Effects of velocity loss during resistance training on athletic performance, strength gains and muscle adaptations

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We compared the effects of two resistance training (RT) programs only differing in the repetition velocity loss allowed in each set: 20% (VL20) vs 40% (VL40) on muscle structural and functional adaptations. Twenty-two young males were randomly assigned to a VL20 (n = 12) or VL40 (n = 10) group. Subjects followed an 8-week velocity-based RT program using the squat exercise while monitoring repetition velocity. Pre- and post-training assessments included: magnetic resonance imaging, vastus lateralis biopsies for muscle cross-sectional area (CSA) and fiber type analyses, one-repetition maximum strength and full load-velocity squat profile, countermovement jump (CMJ), and 20-m sprint running. VL20 resulted in similar squat strength gains than VL40 and greater improvements in CMJ (9.5% vs 3.5%, P < 0.05), despite VL20 performing 40% fewer repetitions. Although both groups increased mean fiber CSA and whole quadriceps muscle volume, VL40 training elicited a greater hypertrophy of vastus lateralis and intermedius than VL20. Training resulted in a reduction of myosin heavy chain IIX percentage in VL40, whereas it was preserved in VL20. In conclusion, the progressive accumulation of muscle fatigue as indicated by a more pronounced repetition velocity loss appears as an important variable in the configuration of the resistance exercise stimulus as it influences functional and structural neuromuscular adaptations.

The adaptive response to resistance training (RT) depends on several variables that configure the resistance exercise stimulus such as loading magnitude, number of sets and repetitions, exercise type and order, rests duration, and movement velocity (Spiering et al., 2008; Sánchez-Medina & González-Badillo, 2011). It has been shown that velocity loss and metabolic stress considerably differ depending on the actual number of repetitions performed in an exercise set in relation to the maximum number that can be completed (Sánchez-Medina & González-Badillo, 2011). Although some studies (Rooney et al., 1994; Ahtiainen et al., 2003; Drinkwater et al., 2005) suggest that performing repetitions to failure may be necessary to maximize muscle mass and strength, others seem to indicate that similar, if not greater, strength gains and improvements in athletic performance can be obtained without reaching muscle failure (Folland et al., 2002; Izquierdo et al., 2006; Izquierdo-Gabarren et al., 2010). It has been hypothesized that RT eliciting high levels of fatigue, as it occurs in typical body-building routines, may induce greater strength adaptations due to an enhanced activation of motor units and secretion of growth-promoting hormones (Schott et al., 1995; Schoenfeld, 2010). However, definitive evidence is lacking and the controversial results found in the literature clearly emphasize the need to conduct further research on this topic.

Experiments with isolated human muscle fibers (Mogensen et al., 2006), as well as in vivo human studies (Aagaard & Andersen, 1998; Sanchis-Moysi et al., 2010) have shown that a high proportion of type II muscle fibers or myosin heavy chain (MHC) II isoforms is associated with high levels of force production during fast muscle contractions. Interestingly, most studies have shown that the percentage of type IIX fibers is reduced following a RT program based on repetitions to failure (Staron et al., 1991; Andersen & Aagaard, 2000; Campos et al., 2002; Andersen et al., 2005). Nevertheless, a study by Harridge et al. (1998) showed that maximal isometric strength (voluntary and electrically evoked) can be significantly increased without a reduction in the
MHC-IIX fiber pool following a 6-week training program based on 4 sessions/week of high-intensity, short-duration, cycling exercise (three 3-s sprints with 30-s recoveries), aimed to avoid a decline in performance during the training session.

During RT muscle fatigue increases with the accumulation of repetitions, and if the exercise is not stopped, task failure eventually occurs. However, prior to task failure, other signs of muscle fatigue are detectable, such as reduced maximal force application, slower shortening velocity and decreased power output (Allen et al., 1995; Sánchez-Medina & González-Badillo, 2011; Gorostiaga et al., 2012). The complexity of fatigue assessment has led to the utilization of procedures that lack specificity. It has been shown that neuromuscular fatigue induced by RT protocols can be monitored by assessing the repetition velocity loss within a set (Sánchez-Medina & González-Badillo, 2011). A novel, velocity-based approach to RT has been proposed in which, rather than prescribing a fixed number of repetitions to perform with a given load, training is configured using two variables: (a) first repetition’s mean velocity, which is intrinsically related to relative loading magnitude (González-Badillo & Sánchez-Medina, 2010); and (b) the velocity loss to be allowed, expressed as a percent loss in mean velocity from the fastest (usually first) repetition of each exercise set (Sánchez-Medina & González-Badillo, 2011). Thus, when the prescribed percent velocity loss limit is exceeded, the set is terminated.

In an attempt to gain further insight into the adaptations brought about by training close to muscle failure vs not to failure, we aimed to compare the effects of two RT programs that only differed in the magnitude of repetition velocity loss allowed in each set (20% vs 40%) on structural and functional adaptations. We hypothesized that, despite a remarkably lower training volume, improvements in strength and performance will be greater with the RT program allowing only a 20% reduction in repetition velocity, whereas a 40% velocity loss will result in greater muscle hypertrophy.

Materials and methods

Subjects

Twenty-four young and healthy men (age 22.7 ± 1.9 years, height 1.76 ± 0.06 m, body mass 75.8 ± 7.0 kg) volunteered to participate in this study. Their initial 1RM strength for the heaviest loads (>50% 1RM), two for medium (50–80% 1RM), and only one for the heaviest loads (>80% 1RM). Strong verbal encouragement was provided to motivate participants to give a maximal effort. Inter-set recoveries ranged from 3 min (light) to 5 min (heavy loads). Only the best repetition at each load, according to the criteria of fastest MPV was considered for subsequent analysis. All velocity measures reported in this study corresponded to the mean velocity of the propulsive phase of each repetition. The propulsive phase was defined as that fraction

Velocity loss as a strength training variable

During the course of the study, two subjects from the VL40 dropped out because of injury (not related to the training intervention) or illness; thus, the complete dataset for the VL40 group was obtained from the remaining 10 subjects. All subjects were informed about the experimental procedures and potential risks before they provided their written informed consent. The study was approved by the institutional review committee of Pablo de Olavide University, and was performed in accordance with the Declaration of Helsinki. No physical limitations, health problems, or musculoskeletal injuries that could affect training were found after a medical examination. None of the subjects were taking drugs, medications, or dietary supplements.

Study design

Subjects trained twice a week (48–72 h apart) during 8-week for a total of 16 sessions. A progressive RT program which comprised only the SQ exercise was used (Table 1). The two groups trained at the same relative loading magnitude (percentage of one-repetition maximum, %1RM) in each session but differed in the maximum percent velocity loss reached in each exercise set (20% vs 40%). As soon as the corresponding target velocity loss limit was exceeded, the set was terminated. Sessions were performed in a research laboratory under the direct supervision of the investigators, at the same time of day (±1 h) for each subject and under controlled environmental conditions (20 °C and 60% humidity). Subjects were required not to engage in any other type of strenuous physical activity, exercise training, or sports competition for the duration of the present investigation. Both VL20 and VL40 groups were assessed on two occasions: 48 h before (Pre) and 72 h after (Post) the 8-week training intervention. Training compliance was 100% of all sessions for the subjects that completed the intervention.

Testing Procedures

Isoinertial strength assessment

A progressive loading test up to the 1RM load was performed in the SQ exercise. The SQ was performed with subjects starting from the upright position with the knees and hips fully extended, parallel feet stance approximately shoulder-width extended, parallel feet stance approximately shoulder-width extended, and the barbell resting across the back at the level of the acromion. Each subject descended in a continuous motion until the top of the thighs was below the horizontal plane, the posterior thighs and shanks making contact with each other (~35–40° knee flexion), then immediately reversed motion and raised back to the upright position. Unlike the eccentric phase that was performed at a controlled mean velocity (~0.50–0.65 m/s), subjects were required to always execute the concentric phase at maximal intended velocity. Initial load was set at 30 kg and was progressively increased in 10-kg increments until the attained mean propulsive velocity (MPV) was ≤0.6 m/s. Thereafter, load was individually adjusted with smaller increments (5 down to 2.5 kg) so that 1RM could be precisely determined. Three repetitions were executed for light (≤50% 1RM), two for medium (50–80% 1RM), and only one for the heaviest loads (>80% 1RM). Strong verbal encouragement was provided to motivate participants to give a maximal effort. Inter-set recoveries ranged from 3 min (light) to 5 min (heavy loads). Only the best repetition at each load, according to the criteria of fastest MPV was considered for subsequent analysis. All velocity measures reported in this study corresponded to the mean velocity of the propulsive phase of each repetition. The propulsive phase was defined as that fraction
| Actually performed | Session 1 | Session 2 | Session 3 | Session 4 | Session 5 | Session 6 | Session 7 | Session 8 | Session 9 |
|--------------------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|
| **Best MPV (%1RM)**|           |           |           |           |           |           |           |           |           |
| VL20               | 0.85 ± 0.05 | 0.85 ± 0.03 | 0.87 ± 0.04 | 0.86 ± 0.05 | 0.84 ± 0.03 | 0.83 ± 0.03 | 0.78 ± 0.03 | 0.78 ± 0.02 | 0.78 ± 0.03 |
|                    | (~69% 1RM) | (~69% 1RM) | (~67% 1RM) | (~68% 1RM) | (~69% 1RM) | (~70% 1RM) | (~73% 1RM) | (~73% 1RM) | (~73% 1RM) |
| VL40               | 0.85 ± 0.04 | 0.86 ± 0.05 | 0.83 ± 0.05 | 0.85 ± 0.03 | 0.82 ± 0.04 | 0.80 ± 0.06 | 0.76 ± 0.03 | 0.76 ± 0.06 | 0.76 ± 0.03 |
|                    | (~69% 1RM) | (~68% 1RM) | (~70% 1RM) | (~69% 1RM) | (~70% 1RM) | (~72% 1RM) | (~75% 1RM) | (~75% 1RM) | (~75% 1RM) |
| **VL (%)**         |           |           |           |           |           |           |           |           |           |
| VL20               | 22.7 ± 2.6 | 21.9 ± 3.0 | 21.4 ± 3.9 | 19.4 ± 1.6 | 21.6 ± 2.3 | 21.2 ± 3.1 | 21.2 ± 2.8 | 19.6 ± 2.3 | 19.9 ± 2.2 |
| VL40               | 23.3 ± 2.1 | 31.9 ± 2.6 | 40.9 ± 2.6 | 49.7 ± 3.9 | 48.6 ± 2.2 | 47.9 ± 7.9 | 36.7 ± 2.0 | 45.6 ± 2.7 | 50.5 ± 2.9 |
| **Rep per set**    |           |           |           |           |           |           |           |           |           |
| VL20               | 5.2 ± 1.0  | 5.0 ± 1.0  | 5.0 ± 1.4  | 5.3 ± 1.2  | 5.4 ± 1.1  | 4.4 ± 0.6  | 3.9 ± 0.6  | 4.3 ± 1.0  | 3.9 ± 0.8  |
| VL40               | 5.3 ± 1.3  | 6.9 ± 1.8  | 7.7 ± 1.4  | 9.4 ± 1.8  | 9.2 ± 1.1  | 9.4 ± 2.3  | 6.1 ± 1.2  | 7.3 ± 1.4  | 7.5 ± 1.0  |

| Actually performed | Session 10 | Session 11 | Session 12 | Session 13 | Session 14 | Session 15 | Session 16 | Overall |
|--------------------|------------|------------|------------|------------|------------|------------|------------|---------|
| **Best MPV (%1RM)**|           |           |           |           |           |           |           |         |
| VL20               | 0.78 ± 0.03 | 0.69 ± 0.03 | 0.70 ± 0.03 | 0.70 ± 0.04 | 0.63 ± 0.02 | 0.62 ± 0.03 | 0.63 ± 0.03 | 0.76 ± 0.01 |
|                    | (~73% 1RM) | (~79% 1RM) | (~78% 1RM) | (~78% 1RM) | (~83% 1RM) | (~84% 1RM) | (~83% 1RM) | (~75% 1RM) |
| VL40               | 0.76 ± 0.03 | 0.69 ± 0.03 | 0.69 ± 0.04 | 0.69 ± 0.04 | 0.61 ± 0.04 | 0.60 ± 0.03 | 0.63 ± 0.04 | 0.75 ± 0.02 |
|                    | (~75% 1RM) | (~79% 1RM) | (~79% 1RM) | (~79% 1RM) | (~84% 1RM) | (~85% 1RM) | (~83% 1RM) | (~75% 1RM) |
| **VL (%)**         |           |           |           |           |           |           |           |         |
| VL20               | 20.6 ± 3.4 | 23.1 ± 6.2 | 22.4 ± 4.4 | 23.2 ± 4.8 | 15.9 ± 4.1 | 15.2 ± 3.1 | 14.3 ± 3.8 | 20.4 ± 1.5 |
| VL40               | 50.1 ± 2.3 | 37.0 ± 3.7 | 44.7 ± 3.4 | 45.9 ± 2.9 | 33.1 ± 5.0 | 43.8 ± 5.1 | 43.8 ± 5.5 | 41.9 ± 1.9 |
| **Rep per set**    |           |           |           |           |           |           |           |         |
| VL20               | 3.8 ± 0.4  | 3.0 ± 0.4  | 3.3 ± 0.8  | 3.3 ± 1.0  | 2.0 ± 0.4  | 2.1 ± 0.4  | 1.9 ± 0.8  | 3.9 ± 0.5  |
| VL40               | 7.3 ± 1.4  | 4.6 ± 1.0  | 5.8 ± 1.8  | 5.2 ± 1.3  | 3.3 ± 0.8  | 3.7 ± 0.6  | 4.5 ± 0.7  | 6.5 ± 0.9  |

Data are mean ± SD. Only one exercise (full squat) was used in training.

VL20: Group that trained with a mean velocity loss of 20% in each set (n = 12); VL40: Group that trained with a mean velocity loss of 40% in each set (n = 10); Best MPV: The fastest mean propulsive velocity attained with the intended load (%1RM); VL: Magnitude of velocity loss expressed as percent loss in mean repetition velocity from the fastest (usually first) to the slowest (last one) repetition of each set; Rep per set: actual number of repetitions performed in each set.
Velocity loss as a strength training variable

Sprint and vertical jump tests

Vertical jump and sprint ability were assessed as indicators of explosive force production and lower limb whole muscle performance. Two maximal 20-m sprints, separated by a 3-min rest, were performed in an indoor running track. Photocell timing gates were placed at 0 and 20 m so that the times to cover 0–20 m (T20) could be determined. A standing start with the lead-off foot placed 1 m behind the first timing gate was used. Subjects were required to give an all-out maximal effort in each sprint and the best of both trials was kept for analysis. The same warm-up protocol which incorporated several sets of progressively faster 30-m running accelerations was followed at Pre and Post. Sprint times were measured using photocells (Polifemo Radio Light, Microgate, Bolzano, Italy). Five maximal countermovement vertical jumps (CMJs), separated by 20-s rests, were performed next. The highest and lowest CMJ height values were discarded, and the resulting average kept for analysis. Jump height was determined using an infrared timing system (Optojump, Microgate, Bolzano, Italy). Test–retest reliability measured by the coefficient of variation (CV) were 0.9% and 1.5% for T20 and CMJ, respectively. The intraclass correlation coefficients (ICCs) were 0.957 (95% confidence interval, CI: 0.903–0.981) for T20, and 0.995 (95% CI: 0.990–0.998) for CMJ.

Muscle biopsy sampling

Subjects reported to the laboratory at 7:00 a.m. after a 12-h overnight fast. The dinner previous to the biopsy day was standardized for Pre and Post tests. After a 10-min rest in the supine position, the middle portion of the vastus lateralis (VL) muscle was anesthetized with 2% lidocaine (2 mL). Thereafter, muscle biopsy samples (80–160 mg) were extracted from the superficial region (2–3 cm depth) of the VL muscle using the Bergstrom technique with suction (Perez-Gomez et al., 2008). In order to minimize sampling error, all biopsies were performed by the same medical doctor and great care was taken to standardize the site and depth of the sample. Upon collection, muscle samples, weighing 75–100 mg, were divided into two. The first half was mounted on cork blocks with the use of Tissue-Tek® O.C.T.™ embedding medium an orientated so that myofibers could be transversely cut. Specimens were systematically frozen by immersion (10–15 s) in isopentane, kept at freezing point in liquid nitrogen. The second half was immediately frozen in liquid nitrogen. Both biopsy pieces were stored at −80 °C until analyzed. Biopsy samples were obtained and frozen in less than 30 s at Pre and Post (48 h after the last testing session). Because of possible variations in fiber type distribution from superficial to deep and proximal to distal (Blomstrand & Ekblom, 1982), we attempted to extract Pre and Post tissue samples from within a small area of the muscle. We used the pre-biopsy scar to identify the location from where the post-biopsy should be obtained, which was located 3 cm away from the pre-biopsy site.

Fiber type and muscle cross-sectional area determinations

Serial sections (10 μm) of the muscle biopsy samples were cut successively in a cryostat (−20 °C) and carefully placed on microscope slides. To determine the muscle fiber type composition, adenosine triphosphatase (ATPase) histochemistry was performed using pre-incubation pH values of 4.37, 4.60, and 10.30 (Brooke & Kaiser, 1970). According to Staron et al. (1991), five different fiber types were defined (types I, IIC, IIA, IIX, and IIX, Fig. 1a). Cross-sections from Pre and Post biopsies from the same individual were placed on the same slide so that they could be processed simultaneously for ATPase histochemistry. Only truly horizontally cut fibers were used in the determination of fiber size. An average of 180 ± 27 fibers was examined in each of the biopsies. The number of IIC fibers was too low in some individuals as to allow a reliable statistical analysis. The serial sections were visualized and analyzed using an Olympus BX40 microscope (Olympus Optical Co., Tokyo, Japan), an Olympus camera (DP 26, Olympus Optical Co.), combined with image analysis software (Olympus CellSens Standard, Tokyo, Japan). Using the 4.60 ATPase staining, a fiber mask was drawn manually following the boundaries of the fibers. This mask was overimposed on the images obtained with the additional ATPase stainings. Fiber types were then identified according to staining properties at different pHs and the cross-sectional area (CSA) was determined. The relative fiber type area was calculated as the product of average fiber type size by percentage distributions.

Protein extraction for MHC

For total protein extraction from human skeletal muscle, a piece of frozen tissue (10–20 mg) was pulverized with grinding balls at 3000 rpm for 1 min in a Mikro-Dismembrator S (Sartorius, Goettingen, Germany). Proteins were extracted in urea-lysis buffer [6 M urea, 1% (wt/vol) SDS, 1X of Complete protease inhibitor, and PhosSTOP phosphatase inhibitor (Roche Diagnostics, Mannheim, Germany)] by the use of sonication (75 W, 3 pulses of 10 s). After centrifugation at 20,000 g to remove tissue debris, total protein extracts were transferred to clean tubes, and protein quantification was performed by bicinchoninic acid assay (Smith et al., 1985).
MHC analysis

MHC analysis was performed on the muscle biopsies using sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) as reported by Larsson et al. (2002). Different amounts of protein extracts (range: 2–4 μL) were loaded on a SDS-PAGE gel. Gels were run in a Protean II unit (Bio-Rad Laboratories Inc., Hercules, California, USA), at 70 V for 40 h followed by 7 h at 120 V, both at 4 °C. Subsequently, the gels were Coomassie stained. This method resolved MHCs in three bands: I, IIA, and IIX (Fig. 1b) from the fastest to the slowest migration band (Biral et al., 1988). Gels were then scanned with a video-scanning densitometric system and quantified with the image analysis software Quantity One (Bio-Rad Laboratories Inc.). The quantification of each MHC isoform was obtained in relative terms for each muscle biopsy sample.

Anatomical muscle CSA and volume

A 1.5-T scanner (General Electric, Milwaukee, Wisconsin, USA) was used to acquire 4-mm axial contiguous slices from the more distal edge of the femur condyles to the iliac spine with the subjects lying supine with extended knees. Axial gradient-echo T1-weighted images of the thigh were collected from both legs simultaneously using a repetition time of 132 ms and an echo time of 4.2 ms, a flip angle of 80° with a 42 cm² field of view, and a matrix of 256 × 256 pixels (in-plane spatial resolution = 1.64 mm × 1.64 mm).

The boundaries of the VL, vastus intermedius (VI), vastus medialis (VM), and rectus femoris (RF) muscles were manually outlined slice-by-slice using a specially designed image analysis software (SliceOmatic 4.3, Tomovision Inc., Montreal, Quebec, Canada), as described elsewhere (Lee et al., 2000). A representative example is depicted in Fig. 1c. Two investigators with expert anatomic knowledge and experience in muscle segmentation analysis manually traced all images (Sanchis-Moysi et al., 2010, 2011, 2012). One observer outlined the VL, the VI, and the VM, and the other one outlined the RF. Examiners were blinded to the allocation of the subjects to the VL20 or VL40 groups.

The total volume of VL, VI, VM and RF was calculated from the most distal slice where the VM could be first identified to the most proximal slice where the RF starts to be seen. Because substantial fusion may be found between VL and VI on some slices (Barnouin et al., 2014), these two muscles were outlined together (VL+VI). Muscle length was calculated as the number of slices from the proximal to the distal reference times the slice thickness. The degree of hypertrophy was calculated by subtracting the muscle volume at Pre from the muscle volume at Post, expressed as a percentage of the Pre value. The intra-observer coefficient of variation for muscle segmentation in our laboratory is below 2% (Sanchis-Moysi et al., 2010, 2011, 2012).

Resistance training program

Descriptive characteristics of the RT program are presented in Table 1. Both VL20 and VL40 groups trained using only the
SQ exercise as previously described. Relative magnitude of training loads (%1RM), number of sets (three), and inter-set recoveries (4 min) were kept identical for both groups in each training session. Relative loads were determined from the load–velocity relationship for the SQ as it has recently been shown that there exists a very close relationship between %1RM and MPV (González-Badillo & Sánchez-Medina, 2010; Sánchez-Medina et al., 2014). Thus, a target MPV to be attained in the first (usually the fastest) repetition of the 1st exercise set in each session was used as an estimation of %1RM, as follows: 0.82 m/s (~70% 1RM), 0.75 m/s (~75% 1RM), 0.68 m/s (~80% 1RM), and 0.60 m/s (~85% 1RM); i.e., a velocity-based training was actually performed, instead of a traditional loading-based RT program (González-Badillo et al., 2014; Pareja-Blanco et al., 2014). The absolute load (kg) was individually adjusted to match the velocity associated (±0.03 m/s) with the %1RM that was intended for that session. Thus, both groups trained using the same loading magnitude in each session (progressively increasing from 70% to 85% 1RM over the time course of the study, Table 1) but differed in the degree of neuromuscular fatigue experienced during the exercise sets, which was objectively quantified by the magnitude of velocity loss attained in each set (Sánchez-Medina & González-Badillo, 2011). A 40% velocity loss limit for the SQ exercise (VL40 group) implies performing repetitions to, or very close to, muscle failure in most exercise sets (Sánchez-Medina & González-Badillo, 2011). In contrast, a 20% velocity loss corresponds to performing approximately half the maximum possible number of repetitions per set in the SQ exercise, as previously reported (Sánchez-Medina & González-Badillo, 2011). During training, subjects received immediate velocity feedback while being encouraged to perform each repetition at maximal intended velocity. Total work was calculated from the data provided by the linear velocity transducer as the sum of the force (weight lifted) did not differ between groups (0.75 ± 0.03 m/s, respectively; P < 0.001), whereas VL40 performed more repetitions (P < 0.001) than VL20 (310.5 ± 42.0 vs 185.9 ± 22.2). The mean fastest repetition during each session (which indicates the relative magnitude of the load being lifted) did not differ between groups (0.76 ± 0.01 m/s, for VL40 and VL20, respectively).

Statistical analyses

Values are reported as mean ± standard deviation (SD). Test–retest absolute reliability was assessed using the CV, whereas relative reliability was calculated by the ICC with 95% confidence interval (CI), using the one-way random effects model. The normality of distribution of the variables at Pre was examined with the Shapiro–Wilk test and the homogeneity of variance across groups (VL20 vs VL40) was verified using the Levene’s test. Data were analyzed using a 2 × 2 factorial ANOVA with Bonferroni’s post-hoc comparisons using one between factor (VL20 vs VL40) and one within factor (Pre vs Post). Statistical significance was established at the P ≤ 0.05 level. All statistical analyses were performed using SPSS software version 18.0 (IBM, Chicago, Illinois, USA).

Results

No significant differences between the VL20 and VL40 groups were found at Pre for any of the variables analyzed. Descriptive characteristics of the training actually performed by both groups are reported in Table 1. The total number of repetitions and the repetitions performed in different velocity ranges by each group are shown in Fig. 2. Subjects from the VL20 group trained at a significantly faster mean velocity than those from VL40 (0.69 ± 0.02 vs 0.58 ± 0.03 m/s, respectively; P < 0.001), whereas VL40 performed more repetitions (P < 0.001) than VL20 (310.5 ± 42.0 vs 185.9 ± 22.2). The mean fastest repetition during each session (which indicates the relative magnitude of the load being lifted) did not differ between groups (0.75 ± 0.03 vs 0.76 ± 0.01 m/s, for VL40 and VL20, respectively).

![Fig. 2. Number of repetitions in the squat exercise performed in each velocity range, and total number of repetitions completed by both training groups. Data are mean ± SD. Statistically significant differences between groups: ***P < 0.01, ****P < 0.001. VL20: group that trained with a mean velocity loss of 20% in each set (n = 12); VL40: group that trained with a mean velocity loss of 40% in each set (n = 10). Warm-up repetitions are excluded.](image-url)
and initial repetition velocities matched the expected target velocities for every training session. Subjects from the VL40 group reached muscle failure during 27.0 ± 4.2 sets (56.3% of total training sets). The mean magnitude of velocity loss matched that intended for each group (41.9 ± 1.7% vs 20.4 ± 1.5% for VL40 and VL20, respectively). Total work was significantly greater for VL40 compared to VL20 (200.6 ± 47.1 vs 127.5 ± 15.2 kJ, \( P < 0.001 \)).

Isoinertial strength assessments

No significant ‘group’ × ‘time’ interactions were observed for any of the isoinertial strength variables analyzed. Following the training intervention, statistically significant increases were observed in 1RM strength (18.0% and 13.4%), AV (12.5% and 6.0%), and AV<1 (21.7% and 13.7%) for VL20 and VL40 groups, respectively (Table 2). AV>1 improved in VL20 (6.2%, \( P < 0.01 \)) but remained unchanged (+1.0%, \( P = 0.62 \)) in VL40 (group × time interaction \( P = 0.09 \)).

Vertical jump and sprint ability

CMJ height was increased by 9.5% in the VL20 group (\( P < 0.001 \)), while it remained unchanged (+3.5% \( P = 0.07 \)) in the VL40 (group × time interaction \( P < 0.05 \)) (Table 2). No statistically significant changes in sprint running performance were observed in any group (Table 2).

Fiber type distribution and MHC content

The mean CSA of muscle fibers increased similarly in both groups (+10.5%, time effect \( P < 0.01 \)). The increase in mean CSA was explained by an increase of type I CSA (+9.9% time effect \( P < 0.01 \)) and la T con minúscula: type II (+11.1% time effect \( P < 0.05 \)) (Table 3). No significant changes were observed in muscle fiber type (ATPase analysis method, Table 3). However, the percentage of MHC-IIx decreased in VL40 (\( P < 0.001 \)) while it remained unchanged in VL20 (group × time interaction \( P < 0.05 \), Table 3).

Anatomical muscle CSA and volume

Total quadriceps femoris volume (Fig. 3a) was increased by 6.0% (time effect \( P < 0.05 \)). This was explained by a significant increase of VM volume (Fig. 3c) in both groups, whereas the VL+VI volume (Fig. 3d) only increased in the VL40 group (group × time interaction, \( P = 0.05 \)). RF muscle volume remained unaltered in both groups (Fig. 3b). The total length of RF (Pre 31.6 ± 2.0 vs Post 31.1 ± 2.2 cm for VL40, and Pre 32.0 ± 2.2 vs Post 31.7 ± 1.9 cm for VL20), VM (Pre 33.2 ± 1.9 vs Post 33.8 ± 2.6 cm for VL40, and Pre 33.7 ± 2.1 vs Post 33.6 ± 1.4 cm for VL20), and VL+VI (Pre 33.3 ± 3.0 vs Post 34.0 ± 2.3 cm for VL40, and Pre 33.4 ± 1.9 vs Post 33.0 ± 2.0 cm for VL20) muscles did not change for any group before and after the RT intervention.

Discussion

To the best of our knowledge, this is the first study that has analyzed the effect of two isoinertial RT programs differing in the magnitude of velocity loss experienced during each exercise set on muscle structure and performance. Although both intervention groups trained using the same relative loading magnitude (%1RM) in each session and performed repetitions at maximal intended velocity, a low mag-

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Table 2. Changes in selected neuromuscular performance variables from pre- to post-training for each group

|            | VL40          |        |         | VL20          |        |         |
|------------|---------------|--------|---------|---------------|--------|---------|
|            | Pre           | Post   | \( P \)-value | Pre           | Post   | \( P \)-value |
| 1RM (kg)   | 104.5 ± 15.1  | 118.6 ± 20.4 | <0.001 | 106.5 ± 12.2  | 125.2 ± 12.3 | <0.001 |
| AV (m/s)   | 0.95 ± 0.06   | 1.01 ± 0.09  | 0.03  | 0.95 ± 0.06   | 1.06 ± 0.06  | <0.001 |
| AV<1 (m/s) | 1.22 ± 0.03   | 1.23 ± 0.08  | 0.62  | 1.21 ± 0.05   | 1.29 ± 0.07  | 0.005 |
| AV>1 (m/s) | 0.72 ± 0.04   | 0.81 ± 0.07  | 0.001 | 0.72 ± 0.04   | 0.87 ± 0.07  | <0.001 |
| CMJ (cm)   | 41.0 ± 4.3    | 42.5 ± 5.8   | 0.06  | 40.5 ± 6.0    | 44.2 ± 6.0  | <0.001 |
| T20 (s)    | 2.99 ± 0.09   | 3.02 ± 0.08  | 0.25  | 3.00 ± 0.11   | 2.99 ± 0.10  | 0.45   |

Data are mean ± SD; \( P \)-values calculated using Bonferroni adjustment.
VL20: group that trained with a mean velocity loss of 20% in each set (\( n = 12 \)); VL40: group that trained with a mean velocity loss of 40% in each set (\( n = 10 \)).

1RM: one-repetition maximum squat strength, AV: average MPV attained against absolute loads common to pre- and post-test in the squat progressive loading test; AV<1: average MPV attained against absolute loads common to pre- and post-test that were moved faster than 1 m/s; AV>1: average MPV attained against absolute loads common to pre- and post-test that were moved slower than 1 m/s; CMJ: countermovement jump height, T20: 20-m sprint time.
Velocity loss as a strength training variable

Table 3. Changes in muscle cross-sectional areas and muscle fiber types percentages, from pre- to post-training for each group, using myofibrillar adenosine triphosphatase histochemistry

|                | Pre  | Post | P-value |                | Pre  | Post | P-value |                | Pre  | Post | P-value |
|----------------|------|------|---------|----------------|------|------|---------|----------------|------|------|---------|
| CSA muscle fibers (ATPase) |      |      |         |                |      |      |         |                |      |      |         |
| CSA (µm²)      | 4935 ± 690 | 5438 ± 788   | 0.02 | 4800 ± 691 | 5217 ± 701 | 0.05 | 0.005 | 0.77 |
| CSA-I (µm³)    | 4314 ± 676 | 4798 ± 804   | 0.01 | 4070 ± 834 | 4346 ± 873   | 0.13 | 0.007 | 0.41 |
| CSA-IIA (µm³)  | 5584 ± 1259 | 6233 ± 998   | 0.05 | 5708 ± 893 | 6169 ± 716   | 0.16 | 0.03  | 0.68 |
| CSA-IIX (µm²)  | 4619 ± 1022 | 5260 ± 962   | 0.04 | 4936 ± 740 | 5146 ± 774   | 0.49 | 0.06  | 0.31 |
| CSA-IIX (µm³)  | 4406 ± 1037 | 4927 ± 1502  | 0.30 | 4130 ± 930 | 4853 ± 1016  | 0.16 | 0.09  | 0.77 |
| Percentage fiber area (ATPase) |      |      |         |                |      |      |         |                |      |      |         |
| Type I (%)     | 44.3 ± 10.4 | 47.5 ± 9.8   | 0.25 | 45.9 ± 15.7 | 43.7 ± 13.4  | 0.39 | 0.78  | 0.15 |
| Type IIC (%)   | 0.1 ± 0.2  | 0.3 ± 0.6   | 0.87 | 0.5 ± 1.1  | 1.6 ± 4.9    | 0.22 | 0.34  | 0.48 |
| Type IIA (%)   | 36.5 ± 9.7 | 36.4 ± 7.6   | 0.98 | 33.8 ± 10.2 | 38.5 ± 11.0  | 0.13 | 0.31  | 0.29 |
| Type IIX (%)   | 11.2 ± 6.1 | 12.0 ± 6.3   | 0.71 | 13.7 ± 11.2 | 10.1 ± 7.6   | 0.07 | 0.32  | 0.13 |
| Type IIX (%)   | 7.8 ± 7.0  | 3.8 ± 5.0   | 0.04 | 6.3 ± 8.9  | 6.1 ± 8.2    | 0.91 | 0.10  | 0.14 |
| Percentage fiber area (ATPase) |      |      |         |                |      |      |         |                |      |      |         |
| Type I (%)     | 38.8 ± 10.0 | 42.5 ± 10.7  | 0.14 | 38.9 ± 16.3 | 37.9 ± 16.2  | 0.68 | 0.43  | 0.18 |
| Type IIC (%)   | 0.1 ± 0.2  | 0.3 ± 0.6   | 0.23 | 0.6 ± 1.2  | 1.8 ± 5.3    | 0.39 | 0.34  | 0.47 |
| Type IIA (%)   | 42.4 ± 11.7 | 41.8 ± 9.1   | 0.59 | 39.4 ± 12.7 | 43.8 ± 12.8  | 0.69 | 0.48  | 0.36 |
| Type IIX (%)   | 11.2 ± 6.8 | 11.9 ± 6.8   | 0.77 | 15.1 ± 11.9 | 10.5 ± 6.8   | 0.06 | 0.24  | 0.12 |
| Type IIX (%)   | 7.5 ± 6.9  | 3.6 ± 4.8   | 0.05 | 6.0 ± 8.2  | 6.1 ± 8.2    | 0.96 | 0.15  | 0.13 |
| MHC percentage |      |      |         |                |      |      |         |                |      |      |         |
| MHC-I (%)      | 42.8 ± 7.9 | 45.5 ± 7.6   | 0.30 | 40.0 ± 8.6 | 39.3 ± 9.3   | 0.77 | 0.56  | 0.33 |
| MHC-IIA (%)    | 42.6 ± 3.8 | 47.3 ± 5.9   | 0.05 | 42.9 ± 5.4 | 45.8 ± 8.6   | 0.18 | 0.02  | 0.56 |
| MHC-IIX (%)    | 14.6 ± 8.9 | 7.2 ± 7.6    | <0.001 | 17.0 ± 7.4 | 14.8 ± 8.2   | 0.18 | 0.001 | 0.04 |

Data are mean ± SD; P-values calculated using Bonferroni adjustment. VL20: group that trained with a mean velocity loss of 20% in each set (n = 12); VL40: group that trained with a mean velocity loss of 40% in each set (n = 10).

CSA, cross-sectional area; MHC, myosin heavy chain.

Drinkwater et al., 2005) reported that RT to failure induced greater strength gains compared with training not leading to failure. However, in these two studies repetition velocity during training was not monitored, nor it was intended to be maximal. Training at maximal voluntary velocity has recently been shown to be of paramount importance for maximizing strength gains and athletic performance (jumping ability) (González-Badillo et al., 2014; Pareja-Blanco et al., 2014). In agreement with the present findings, it has been reported that muscle failure might not be necessary to attain greater strength gains (Folland et al., 2002; Izquierdo et al., 2006; Izquierdo-Gabarren et al., 2010; Sampson & Groeller, 2015). In the present study, similar gains in 1RM were observed for VL20 vs VL40 (18.0% vs 13.4%). In the same line, Izquierdo et al. (2006) reported that 16 weeks of RT to failure vs not to failure resulted in similar 1RM gains (~22%) in the bench press and parallel squat. However, to the best of our knowledge, in previous studies on this topic, the impact of RT on dynamic muscle performance was limited to the evaluation of maximum strength disregarding potential effects at different regions of the load–velocity relationship.

In the present investigation, we assessed changes in average velocity attained against all absolute loads common to Pre and Post SQ tests, from light to heavy, as well as changes in CMJ height. The VL20 group obtained similar squat strength gains and even...
greater improvements in CMJ height than VL40 (Table 2). Interestingly, VL40 did not improve in the fastest actions, such as velocity developed against lighter loads and CMJ height. A plausible explanation for these findings might be the significantly greater number of slow repetitions (MPV <0.6 m/s) performed by VL40 (Fig. 2), which could be responsible for the significant reduction of IIX fiber type observed only in this group. It is known that force, velocity, and power output gradually decrease as the number of repetitions increase during a set performed to failure, concomitant with an increased purine nucleotide degradation (Sánchez-Medina & González-Badillo, 2011; Gorostiaga et al., 2012), which is likely to impair the capacity for maximal force production or maximal rate of force development (RFD) (Allen et al., 1995), and may increase the time needed for recovery after training. Conversely, if only low or moderate repetition velocity losses (~20%) are experienced, higher forces and faster velocities will be achieved during training, while minimizing fatigue. It is for these reasons that setting a certain repetition velocity loss limit during RT has been proposed as a strategy to avoid performing unnecessarily slow and fatiguing repetitions that might result in a higher degree of muscle hypertrophy, but could be counterproductive for obtaining the rapid force production adaptations required by many sports and athletic disciplines.

Despite the common assumption that training to muscle failure is needed to maximize the hypertrophic response (Willardson, 2007), only one recent study (Sampson & Groeller, 2015) has analyzed the effect of RT leading or not leading to failure on muscle CSA. Sampson and Groeller (2015) observed similar increases in elbow flexors CSA after a 12-week RT program leading or not to failure, concluding that reaching repetition failure is not critical to elicit significant neural and structural muscle changes. In the present study, we analyzed changes in whole quadriceps femoris muscle volume using MRI (79.1 ± 1.1 slices per leg), whereas Sampson and Groeller (2015) only calculated the CSA as the mean across three slices central to the muscle belly. It has

Fig. 3. Changes in muscle volume following 8 weeks of velocity-based resistance training for: (a) Whole quadriceps femoris; (b) rectus femoris (RF); (c) vastus medialis (VM); and (d) vastus lateralis plus vastus intermedius (VL+VI). Data are mean ± SD. VL20: group that trained with a mean velocity loss of 20% in each set (n = 12); VL40: group that trained with a mean velocity loss of 40% in each set (n = 10). Intra-group significant differences from Pre to Post: **P < 0.01, ***P < 0.001. Significant group × time interaction: #P < 0.05.

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been suggested that the multiple-slice MRI scanning allows a more accurate quantification of changes in whole muscle volume (Folland & Williams, 2007). Limiting the analysis to a few slices precludes any definitive conclusion regarding muscle volume changes (Aagaard et al., 2001) or potential differences in regional adaptations (Folland & Williams, 2007; Sanchis-Moysi et al., 2010, 2011, 2012). In the present study, VL40 resulted in greater hypertrophy (VL40: 9.0% vs 3.4%, \( P < 0.05 \)) than VL20. However, VL20 also increased quadriceps volume (4.6%) and fiber size (9.8%), despite performing ~58% of the training volume done by VL40. The greater mechanical and metabolic stress (Sánchez-Medina & González-Badillo, 2011), hormonal response, and muscle damage associated to typical RT to failure protocols (Willardson, 2007; Schoenfeld, 2010) might explain the greater hypertrophic response observed for VL40, as it has been reported that mechanical tension, metabolic stress, and muscle damage may mediate hypertrophic adaptations (Schoenfeld, 2010).

Resistance training to failure is known to elicit IIX to IIA fiber transformation (Staron et al., 1991; Kraemer et al., 1995; Andersen & Aagaard, 2000; Campos et al., 2002; Andersen et al., 2005, 2010). The present study shows that the magnitude of velocity loss experienced during a training set may be a critical factor in inducing a fast-to-slow phenotypic remodeling in muscle fiber type, as VL40 showed greater reduction of IIX fiber type than VL20 (detected MHC analysis, Table 3). When resistance exercise sets are performed to or very close to muscle failure, actual training velocities end up being slow resulting in high levels of metabolic and mechanical fatigue (Sánchez-Medina & González-Badillo, 2011; Gorostiaga et al., 2012). As the neuromuscular system specifically adapts to the stimuli it is faced with (Spiering et al., 2008), it is likely that the stress associated to RT to failure induces greater adaptations in the slower and more resistant to fatigue muscle fiber types. As the MHC-IIx isoform is the fastest and most powerful one (Hartridge, 2007), a reduction in the percentage of IIX fibers and in the percent area of type IIX muscle fibers (as observed for VL40 in this study) might be detrimental for sports where very rapid movements and explosive force production are decisive for performance. A previous study (Andersen et al., 2010) observed a decrease in the early phase of the relative RFD following 14 weeks of RT to failure. This decrease in the early relative RFD was positively correlated \( (r = 0.61, P < 0.05) \) with the decrease in the area percentage of type IIX muscle fibers (Andersen et al. 2010). These findings might explain the lower gains in vertical jump (CMJ) performance observed for VL40 compared to VL20 in the present study (Table 2).

In summary, this study shows that the magnitude of velocity loss experienced during RT is a variable that should be taken into account when configuring the resistance exercise stimulus. Our findings suggest that a higher loss of repetition velocity during training (VL40) seems suitable to maximize the hypertrophic response, but tends to induce a fast-to-slow shift in muscle phenotype. Despite the greater hypertrophic adaptations observed in VL40, VL20 training resulted in similar strength squat gains and superior improvements in vertical jump performance. These results were obtained despite the fact that VL20 only performed 60% of the total training volume carried out by VL40.

Limitations

Due to the small number of subjects studied, we cannot rule out a type II error when comparing the two types of training. In fact, several variables show interaction \( P \)-values above 0.05 but close to 0.10, indicating that training with a lower loss of repetition velocity, i.e. with less fatigue at the end of each set, may result in more favorable adaptations to enhance performance in dynamic exercises than here reported. Another limitation is that the study did not include a control group and as a consequence the influence of environmental variables cannot be ruled out.

Perspectives

The magnitude of velocity loss experienced during RT appears to influence functional and structural muscle adaptations. Once a moderate velocity loss is achieved during a training set, performing more repetitions does not seem to elicit further strength gains and may even be detrimental for improving explosive strength. This is particularly relevant for many athletes for whom resistance training is not only focused on maximizing muscle hypertrophy but rather it is mainly aimed at improving dynamic performance in the most efficient way.

Key words: Muscle strength, training to failure, muscle hypertrophy, fiber type, magnetic resonance imaging.

Conflicts of interest

The authors declare no conflicts of interest.

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