Effect of Resistance Training to Muscle Failure vs. Volitional Interruption at High- and Low-Intensities on Muscle Mass and Strength

Sanmy R. Nóbrega,[1] Carlos Ugrinowitsch,[2] Lucas Pintanel,[1] Cintia Barcelos,[1] and Cleiton A. Libardi[1]

[1]*Laboratory of Neuromuscular Adaptations to Resistance Training, Department of Physical Education, Federal University of São Carlos, São Carlos, Brazil; and* [2]*Department of Sport, School of Physical Education and Sport, University of São Paulo–USP, São Paulo, Brazil*

Abstract

Nóbrega, SR, Ugrinowitsch, C, Pintanel, L, Barcelos, C, and Libardi, CA. Effect of resistance training to muscle failure vs. volitional interruption at high- and low-intensities on muscle mass and strength. *J Strength Cond Res* 32(1): 162–169, 2018—The purpose of this study was to investigate the effects of resistance training (RT) at high- and low-intensities performed to muscle failure or volitional interruption on muscle strength, cross-sectional area (CSA), pennation angle (PA), and muscle activation. Thirty-two untrained men participated in the study. Each leg was allocated in 1 of 4 unilateral RT protocols: RT to failure at high and low intensities, and RT to volitional interruption (repetitions performed to the point in which participants voluntarily interrupted the exercise) at high (HIRT-V) and low (LIRT-V) intensities. Muscle strength (1 repetition maximum [1RM]), CSA, PA, and muscle activation by amplitude of the electromyography (EMG) signal were assessed before (Pre), after 6 (6W), and 12 (12W) weeks. 1RM increased similarly after 6W (range: 15.8–18.9%, effective size [ES]: 0.41–0.58) and 12W (range: 25.6–33.6%, ES: 0.64–0.98) for all protocols. All protocols were similarly effective in increasing CSA after 6W (range: 3.0–4.6%, ES: 0.10–0.24) and 12W (range: 6.1–7.5%, ES: 0.22–0.26). PA increased after 6W (~3.5) and 12W (~9%; main time effect, $p < 0.0001$), with no differences between protocols. EMG values were significantly higher for the high-intensity protocols at all times (main intensity effect, $p < 0.0001$). In conclusion, both HIRT-V and LIRT-V are equally effective in increasing muscle mass, strength, and PA when compared with RT performed to muscle failure.

Key Words volitional fatigue, muscle cross-sectional area, pennation angle, ultrasound, electromyography

Address correspondence to Dr. Cleiton A. Libardi, c.libardi@ufscar.br.

32(1)/162–169

Journal of Strength and Conditioning Research

Introduction

Resistance training (RT) increases muscle strength, muscle cross-sectional area (CSA) (i.e., muscle hypertrophy), and may be followed by increases in muscle fiber pennation angle (PA) (1,2). It has been suggested that performing high-intensity RT (HIRT) to muscle failure can maximize gains in muscle strength (12,32) and hypertrophy (37), as it allows greater recruitment of motor units (MUs) when compared with no-failure HIRT programs (44). However, a recent meta-analysis showed that HIRT to failure (HIRT-F) does not result in additional increases in muscle strength compared with non-failure HIRT (11). On the other hand, the effect of RT to muscle failure on muscle mass is still poorly understood.

Although the advantages of muscle failure in HIRT programs are equivocal, it has been demonstrated that muscle failure can be important to muscle adaptation when RT is performed at low-intensity ([LIRT]; e.g., 30% of 1 repetition maximum [1RM]) (8,25,27,36). Recent studies have demonstrated that LIRT to failure (LIRT-F) produces similar myofibrillar protein synthesis and muscle hypertrophy to HIRT (8,25,27,36). It has been hypothesized by intramuscular electromyography (EMG) analyses that, when one performs several low-intensity contractions, initially recruited MUs fatigue and/or cease firing, which may eventually recruit the available MU pool to maintain force output (13,16). As a consequence, LIRT performed to muscle failure may recruit a similar number of MUs to HIRT, and, therefore, result in comparable increases in muscle strength and hypertrophy (25–27,44).

Despite being commonly recommended (12,25,27,32,36,37), some studies have suggested that performing RT to failure for prolonged periods of time (i.e., months or years) may result in overtraining (39), increased risk of musculoskeletal injury (39), and peaks of blood pressure (23). Accordingly, some guidelines recommend performing RT to volitional interruption (i.e., when the individual voluntarily interrupts the exercise) and not to muscle failure (17,24,29,30). However, no study has investigated if HIRT and LIRT performed to volitional interruption result in similar gains in muscle strength and hypertrophy compared with protocols performed to muscle failure. Considering the current literature, it seems possible to reach high levels of muscle activity without going to muscle failure, as Sundstrup et al. (40) demonstrated an activation plateau occurring 3–5 repetitions before muscle failure. Therefore, HIRT and LIRT performed to volitional interruption may result in similar muscle activation compared with HIRT and LIRT performed to muscle failure, pointing towards similar increases in muscle strength and hypertrophy between these training protocols.

Therefore, the aim of this study was to investigate the effects of HIRT and LIRT performed to failure and volitional interruption on muscle strength and CSA. Additionally, we verified the effects of these RT protocols on PA and muscle activation. We hypothesized that increases in muscle strength and CSA would be similar between all 4 protocols, regardless of RT condition (failure or volitional interruption) and intensity (80 or 30% 1RM).

Methods

Experimental Approach to the Problem

Before the training period (Pre), all participants were familiarized with the 1RM test and exercise protocols. Seventy-two hours after familiarization, a new 1RM test was performed. If 1RM values differed more than 5% from the previous test, a subsequent test was performed after 72 hours. On average, each participant performed 3 1RM tests. At least 72 hours after the last 1RM test, vastus lateralis (VL) muscle CSA and PA were assessed. To reduce inter-subjects variability, we used a design in which each participant's leg was allocated in a randomized and balanced way, according to 1RM and CSA values, to 1 of the 4 training protocols: HIRT-F; HIRT to volitional interruption (HIRT-V); LIRT-F; and LIRT to volitional interruption (LIRT-V), for a total of 16 legs in each group ($n = 16$). Before training period initiation, VL muscle activation was assessed through EMG, with participants performing their respective training protocol. Training period was then initiated and all groups performed their respective protocols for 12 weeks (12W). After 6 weeks of training (6W), 1RM was reassessed 72 hours after the twelfth training session, with reassessments of muscle CSA and PA 72 hours after the 1RM test. Seventy-two hours later, load was adjusted according to the new 1RM value, and a new EMG assessment was performed. Training continued for another 6 weeks with the adjusted load. Seventy-two hours after the last training session, final 1RM was assessed, with subsequent assessments of CSA and PA 72 hours later. A final EMG assessment was performed 72 hours after the final CSA and PA assessment (12W).

Subjects

Thirty-two young men between 18 and 30 years old (Mean +/− SD age: 23.0 ± 3.6 years; height: 176.0 ± 0.6 cm; body mass index: 24.3 ± 3.9 $kg \cdot m^{-2}$) were recruited. None of the participants had performed RT in the last 6 months. Those receiving care for any lower-body musculoskeletal disorder at the time of the study were excluded from participation. All procedures performed in the study were in accordance with the ethical standards of the Federal University of São Carlos' research committee and with the 1964 Helsinki declaration. Before the study initiation, all participants were informed of the procedures and were asked to provide written informed consent.

Only the participants who completed 100% of the training sessions were included. Twenty-seven participants completed the study, for a total of n = 54 legs (HIRT-F = 14; HIRT-V = 14; LIRT-F = 13; LIRT-V = 13). Five participants did not complete all sessions or dropped out for personal reasons, thus they were not included in the analyses.

Procedures

Maximal Dynamic Strength Test. Unilateral 1RM tests were performed on a knee extension machine, according to the Brown and Weir (6) protocol. Initially, participants warmed up for 5 minutes on a cycle ergometer at 20 $km \cdot h^{-1}$. Then, participants performed 8 repetitions at 50% of the estimated 1RM, followed by 3 repetitions at 70% of the estimated 1RM, with a rest interval of 2 minutes between sets. After warm-up, participants initiated the 1RM test at full knee extension (~180°), performing both eccentric and concentric exercise phases at a range of 90°. Up to 5 attempts were allowed, with a rest interval of 3 minutes between attempts. The coefficient of variation (CV) and typical error (TE) between maximal dynamic strength tests were 1.01% and 0.5 kg, respectively.

Muscle Activation. Activation of the VL muscle was assessed by the amplitude of the EMG signal. Initially, participants performed a maximal voluntary isometric contraction (MVIC) test (35). Before electrode placement, participants were prepared by shaving the desired area, followed by skin abrasion and skin cleansing with isopropyl alcohol to ensure low skin impedance. Self-adhesive disposable electrodes were then placed over the VL according to SENIAM (38), with an interelectrode distance of 2 cm. A reference electrode was fixed on the opposite ankle. For better stability, micropore tape was applied over the electrodes. After a 5-minute warm-up on a cycle ergometer at 20 $km \cdot h^{-1}$, participants were positioned in a knee extension machine (FISIOMAQ, Paraná, Brazil) with knees fixed at 90° of knee flexion. The knee extension machine arm was locked at 90°. Participants were asked to gradually build force and hold it for 3 seconds at maximal force. Three trials were performed, with 1 minute rest between trials, and the highest root mean square (RMS) value attained was used for normalizing EMG signals. To differentiate concentric and eccentric EMG signals, an electrogoniometer (EMG System; São José dos Campos, São Paulo, Brazil) was placed on the estimated center of rotation of the knee joint (i.e., intercondilar line). EMG and electrogoniometer signals were acquired using the EMG832C electromyographic device

(EMG System; São José dos Campos) and active bipolar surface electrodes with preamplifier gains of 20-fold and common-mode rejection rate >100 db. After electrodes placement, participants were instructed to perform the MVIC as instructed. For EMG acquisition, participants were instructed to exercise each leg after the RT protocols to which they were allocated. In short, the legs allocated to the HIRT-F and LIRT-F performed 3 sets to muscle failure, whereas those allocated in the HIRT-V and LIRT-V protocols performed 3 sets to volitional interruption, with either 30 or 80% 1RM, according to their respective protocol. The loads used in the EMG tests were adjusted according to the volunteers' most recent 1RM value. The training protocols are described in detail in the "resistance training protocols" section. A 2-minute rest interval was timed between sets. Signals were collected at 2,000 Hz and filtered with an eighth order Butterworth bandpass filter set at 20–500 Hz. Data processing was performed offline using a custom MATLAB routine (MathWorks, Natick, MA, USA). Initially, EMG data were normalized using MVIC data. After data normalization, the beginning and ending of each repetition were manually identified on the MATLAB routine for each set. Minimal and maximal angle values were used to define the end of the eccentric and concentric phases, respectively. The RMS of the EMG signal of the concentric phase of the last 3 repetitions was used to calculate the mean muscle activation.

Muscle Cross-Sectional Area. Vastus lateralis CSA was assessed using an ultrasound (US) machine. Procedures similar to Lixandrão et al. (22) were adopted. Participants were asked to refrain from vigorous physical activities for at least 72 hours before the image acquisitions. After arrival, participants laid in a supine position for 15 minutes to allow fluid shifts to occur. Images were collected using the US B-mode with a 7.5 MHz probe (Samsung, MySono U6; industria e comércio Ltda., São Paulo, Brazil). Surface gel was applied to promote acoustic coupling while avoiding dermal deforming. The CSA was obtained at 50% of the femur length, manually measured as the midway point between the greater trochanter and the lateral epicondyle. The skin was transversally marked every 2 cm from the reference point toward the medial and lateral aspects of the thigh to guide probe displacement. Sequential images were acquired aligning the superior edge of the probe with the ink marking, moving in a middle-to-lateral direction. After data acquisition, VL CSA was reconstructed according to Reeves et al. (31), in which images were sequentially opened and rotated using PowerPoint version 2007 (Microsoft, Redmond, WA, USA), until full muscle area was visible. CSA value was assessed using the *ImageJ* polygonal tool. Each muscle area was reconstructed 3 times, and the mean was assumed as the true CSA value. Great care was taken to avoid the surrounding connective and bone tissue. The CV and TE between 2 repeated measures performed in different days (72 hours apart) for the CSA were 1.38% and 0.33 cm, respectively.

Pennation Angle

Vastus lateralis PA was assessed using B-mode US at the same site as CSA. The transducer was placed longitudinally to the muscle tissue and, when necessary, was laterally tilted to better allow fascicle visualization (4). Muscle fiber PA was determined as the intersection of the fascicles with the deep aponeurosis as assessed using the *ImageJ* angular tool. Mean value was averaged from 3 images. CV and TE were 1.87% and 0.35°, respectively.

Resistance Training Protocols. All RT protocols were performed unilaterally using a conventional leg-extension machine, twice a week for 12 weeks (total of 24 training sessions). At the beginning of each RT session, participants performed a general warm-up on a cycle ergometer (Ergo-Fit, Ergo-cycle 167; Ergo-Fit GmbH & Co KG; Pirmasens, Germany) pedaling at 20 $km \cdot h^{-1}$ for 5 minutes. After, HIRT-F and HIRT-V protocols performed 3 sets of 80% 1RM. The LIRT-F and LIRT-V protocols performed the same number of sets, but with a load corresponding to 30% 1RM. A 2-minute rest interval was timed between sets. For the muscular failure protocols, repetitions were performed to the point of inability to perform a repetition with full range of motion (i.e., 90°) (19,20,35,36), as evaluated by researchers familiar with the protocols. For the volitional interruption protocols, repetitions were performed to the point in which participants voluntarily interrupted the exercise (29) before muscle failure. Thus, all participants were previously instructed on the criteria for muscular failure.

Total Training Volume and Number of Repetitions. Training loads were recorded for all 24 training sessions. Total training volume (TTV) was calculated as the sum of the training volume (sets × load × repetitions) performed in sessions 1–12 (1–6W) and 13–24 (7–12W). To calculate the number of repetitions (Nrep) performed in the muscle failure and voluntary interruption conditions, repetitions performed over the experimental period were summed per set. Then, we calculated the absolute and relative differences between the third and the first set (Nreps = third set − first set; % Nreps = ([third set − first set]/first set), respectively).

Statistical Analyses

After visual inspection, data normality was assessed using the Shapiro-Wilk test. Initially, a one-way repeated measures analysis of variance was implemented to test for differences in baseline values. Then, a mixed model was applied for each dependent variable, having intensity (30 and 80% 1RM), condition (muscular failure or volitional interruption), and time (Pre, 6W and 12W) as fixed factors, and participants as random factors. Only EMG values were significantly different at baseline, and therefore, were considered as a covariate, and an analysis of covariance (ANCOVA) was implemented using a mixed model also having intensity, condition, and time as fixed factors, and subjects as a random factor. In case of significant *F* values, Tukey's adjustment was used for multiple comparison purposes. An independent *t* test was used to compare absolute and relative mean differences in Nrep between the third and first sets for the HIRT-F vs. HIRT-V and LIRT-F vs. LIRT-V protocols. Finally, within-group effect sizes (ESs) (1–6W and 1–12W changes) were calculated using Cohen's *d* (10) to 1RM and muscle CSA. Statistical analysis was carried out using SAS 9.3 software (SAS institute Inc., Cary, NC, USA).

Results

Total Training Volume and Number of Repetitions

Significant differences were found for TTV, with HIRT-F and HIRT-V resulting in a greater TTV compared with LIRT-F and LIRT-V protocols at 1–6W (12,795 ± 3,654 kg; 12,619 ± 2,950 kg; 9,923 ± 3,144 kg; and 9,755 ± 2,723 kg, respectively; main intensity effect, $p = 0.008$) and 7–12W (13,899 ± 3,236 kg; 13,423 ± 2,863 kg; 11,191 ± 3,346 kg; and 10,888 ± 2,515 kg, respectively; main intensity effect, $p = 0.008$). Additionally, 7–12W resulted in a significantly higher TTV compared with 1–6W for all protocols (main time effect, $p < 0.0001$). There were significant differences in Nrep within intensities (HIRT-F vs. HIRT-V; LIRT-F vs. LIRT-V) ($p \leq 0.05$). Failure protocols resulted in larger decreases in Nrep compared with the volitionally-interrupted counterpart (HIRT-F = −78 ± 20 reps, −30.3 ± 4.9% vs. HIRT-V = −67 ± 27 reps, −25.3 ± 9.7%; $p = 0.001$; LIRT-F = −225 ± 68 reps, −39.9 ± 8.3% vs. LIRT-V = −184 ± 75 reps, −33.8 ± 7.8%; $p = 0.03$).

Maximal Dynamic Strength (1RM)

No significant differences between groups were found ($p > 0.05$). All training protocols showed significant increases in 1RM after 6W (main time effect, $p < 0.0001$; ES: HIRT-F = 0.41, HIRT-V = 0.58, LIRT-F = 0.44, and LIRT-V = 0.43) and 12W (main time effect, $p < 0.0001$; ES: HIRT-F = 0.65, HIRT-V = 0.98, LIRT-F = 0.64, and LIRT-V = 0.66) compared with Pre. Additionally, 1RM values were significantly higher at 12W compared with 6W (main time effect, $p < 0.0001$) (Figure 1).

Muscle Cross-Sectional Area

Vastus lateralis muscle CSA values increased for all training protocols after 6W (main time effect, $p < 0.0001$; ES: HIRT-F = 0.17, HIRT-V = 0.11, LIRT-F = 0.24, and LIRT-V = 0.10) and 12W (main time effect, $p < 0.0001$; ES: HIRT-F = 0.26, HIRT-V = 0.23, LIRT-F = 0.26, and LIRT-V = 0.22) compared with initial values (Pre), with no significant differences between groups ($p > 0.05$). In addition, muscle CSA at 12W was significantly greater than that at 6W for all training protocols (main time effect, $p < 0.0001$) (Figure 2), with no differences between groups ($p > 0.05$).

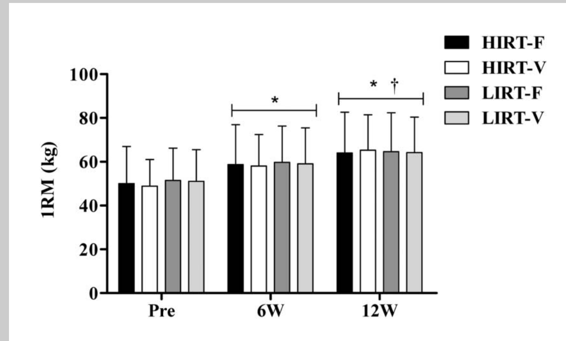

Figure 1. Maximal dynamic strength (1RM) at baseline (Pre), and after 6 (6W) and 12 weeks (12W) of high-intensity resistance training to muscular failure (HIRT-F), high-intensity resistance training to volitional interruption (HIRT-V), low-intensity resistance training to muscular failure (LIRT-F), and low intensity resistance training to volitional interruption (LIRT-V). Results are presented as mean ± *SD*. *Significant difference compared with Pre (main time effect, $p < 0.0001$); †Significant difference compared with 6W (main time effect, $p < 0.0001$).

Pennation Angle

PA values increased significantly for all training groups from Pre (HIRT-F = 19.60 ± 2.02°; HIRT-V = 20.00 ± 2.65°; LIRT-F = 20.52 ± 3.78°; LIRT-V = 20.90 ± 2.87°) to 6W (HIRT-F = 20.41 ± 2.14°, 4.15%; HIRT-V = 20.31 ± 2.71°, 1.56%; LIRT-F = 21.80 ± 3.31°, 6.21%; LIRT-V = 21.33 ± 2.88°, 2.03%; main time effect, $p = 0.0192$) and 12W (HIRT-F = 22.05 ± 1.74°, 12.51%; HIRT-V = 21.44 ± 2.64°, 7.24%; LIRT-F = 2.48 ± 3.32°, 9.53%; LIRT-V = 22.28 ± 3.84°, 6.60%; main time effect, $p < 0.0001$). Furthermore, PA was significantly higher at 12W compared with 6W (main time effect, $p = 0.001$). No significant differences between groups were found ($p > 0.05$).

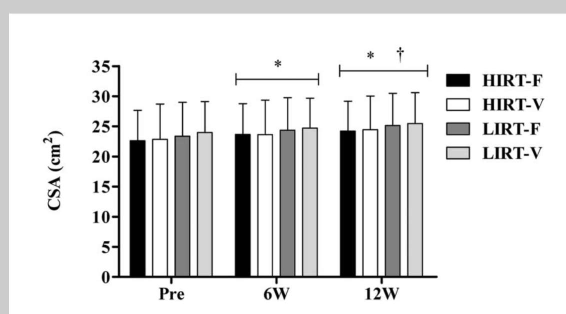

Figure 2. Muscle cross-sectional area (CSA) before (Pre), and after 6 (6W) and 12 weeks (12W) of high-intensity resistance training to muscular failure (HIRT-F), high-intensity resistance training to volitional interruption (HIRT-V), low-intensity resistance training to muscular failure (LIRT-F), and low intensity resistance training to volitional interruption (LIRT-V). Results are presented as mean ± *SD*. *Significant difference compared with Pre (main time effect, $p < 0.0001$); †Significant difference compared with 6W (main time effect, $p < 0.0001$).

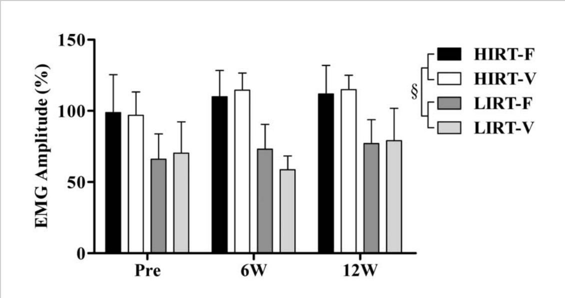

Figure 3. Peak electromyography (EMG) amplitude normalized by maximal voluntary isometric contraction at baseline (Pre), and after 6 (6W) and 12 weeks (12W) of high-intensity resistance training to muscular failure (HIRT-F), high-intensity resistance training to volitional interruption (HIRT-V), low-intensity resistance training to muscular failure (LIRT-F), and low intensity resistance training to volitional interruption (LIRT-V). Results are presented as mean ± *SD*. §Significant difference compared with LIRT-F and LIRT-V (main group effect, $p \leq 0.05$).

Electromyography Activity

There were significant differences in EMG amplitude at baseline (Pre; $p \leq 0.05$). When baseline differences in EMG values were taken into account (repeated measures ANCOVA), a significant main intensity effect ($p < 0.0001$) was found (Figure 3), with significantly higher EMG values for the high-intensity protocols.

Discussion

Our main findings show that both muscle failure and volitional interruption protocols were similarly effective at inducing increases in muscle hypertrophy and strength gains, regardless of intensity (i.e., high and low intensity), confirming our initial hypothesis.

In this regard, it has been suggested that LIRT can promote similar muscle hypertrophy to HIRT only when performed to muscle failure (14,25–27,36). Our results show otherwise, as we observed similar increases in CSA after 6W (range: 3.0–4.6%; ES: 0.10–0.24) and 12W (range: 6.1–7.5%; ES: 0.22–0.26), regardless of RT intensity (i.e., high or low intensity) and condition (i.e., failure or volitional interruption) of the training protocol. It has been suggested that LIRT performed to muscle failure can recruit MU pool to maintain muscle tension as fatigue develops (8,25). However, despite these suggestions and the positive relationship between EMG activity and muscle hypertrophy (42), our results show that VL EMG activity was significantly higher for the HIRT protocols at all time points (i.e., main group effect, $p < 0.0001$), despite similar muscle hypertrophy between protocols. Therefore, EMG signal may not truly reflect MUs' recruitment when high- and low-intensity protocols are compared. Some researchers state that, during submaximal fatiguing contractions, MUs' recruitment threshold declines to maintain a constant force production (3). Such decline could allow a momentary MUs derecruitment (i.e., MUs cycling) to reduce fatigue (43). Thereby, both LIRT-F and LIRT-V could result in a significant MUs recruitment, but because of MUs cycling, along with a different recruitment pattern (i.e., simultaneous MUs recruitment in HIRT vs. progressive recruitment in LIRT), MU recruitment would be lower in this condition compared with HIRT-F and HIRT-V recruitment (41), resulting in lower surface EMG amplitude. It should also be considered that HIRT protocols might produce a higher degree of MUs synchronization than LIRT protocols, as the former requires a higher number of MUs firing simultaneously than the last. Also, as artificially demonstrated using a computer model, a high MU synchronization can increase EMG amplitude without activating a higher number of MUs (45). Some authors have also suggested that similar increases in muscle CSA between HIRT and LIRT may be induced by a higher TTV when LIRT is performed to failure compared with HIRT (25–27,36). Interestingly, in our study, the HIRT protocols resulted in a significantly higher TTV than the LIRT protocols. This difference in TTV between our and their studies might be due to either differences in protocols (uni- vs. multi-joint exercises) (26,27), a higher number of sets for the LIRT protocol compared with HIRT (27), participants' training levels (untrained vs. trained) (26,36), or even researchers' subjectivity in determining the point of muscle failure during protocol execution. Despite the differences in TTV, both HIRT and LIRT resulted in similar increases in muscle strength and CSA. Our results are in agreement with other studies that found similar muscle adaptations after protocols of different TTV (5,25,28). For example, Mitchell et al. (25) found no difference in muscle strength (1RM) after protocols of different intensities and volumes (30%-3 sets vs. 80%-3 sets vs. 80%-1 set), despite the smaller RT volume performed by the 80%-1 protocol. Interestingly, the increases in muscle CSA were 2 times the increase after the single set protocol. However, no statistically significant differences were found for muscle CSA, which might indicate a possible type II error, especially when the ES is considered (30%-3 = 0.34, 80%-3 = 0.47, and 80%-1 = 0.19). On the other hand, in our study, all protocols resulted in similar hypertrophy with ES ranging from 0.22 to 0.26, regardless of the differences in protocols' TTV. This way, if a higher TTV was really necessary for LIRT to result in increased muscle hypertrophy, one would expect that, because of the smaller TTV, LIRT would have resulted in smaller increases in muscle CSA compared with HIRT protocols, which did not occur. Thus, our findings suggest that neither a higher TTV nor the occurrence of muscular failure is necessary to maximize gains in muscle mass and strength after LIRT protocols. Further studies are needed to elucidate the mechanisms responsible for such adaptations.

Regarding muscle strength, 1RM values increased after 6W (range: 15.8–18.9%; ES: 0.41–0.58) and 12W (range: 25.6–33.6%; ES: 0.64–0.98) for all training protocols, without significant differences between them. Our results are in line with Morton et al. (26), but diverge from others that showed greater increases in 1RM after HIRT compared with LIRT (9,25,36). It is also important to consider that of the studies cited above, only Morton et al. (26) reported performing familiarization sessions for the 1RM test. By contrast, participants in our study performed, on average, 3 familiarization sessions with the 1RM test before training initiation. As stated by Buckner et al. (7), performance in 1RM tests may be influenced by protocol specificity, with better outcomes for training protocols that most closely resemble the test. Thus, HIRT protocols would most likely result in greater 1RM values as compared to low-load protocols. However, familiarization sessions to 1RM test may mitigate the differences in test performance between HIRT and LIRT protocols (7). Thus, 1RM test familiarization may be considered of utmost importance when comparing strength gains between high- and low-load training protocols. Another factor that could explain the similar increases in muscle strength between HIRT and LIRT was the increase in fiber PA after 6W (~3.5%) and 12W (~9%) for all protocols, without significant difference between them, independently of RT condition (i.e., muscle failure or volitional interruption protocols). To the authors' knowledge, this is the first study comparing the effects of HIRT and LIRT performed or not until muscle failure. It is well known that increases in PA, which determines the component of force of fibers to the line of pull of the muscle, reduce force output generation because of a decrease in specific tension and force transmission (21). On the other hand, increases in muscle PA allow for an increase in contractile material, with a possible increase in crossbridges formation and the number of crossbridges simultaneously activated during a muscle contraction, overcoming the decrease in force production due to greater PAs (1,21). Thus, maximal muscle force is expected to increase with increases in muscle PA up to 45° (33). If PA increases were to affect the final increases in muscle strength, all protocols would be equally affected in our study, considering the similar increases in PA between them.

Even though previous studies have suggested RT to failure to maximize increases in muscle strength and hypertrophy (12,14,26,32,36,37), our results show similar increases in both 1RM and CSA when comparing failure vs. no-failure (i.e., volitional interruption) conditions. In regard to muscle strength, our results are in agreement with other studies that found no differences in strength gains when HIRT was performed to failure or no-failure (15,19). Furthermore, a recent meta-analysis by Davies et al. (11) on the effects of muscle failure on strength gains found no advantage when RT was performed to failure. Interestingly, results held true even for studies that did not equalize TTV (11). On the other hand, little is known on how muscle failure affects hypertrophy in protocols of same intensity (e.g., HIRT-F vs. HIRT-V). A recent study by Sampson and Groeller (34) investigated the effects of HIRT (85% 1RM) to failure and no-failure on elbow flexors CSA and found similar increases in muscle CSA after 12 weeks for all groups (~11.4%), despite the differences in repetition velocities between the muscle failure and no-failure protocols. Accordingly, our results show similar increases in muscle CSA after both HIRT-F and HIRT-V, indicating no advantage in performing HIRT to muscle failure. In regard to LIRT, we are the first to show that training to failure does not result in greater increases in muscle strength and mass compared with volitional interruption, refuting the suggestion that LIRT only results in increased muscle strength and hypertrophy when performed to muscle failure (18,25,26). Considering our results, protocols of same intensity (i.e., HIRT-F vs. HIRT-V and LIRT-F vs. LIRT-V) performed to volitional interruption or muscle failure may result in a similar MUs recruitment, which could explain the similar gains in muscle strength and mass between them. In this sense, Sundstrup et al. (40) verified that EMG amplitude was maximal before muscular failure, and further repetitions did not result in increased EMG amplitude. Our EMG results support this hypothesis as there were no significant differences when protocols of same intensity performed to muscle failure or volitional interruption were compared.

Finally, some studies suggest that training to failure for a long period might result in overtraining, increased risk of muscle injury, and increased hemodynamic responses with blood pressure peaks near muscle failure (23,39). In our study, both failure conditions (HIRT-F and LIRT-F) resulted in a significant decrease in Nrep throughout the training period compared with their non-failure counterpart (HIRT-V and LIRT-V), which might indicate that the failure conditions resulted in significant higher levels of fatigue.

In conclusion, both high-intensity (80% 1RM) and low-intensity (30% 1RM) RT performed to volitional interruption are equally effective in promoting increases in muscle strength, muscle CSA, and PA as RT performed to muscle failure.

Practical Applications

Considering the similar adaptations among the muscle failure and volitional interruption groups, our results support performing RT to volitional interruption as recommended by some studies (17,24,29,30). These findings might be especially important for impaired populations, such as the elderly and cardiac patients, who are unable to perform neither HIRT nor LIRT to muscle failure, in which performing RT to a substantial fatigue level (i.e., volitional interruption) would result in maximal gains in muscle strength and hypertrophy, regardless of RT intensity.

Acknowledgments

We would like to acknowledge all participants of this study. This work was supported by São Paulo Research Foundation

(FAPESP) Grants (2013/21218-4 to CAL and 2015/05650-9 to LP) and National Council for Scientific and Technological Development (CNPq) Grant (304205/2011-7 to CU). The results of the present study do not constitute endorsement of the product by the authors or the National Strength and Conditioning Association.

References

1. Aagaard, P, Andersen, JL, Dyhre-Poulsen, P, Leffers, AM, Wagner, A, Magnusson, SP, Halkjaer-Kristensen, J, and Simonsen, EB. A mechanism for increased contractile strength of human pennate muscle in response to strength training: Changes in muscle architecture. *J Physiol* 534: 613–623, 2001.
2. ACSM. American College of Sports Medicine position stand. Progression models in resistance training for healthy adults. *Med Sci Sports Exerc* 41: 687–708, 2009.
3. Adam, A and De Luca, CJ. Recruitment order of motor units in human vastus lateralis muscle is maintained during fatiguing contractions. *J Neurophysiol* 90: 2919–2927, 2003.
4. Blazevich, AJ, Cannavan, D, Coleman, DR, and Horne, S. Influence of concentric and eccentric resistance training on architectural adaptation in human quadriceps muscles. *J Appl Physiol (1985)* 103: 1565–1575, 2007.
5. Bottaro, M, Veloso, J, Wagner, D, and Gentil, P. Resistance training for strength and muscle thickness: Effect of number of sets and muscle group trained. *Sci Sports* 26: 259–264, 2011.
6. Brown, LE and Weir, JP. ASEP procedures recommendation I: Accurate assessment of muscular strength and power. *J Exerc Physiol Online* 4: 1–21, 2001.
7. Buckner, SL, Jessee, MB, Mattocks, KT, Mouser, JG, Counts, BR, Dankel, SJ, and Loenneke, JP. Determining strength: A case for multiple methods of measurement. *Sports Med* 47: 193–195, 2017.
8. Burd, NA, West, DW, Staples, AW, Atherton, PJ, Baker, JM, Moore, DR, Holwerda, AM, Parise, G, Rennie, MJ, Baker, SK, and Phillips, SM. Low-load high volume resistance exercise stimulates muscle protein synthesis more than high-load low volume resistance exercise in young men. *PLoS One* 5: e12033, 2010.
9. Campos, GE, Luecke, TJ, Wendeln, HK, Toma, K, Hagerman, FC, Murray, TF, Ragg, KE, Ratamess, NA, Kraemer, WJ, and Staron, RS. Muscular adaptations in response to three different resistance-training regimens: Specificity of repetition maximum training zones. *Eur J Appl Physiol* 88: 50–60, 2002.
10. Cohen, J. *Statistical Power Analysis for the Behavioral Sciences.* Hillsdale, NJ: Lawrence Erlbaum, 1988.
11. Davies, T, Orr, R, Halaki, M, and Hackett, D. Effect of training leading to repetition failure on muscular strength: A systematic review and meta-analysis. *Sports Med* 46: 487–502, 2016.
12. Drinkwater, EJ, Lawton, TW, Lindsell, RP, Pyne, DB, Hunt, PH, and McKenna, MJ. Training leading to repetition failure enhances bench press strength gains in elite junior athletes. *J Strength Cond Res* 19: 382–388, 2005.
13. Fallentin, N, Jorgensen, K, and Simonsen, EB. Motor unit recruitment during prolonged isometric contractions. *Eur J Appl Physiol Occup Physiol* 67: 335–341, 1993.
14. Fink, J, Kikuchi, N, Yoshida, S, Terada, K, and Nakazato, K. Impact of high versus low fixed loads and non-linear training loads on muscle hypertrophy, strength and force development. *Springerplus* 5: 698, 2016.
15. Folland, JP, Irish, CS, Roberts, JC, Tarr, JE, and Jones, DA. Fatigue is not a necessary stimulus for strength gains during resistance training. *Br J Sports Med* 36: 370–373, 2002. Discussion 374.
16. Fuglevand, AJ, Zackowski, KM, Huey, KA, and Enoka, RM. Impairment of neuromuscular propagation during human fatiguing contractions at submaximal forces. *J Physiol* 460: 549–572, 1993.
17. Haskell, WL, Lee, IM, Pate, RR, Powell, KE, Blair, SN, Franklin, BA, Macera, CA, Heath, GW, Thompson, PD, and Bauman, A. Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Exerc* 39: 1423–1434, 2007.
18. Holm, L, Reitelseder, S, Pedersen, TG, Doessing, S, Petersen, SG, Flyvbjerg, A, Andersen, JL, Aagaard, P, and Kjaer, M. Changes in muscle size and MHC composition in response to resistance exercise with heavy and light loading intensity. *J Appl Physiol* 105: 1454–1461, 2008.
19. Izquierdo, M, Ibanez, J, Gonzalez-Badillo, JJ, Hakkinen, K, Ratamess, NA, Kraemer, WJ, French, DN, Eslava, J, Altadill, A, Asiain, X, and Gorostiaga, EM. Differential effects of strength training leading to failure versus not to failure on hormonal responses, strength, and muscle power gains. *J Appl Physiol (1985)* 100: 1647–1656, 2006.
20. Jenkins, ND, Housh, TJ, Bergstrom, HC, Cochrane, KC, Hill, EC, Smith, CM, Johnson, GO, Schmidt, RJ, and Cramer, JT. Muscle activation during three sets to failure at 80 vs. 30% 1RM resistance exercise. *Eur J Appl Physiol* 115: 2335–2347, 2015.
21. Kawakami, Y, Abe, T, Kuno, SY, and Fukunaga, T. Training-induced changes in muscle architecture and specific tension. *Eur J Appl Physiol Occup Physiol* 72: 37–43, 1995.
22. Lixandrão, ME, Ugrinowitsch, C, Bottaro, M, Chacon-Mikahil, MP, Cavaglieri, CR, Min, LL, Oliveira de Souza, E, Laurentino, GC, and Libardi, CA. Vastus lateralis muscle cross sectional area ultrasonography validity for image-fitting in humans. *J Strength Cond Res* 28: 3293–3297, 2014.
23. MacDougall, JD, Tuxen, D, Sale, DG, Moroz, JR, and Sutton, JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol* 58: 785–790, 1985.
24. Mazzeo, RS and Tanaka, H. Exercise prescription for the elderly: Current recommendations. *Sports Med* 31: 809–818, 2001.
25. Mitchell, CJ, Churchward-Venne, TA, West, DW, Burd, NA, Breen, L, Baker, SK, and Phillips, SM. Resistance exercise load does not determine training-mediated hypertrophic gains in young men. *J Appl Physiol (1985)* 113: 71–77, 2012.
26. Morton, RW, Oikawa, SY, Wavell, CG, Mazara, N, McGlory, C, Quadrilatero, J, Baechler, BL, Baker, SK, and Phillips, SM. Neither load nor systemic hormones determine resistance training-mediated hypertrophy or strength gains in resistance-trained young men. *J Appl Physiol (1985)* 121: 129–138, 2016.
27. Ogasawara, R, Loenneke, J, Thiebaud, R, and Abe, T. Low-load bench press training to fatigue results in muscle hypertrophy similar to high-load bench press training. *Int J Clin Med* 4: 114–121, 2013.
28. Ostrowski, KJ, Wilson, GJ, Weatherby, R, Murphy, PW, and Lyttle, AD. The effect of weight training volume on hormonal output and muscular size and function. *J Strength Cond Res* 11: 148–154, 1997.
29. Pitcher, JB and Miles, TS. Influence of muscle blood flow on fatigue during intermittent human hand-grip exercise and recovery. *Clin Exp Pharmacol Physiol* 24: 471–476, 1997.
30. Pollock, ML, Franklin, BA, Balady, GJ, Chaitman, BL, Fleg, JL, Fletcher, B, Limacher, M, Pina, IL, Stein, RA, Williams, M, and Bazzarre, T. AHA Science Advisory. Resistance exercise in individuals with and without cardiovascular disease: Benefits, rationale, safety, and prescription: An advisory from the Committee on Exercise, Rehabilitation, and Prevention, Council on Clinical Cardiology, American Heart Association. *Circulation* 101: 828–833, 2000.
31. Reeves, ND, Maganaris, CN, and Narici, MV. Ultrasonographic assessment of human skeletal muscle size. *Eur J Appl Physiol* 91: 116–118, 2004.

32. Rooney, KJ, Herbert, RD, and Belnave, RJ. Fatigue contributes to the strength training stimulus. *Med Sci Sports Exerc* 26: 1160–1164, 1994.

33. Rutherford, OM and Jones, DA. Measurement of fibre pennation using ultrasound in the human quadriceps in vivo. *Eur J Appl Physiol Occup Physiol* 65: 433–437, 1992.

34. Sampson, JA and Groeller, H. Is repetition failure critical for the development of muscle hypertrophy and strength? *Scand J Med Sci Sports* 26: 375–383, 2016.

35. Schoenfeld, BJ, Contreras, B, Willardson, JM, Fontana, F, and Tiryaki-Sonmez, G. Muscle activation during low- versus high-load resistance training in well-trained men. *Eur J Appl Physiol* 114: 2491–2497, 2014.

36. Schoenfeld, BJ, Peterson, MD, Ogborn, D, Contreras, B, and Sonmez, GT. Effects of low- vs. high-load resistance training on muscle strength and hypertrophy in well-trained men. *J Strength Cond Res* 29: 2954–2963, 2015.

37. Schott, J, McCully, K, and Rutherford, OM. The role of metabolites in strength training. II. Short versus long isometric contractions. *Eur J Appl Physiol Occup Physiol* 71: 337–341, 1995.

38. Available at: http://seniam.org/sensor_location.htm. Accessed: January 12, 2016.

39. Stone, MH, Chandler, TJ, Conley, MS, Kramer, JB, and Stone, ME. Training to muscular failure: Is it necessary? *Strength Cond J* 18: 44–48, 1996.

40. Sundstrup, E, Jakobsen, MD, Andersen, CH, Zebis, MK, Mortensen, OS, and Andersen, LL. Muscle activation strategies during strength training with heavy loading vs. repetitions to failure. *J Strength Cond Res* 26: 1897–1903, 2012.

41. Vigotsky, AD, Beardsley, C, Contreras, B, Steele, J, Ogborn, D, and Phillips, SM. Greater electromyographic responses do not imply greater motor unit recruitment and "hypertrophic potential" cannot be inferred. *J Strength Cond Res* 31: e1–e4, 2017.

42. Wakahara, T, Fukutani, A, Kawakami, Y, and Yanai, T. Nonuniform muscle hypertrophy: Its relation to muscle activation in training session. *Med Sci Sports Exerc* 45: 2158–2165, 2013.

43. Westad, C, Westgaard, RH, and De Luca, CJ. Motor unit recruitment and derecruitment induced by brief increase in contraction amplitude of the human trapezius muscle. *J Physiol* 552: 645–656, 2003.

44. Willardson, JM. The application of training to failure in periodized multiple-set resistance exercise programs. *J Strength Cond Res* 21: 628–631, 2007.

45. Yao, W, Fuglevand, RJ, and Enoka, RM. Motor-unit synchronization increases EMG amplitude and decreases force steadiness of simulated contractions. *J Neurophysiol* 83: 441–452, 2000.