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Original Article

Four weeks of high- versus low-load resistance training to failure on the rate of torque development, electromechanical delay, and contractile twitch properties

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Abstract

The purpose of this study was to investigate the effects of 4-weeks of high- versus low-load resistance training to failure on rate of torque development (RTD), electromechanical delay (EMD), and contractile twitch characteristics. Fifteen men (mean±SD; age=21.7±2.4 yrs) were randomly assigned to either a high- (80% 1RM; n=7) or low-load (30% 1RM; n=8) training group and completed elbow flexion resistance training to failure 3 times per week for 4 weeks. The participants were tested at baseline, 2-, and 4-weeks of training. Peak RTD ($pRTD_V$) and RTD at 0-30 ($RTD30_V$), 0-50 ($RTD50_V$), 0-100 ($RTD100_V$), and 0-200 ($RTD200_V$) ms, integrated EMG amplitude (iEMG) at 0-30, 0-50, and 0-100 ms, and EMD were quantified during maximal voluntary isometric muscle actions. Peak twitch torque, peak RTD, time to peak twitch, 1/2 relaxation time and the peak relaxation rate were quantified during evoked twitches. Four weeks of high-load, but not low-load resistance training, increased $RTD200_V$. There were also increases in iEMG during the first 30 ms of muscle activation for the high- and low-load groups, which may have indirectly indicated increases in early phase motor unit recruitment and/or firing frequency. There were no significant training-induced adaptations in EMD or contractile twitch properties.

Keywords: Electromyography, Rapid Torque Production, Skeletal Muscle

Introduction

Several recent studies[1-5] have challenged the current recommendation that resistance exercise loads of 60-85% of the one repetition maximum (1RM) are optimal for maximizing muscle hypertrophy[6,7]. For example, Burd and colleagues[1] observed similar muscle protein synthetic and anabolic signaling responses following resistance exercise to failure at 30% versus 90% 1RM. In a follow up study, Mitchell et al.[2] demonstrated that 10 weeks of leg extension resistance

training to failure at 30% 1RM and 80% 1RM were equally effective for causing muscle hypertrophy. Similarly, Ogasawara et al.[3] observed comparable muscle hypertrophy in response to 30% and 80% 1RM bench press resistance training to failure in the pectoralis major and triceps brachii. These recent experimental results have sparked a debate regarding the recommended resistance-training load to augment muscle size[8,9].

Despite the similar hypertrophic adaptations to high- versus low-load training, several studies have shown that high-load training is superior for enhancing muscle strength[2-5]. Mitchell et al.[2] demonstrated that 10 weeks of training at 80% 1RM increased 1RM strength to a greater degree than training at 30% 1RM, although both intensities increased maximal voluntary isometric contraction (MVIC) strength to a similar degree. Ogasawara et al.[10], however, observed greater improvements in both 1RM and MVIC strength following training at 80% versus 30% 1RM. In a 4 week training study, Jenkins et al.[5] also observed similar hypertrophy of the elbow flexors following training at 80% versus 30%

1RM. However, MVIC and 1RM strength significantly improved in the 80% 1RM group, but did not significantly improve in the 30% 1RM group. Therefore, studies are needed to help understand the neuromuscular adaptations that may facilitate strength improvements following high- but not low-load training, even though they may enhance muscle size to a similar degree[2,5,10].

The rate of torque development (RTD), calculated during the onset of a maximal isometric muscle action, is thought to provide important information regarding neural and mechanical adaptations to training dependent on the time interval in which it is calculated[11-13]. Specifically, RTDs calculated in early time intervals may provide information regarding neuromuscular activation characteristics or contractile speed, while RTDs calculated during later time intervals may be more related to maximal strength[12-15]. Therefore, an examination of RTD and EMG during the onset of torque production and muscle activation can provide information about the neuromuscular adaptations that occur in response to resistance training at 80% versus 30% 1RM.

The electromechanical delay (EMD) is the time lag between the onset of electrical activity in a muscle and the onset of a measureable torque response[16-18]. Although physiological factors such as the propagation of action potentials along the sarcolemma and excitation-contraction coupling may influence the EMD, it has been suggested that the time required to stretch the series elastic component (SEC) represents the major portion of the measured EMD[17]. Consequently, the EMD has been used as an indicator of musculotendinous stiffness[19,20]. For example, Grosset et al.[19] and Kubo et al.[21] demonstrated changes in EMD with concurrent changes in musculotendinous stiffness following endurance, plyometric, and/or isometric training. Kubo et al.[21] suggested that training-induced increases in musculotendinous stiffness are an "advantage for increasing the RTD and shortening the EMD" (Table 3). However, Malliaras et al.[22] demonstrated that changes in tendon stiffness following resistance training may be load-dependent. Consequently, the EMD may provide information regarding load-dependent adaptations related to musculotendinous stiffness.

It has been hypothesized[23,24] that there are fiber specific adaptations to high- versus low-load training. For example, Mitchell et al.[2] reported a (non-significant) 7% greater increase in type I fiber size of the vastus lateralis (VL) after 10 weeks of 30% versus 80% 1RM resistance training. Netreba et al.[25] showed a greater increase in type II fiber size of the VL following 8 weeks of resistance training at 85% versus 25% 1RM. It is known that fibers with different myosin isoform contents display different functional properties[26-28]. Accordingly, examination of a muscle or muscle group's contractile twitch properties may provide insight into the specific adaptations that occur following high- versus low-load resistance training programs.

Together, the quantification of RTD, EMD, and contractile twitch properties may provide information on the neuromuscular adaptations that are facilitating greater strength improvements during high- versus low-load training. There-

fore, the purpose of this study was to investigate the effects of 4-weeks of high- (80% 1RM) versus low-load (30% 1RM) resistance training on voluntary RTD, voluntary EMD, and contractile twitch characteristics in untrained men. We hypothesized that there would be greater changes in RTD, EMD, and time-dependent contractile twitch properties (i.e., RTD, time to peak torque, etc.) in response to training at 80% 1RM, but that peak twitch torque would increase similarly following training at 80% and 30% 1RM[29].

Materials and methods

Participants

Eighteen untrained men were enrolled in this study; however, 3 men did not complete this study for the following reasons: 1 participant did not wish to continue the study due to discomfort during the testing sessions, 1 participant did not wish to continue the study due to the time commitment, and 1 participant withdrew to begin a resistance training program outside of the study. Therefore, only the data from 15 men (mean±SD; age = 21.7±2.4 yrs; height= 181.6±7.5 cm; weight= 84.7±23.5 kg) were analyzed and reported. This study was approved by the university's Institutional Review Board for the protection of human participants (IRB Approval #: 20140314046FB). Prior to any data collection, all participants signed an informed consent form and completed a health history questionnaire. To be eligible, each participant must have been between the ages of 19 and 29, free from any current or ongoing musculoskeletal injuries or neuromuscular disorders involving the shoulders, elbows, or wrists, and could not have completed any regular or formal resistance training for at least 6 months prior to the start of the study.

Experimental design

A randomized, between-group, repeated measures, parallel design was used for this study. Participants were randomly assigned to either a high- (80% of 1RM; n = 7) or low-load (30% of 1RM; n = 8) resistance training group and completed elbow flexion resistance training to failure 3 times per week for 4 weeks. The participants were familiarized with the testing procedures prior to baseline testing, and testing was completed at baseline, 2-, and 4-weeks of training. All participants completed a total of fourteen visits, and each visit was separated by 48–72 hours at the same time of day (±2 h). During each testing session, participants completed maximal voluntary and evoked muscle actions, during which torque and electromyographic (EMG) signals were recorded. The participants were asked to refrain from any outside resistance exercise for the duration of the study.

Resistance training

During all 11 training visits, subjects completed 3 sets of dynamic constant external resistance elbow flexion resistance training (e.g., dumbbell biceps curls) to failure with loads corresponding (to the nearest 1.1 kg) to either 80% or 30%

of 1RM. The subjects stood with their backs against a wall and their elbows supported by a brace (Bicep Bomber, Body Solid, Inc., Forest Park, IL, USA) to eliminate swinging of the torso or arms. Subjects were instructed to perform all repetitions through a complete range of motion. A metronome (Pro Metronome, EUMLab, Berlin, Germany) was set to 1 Hz, and subjects were instructed to perform the concentric and eccentric phases corresponding with each tick of the metronome so that the concentric and eccentric phases were approximately 1 s. Verbal instruction and encouragement were provided during each set. Failure was defined as the inability to complete another concentric muscle action through the full range of motion. Two min of rest was provided between sets for both conditions (80% and 30% 1RM). The weight utilized during training was adjusted based on the new 1RM established at the 2 week testing session. Because it has been suggested that the timing and type of protein ingested surrounding resistance training may augment the magnitude and duration of the muscle protein synthetic response to training[30], each participant consumed a protein shake mixed with water in the laboratory (EAS 100% Whey Protein, EAS Sports Nutrition, Abbott Laboratories, Columbus, OH, USA) that provided 150 kcals and 26 g of protein immediately following each resistance training session.

Isometric testing

For isometric testing, the participants were seated with straps securing the trunk and pelvis on a calibrated isokinetic dynamometer (Biodex System 3; Biodex Medical Systems, Inc. Shirley, NY, USA) with a custom-built apparatus (Omega-dyne, model LC402, range 0–500 lbs, Stamford, CT, USA). The participants' wrists were secured with a velcro strap, the axis of rotation of the dynamometer head was aligned with the axis of rotation of the elbow joint, and the arm was positioned in 10° of abduction to better expose the musculotaneous nerve for transcutaneous nerve stimulation. The joint angle between the arm and the forearm was set at 90°, which was used for both voluntary and evoked isometric muscle actions.

Transcutaneous electrical stimuli were delivered via bipolar surface electrodes placed over the musculotaneous nerve just medial to the anterior deltoid using a high voltage (maximal voltage= 400 V), constant-current stimulator (Digitimer DS7AH, Hertfordshire, UK). Optimal stimulation electrode location was determined by delivering single low-amperage exploratory stimuli (20 mA) using a hand-held stimulation probe (Digitimer Bipolar Felt Pad Electrodes). Electrode location was selected based on visual inspections of the twitch force and the compound muscle action potential (M-wave) amplitudes that were displayed on an external computer screen. Once the location was determined and marked, disposable 20 mm diameter adhesive surface electrodes (Plaquette Disposable 4-Disk Electrodes, Technomed Europe, the Netherlands) were taped to the skin with an interelectrode distance of 25.4 mm (distance between the anode and cathode of the hand-held probe). Maximal peak-to-peak M-wave amplitude (M_{PP}) was achieved by increasing amperage in 20-40 mA increments until a plateau in M_{PP} and twitch force was observed after three consecutive amperage increases. To ensure a supramaximal stimulus, 120% of the stimulus used to evoke the maximal M_{PP} was used to evoke the elbow flexor muscles with 1 singlet and doublet stimuli (200 ms duration square-wave impulse at 100 Hz) with 1 minute of rest between each stimulus.

Participants completed 2, 4–5 s MVICs of the elbow flexors with 2 min of rest between each muscle action. For each attempt, subjects were instructed to contract as "fast and hard as possible" when the investigator said "go!" Loud verbal encouragement was provided during each MVIC.

Electromyography

Pre-gelled bipolar surface electrodes (Ag/AgCl, AccuSensor, Lynn Medical, Wixom, MI, USA) were placed on the biceps brachii (BB) muscle of the right arm with an inter-electrode distance of 30 mm. The center of the bipolar electrode pair was placed at 33% of the distance between the cubital fossa and the acromion process[31]. A single pre-gelled surface electrode (Ag/AgCl, AccuSensor, Lynn Medical, Wixom, MI, USA) was placed on the lateral epicondyle of the humerus to serve as the reference electrode. All electrode locations were marked with a permanent marker and were kept throughout the duration of the study. To reduce inter-electrode impedance and increase the signal-to-noise ratio[32], local areas of the skin were shaved, abraded, and cleaned with isopropyl alcohol prior to the placement of the electrodes. Interelectrode impedance was measured using a digital multimeter (Fluke 179 True RMS Multimeter, Everett, WA, USA) and was kept below 2000 Ω[32].

Signal processing

The torque and EMG signals were sampled simultaneously at 2kHz with a BIOPAC data acquisition system (MP150WSW, Biopac Systems, Inc., Santa Barbara, CA, USA). The signals were recorded and stored on a personal computer and processed off-line with custom written software (LabVIEW 12.0, National Instruments, Austin, TX, USA).

The torque signals were low-pass filtered with a 20 Hz cutoff (zero-phase shift 4th-order Butterworth filter) and all analyses were completed on the filtered signals. For the voluntary muscle actions, RTDs were quantified as averages of the first derivative (i.e., instantaneous slopes) of the torque signal in time intervals of 0-30 ($RTD30_V$), 0-50 ($RTD50_V$), 0-100 ($RTD100_V$), and 0-200 ($RTD200_V$) ms from the onset of torque production, and peak RTD ($pRTD_V$) was calculated as the highest 10 ms average of the first derivative of the torque signal[12,33].

As described previously[34], contractile twitch properties were calculated from the evoked singlet (denoted by a subscript 'S') and doublet (denoted by a subscript 'D') muscle actions. Specifically, peak twitch torque (PTT) was calculated as the highest 2.5 ms torque value (Nm) obtained after the onset of the evoked twitch. Peak RTD (pRTD) was calculated

as the highest 2.5 ms average of the first derivative of the torque signal ($Nm·s^{-1}$) between the onset of the evoked twitch and PTT. Time to peak twitch (TPT) was calculated as the time (ms) from the onset of the evoked twitch to PTT. The 1/2 relaxation time (HRT) and the peak relaxation rate (pRR) were calculated as the time (ms) from PTT to 1/2 of PTT and as the lowest 2.5 ms average of the first derivative of the torque signal ($Nm·s^{-1}$) after the attainment of PTT, respectively.

The EMG signals were amplified (gain 1000) using a differential amplifier (EMG100C, Biopac Systems, Inc., Santa Barbara, CA, USA, bandwidth 1–5000 Hz) with a common mode rejection ratio of 110 dB min and an input impedance of 2 MΩ, sampled at 2 kHz, and digitally filtered (zero phase-shift 4th-order Butterworth) with a bandpass of 10-999 Hz for the voluntary and evoked muscle actions. For the voluntary muscle actions, the EMG signals were full-wave rectified, and the time-averaged integrated EMG amplitude (μV)[35] was calculated during the first 0-30 (iEMG30), 0-50 (iEMG50), and 0-100 (iEMG100) ms relative to the onset of EMG activity[12]. During the evoked singlet and doublet muscle actions, the M-wave amplitude was quantified as the peak-to-peak amplitude (M_{PP}) in μV. The M-wave duration was quantified as the time (ms) from the onset to cessation of the M-wave. For the voluntary and evoked muscle actions, the electromechanical delay (EMD_V and $EMD_{S/D}$, respectively) was calculated as the time (ms) from the onset of the EMG signal to the onset of torque production.

The onsets of the voluntary and evoked torque and EMG signals were determined manually via visual inspection of the filtered torque and EMG signals where they first deflected from the baseline when viewed in a 20 ms window that provided a precise visual illustration[20,33,36]. All signal onsets were determined using custom written software (LabView 12.0, National Instruments, Austin, TX, USA).

Statistical analyses

Twenty-five two-way mixed factorial analyses of variance (ANOVAs) (time [Baseline vs. Week 2 vs. Week 4] x group [80% 1RM vs. 30% 1RM]) were used to analyze $pRTD_V$, $RTD30_V$, $RTD50_V$, $RTD100_V$, $RTD200_V$, EMD_V, iEMG30, iEMG50, iEMG100, PTT_S, $pRTD_S$, TPT_S, pRR_S, HRT_S, EMD_S, M_{PPS}, M_{DURS}, PTT_D, $pRTD_D$, TPT_D, pRR_D, HRT_D, EMD_D, M_{PPD}, and M_{DURD}. Partial eta squared (η_p^2) and Cohen's *d* effect sizes (*d*) were calculated for each ANOVA and t-test, respectively. Significant interactions were decomposed with follow-up repeated measures ANOVAs and dependent and/or independent samples t-tests on the simple main effects. Significant main effects that were not involved in an interaction were analyzed with dependent samples t-tests on the marginal means. All statistical analyses were completed using IBM SPSS Statistics (v. 22; Armonk, NY) and a type-I error rate was set *a priori* at 5%.

Test-retest reliability for $pRTD_V$, $RTD30_V$, $RTD50_V$, $RTD100_V$, $RTD200_V$, EMD_V, iEMG30, iEMG50, iEMG100, PTT_S, $pRTD_S$, TPT_S, pRR_S, HRT_S, EMD_S, M_{PPS}, and M_{DURS} were assessed from familiarization to baseline. Repeated measures ANOVAs were used to assess systematic error, and model
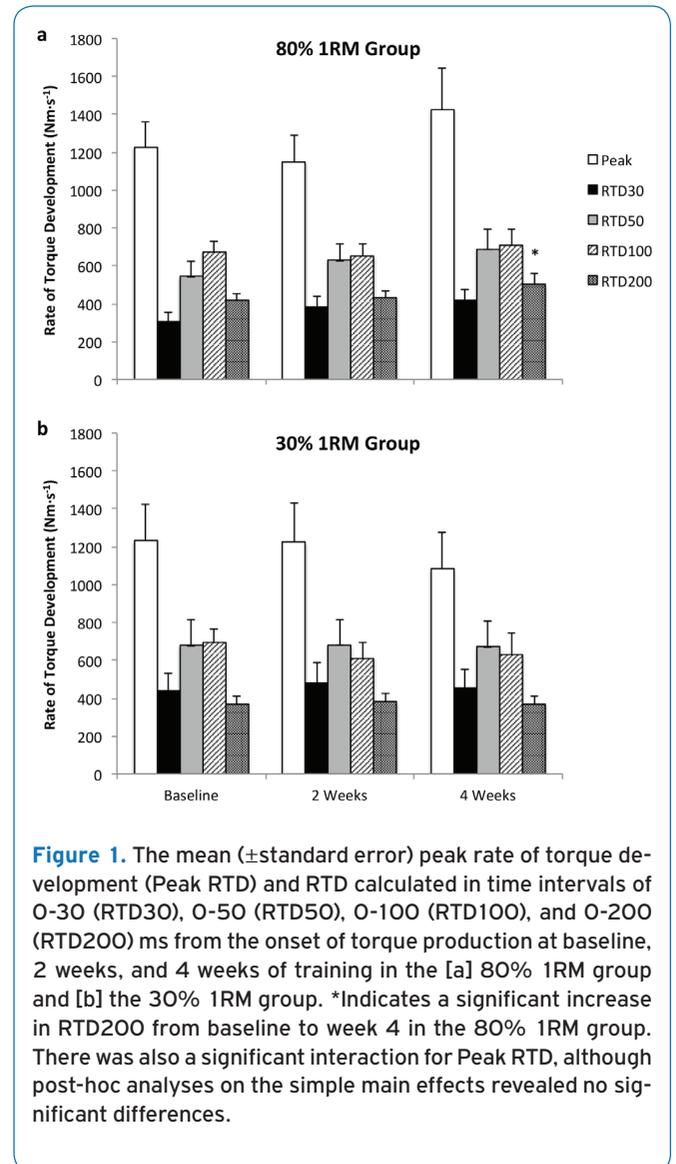

Figure 1. The mean (±standard error) peak rate of torque development (Peak RTD) and RTD calculated in time intervals of 0-30 (RTD30), 0-50 (RTD50), 0-100 (RTD100), and 0-200 (RTD200) ms from the onset of torque production at baseline, 2 weeks, and 4 weeks of training in the [a] 80% 1RM group and [b] the 30% 1RM group. *Indicates a significant increase in RTD200 from baseline to week 4 in the 80% 1RM group. There was also a significant interaction for Peak RTD, although post-hoc analyses on the simple main effects revealed no significant differences.

2,k[37,38] was used to calculate intraclass correlation coefficients (ICCs) and standard errors of measurement (SEMs). The SEMs were expressed as a percentage of the grand mean and were reported as coefficients of variation (CV)[39]. The 95% confidence intervals for the ICCs were calculated according to the procedure described by Shrout and Fleiss[37] in order to test whether each ICC was greater than zero[33].

Results

The range of repetitions completed in the first training session during sets 1, 2, and 3 were 37–58, 17–28, and 15–29 repetitions, respectively in the 30% 1RM group and 8–15, 7–11, and 2–8 repetitions, respectively in the 80% 1RM group. The range of repetitions completed in the last training session during sets 1, 2, and 3 were 41–97, 22–49, and

Figure 2. Mean (±standard error) voluntary electro-mechanical delay at baseline, 2 weeks, and 4 weeks, in the 80% and 30% 1RM groups.
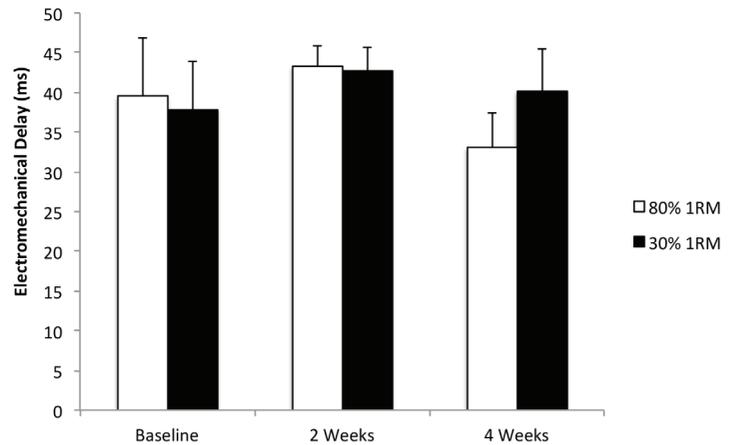
Figure 3. Mean (±standard error) voluntary electromyographic (EMG) amplitude integrated in time intervals of 0-30 (iEMG30), 0-50 (iEMG50), and 0-100 (iEMG100) ms after the onset of EMG activity collapsed across the 80% and 30% 1RM groups. *Indicates that iEMG30 is significantly greater at weeks 2 and 4 than at baseline.
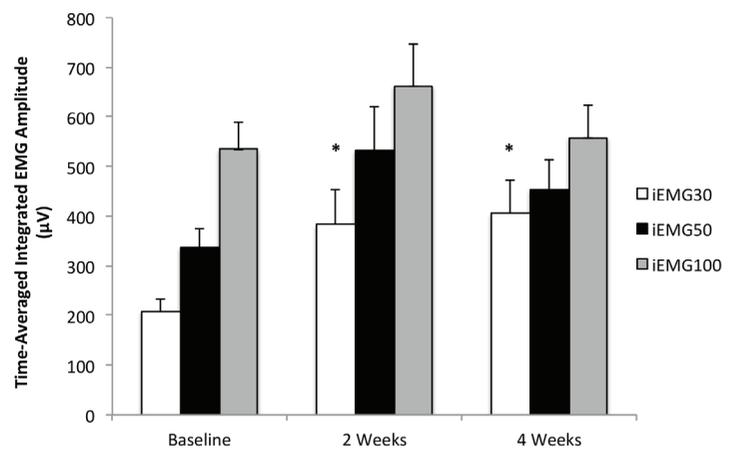
17−46 repetitions, respectively in the 30% 1RM group and 6−18, 5−14, and 5−10 repetitions, respectively in the 80% 1RM group.

Figure 1 illustrates the means (± standard errors) for voluntary RTD at baseline, week 2, and week 4 in the 80% and 30% 1RM training groups. There were no group × time interactions (p=0.16−0.68; (η_p^2)=0.03−0.13) or main effects for time (p=0.38−0.53; (η_p^2)=0.05−0.07) or group (p=0.34−0.90; (η_p^2)=<0.01−0.06) for RTD30$_V$, RTD50$_V$, or RTD100$_V$. However, there were group × time interactions for pRTD$_V$ (p=0.02; (η_p^2)=0.27) and RTD200$_V$ (p=0.04; (η_p^2)=0.22). Post-hoc analyses revealed no significant changes across time for pRTD$_V$ in either the 80% (p=0.11; (η_p^2)=0.28) or 30% 1RM (p=0.06; (η_p^2)=0.37) groups, nor any significant differences between groups (p>0.05) at baseline (d=0.02), week 2 (d=0.15), or week 4 (d=0.62). RTD200$_V$ did not change from baseline to week 2 (d=0.46), but increased from baseline to week 4 (d=1.11) in the 80% 1RM group (Figure 1a). In contrast, RTD200$_V$ did not change from baseline to week 2 (d=0.04) or week 4 (d=0.22) in the 30% 1RM group (Figure 1b).

For EMD, there was no group × time interaction (p=0.62; n=0.04) or main effects for time (p=0.40; (η_p^2)=0.07) or group (p=0.72; (η_p^2)=0.01) (Figure 2). For iEMG30, there was no group × time interaction (p=0.95; (η_p^2)<0.01) or main effect for group (p=0.54; (η_p^2)=0.03), but there was a main effect for time (p=0.03; (η_p^2)=0.24). iEMG30 was greater at weeks 2 (d=0.91) and 4 (d=0.85) than at baseline (Figure 3). There were, however, no group × time interactions (p=0.86-0.96; (η_p^2)=<0.01-0.01), main effects for time (p=0.07−0.26; (η_p^2)=0.10-0.18), or main effects for group (p=0.44-0.56; (η_p^2)=0.03-0.05) for iEMG50 or iEMG100.

Table 1 displays the means (± standard errors) for the contractile characteristics calculated during the evoked singlet twitches at baseline, week 2, and week 4. There were no group × time interactions (p=0.31−0.88; (η_p^2)=0.01−0.09), main effects for time (p=0.16−0.81; (η_p^2)=0.02−0.13), or main effects for group (p=0.11−0.81; (η_p^2)=<0.01−0.19) for PTT$_S$, pRTD$_S$, TPT$_S$, pRR$_S$, HRT$_S$, EMD$_S$, M$_{PPS}$, M$_{DURS}$.

Table 2 displays the means (± standard errors) for the contractile characteristics calculated during the evoked dou-

Table 1. The mean (±standard error) evoked singlet twitch characteristics at baseline, week 2, and week 4 in the 80% 1RM and 30% 1RM groups.

		Baseline	Week 2	Week 4
80% 1RM	PTT (Nm)	10.3 (±1.7)	10.8 (±2.8)	11.0 (±2.1)
	pRTD (Nm·s⁻¹)	298.0 (±72.4)	328.3 (±91.4)	338.0 (±62.1)
	TPT (ms)	65.3 (±3.1)	62.3 (±4.4)	61.4 (±3.2)
	pRR (Nm·s⁻¹)	-187.1 (±40.7)	-196.0 (±56.5)	-193.8 (±33.8)
	HRT (ms)	45.9 (±7.9)	61.3 (±17.6)	39.1 (±5.2)
	EMD (ms)	7.6 (±1.7)	7.8 (±2.4)	7.1 (±2.7)
	M_{PP} (μV)	11810.1 (±1554.5)	11747.2 (±931.8)	11924.2 (±1455.5)
	M_{Dur} (ms)	30.2 (±2.2)	30.2 (±1.6)	26.3 (±2.4)
30% 1RM	PTT (Nm)	10.1 (±1.2)	9.4 (±1.5)	10.5 (±1.9)
	pRTD (Nm·s⁻¹)	263.3 (±24.2)	276.0 (±50.3)	311.4 (±53.1)
	TPT (ms)	79.4 (±11.4)	75.6 (±5.6)	63.0 (±4.2)
	pRR (Nm·s⁻¹)	-118.7 (±6.3)	-122.7 (±29.5)	-152.3 (±31.8)
	HRT (ms)	62.7 (±13.9)	55.9 (±6.0)	63.1 (±19.2)
	EMD (ms)	8.9 (±1.4)	5.6 (±1.1)	6.9 (±1.1)
	M_{PP} (μV)	10245.8 (±1638.8)	10604.9 (±1589.9)	11401.5 (±1647.5)
	M_{Dur} (ms)	27.0 (±3.0)	28.3 (±1.9)	28.2 (±1.8)

PTT = peak twitch torque; pRTD = peak rate of torque development; TPT = time to peak twitch torque; pRR = peak relaxation rate; HRT = half relaxation rate; EMD = electromechanical delay; M_{PP} = peak-to-peak M-wave amplitude; M_{Dur} = M-wave duration.

Table 2. The mean (±standard error) evoked doublet twitch characteristics at baseline, week 2, and week 4 in the 80% 1RM and 30% 1RM groups.

		Baseline	Week 2	Week 4
80% 1RM	PTT (Nm)	19.7 (±4.8)	20.3 (±7.3)	21.1 (±5.5)
	pRTD (Nm·s⁻¹)	544.4 (±16.6)	636.3 (±258.7)	574.0 (±145.4)
	TPT (ms)	54.2 (±2.3)	54.7 (±4.1)	56.1 (±2.3)
	pRR (Nm·s⁻¹)	-303.4 (±43.8)	-319.4 (±77.3)	-339.9 (±49.5)
	HRT (ms)	55.1 (±13.3)	44.5 (±6.4)	43.9 (±5.0)
	EMD (ms)	6.9 (±0.8)	5.6 (±0.8)	5.2 (±0.6)
	M_{PP} (μV)	13656.7 (±1301.8)	13366.5 (±832.5)	13114.0 (±1220.8)
	M_{Dur} (ms)	32.0 (±1.7)	28.9 (±1.8)	30.3 (±1.9)
30% 1RM	PTT (Nm)	15.1 (±1.5)	13.0 (±1.4)	14.5 (±1.5)
	pRTD (Nm·s⁻¹)	404.8 (±33.3)	379.2 (±34.8)	422.7 (±32.9)
	TPT (ms)	59.1 (±5.9)	57.7 (±3.4)	53.2 (±2.0)
	pRR (Nm·s⁻¹)	-179.5 (±21.3)	-179.4 (±31.3)	-192.5 (±26.2)
	HRT (ms)	67.4 (±12.0)	69.3 (±12.4)	53.8 (±9.7)
	EMD (ms)	6.4 (±0.9)	5.1 (±1.4)	4.9 (±1.0)
	M_{PP} (μV)	9796.1 (±1779.4)	11998.8 (±1771.6)	12586.1 (±1332.3)
	M_{Dur} (ms)	28.1 (±2.8)	28.2 (±2.0)	28.7 (±2.0)

PTT = peak twitch torque; pRTD = peak rate of torque development; TPT = time to peak twitch torque; pRR = peak relaxation rate; HRT = half relaxation rate; EMD = electromechanical delay; M_{PP} = peak-to-peak M-wave amplitude; M_{Dur} = M-wave duration.

Table 3. The reliability statistics (intraclass correlation coefficient [ICC], standard error of measurement [SEM], and coefficient of variation [CV]) for voluntary RTD, EMG, and muscle activation, and the evoked singlet contractile characteristics.

		Grand Mean	ICC	SEM	CV (%)
Voluntary	pRTD (Nm·s⁻¹)	1264.3	0.76	203.8	16.1
	RTD30 (Nm·s⁻¹)	402.1	0.65	139.4	34.7
	RTD50 (Nm·s⁻¹)	635.7	0.76	156.8	24.7
	RTD100 (Nm·s⁻¹)	667.8	0.55	128.1	19.2
	RTD200 (Nm·s⁻¹)	392.6	0.68	54.8	14.0
	EMD (ms)	38.5	0.74	7.8	20.4
	iEMG30 (µV)	202.1	0.76	41.3	20.4
	iEMG50 (µV)	342.5	0.75	70.6	20.6
	iEMG100 (µV)	558.5	0.81	88.3	15.8
Evoked	PTT (Nm)	10.1	0.92	1.2	11.9
	pRTD (Nm·s⁻¹1)	284.6	0.88	49.5	17.4
	TPT (ms)	71.3	0.64	11.3	15.9
	pRR (Nm·s⁻¹)	-147.6	0.91	25.1	17.0
	HRT (ms)	56.3	0.85	10.5	18.7
	EMD (ms)	8.1	0.70	2.5	31.3
	M_{PP} (µV)	10768.8	0.88	1672.2	15.5
	M_{Dur} (ms)	28.4	0.91	2.3	8.0

pRTD = peak rate of torque development; RTD30 = rate of torque development from 0 -30 ms; RTD50 = rate of torque development from 0 − 50 ms; RTD100 = rate of torque development from 0 − 100 ms; RTD200 = rate of torque development from 0 − 200 ms; EMD = electro-mechanical delay; iEMG30 = time-averaged integrated EMG amplitude from 0 - 30 ms; iEMG50 = time-averaged integrated EMG amplitude from 0 − 50 ms; iEMG100 = time-averaged integrated EMG amplitude from 0 − 100 ms; PTT = peak twitch torque; TPT = time to peak twitch torque; pRR = peak relaxation rate; HRT = half relaxation rate; M_{PP} = peak-to-peak M-wave amplitude; M_{Dur} = M-wave duration.

blet twitches at baseline, week 2, and week 4. There were no group × time interactions (p=0.16−0.99; (η_p^2)=<0.01−0.13), main effects for time (p=0.11−0.87; (η_p^2)=0.01−0.16), or main effects for group (p=0.17−0.66; (η_p^2)=0.02−0.14) for PTT_D, $pRTD_D$, TPT_D, HRT_D, EMD_D, M_{PPD}, M_{DURD}. For pRR_D there was no group × time interaction (p=0.88; (η_p^2)=0.01) or main effect for time (p=0.56; (η_p^2)=0.04), but there was a main effect for group (p=0.04; (η_p^2)=0.29). The pRR_D was greater (d=1.31) in the 80% than the 30% 1RM group.

There was no systematic variability from familiarization to baseline for any of the variables (p>0.05). All of the ICCs were significantly greater than zero (p<0.05) according to the 95% confidence intervals. Table 3 displays the reliability statistics for the voluntary RTD, EMG, and muscle activation, as well as the evoked singlet contractile characteristics.

Discussion

To our knowledge, this was the first study to investigate the effects of high- versus low-load resistance training on RTD, EMD, and contractile twitch properties. The primary findings of the present study were that: (1) RTD200ᵥ increased from baseline to week 4 in the 80% 1RM group, (2) there

was an interaction for pRTD that may have reflected an increase for the 80% 1RM group and no change or a decrease for the 30% 1RM group, (3) iEMG30 increased from baseline to week 2 in both groups, and (4) there were no significant changes in voluntary EMD or contractile twitch properties following 4 weeks of training at 80% or 30% 1RM.

In a previous study, we observed a significant 23% increase in MVIC strength after 4 weeks of training at 80% but not 30% 1RM[5]. In the present study, RTD200 increased from baseline to week 4 for the 80%, but not the 30% 1RM group. Andersen and Aagaard[14] previously reported that RTD200 explained 80% of the variance in MVIC strength, while Jenkins et al.[15] suggested that RTD200 responds similarly to MVIC strength following eccentric-induced muscle damage. Therefore, RTD200 responses in the present study mirrored the previously reported MVIC strength responses following training at 80% versus 30% 1RM[5], and supported the hypothesis that RTD200 is influenced by and/or reflects the same physiological information that is provided by MVIC strength[14,15]. Unlike earlier phase RTD measurements (i.e., RTD30, RTD50, RTD100), these findings collectively suggest that RTD200 and MVIC may provide redundant information.

There was also an interaction for pRTD in the present

study, which may have been due to an increase for the 80% 1RM group and a decrease for the 30% 1RM group. However, post-hoc analyses revealed no significant differences from pre- to post-training or between groups, although a moderate (d=0.62) difference was observed between the 80% and 30% 1RM groups at week 4. Jenkins et al.[15] demonstrated that pRTD behaves similarly to RTD100 following eccentric-induced muscle damage. Although RTD100 did not change significantly following training, the pattern of change does appear to be similar to pRTD. Regardless, our results were inconclusive concerning the effects of short-term high- versus low-load training on pRTD.

Previous studies have identified increases in motor unit firing rate and/or earlier motor unit recruitment as possible mechanisms for training- or population-related differences in iEMG or the rate of rise in EMG at the onset of muscle activation[12,33,40,41]. In the present study, iEMG30 increased by 95% and 76% from baseline to week 2 in the 80% and 30% 1RM groups, respectively, with no differences between groups. Although these adaptations did not result in significant increases in the early phase RTD measurements, there were non-significant 17% and 8% increases in RTD30 and RTD50 from baseline to week 2 (collapsed across group). Early phase RTD adaptations are also thought to reflect changes in motor unit firing rate[12,13,16,42]. Therefore, the significant increase in iEMG30 and non-significant increases in the early phase RTDs that occurred, independent of the different training protocols, may reflect increases in motor unit firing rate, occurrence of doublet discharges, and/or earlier motor unit recruitment that occurred during the first 4 weeks of training. Future studies may wish to continue examining the effects of high- versus low-load training on RTD and iEMG during the initial phases of torque production and muscle activation, respectively.

The EMD has been used as an indirect indicator of musculotendinous stiffness[20]. Theoretically, a stiffer muscle-tendon unit would result in a decrease in the EMD and result in enhanced transmission of forces from the muscle to the bone[43-45]. Muscle stiffness has been shown to be related to muscle size[43], and muscle hypertrophy is similar in response to high- versus low-load training[2,3,5,46]. However, resistance-training mediated increases in tendon stiffness have been shown to be load-dependent[22]. Consequently, it may be hypothesized that load-dependent alterations in musculotendinous stiffness may influence the strength adaptations observed previously following high- versus low-load resistance training[3-5]. In the present study, however, there were no changes in voluntary or evoked EMD for either the 80% or 30% 1RM training groups. The length of training (4 weeks) may have been insufficient to observe changes in EMD, however, since previous studies[19,22] have observed changes in musculotendinous stiffness following 10-12 weeks of training. Future studies should evaluate the effects of high- versus low-load resistance training on the EMD over longer training periods.

We observed no significant changes in the evoked contractile twitch characteristics measured in the current study. Since it is thought that most of the adaptations during the ini-

tial stages of resistance training are neurally mediated[47], the lack of observed changes in peripheral contractile properties may be unsurprising. However, recent studies have shown 4-6% increases in muscle size[48,49] and a 5% increase in fascicle length[48] in as few as 20-28 days of resistance training. These findings suggested[48,49] that peripheral adaptations may occur parallel to neural adaptations and earlier in a resistance training program than previously suspected[47,50,51]. Future studies are needed to more clearly characterize the time course of peripheral adaptations (i.e., muscle hypertrophy, architecture, and contractile twitch properties) in response to resistance training.

This study had several limitations. First, due to the testing procedures (i.e., peripheral nerve stimulation), our sample size was limited. In addition, this study investigated the effects of 80% versus 30% 1RM resistance training during elbow flexion. Therefore, these results may not be generalizable to other muscle groups (i.e., leg extensors/flexors, plantar flexors, etc.) or to multi-joint movements. Finally, training was performed over the course of 4 weeks. Future studies may wish to study the adaptations to 80% versus 30% 1RM resistance training over longer periods of training.

Overall, the results of the present study indicated that 4 weeks of resistance training at 80% 1RM, but not 30% 1RM, caused an in increase in RTD200, which likely reflected similar increases in MVIC. There were also increases from baseline to week 4 in iEMG during the first 30 ms of muscle activation for the 80% and 30% 1RM groups, which may have indicated increases in early phase recruitment or motor unit firing frequency. However, there were no significant training-induced adaptations in EMD or contractile twitch properties. Future longer-term studies are needed to continue our understanding of the changes in RTD, EMD, and contractile twitch properties in response to high- versus low-load resistance training.

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