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Is repetition failure critical for the development of muscle hypertrophy and strength?

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This investigation sought to determine the effect of resistance training to failure on functional, structural and neural elbow flexor muscle adaptation. Twenty-eight males completed a 4-week familiarization period and were then counterbalanced on the basis of responsiveness across; non-failure rapid shortening (RS; rapid concentric, 2 s eccentric), non-failure stretch-shortening (SSC; rapid concentric, rapid eccentric), and failure control (C, 2 s concentric, 2 s eccentric), for a 12-week unilateral elbow flexor resistance training regimen, 3 × week using 85% of one repetition maximum (1RM). 1RM, maximal voluntary contraction (MVC), muscle cross-sectional area (CSA), and muscle activation (EMG_{RMS}) of the agonist, antagonist, and stabilizer muscles were assessed before and after the 12-week training period. The average number of repetitions per set was significantly lower in RS 4.2 [confidence interval (CI): 4.2, 4.3] and SSC 4.2 (CI: 4.2, 4.3) compared with C 6.1 (CI: 5.8, 6.4). A significant increase in 1RM (30.5%), MVC (13.3%), CSA (11.4%), and agonist EMG_{RMS} (22.1%) was observed; however, no between-group differences were detected. In contrast, antagonist EMG_{RMS} increased significantly in SSC (40.5%) and C (23.3%), but decreased in RS (13.5%). Similar adaptations across the three resistance training regimen suggest repetition failure is not critical to elicit significant neural and structural changes to skeletal muscle.

First proposed as a means of accelerating the rehabilitation of injured World War II soldiers (Delorme, 1945), the use of repetition failure is now an established cornerstone of modern resistance training regimen (Anderson & Kearney, 1982; Hakkinen et al., 1985; Campos et al., 2002). Resistance exercise performed to failure elevates muscle protein synthesis independent of volume (sets × reps) or % one repetition maximum (1RM) load (Burd et al., 2010b; Mitchell et al., 2012). For example, low-load blood flow-restricted exercise has been shown to elicit significant increases in muscle hypertrophy and strength (Fujita et al., 2007; Takada et al., 2012). Furthermore, investigations that controlled relative %1RM training load and volume reported that repetition failure led to significantly greater gains in muscular strength (Rooney et al., 1994; Drinkwater et al., 2005). Thus, collectively, the evidence appears to suggest that repetition failure is an essential characteristic of resistance training regimen (Phillips, 2009).

However, accumulation of intramuscular metabolites or elevated endogenous circulating hormones, physiological responses associated with resistance exercise to failure, are not necessarily required to elicit significant changes in skeletal muscle structure or function (Wilkinson et al., 2006; West et al., 2009), suggesting that there are multiple signaling pathways that may promote muscular hypertrophy and strength in the absence of repetition failure (Goldberg, 1967; Spangenburg et al., 2008). For example, when experimental groups were matched for total work, both Folland et al. (2002) and Izquierdo et al. (2006) observed isometric force production, single repetition maximum strength, local muscle endurance, and explosive power gains were similar regardless of the level of local muscle fatigue induced by the resistance training regimen (Folland et al., 2002; Izquierdo et al., 2006). Furthermore, greater gains in muscular strength have been reported with increased resistance exercise volume but in the absence of repetition failure (Kramer et al., 1997; Sanborn et al., 2000). Collectively, these findings suggest repetition failure may not be important to elicit changes in skeletal muscle function. However, to our knowledge, no current investigation has reported upon the effect of repetition failure on skeletal muscle cross-sectional area (CSA).

Mechanical force is a factor that regulates protein function (Seifert & Gräter, 2013) and has direct effects upon the nucleus of the cell (Fedorchak et al., 2014), and

skeletal muscle is sensitive to changes in mechanical tensile loading (Martineau & Gardiner, 2001). At an integrated level, rapid muscle activations have been shown to increase exposure of skeletal muscle to peak mechanical force (Newton et al., 1997; Sampson et al., 2014) and simultaneously elevate motor unit recruitment via decreased recruitment thresholds and increased rate of motor unit discharge (Desmedt & Godaux, 1977). Munn et al., 2005, clearly showed that rapid muscle activations performed to failure led to similar gains in elbow flexor strength than was observed with a twofold increase in resistance training volume at slower movement speeds. These findings suggest that rapid muscle activation may increase adaptive sensitivity to resistance training independent of training volume and repetition failure.

This investigation therefore determined if repetition failure was a critical characteristic for skeletal muscle adaptation to resistance training. A novel loading strategy was used, where the experimental groups performed only four of the six elbow flexor repetitions required for repetition failure and thus also, these groups had a reduced resistance training volume. Furthermore, to minimize heterogeneity in responsiveness to resistance training, all subjects completed a 4-week familiarization period prior to commencing the investigation.

Methods

Subjects

Twenty-eight males, who had not participated in resistance exercise for a minimum of 6 months, volunteered to participate in this investigation. All subjects completed a physical activity readiness questionnaire and provided written informed consent. All procedures were approved by the University of Wollongong Human Ethics Research Committee.

Experimental familiarization and randomization

All subjects completed a 4-week familiarization phase that consisted of controlled (2 s concentric, 2 s eccentric) resisted unilateral elbow flexor exercise to repetition failure (Sampson et al., 2013). Resistance loading commenced at 50% of 1RM and increased by 10% each week, thus, in the fourth and final week of familiarization, a load of 80% of 1RM was used. The relative gain (%) in 1RM during the familiarization period was calculated for each subject, and using a triplet method, subjects were counterbalanced across the three experimental training conditions on the basis of responsiveness to the familiarization period. Thus, higher and lower responders to the 4-week elbow flexor resistance exercise period were evenly distributed between the three conditions prior to the 12-week experimental training regimen. The 1RM strength gain during the familarization period was similar between rapid shortening (RS) 19.0% [confidence interval (CI): 13.2, 24.8], stretch-shortening cycle (SSC) 17.3% (CI: 8.3, 26.4), and control (C) 15.6% (CI: 7.7, 23.4). This investigation focuses upon reporting physiological changes from participation in the 12-week training regimen, and changes related to the familiarization period have already been reported (Sampson et al., 2013).

Experimental protocol and regimen

Subjects were assessed for elbow flexion performance in dynamic 1RM, isometric maximal voluntary contraction (MVC), agonist muscle CSA, and agonist and antagonist muscle activation (EMG) at four time points, prior to commencing the 12-week regimen (week 1) in the fourth and eighth week (weeks 4 and 8) and at the completion of the training regimen (week 12). All training sessions comprised 85% 1RM unilateral dominant limb elbow flexion-extension exercise, commencing at 60° and terminating at 160° of flexion in a supine position with the hips and knees flexed at 90° as described and illustrated elsewhere (Sampson et al., 2013, 2014). Each experimental group was required to complete four sets of resistance exercise with a 3-min rest period between each set three times per week on alternate days.

The treatment groups were differentiated in two ways: (a) the speed in which the elbow flexion extension movement was performed, and, (b) the number of completed repetitions within each set. The control training regimen (C) performed a 2-s flexion and 2-s extension movement controlled via a metronome, the RS performed maximal acceleration during elbow flexion followed by a 2-s extension, and the SSC regimen completed maximal acceleration during both elbow flexion and extension movements. Participants in C were required to exercise to repetition failure for each set (six repetitions). In contrast, participants in the RS and SSC completed only four repetitions in each set, thus these regimens did not require repetitions to be completed to failure. To ensure the relative loading was comparable with C, RS and SSC performed a single set of elbow flexion to failure once each week. The training load was then adjusted for the week on the basis of this assessment.

Experimental assessment

Elbow flexor 1RM

Dominant limb dynamic elbow flexor 1RM strength was assessed from the experimental training position (60–160°) before (week 1), during (weeks 4 and 8), and after (week 12) the 12-week intervention. During these assessments, the dominant and contralateral glenohumeral joints were secured to prevent unwanted movement. A minimum 2-min rest period was given between successive attempts, and 1RM was recorded as the highest successful repetition completed to the closest 0.25 kg. 1RM was obtained within six trials.

Elbow flexor MVC torque

A 5-s MVC at 90° of elbow flexion was also performed in weeks 1 and 12. In the experimental position, the forearm was supinated and strapped to a platform and subjects were instructed to produce maximal force as rapidly as possible at the illumination of an LED light. Visual feedback via an oscilloscope, and verbal encouragement to reach maximal force was provided. Peak torque (Nm) over 250 ms was determined from a 1000 N load cell (Applied Measurement, X-TRAN, 51W-1kN, Eastwood, NSW, Australia) fixed in series with the experimental equipment, recorded by a DC pressure amplifier (Neurolog, 108A, Digitimer Neurolog, Hertfordshire, UK) collecting data at 200 Hz. After 5 min of rest, MVC tests were repeated to confirm maximal effort; if the difference was >5%, a third MVC was performed.

Elbow flexor muscle activation

During 1RM and MVC strength assessments, muscle activity was recorded. The surface of the skin was shaved, abraded, and cleansed with alcohol at the electrode sensor placement site. Surface electrodes (Ag/AgCL contact diameter 15 mm) were adhered central to the muscle belly of the biceps brachii and triceps brachii midway between the acromion process and elbow crease. Movement of the proximal radioulnar joint was controlled

by maintaining the forearm in supination. Surface electrodes were also applied to monitor shoulder stabilization during 1RM assessments at the anterior deltoid 40 mm below the clavicle, and upper trapezius, along the ridge of the shoulder, halfway between the cervical spine and the acromion. A reference electrode was adhered to the most prominent portion of the right clavicle with the intra-electrode distance set at 20 mm. Electrode positions were marked with henna dye and maintained throughout the 12-week training period to ensure reliable placement of electrodes during each subsequent trial. Electromyographic signals were pre-amplified with a low-frequency cutoff (3 Hz), amplified 1000 times, and high- and low-band pass filtered (10–500 Hz, Neurolog 844, 820, 144, 135, Digitimer Neurolog). This system provides a 100 Ω input impedance and common mode rejection ratio >120 dB. Data were collected at 2000 Hz per channel, processed via an analog-to-digital converter (Power 1401, Cambridge Electronic Design, Cambridge, UK) and assessed via a series of 250-ms windows with a 50% overlap using Spike 2 software (Ver 5.13, Cambridge Electronic Design, Cambridge, UK). Elbow flexion and extension was identified from a shaft encoder with a resolution of ~0.07 mm (E6C2-CWZ6C-1000, Omron, Minato-ku, Tokyo, Japan), acting as the first pulley wheel within the experimental equipment to provide distance, time, and direction data during muscular contractions. Shaft encoder data were processed through the Power 1401 analog-digital converter and synchronized with EMG through the Spike 2 software program. In this investigation, average electromyographic root mean square amplitude (EMG_{RMS}, mV) was calculated over concentric and eccentric phases of the contraction during 1RM assessments. Peak EMG_{RMS} (mV) during the MVC was recorded over 250 ms central to peak isometric torque (Nm) developed by each subject. EMG_{RMS} values recorded in week 12 were normalized to the respective 1RM or MVC value recorded in week 1 (Newton et al., 1996, 1997; Sampson et al., 2014).

Elbow flexor muscle CSA

Elbow flexor CSA was recorded by an experienced radiologist at week 1 and week 12. Scans were performed a minimum of 24 h after the final training session of each respective week. A total of 44 muscle slices were recorded (thickness: 6.35 mm, 1 mm inter-slice gap) via magnetic resonance imaging (MRI), Turbo Spin Echo, T2 images, (1.5 T Philips Intera, Philips Healthcare, Da Best, the Netherlands). Participants were supine for these scans with the superior margin of the coil positioned level with the acromioclavicular joint. Imaging commenced at the superior portion of the humeral head, extending distally along the length of the muscle. The biceps brachii and brachialis (Fig. 1) were traced individually using commercially available software (3d-Doctor, Able Software Corporation, Lexington, Massachusetts, USA) with care taken to trace round any visible intramuscular fat and connective tissue. CSA was calculated as the mean across three images central to the muscle belly (slices 21–23).

Elbow flexion kinematics

Kinematic data in each of the three exercise conditions was captured during one training session in week 11. Limb movement velocity ($m \cdot s^{-1}$, $\Delta d/\Delta t^{-1}$) was calculated from time and displacement data provided by the shaft encoder by setting cursors to count the number of pulses delivered from the start of movement, to the end of the last completed repetition prior to task failure. A calibration reference was gained by moving the arm of each subject passively through a 100° range of motion prior to exercise. Significant kinematic differences between the C, RS, and SSC groups have been reported previously (Sampson et al., 2014).

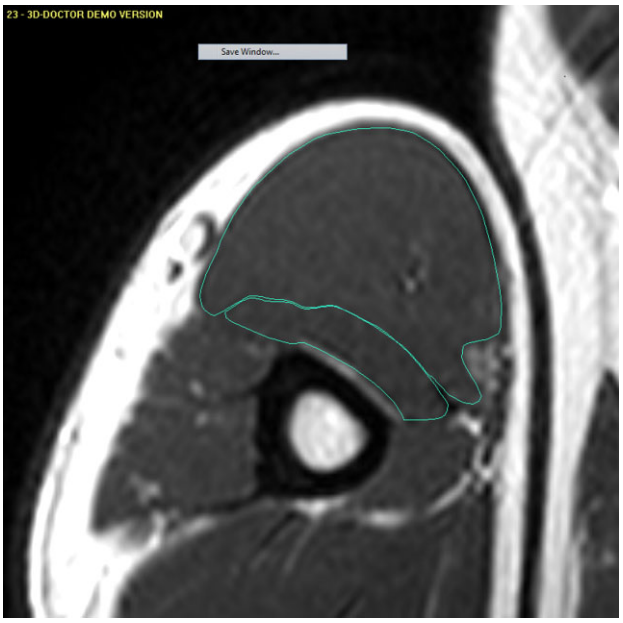

Fig. 1. Representation of an MRI trace from one subject highlighting the combined biceps and brachialis area assessed for measures of muscle cross-sectional area in this investigation.

Psychophysical response

At the end of each set, subjects were asked "how hard were you exercising" to provide a rating of perceived exertion (Borg, 1970) and the total resistance exercise volume (repetitions × load) was quantified over the 12-week training regimen.

Statistical analysis

A two-way repeated measures analysis of variance (ANOVA) examined treatment effects over time, and where interactions were observed, a post-hoc Tukey's was applied (Prism Ver. 6.00, GraphPad Software, San Diego, California, USA). Where significant differences over time were observed and no between groups interactions were detected, data were also pooled, and one-way ANOVA, or paired *t*-test analysis was performed. Data are reported as means ± 95% confidence intervals (CI), unless otherwise stated as standard deviation (SD). Significance is set at an alpha level of < 0.05 for all statistical analyses.

Results

Baseline characteristics

Twenty-eight subjects completed the investigation, C ($n = 10$), RS ($n = 10$), SSC ($n = 8$), and their results are reported herein. Group characteristics at week 1 are reported in Table 1. No significant difference in the age, stature, mass, and elbow flexor CSA was observed between RS, SSC, and C. Force production characteristics at week 1 were also similar for elbow flexor 1RM and MVC in RS, SSC, and C, respectively.

Training regimen characteristics and compliance

Characteristics of the training regimen for each treatment group are highlighted in Table 2. Each exercise

Table 1. Characteristics of participants in the rapid shortening (RS), stretch-shortening cycle (SSC), and control (C) groups prior to the 12-week training period

Group	Age (years)	Stature (cm)	Mass (kg)	CSA (cm^2)	1RM (kg)	MVC (Nm)
RS	23.7	179.1	85	13.3	22.3	91.4
	SD6.2	SD7.5	SD13.7	SD2.0	SD3.6	SD13.7
SSC	24.3	179.0	77.9	11.9	19.2	93.9
	SD7.0	SD8.8	SD12.1	SD2.3	SD1.8	SD28.8
C	23.4	180.3	76.9	12.0	19.9	80.1
	SD6.6	SD5.8	SD0.2	SD1.8	SD3.7	SD26.6

Data are displayed as within-group averages and standard deviation (SD).
1RM, one repetition maximum; CSA, cross-sectional area; MVC, maximal voluntary contraction.

Table 2. Characteristics of the 12-week training regimen in the rapid shortening (RS), stretch-shortening cycle (SSC), and control (C) groups

Variable	Group		
	RS	SSC	C
Training load (kg)	22.6 (20.2,25.0)	19.3 (17.4,21.3)	21.0 (18.8,23.3)
Repetitions (reps/set)	4.2 (4.2,4.3)*	4.2 (4.2,4.3)*	6.1 (5.8,6.4)
Training volume (reps × sets)	17.0 (16.8,17.2)*	17.0 (16.9,17.1)*	24.4 (23.4,25.4)
RPE	16.0 (15.3,16.7)*	15.5 (14.9,16.2)*	17.0 (16.6,17.4)
Attendance (%)	99.3 (98.1,100)	98.8 (97.0,100)	99.1 (98.1,100)

Data are reported as means and 95% confidence intervals.
*Significantly different from control (C).
RPE, Rating of perceived exertion.

regimen required subjects to perform resistance exercise with a relative 85% 1RM load. Over the 12 weeks of training, no significant difference was observed in the average load lifted between groups. In contrast, and in line with the experimental design, the average number of repetitions completed per set throughout the 12-week regimen was significantly greater ($P < 0.0001$) in C compared with RS and SSC. Thus, the training volume (repetitions × sets) was significantly ($P < 0.0001$) lower in RS and SSC than C. In week 11, during kinematic assessment, the control group (26.5 s, CI: 25.5, 27.5) spent significantly greater time under elbow flexor tension compared with RS (13.2 s, CI: 12.5, 13.8) and SSC (8.0 s, CI: 7.3, 8.8). The significant difference in time under tension between the groups was explained by the marked increase in movement velocity (Fig. 2) in SSC than RS than C. Overall, participants reported significantly lower ($P = 0.0013$) ratings of perceived exertion when performing the RS and SSC training regimen compared with C. We observed similar levels of compliance to the training regimen between RS, SSC, and C, with 94% of the training sessions attended by participants.

Physiological adaptations to the training regimen

Despite the significant difference in the volume of the training stimulus, no significant difference in 1RM, MVC, MRI, or agonist EMG_{RMs} was observed between groups (Table 3). A significant ($P < 0.001$) 30.5% increase in pooled 1RM strength was observed over the 12-week training period and significant ($P < 0.001$) gains in 1RM strength were expressed throughout the training period with 11.4% (CI: 8.7, 14.2), 9.4% (CI: 7.2, 11.6), and 7.3% (CI: 5.1, 9.5) increase detected between weeks 1–4, 4–8, and 8–12, respectively (Fig. 3). Maximal voluntary contractile strength was similar at week 1 between SSC (93.9 Nm, CI: 69.8, 118), RS (91.4 Nm, CI: 81.6, 101.2), and C (80.9 Nm, CI: 61.8, 99.9), and increased by week 12 in SSC (105.1 Nm, CI: 79.3, 130.1), RS (103.6 Nm, CI: 83.9, 123.2), and C (90.3 Nm, CI: 72.4, 108.2) with no between-group interaction detected. Over the duration of the 12-week regimen a significant ($P = 0.003$) 13.3% (CI: 5.9, 20.7) increase in pooled MVC elbow flexor torque was observed. Similarly, no significant difference was observed in elbow flexor muscle CSA at week 1 SSC (14.4 cm^2, CI: 12.6, 16.7), RS (12.2 cm^2, CI: 10.8, 13.5), and C (13.0 cm^2, CI: 10.9, 15.0), and increased by week 12 in SSC (15.8 cm^2, CI: 13.8, 17.8), RS (13.4 cm^2, CI: 12.0, 14.8), and C (14.6 cm^2, CI: 12.9.7, 16.5) with no between-group interaction detected. Over the duration of the 12-week regimen, a significant ($P < 0.001$) pooled increase of 11.4% (CI: 8.7, 14.1) was observed in all participants in muscle CSA.

A significant interaction in antagonist EMG_{RMS} activity was observed ($P = 0.029$) with a relative increase observed in SSC and C, and a decrease in RS during the MVC assessment (Table 3). No significant between-group interaction was observed in 1RM flexor muscle activation; however, a significant ($P = 0.005$) 22.1% (CI: 5.9, 38.4) pooled increase in biceps brachii average

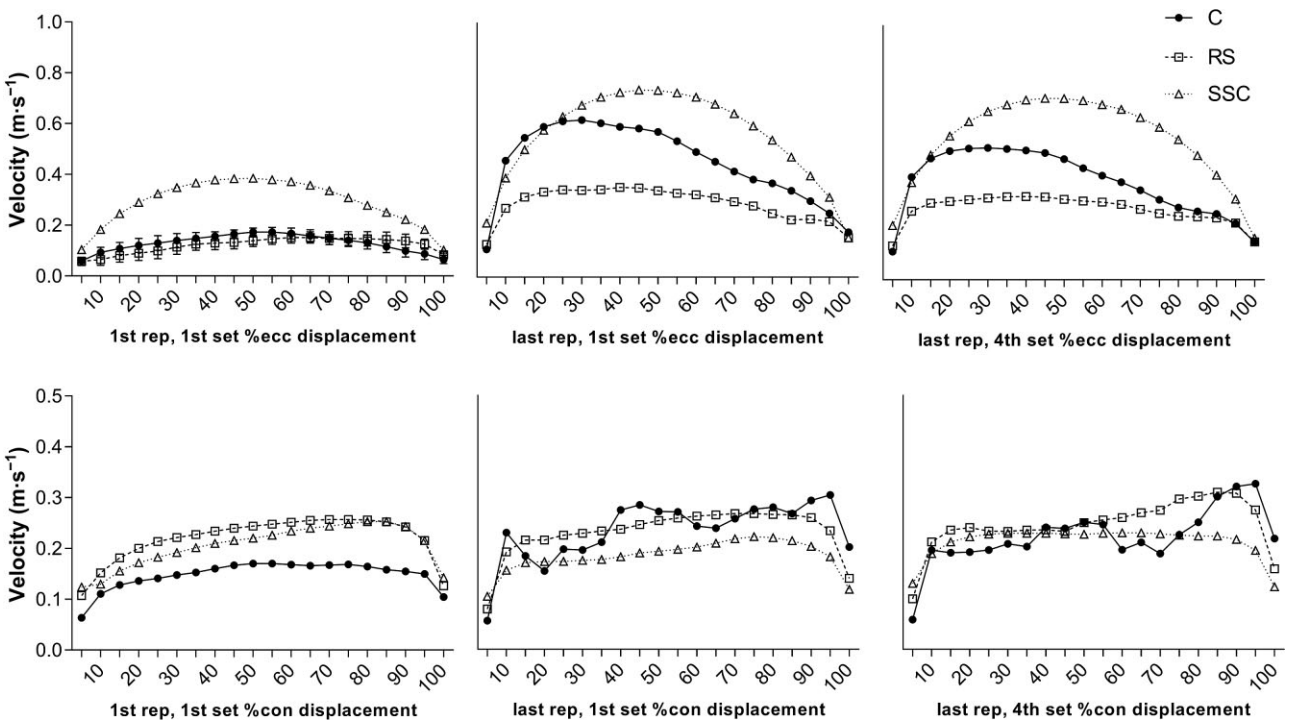

Fig. 2. Movement profiles depicting the average movement velocity ($m \cdot s^{-1}$) calculated and displayed as mean for every 5% of movement relative to displacement during lengthening and shortening muscle contractions within rapid shortening (RS; dotted line, squares), stretch-shortening cycle (SSC; dashed line, triangles), and control (C; solid line, circles) groups. Data were collected during the first and last repetitions of the first set of exercise performed in the final week of training.

Table 3. Change in one repetition maximum (1RM), maximal voluntary contraction (MVC), muscle cross-sectional area (CSA), raw (mV) and normalized (%) agonist and antagonist EMG_{RMS} activity in the rapid shortening (RS), stretch-shortening cycle (SSC), and control (C) groups over the 12-week experimental training period

Variable	Group		
	RS	SSC	C
1RM (%)	28.6 (23.6, 33.5)	32.8 (29.2, 36.4)	30.6 (22.1, 39.1)
MVC (%)	12.7 (–2.6, 28.0)	12.8 (2.7, 22.9)	14.3 (–2.0, 30.6)
CSA (%)	10.9 (7.4, 14.4)	7.1 (0.8, 13.5)	11.6 (5.7, 17.4)
1RM EMG_{RMS} (mV)			
Biceps pretraining	0.63 (0.49, 0.76)	0.62 (0.36, 0.88)	0.60 (0.51, 0.68)
Biceps post-training	0.69 (0.49, 0.89)	0.81 (0.64, 0.99)	0.69 (0.53, 0.84)
Triceps pretraining	0.07 (0.06, 0.09)	0.09 (0.06, 0.12)	0.08 (0.06, 0.09)
Triceps post-training	0.06 (0.05.0.08)	0.12 (0.08, 0.17)	0.09 (0.08, 0.09)
1RM EMG_{RMS} (%)			
Biceps	7.7 (–4.0, 19.3)†	47.0 (–4.5, 98.5)†	16.7 (–8.5, 41.9)†
Triceps	–13.45 (–29.3, 2.4)	40.5 (–7.6, 88.7)*	23.3 (2.0, 44.6)†*
MVC EMG_{RMS} (mV)			
Biceps pretraining	0.89 (0.60, 1.18)	1.19 (0.69, 1.70)	0.82 (.062, 1.03)
Biceps post-training	0.92 (0.68, 1.12)	1.33 (0.73, 1.93)	0.87 (0.59, 1.12)
Triceps pretraining	0.09 (0.07, 0.11)	0.11 (0.08, 0.14)	0.10 (0.08, 0.12)
Triceps post-training	0.07 (0.05, 0.08)	0.12 (0.07, 0.18)	0.10 (0.09, 0.12)
MVC EMG_{RMS} (%)			
Biceps	10.1 (–11.1, 31.2)	22.5 (–43.4, 88.6)	5.5 (–18.6, 29.7)
Triceps	–20.4 (–41.1, 0.35)	12.4 (–11.4, 36.2)*	8.0 (–9.2, 25.2)*

Data are reported as means and 95% confidence intervals.

*Significantly different from RS.

†Significant change over time.

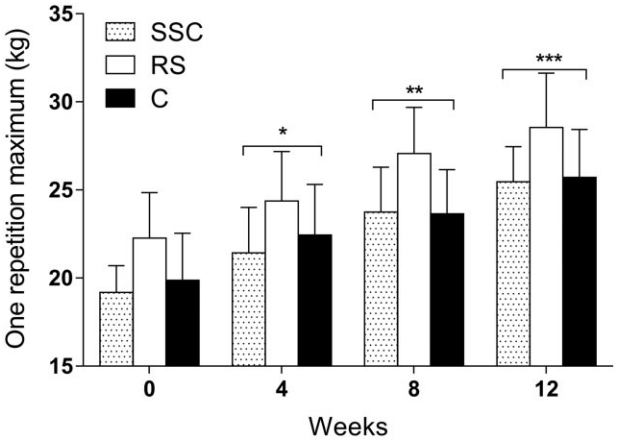

Fig. 3. Dominant limb one repetition maximum strength gain (kg) recorded in control (C), stretch-shortening cycle (SSC), and rapid shortening (RS) resistance exercise groups. Strength assessments performed at baseline (week 0), during (weeks 4 and 8), and following the 12-week training intervention (week 12). Data represent means and 95% confidence intervals. *Significant within-group difference from baseline. **Significant within-group difference from week 4. ***Significant within-group difference from week 8. Significance is set at $P < 0.05$.

EMG_{RMS} was observed over the 12-week regimen. Antagonist muscle activation also changed significantly ($P = 0.028$) during a single repetition maximum after 12 weeks of training. Similarly in MVC, a significant interaction was seen ($P = 0.016$) as triceps brachii average EMG_{RMS} amplitude increased following SSC and C, but declined following RS (Table 3). Shoulder stabilizers, anterior deltoid and upper trapezius showed no change in average EMG_{RMS} amplitude during 1RM assessments following the 12-week experimental training period, suggesting subjects successfully maintained shoulder joint stabilization throughout assessment and training.

Discussion

A 30% decrease in training volume and 90% reduction in the number of sets performed to repetition failure had no significant effect on gains in 1RM (~30%) and maximal voluntary force production (~15%) after 12 weeks of heavy 85% 1RM unilateral resistance exercise. Indeed, the regimen used within this investigation was very effective in developing elbow flexor strength when considered in light of a 17% increase observed in the preceding 4-week familiarization period (Rooney et al., 1994; Munn et al., 2005). These are interesting findings as they suggest that repetition failure and training volume may be of less importance for the development of muscle hypertrophy and strength when the characteristics of the muscle activation are manipulated.

Non-ballistic rapid elbow flexor movement is associated with a significant increase in muscle activation and near twofold increase in peak force (Sampson et al., 2014). It was this modification of transient tensile loading and muscle recruitment that this investigation sought to manipulate. It is well known that eccentric muscle activations are very effective in eliciting muscle hypertrophy (Higbie et al., 1996; Farthing & Chilibeck, 2003) and that these rapid or explosive movements are associated with the facilitation of skeletal muscle work (Komi & Bosco, 1978; Newton et al., 1997), declining motor unit recruitment thresholds (Desmedt & Godaux, 1977) and increased muscle activation (Newton et al., 1997; Sampson et al., 2014).

Our findings, with respect to the manipulation of resistance training volume, are supported by Munn et al. (2005) who observed no difference in 1RM strength gain in subjects that completed a single set of fast elbow flexor training than those subjects that completed three slower sets. The authors also observed that the training regimen utilizing a single set of slower joint speeds resulted in attenuated elbow flexor strength gains in comparison to other regimens (Munn et al., 2005). However, the investigators ensured all regimens were completed to repetition failure. The regimen utilized within the current investigation required two of the experimental groups (RS and SSC) to complete one set to repetition failure each week, in contrast to C who completed 12 sets to repetition failure.

Significant gains in muscular strength have been reported when resistance exercise is performed to repetition failure (Rooney et al., 1994; Drinkwater et al., 2005). These gains in muscle strength and hypertrophy have been found to be independent of exercise load and volume (Mitchell et al., 2012; Takada et al., 2012). Fatiguing bouts of resistance exercise are associated with increased metabolite accumulation, motor unit recruitment and endogenous hormone secretion, physiological signals that may contribute to muscle hypertrophy and enhanced force production capacity (Burd et al., 2010a, 2012). The hypertrophic response of skeletal muscle may be dependent on muscle fatigue with greater gains observed when higher volumes are applied (Mitchell et al., 2012). However, in this investigation, despite the relative absence of fatiguing bouts of resistance exercise to failure and significant reduction in the total exercise volume, our investigation detected no significant difference in agonist muscle CSA between the experimental groups after 12 weeks of resistance training.

Furthermore, an 11% change in muscle CSA observed within our investigation was consistent with the increase in muscle hypertrophy reported by other investigations using a similar training duration and loading strategy (Holm et al., 2008; Mitchell et al., 2012). Thus, the loading strategies adopted within this investigation did not compromise muscle hypertrophy. Given the unique experimental design adopted within this investigation, our findings suggest performing additional repetitions to failure maybe superfluous when the %1RM resistance training load is high. While other researchers have

assessed the effect of repetition failure upon dynamic and static force production capacity (Rooney et al., 1994; Folland et al., 2002; Izquierdo et al., 2006), this is the first investigation to our knowledge to also consider the effect of repetition failure upon muscle CSA. Significantly, these previous investigations had utilized a model that ensured total training volume was identical between the experimental groups (Rooney et al., 1994; Folland et al., 2002; Izquierdo et al., 2006). In contrast, the relative absence of repetition failure in RS and SSC within this investigation was deliberately used to reduce the training volume by over 30%, a decline that did not compromise structural or functional changes in the elbow flexor skeletal muscles. Thus, while our experimental design cannot confirm if the rapid muscle activations associated with RS and SSC regimen lead to an enhanced sensitivity to the training stimulus, they are encouraging and suggest further investigation is warranted.

Significant gains in strength within the first 4 weeks of resistance training are apportioned to neurally mediated adaptation (Moritani & DeVries, 1979). In this investigation, a 22% increase in biceps brachii muscle activity was observed during dynamic 1RM assessment in all three groups. Thus, although all subjects had participated in a 4-week familiarization period where significant neural adaptation was observed (Sampson et al., 2013), significant improvements in agonist muscle activation continued to occur during the subsequent 12-week regimen. These adaptations explain much of the 30% improvement observed in 1RM elbow flexor strength within our 12-week training regimen. However, no change in agonist muscle activation was observed during maximal isometric elbow flexor force production, suggesting changes observed in muscle activation were specific to those utilized within the training regimen (Higbie et al., 1996). However, we did observe a significant increase in antagonist muscle activation in SSC and C during dynamic assessment of strength. An absolute or relative decline in antagonist muscle activation is typically observed during 1RM and MVC assessments (Andersen et al., 2005; Tillin et al., 2011) following resistance training. The increase in antagonist activity may be due to increased co-contraction permitting improved joint stability for the development of force (Hagood et al., 1990; Bennett, 1993). However, we observed no change in anterior deltoid or upper trapezius muscle activation, suggesting changes in co-contraction where isolated only to the elbow joint and not more broadly to stabilization of the shoulder complex.

One of the challenges of inter-subject experimental designs is accounting for the large heterogeneity in responsiveness to resistance training within the sample population (Hubal et al., 2005). Some authors have suggested given the magnitude of this biological variance intra-subject designs should be utilized as an experimental design alternative (Folland et al., 2002). We adopted a different approach. This investigation acknowledged *a priori* the heterogenic nature of adaptive responsiveness to resistance training by incorporating a 4-week familiarization period prior to the 12-week regimen. Uniquely, this investigation also counterbalanced subjects to C, RS and SSC on the basis of 1RM strength gain (responsiveness) obtained during the familiarization period. While a significant difference was observed in 1RM strength gain in higher and lower responders during the familiarization period, no significant difference was observed during the subsequent 12-week training regimen, demonstrating the effectiveness of allocating subjects to experimental groups on the basis of responsiveness to a familiarization period (Sampson et al., 2013).

In conclusion, strength gains following a 12-week resistance training regimen are not dependent on repetitions performed to failure, nor in such conditions, is it necessary to equalize the training volume. Similar skeletal muscle adaptations can be gained with rapid muscle activation in the absence of repetition failure and a concurrent reduction in the total exercise volume.

Perspectives

Repetition failure is considered an essential characteristic of resistance training for over 70 years (Delorme, 1945), with more recent evidence supporting this view (Rooney et al., 1994; Drinkwater et al., 2005). However, others have observed equivalent strength gain when exercise volume were matched in repetition failure and non-failure resistance training regimen (Folland et al., 2002; Izquierdo et al., 2006). This investigation has shown that a reduced volume, non-failure resistance training regimen can elicit equivalent gains in strength, muscle activation, and muscle CSA than increased training volume regimen to failure. Rapid muscle activation may be the distinguishing feature that lead to similar adaptive changes in muscle structure and function despite marked differences in resistance training volume (Munn et al., 2005). However, the current research design cannot confirm this outcome particularly for compound multi-joint movements. Thus, further investigations appear warranted to determine the influence of rapid muscle activation, repetition failure, and resistance training volume.

Key words: Resistance training, fatigue, electromyography, velocity, one repetition maximum, neural.

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