was discarded. All statistical analyses were conducted using SPSS Version 22.0 (IBM Corp., Armonk, NY, USA). Paired *t* tests were used for the absolute data to determine if within group pre- to post-changes occurred at a cohort level. Significance was defined as $P < 0.05$ and group data are expressed as mean \pm standard deviation (SD), including whole cohort percentage changes for each variable.

Individual pre- to post-training percentage changes (Δ) for MVT, neural and morphological variables were calculated for each RT group participant. The initial statistical analysis involved Pearson's product moment bivariate correlations between ΔMVT and the different predictor variables: $\Delta QEMG_{MVT}$, $\Delta HEMG_{ANTAG}$, $\Delta QUADS_{VOL}$, $\Delta QUADS\theta_p$ and pre-training MVT. Thereafter, stepwise multiple linear regression was conducted including all the predictor variables that were significantly correlated with ΔMVT to reveal those that independently explained a significant proportion of the total variance in ΔMVT . Adjusted R^2 values from the stepwise multiple linear regression are reported, as well as variance inflation factor (VIF) to confirm the limited multicollinearity.

Results

Cohort adaptations after resistance training

Pre-training MVT (234 ± 40 Nm) within the RT group ranged from 173 to 347 Nm and demonstrated a between-participant coefficient of variation value of 16.9% [(cohort SD/cohort mean) \times 100]. MVT increased from pre-training to 283 ± 43 Nm post-training (paired *t* test $P < 0.001$; $+21.7 \pm 11.5\%$, range -4.9 to $+48.9\%$). In the RT group $QEMG_{MVT}$ increased from 12.3 ± 3.8 to $15.1 \pm 3.5 M_{MAX}$ area s^{-1} (paired *t* test $P < 0.001$; $+29.1 \pm 31.2\%$, range -29.2 to $+91.3\%$) and $HEMG_{ANTAG}$ decreased, pre- to post-training, from 23.9 ± 13.0 to $19.5 \pm 10.8\%$ $HEMG_{MAX}$ ($P = 0.046$; $-6.0 \pm 49.0\%$; range -72.8 to $+99.5\%$). $QUADS_{VOL}$ increased from 1797 ± 260 to 1897 ± 306 cm^3 (paired *t* test $P < 0.001$; $+5.6 \pm 8.1\%$, range -9.9 to $+23.6\%$) and $QUADS\theta_p$ also increased from 14.1 ± 2.3 to $16.0 \pm 2.6^\circ$ ($P < 0.001$; $+13.8 \pm 12.8\%$; range -17.8 to $+37.3\%$).

The CON group's MVT (pre 257 ± 49 N, post 259 ± 57 Nm; paired t test $P = 0.739$), QEMG_{MVT} (pre 12.5 ± 4.7 M_{MAX} area s⁻¹, post 11.6 ± 3.5 M_{MAX} area s⁻¹; $P = 0.298$), HEMG_{ANTAG} (pre $17.1 \pm 10.1\%$ HEMG_{MAX}, post $13.5 \pm 7.6\%$ HEMG_{MAX}; $P = 0.090$), QUADS_{VOL} (pre 1891 ± 272 cm³, post 1906 ± 261 cm³; $P = 0.550$), and QUADS_{θ_p} (pre $15.4 \pm 2.0^\circ$, post $15.3 \pm 1.8^\circ$; $P = 0.810$) did not change significantly from pre to post. As CON did not engage in RT they were not included in further bivariate correlation or multiple regression analyses.

Neural, morphological, and pre-training strength contribution to MVT changes after resistance training

In the RT group individual Δ MVT were correlated with Δ QEMG_{MVT} ($r = 0.576$, $P = 0.001$; Fig. 1a), Δ QUADS_{VOL} ($r = 0.461$, $P = 0.014$; Fig. 2a), and pre-training MVT

($r = -0.429$, $P = 0.023$; Fig. 3), but were not associated with Δ HEMG_{ANTAG} ($r = 0.298$, $P = 0.123$; Fig. 1b) or Δ QUADS_{θ_p} ($r = -0.207$, $P = 0.291$; Fig. 2b). Multiple regression analysis revealed that 59.9% of the variance in the Δ MVT was explained by Δ QEMG_{MVT} [$R^2 = 0.306$ (30.6%); VIF = 1.000], Δ QUADS_{VOL} [$R^2 = 0.187$ (18.7%); VIF = 1.001], and pre-training MVT [$R^2 = 0.106$ (10.6%); VIF = 1.030; Fig. 4).

Discussion

This study investigated the contribution of individual adaptations in neural and morphological factors, as well as pre-training strength, to the strength changes after 12 weeks of RT. Three variables (Δ QUADS_{VOL}, Δ QEMG_{MVT}, and absolute pre-training strength) were moderately correlated

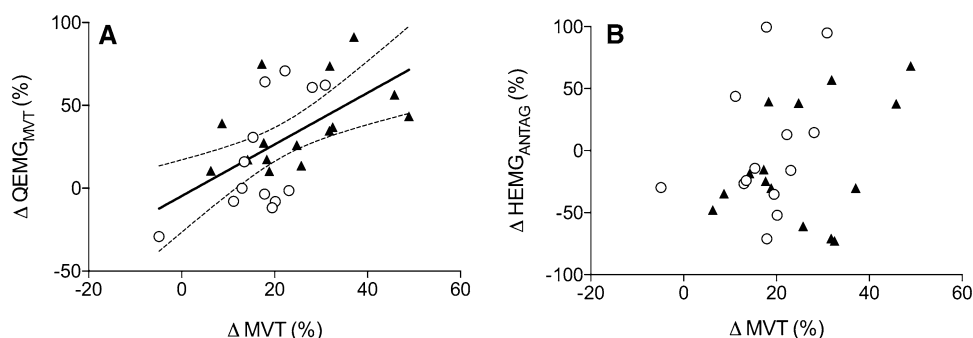


Fig. 1 The relationships between the percentage change (Δ) in knee extension maximum voluntary torque (MVT) and: **a** Δ quadriceps EMG at knee extension MVT (QEMG_{MVT}; $r = 0.576$, $P = 0.001$); **b** Δ antagonist hamstrings EMG during knee extension MVT (HEMG_{ANTAG}; $r = 0.298$, $P = 0.123$), after 12 weeks of resistance training. QEMG_{MVT} values pre- and post-training were normalized to M_{MAX} area and HEMG_{ANTAG} was normalized to EMG at knee flexion

MVT, prior to calculating pre- to post-training percentage changes. *Solid and dashed lines* indicate the trend of the relationship between variables and 95% confidence intervals, respectively. *Black triangles* denote sustained-contraction resistance training participants ($n = 15$); *white circles* denote explosive-contraction resistance training participants ($n = 13$)

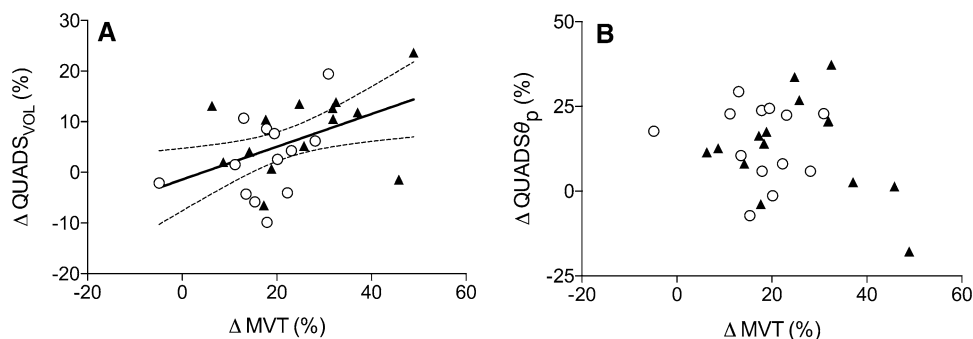


Fig. 2 The relationships between the percentage change (Δ) in knee extension maximum voluntary torque (MVT) and **a** Δ quadriceps muscle volume (QUADS_{VOL}; $r = 0.461$, $P = 0.014$), **b** Δ quadriceps muscle fascicle pennation angle (QUADS_{θ_p}; $r = -0.207$, $P = 0.291$), after 12 weeks of resistance training. *Solid and dashed lines* indicate

the trend of the relationship between variables and 95% confidence intervals, respectively. *Black triangles* denote sustained-contraction resistance training participants ($n = 15$); *white circles* denote explosive-contraction resistance training participants ($n = 13$)

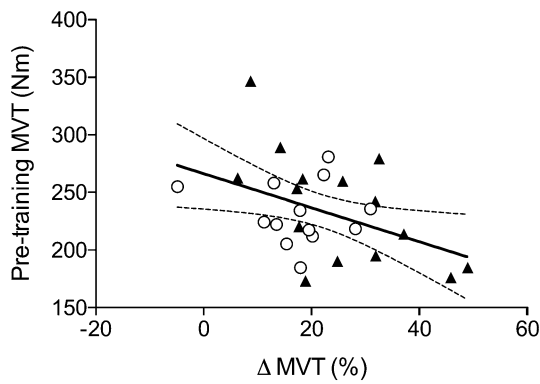


Fig. 3 The relationships between the percentage change (Δ) in knee extension maximum voluntary torque (MVT) and pre-training knee extension MVT ($r = -0.429$, $P = 0.023$) after 12 weeks of resistance training. *Solid and dashed lines* indicate the trend of the relationship between variables and 95% confidence intervals, respectively. *Black triangles* denote sustained-contraction resistance training participants ($n = 15$); *white circles* denote explosive-contraction resistance training participants ($n = 13$)

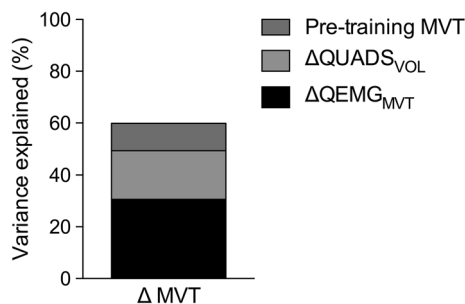


Fig. 4 Contribution of predictor variables that independently explained a significant proportion of the total variance in the individual percentage changes (Δ) in maximum voluntary torque (MVT) after 12 weeks of resistance training assessed with stepwise multiple regression analysis. $QUADS_{VOL}$ total quadriceps muscle volume, $QEMG_{MVT}$ quadriceps EMG measured during MVT production

with strength gains. Subsequent multiple regression analysis found for the first time that these three variables simultaneously contributed to the total explained variance in strength, in combination explaining 60% of the total variance in strength gains. In contrast, changes in θ_p and antagonist EMG were unrelated to changes in strength. Interestingly, agonist neural drive was the primary contributor to strength changes, accounting for more than half of the total explained variance after 12 weeks of RT (30.6%), whereas $QUADS_{VOL}$ changes (18.7%) and pre-training MVT (10.6%) made smaller contributions to the explained variance.

The moderate bivariate correlations between the changes in MVT and agonist EMG in the current study ($r = 0.576$) were consistent with several previous EMG studies of lower

body RT [$r = 0.69$ (Shima et al. 2002); $r = 0.74$ (Häkkinen and Komi 1986); $r = 0.66$ (Häkkinen et al. 1985)], but not an elbow flexor RT study ($r = 0.187$; Erskine et al. 2014), which may be significant as the elbow flexors have been found to have a very high level of activation even in the pre-training state (Allen et al. 1998; Gandevia et al. 1998). In addition, two studies that measured the changes in agonist activation with ITT also found no relationship with individual strength gains after RT (Shima et al. 2002; Erskine et al. 2010b), although several studies have queried the sensitivity of the ITT to detect changes in activation after RT (Herbert et al. 1998; Harridge et al. 1999; Cannon et al. 2007; Del Balso and Cafarelli 2007; Tillin et al. 2011). The EMG procedures of the current study (duplicate measurements of function and agonist activation both pre- and post-training, two EMG sensors on each superficial quadriceps muscle, M_{MAX} area normalization) may have improved the sensitivity of both agonist EMG and strength measurements and thus provided a clearer reflection of the importance of individual changes in agonist activation. The increased $QEMG_{MVT}$ amplitude following training in the current study was likely a result of increased motor unit firing rate (Kamen and Knight 2004; Knight and Kamen 2008). It is also possible that recruitment of additional motor units (Jones et al. 1989; Folland and Williams 2007) and/or increased motor unit synchronization (Milner-Brown et al. 1975; Semmler and Nordstrom 1998) could also enhance EMG amplitude, although it is unclear if the later can contribute to an increase in contractile force (Lind and Petrofsky 1978). The observation of agonist activation being the largest contributor to the strength changes after RT (30.6% of the total variance explained) is consistent with the apparently much larger cohort level changes in agonist activation (+29.1%) than muscle hypertrophy (+5.6%) in the current investigation. We suspect that adaptations in agonist neural drive may account for more of the explained variance in strength changes than reported here, although the difficulty in assessing complex and subtle neural adaptations in vivo remains a challenge.

The relationship between changes in $QUADS_{VOL}$ and MVT in the current investigation (bivariate correlation $r = 0.461$; 21% of the total variance explained) corroborates the contribution of muscle hypertrophy to individual strength gains found by our earlier upper body study (bivariate correlation $r = 0.527$; 28% of the variance explained; Erskine et al. 2014), whilst also extending this finding to the lower body (knee extensors). The slightly lower correlation and regression values in the current study compared to our previous work is likely due to the smaller hypertrophic response following lower body RT (5.6 vs. 15.9%) that is a common observation when comparing lower and upper body muscle size adaptations (Cureton et al. 1988; Welle et al. 1995). Although some

earlier studies did not find a relationship between hypertrophy and strength gains, they involved smaller cohorts (≤ 12) or less precise measurements of muscle size (e.g. single slice scans; Jones and Rutherford 1987; Davies et al. 1988). The simple comparison of group level changes in MVT (+21.7%) and QUADS_{VOL} (+5.6%) suggest that muscle hypertrophy accounts for at most 25% of the change in strength following isometric knee extensor RT over 12 weeks. Therefore, the 18.7% of the total variance in MVT explained by QUADS_{VOL} appears to be capturing the majority of the potential contribution of muscle hypertrophy to strength changes after RT. Whilst the contribution of muscle hypertrophy in the current study was secondary to neural adaptations during this relatively short-term intervention it clearly did contribute to the explained variance in strength and further negates the suggestion that strength and hypertrophy are entirely separate phenomena (Buckner et al. 2016). It seems likely that hypertrophy would be a more important contributor with longer periods of RT.

The combination of agonist neural drive and muscle size adaptations in the current study explained 49.3% of the total variance in strength change, which was substantially less than one previous report (65.0%; Higbie et al. 1996; discussed above). In the current lower body study, pre-training strength was also inversely related to the changes in strength after RT (bivariate correlation $r = -0.429$) which extends the similar findings of previous upper body studies (Erskine et al. 2014: $r = -0.52$; Hubal et al. 2005: $r = -0.55$). Based on these collective findings it seems likely that pre-training strength influences the strength gains of all muscle groups to RT. Furthermore pre-training strength also explained 10.6% of the variance in strength changes, and in combination with agonist neural drive and hypertrophy explained 59.9% of the total variance in strength. Pre-training strength may in turn reflect individual differences in physical activity and/or potentially body mass. Whilst participants in the current study were recruited on the basis of no RT in the preceding 18 months, it seems likely that their previous exposure to strength and power activities, including any RT as well as whole body activities (e.g. sprinting, jumping), may have contributed to differences in pre-training strength and, therefore, also their responsiveness to RT. In addition, theoretically since pre-training strength is known to be related to body mass (Folland et al. 2008), as was also the case in the current study ($n = 42$: $r = 0.455$, $P = 0.002$), this might imply that lighter individuals with lower pre-training strength were more responsive to RT. However the strength gains in this study were unrelated to pre-training body mass ($n = 28$: $r = 0.003$, $P = 0.988$) so it seems more likely that the influence of pre-training strength on strength gains was due to prior physical activity.

Antagonist co-activation was not significantly related to the extent of individual MVT changes. The lack of correlation between individual changes in MVT and HEMG_{ANTAG} may be due to the small (-6.0%) cohort level changes in antagonist co-activation and the relatively poor reliability of this measurement (Tillin et al. 2011). Assuming a linear relationship between hamstring EMG and knee flexion torque, the estimated antagonist torque during knee extension MVT (knee flexion MVT \times HEMG_{ANTAG}) was reduced from 14 Nm (pre) to 13 Nm (post) for the average participant. Therefore, suggesting only ~ 1 Nm of the mean 49 Nm increase in knee extension MVT was due to reduced antagonist co-activation. The current study attempted to improve the HEMG_{ANTAG} measurement with recordings of both the BF and ST, and not just the BF (Carolan and Cafarelli 1992; Pucci et al. 2005; Erskine et al. 2010b), as well as performing duplicate measurement sessions both pre- and post-training. Nevertheless, the measures of antagonist activation were not sensitive enough to capture any influence of individual adaptations in antagonist co-activation on strength gains.

Individual changes in θ_p were not related to changes in MVT, which was consistent with earlier findings (Erskine et al. 2010b, 2014). Whilst increases in θ_p permit more contractile material to be attached to the aponeurosis and thus are assumed to contribute to strength gains it has so far not been possible to detect a relationship between θ_p changes and individual strength gains. The inability to capture the relationship of θ_p adaptations and strength changes on an individual basis may be due to the limited assessment of complex muscle architecture via 2D ultrasonography and the use of 3D imaging techniques, such as diffusion tensor MRI (Damon et al. 2012), seems warranted to further investigate the role of θ_p adaptations in relation to strength changes.

Despite the current study explaining a large proportion of the total variance in MVT changes following 12 weeks of RT, 40% of the variance remained unexplained. This unexplained variance may be in part due to the additive measurement noise/error from two data collection time points combining to influence the change data in this type of longitudinal study design. In terms of the specific predictor, whilst antagonist co-activation and θ_p were not found to contribute to the explained variance in strength changes in the current study, it is possible that with more sensitive measurement techniques they could account for some of the unexplained variance. Theoretically it is possible that other measures of muscle size, e.g. physiological cross-sectional area (PCSA), may be better indices of contractile force generating capacity and potentially explain a greater proportion of the changes in strength after RT. However, muscle volume was selected as our measure of muscle hypertrophy as pre-training QUADS_{VOL} was found

to demonstrate a stronger relationship with pre-training strength ($n = 42$: $r = 0.794$, $P < 0.001$) than quadriceps PCSA ($n = 42$: $r = 0.608$, $P < 0.001$) or quadriceps anatomical cross-sectional area ($n = 42$: $r = 0.680$, $P < 0.001$). In addition, other morphological changes that could also have contributed to the total explained variance in MVT change after RT were not included in the current study (e.g. muscle-fibre composition, contractile protein density). As mentioned previously it is also possible that agonist activation explained more than 30.6% of the total variance in strength gains that we have measured. For example, the current study assessed agonist activation in three of the superficial quadriceps, but not the VI and it is possible that the addition of this measure may have helped to explain more of the total variance in strength changes following RT. Finally, the current study pooled data from two different training interventions to provide a suitable sample size for correlation and regression analysis ($n = 28$). This additional variable (training type) might have been expected to confound the influence of the physiological predictors in the current study. Thus, it is possible that without this potential confounder that the current study might have been able to explain >60% of the variance in strength gains with a uniform RT intervention.

In conclusion, changes in agonist neural drive and muscle volume, as well as pre-training strength, explained 60% of the total variance in strength changes after RT, with agonist neural drive the most important single determinant. In contrast, antagonist co-activation and muscle fascicle pennation angle, were unrelated to strength gains possibly due to the limited sensitivity of these measurements to detect individual changes.

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