

Comparison of the recovery response from high-intensity and high-volume resistance exercise in trained men

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Abstract

Purpose The purpose of this study was to compare the physiological responses of a high-volume (HV; 8 sets of 10 repetitions) versus high-intensity (HI; 8 sets of 3 repetitions) exercise protocol in resistance-trained men.

Methods Twelve men (24.5 ± 4.2 years; 82.3 ± 8.4 kg; 175.2 ± 5.5 cm) with 6.3 ± 3.4 years of resistance training experience performed each protocol in a counterbalanced, randomized order. Performance [counter movement jump peak power (CMJP), isokinetic (ISOK) and isometric leg extension (MVIC), isometric mid-thigh pull (IMTP), and isometric squat (ISQ)] and muscle morphological [cross-sectional area (CSA) of vastus lateralis] assessments were performed at baseline (BL), 30-min (P-30 min), 24-h (P-24 h), 48-h (P-48 h), and 72-h (P-72 h) post-exercise for each testing session. In addition, endocrine (testosterone and cortisol), inflammatory [interleukin-6 (IL-6) and C-reactive protein (CRP)], and markers of muscle damage [creatinase kinase (CK), lactate dehydrogenase (LDH), and myoglobin (Mb)] were assessed at the same time points.

Results Significantly greater reductions in CMJP ($p < 0.001$), and peak torque during both ISOK ($p = 0.003$) and MVIC ($p = 0.008$) at P-30 min were detected in HV

compared to HI protocol. MVIC was still impaired at P-72 h following the HV protocol, while no differences were noted following HI. Markers of muscle damage (LDH, CK, and Mb) were significantly elevated following both HV and HI ($p < 0.05$), while cortisol and IL-6 concentrations were significantly elevated at P-30 min following HV only ($p < 0.001$ and $p < 0.05$, respectively).

Conclusions Results indicate that high-volume resistance exercise results in greater performance deficits, and a greater extent of muscle damage, than a bout of high-intensity resistance exercise.

Keywords Resistance training · Performance · Testosterone · Cortisol · Inflammation · Muscle damage

Abbreviations

| | |
|---------|---|
| HV | High volume |
| HI | High intensity |
| 1-RM | One-repetition maximum |
| CMJP | Counter movement jump peak power |
| MVIC | Maximum voluntary isometric contraction |
| IMTP | Isometric mid-thigh pull |
| ISOK60 | Isokinetic leg extension at 60°/s |
| ISOK180 | Isokinetic leg extension at 180°/s |
| ISQ | Isometric squat |
| pRFD20 | Peak rate of force development |
| VL | Vastus lateralis |
| MT | Muscle thickness |
| CSA | Cross-sectional area |
| EI | Echo intensity |
| Mb | Myoglobin |
| LDH | Lactate dehydrogenase |
| CK | Creatine kinase |
| CRP | C-reactive protein |
| T/C | Testosterone/cortisol |

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Introduction

Recovery from a workout can be impacted by nutrition, sleep, or other physiological or psychological factors, and represents a key factor in optimizing training adaptation (Barnett 2006). However, the extent of fatigue is dependent upon the initial stimulus, which is the exercise or training session itself. Furthermore, such resistance training protocols can induce muscle damage and a reduction in force production (Byrne and Eston 2002; Flores et al. 2011; Nguyen et al. 2009). The magnitude of these deficits may be dependent upon the type of exercise or training program utilized (Aboodarda et al. 2011; Villanueva et al. 2012), and the rate of recovery can impact subsequent workouts affecting the physiological stimulus enhancing or attenuating muscle adaptation. A high-volume (HV) resistance training workout, commonly used to maximize muscle growth, is characterized by high number of repetitions (≥ 10) performed per set, at a moderate intensity ($\sim 70\%$ of 1 repetition maximum; 1-RM) and using short rest (~ 1 min) intervals. In contrast, high-intensity (HI) protocols, generally used to maximize strength gains, are characterized by a lower number of repetitions (4–6) per set at 85–90% of 1-RM with longer rest intervals (2–3 min) (Gonzalez et al. 2015).

Strength loss may be considered one of the most important indicators of muscle fatigue (Behm et al. 2004) and muscle damage (Warren et al. 1999). Isometric and dynamic strength and power measurements have often been used to assess the recovery process following exercise intervention. Byrne and Eston (2002) reported lower body strength and power reductions 72 h following an HV resistance exercise workout in inexperienced lifters, while others reported a significant reduction in elbow flexor strength for 5 days following an HV resistance exercise session that was also performed in novice individuals (Flores et al. 2011). In contrast, HI resistance training has been associated with a short-term increase in power expression (Young et al. 1998), but has been associated with a decrease in performance in others (Scott and Doherty 2004). The differences in these studies may be dependent on the protocol used (Behm et al. 2004) and by the participant's strength levels (Gonzalez et al. 2015).

Scientific studies comparing acute effects of HI to HV resistance training protocols in trained men are limited. Wells et al. (2016) and Gonzalez et al. (2015) recently examined the immune and endocrine responses following different resistance training protocols, but the acute effects on performance were not reported. The acute effects of different training schemes on strength and power performances in experienced, resistance-trained individuals are not well-understood. Therefore, the purpose of this investigation was to compare the acute

responses of two common resistance exercise paradigms: HV, moderate intensity and HI, low volume on performance recovery post-exercise in experienced, resistance-trained men. In addition, markers of muscle damage, inflammation, and endocrine markers of recovery were also examined.

Methods

Experimental design

The experimental protocol consisted of a counterbalanced cross-over research design. Figure 1 shows the experimental design followed in each trial by the participants. Then, they were requested to report back to the Human Performance Laboratory on ten separate occasions. During the first visit, participants were assessed for one-repetition maximal strength (1-RM) on the squat exercise. In addition, they performed several lower body maximal isometric force and power assessments. Baseline (BL) anthropometric measures were also determined. Participants reported back to the laboratory at least 72 h following their initial visit and were randomized into either the HV or HI exercise protocol. BL measures of all blood variables were performed prior to each exercise protocol. Following the workout, participants were tested 30-min post-exercise (P-30 min) to assess the acute fatiguing effect of the workout. Participants reported back to the laboratory 24-h (P-24 h), 48-h (P-48 h), and 72-h (P-72 h) post-exercise for additional performance assessments. Blood samples and muscle ultrasonography were obtained at each time point. Following at least 10 days of rest (average 15.4 ± 5.1 days) from the end of the first interventional session, participants reported back to the laboratory and performed the opposite workout. Participants were also tested for lower body maximal force and power 72 h prior to the second trial.

Both resistance protocols were comprised of the squat exercise only. During HI, participants were asked to perform eight sets of three repetitions at 90% of the previously measured 1-RM. Recovery time between sets was 3 min. During HV, participants were asked to perform eight sets of ten repetitions at 70% of the previously measured 1-RM. Recovery time between sets was 1.25 min. During both trials, if the required number of repetitions per set was not completed, then the load was reduced in the subsequent set to enable the participant to complete the required number of repetitions. No forced repetitions were performed in either protocol. All resistance and assessment sessions were supervised by the same strength and conditioning coaches.

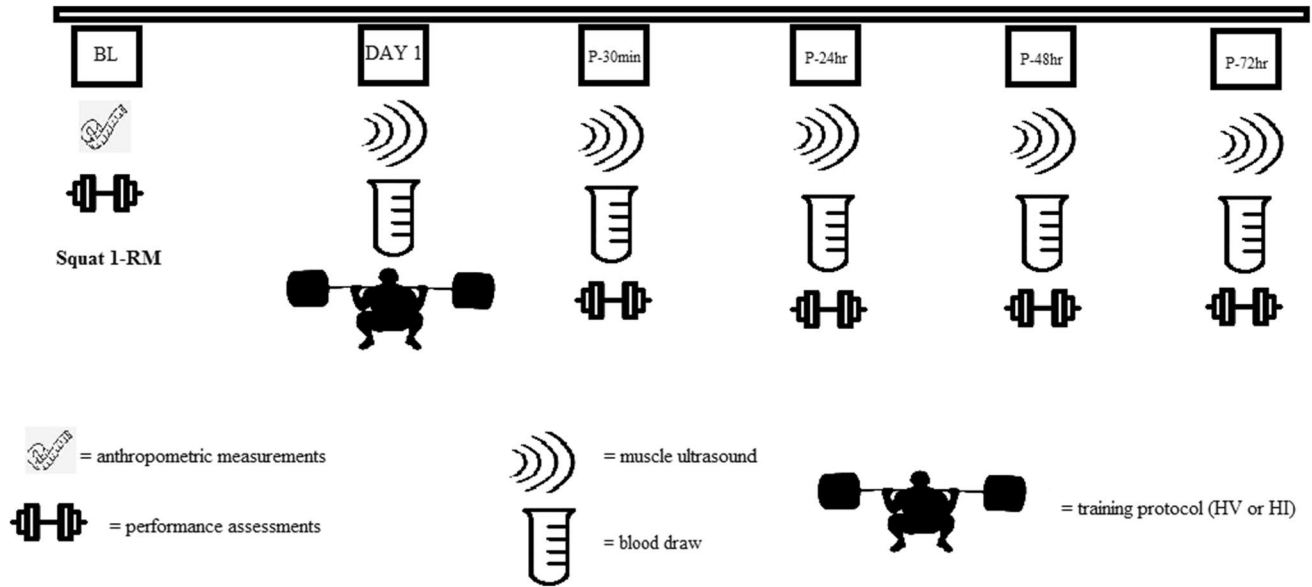


Fig. 1 Experimental protocol of a single trial of the counterbalanced cross-over research design. *BL* baseline, *P-30 min* 30-min post, *P-24 h* 24-h post, *P-48 h* 48-h post, *P-72 h* 72-h post

Participants

Twelve experienced, resistance-trained men (Mean \pm SD: age = 24.5 ± 4.2 years; body mass = 82.3 ± 8.4 kg; height = 175.2 ± 5.5 cm; body fat composition = $13.5 \pm 3.4\%$) volunteered to participate in this study. Inclusion criteria required participants to be between the ages of 18 and 35 years, with a minimum of 2 years of resistance training experience (mean \pm SD; 6.3 ± 3.4 years), and the ability to squat at least 1.5 times their body mass (173.4 ± 31.7 kg). Participants were not permitted to use any additional dietary supplementation, and did not consume any androgens or other performance enhancing drugs. Screening for performance enhancing drug use and additional supplementation was accomplished via a health questionnaire completed at recruitment stage. The study was approved by the University's Institutional Review Board. Testing procedures were fully explained to each participant before obtaining individual written informed consent.

Strength and power testing

Prior to 1-RM squat testing, maximal strength testing, participants performed a standardized warm-up consisting of 5 min on a cycle ergometer against a light resistance, ten body weight squats, ten body weight walking lunges, ten dynamic walking hamstring stretches, and ten dynamic walking quadriceps stretches. The 1-RM test for the barbell back squat was performed using methods previously described by Hoffman (2014). Briefly, each participant

performed two warm-up sets using a resistance of approximately 40–60 and 60–80% of his perceived maximum, respectively. For each exercise, 3–4 subsequent trials were performed to determine the 1-RM. A 3–5-min rest period was provided between each trial. Trials not meeting the range of motion criteria for each exercise or where technique was not appropriate were discarded.

During all other visits, the same standardized warm-up, as described above, was repeated. During each visit, participants were required to perform a CMJ peak power (CMJP) on a force plate (AMTI, Watertown, USA, 1000 Hz). Participants were instructed to maximize the height of each jump keeping the hands on their hips. Flight time was calculated as the time interval from toe-off to landing. The jump height was estimated as $9.81 \times \text{flight time}^2 / 8$ (Bosco et al. 1983). Peak power (CMJP) was calculated (in W) by the jump height and the participant's body mass using the following equation (Sayers et al. 1999): peak Power = $60.7 \times \text{jump height} + 45.3 \times \text{body mass} - 2055$. Participants performed three jumps with a 3-min rest between each jump. The intraclass coefficient calculated for the CMJP was 0.96 (SEM = 122.0 W).

Isometric and isokinetic strength measurements were performed following the CMJP on the participant's right leg using a Biodex (Biodex Medical System, Shirley, NY) isokinetic dynamometer. Participants were seated and stabilized to the device, with the right leg attached to the lever arm. Maximal isometric strength was measured by performing two maximal unilateral voluntary isometric contractions (MVIC) for 6 s at 70° knee flexion. A 3-min rest was provided between each MVIC. Intraclass coefficient

was determined to be 0.79 (SEM=29.16 N m). Isokinetic concentric measurements were performed at an angular velocity of 60°/s (ISOK60) and 180°/s (ISOK180). Three maximal leg extensions were performed for each velocity with a 3-min rest between each set. The highest peak torque value was recorded. Intraclass coefficients were 0.78 (SEM=24.86 N m) and 0.76 (SEM=20.84 N m) for ISOK60 and ISOK180, respectively.

An isometric mid-thigh pull (IMTP) assessment was also performed using a power rack that permitted fixation of the bar at a height that corresponded to the participant's mid-thigh while standing on a force plate (AMTI, Watertown, USA, 1000 Hz). Participants were instructed to assume a body position similar to the second pull of the snatch and clean. Knee angle, hip angle, and grip width were measured to reproduce the same position for all testing sessions (140° and 125° angles for knees and hips, respectively). Participants were secured to the bar using lifting straps and subsequently performed 2 IMTP with a recovery time of 3 min between attempts. Each maximal contraction lasted for 6 s.

The same adjustable rack and force plate was used for the isometric half squat (ISQ). The isometric half squat was performed at a knee flexion angle of 90° between the femur and the tibia. Participants were required to perform two maximal 6-s isometric contractions with a 3-min recovery time between each attempt. Intraclass coefficients were 0.85 (SEM=267.1 N) and 0.73 (SEM=219.6 N) for IMTP and ISQ, respectively.

For both IMTP and ISQ, peak force was measured and the peak rate of force development using a 20-ms window (pRFD20) was calculated as previously described by Haff et al. (2015). Intraclass coefficients were 0.85 (SEM=822.5 N s⁻¹) and 0.51 (SEM=1010.3 N s⁻¹) for pRFD20 expressed at IMTP and ISQ, respectively. During all isometric and isokinetic measurements, participants were verbally encouraged by the study investigators.

Ultrasonography measurements

Non-invasive skeletal muscle ultrasound images were collected from the participant's right thigh. Prior to image collection, all anatomical locations of interest were identified using standardized landmarks for the vastus lateralis (VL muscle). The landmark for the VL was identified along its longitudinal distance at 50% from the proximal insertion of the muscle. The length of the VL encompassed the distance from the lateral condyle of the tibia to the most prominent point of the greater trochanter of the femur. VL measurement required the participants to lay on their side on the examination table for a minimum of 15 min before images were collected. The same investigator performed all landmark measurements for each participant.

A 12-MHz linear probe scanning head (General Electric LOGIQ P5, Wauwatosa, WI, USA) was coated with water soluble transmission gel to optimize spatial resolution and used to collect all ultrasound images. The probe was positioned on the surface of the skin without depressing the dermal layer and the view mode (gain=50 dB; image depth=5 cm) was used to take panoramic pictures of the VL. During the measurements, participants were asked to relax their leg muscles and maintain the left lateral decubitus position. Legs were positioned together, with a 10° bend angle in the knees (Bemben 2002). The muscle CSA was calculated using the polygon tracking tool of Image J software (National Institute of Health, Bethesda, MD, version 1.45 s). All ultrasound images were taken and analyzed by the same technician. Muscle thickness (MT) measures were obtained using a longitudinal B-mode image. Three consecutive MT images were captured and analyzed for each muscle and leg, respectively. For each image, MT was measured with a single perpendicular line from the superficial aponeurosis to the deep aponeurosis. The average of the three MT measures was used for statistical analyses. Intraclass correlation coefficients (ICCs) for MT was 0.78 (SEM=0.17 cm).

Cross-sectional area (CSA) measures were obtained using a transverse sweep in the extended field of view mode. Three consecutive CSA images were captured and analyzed for each muscle and leg, respectively. For each image, CSA was assessed using the polygon tracking tool to include as much lean tissue as possible while excluding bone and surrounding fascia. The average of the three CSA measures was used for statistical analyses. Echo intensity (EI) was determined by gray-scale analysis using the standard histogram function in Image-J. Intraclass coefficients were 0.98 (SEM=0.90 cm²) and 0.97 (SEM=1.40 au) for CSA and EI, respectively.

Blood sampling

During each experimental trial, blood samples were obtained from a superficial forearm vein using a single use disposable needle with the participant placed in a supine position for at least 15 min prior to sampling. All blood draws were performed by personnel trained in phlebotomy. Following the BL blood sample, participants were provided a standardized breakfast consisting of two low protein, low carbohydrate bars (Atkins Nutritionals, Inc, Denver, CO: 7-g protein, 3-g carbohydrate, and 3-g fat each).

All blood samples were collected into two Vacutainer® tubes, one containing no anticlotting agent (6 mL) and the second containing K₂EDTA (6 mL). A small aliquot (50 mL) of whole blood was removed and used for determination of hematocrit and hemoglobin concentrations. The blood in the first tube was allowed to clot at room

temperature for 30 min and subsequently centrifuged at 3000g for 15 min along with the remaining whole blood from the second tube. The resulting plasma and serum were placed into separate microcentrifuge tubes and frozen at -80°C for later analysis.

Biochemical analyses

Blood lactate concentrations were analyzed using an automated analyzer (Analox GM7 enzymatic metabolite analyzer; Analox Instruments USA, Lunenburg, MA). Hematocrit concentrations were analyzed from whole blood via microcentrifugation (CritSpin, Westwood, MA) and microcapillary technique. Hemoglobin concentrations were analyzed from whole blood using an automated analyzer (HemoCue, Cypress, CA). Plasma volume shifts were calculated using the formula established by Dill and Costill (1974). To eliminate interassay variance, all samples were analyzed in duplicate by a single technician. Coefficients of variation were 3.81% for blood lactate, 1.51% for hematocrit, and 0.63% for hemoglobin.

Serum concentrations of testosterone, cortisol, myoglobin (Mb), lactate dehydrogenase (LDH) activity, and creatine kinase (CK), as well as plasma interleukin-6 (IL-6) and C-reactive protein (CRP) were assayed using commercial enzyme-linked immunosorbent assays. Assay absorbance was read according to the manufacturer specifications on a BioTek[®] Eon[™] Microplate Spectrophotometer (BioTek Instruments, Inc., Winooska, VT, USA). To eliminate interassay variance, all samples for a particular assay remained frozen until analysis, were thawed only once, and were measured in duplicate by a single technician. Testosterone and cortisol concentrations were used to calculate individual values of the Testosterone/Cortisol (T/C) ratio. Coefficients of variation for each assay were 2.18, 1.60, 5.29, 2.89, 5.45, 4.48, and 3.29% for testosterone, cortisol, IL-6, CRP, Mb, LDH, and CK, respectively.

Muscle pain and soreness score

Participants were instructed to assess their subjective feelings of pain and soreness intensity using a 100-mm visual analog scale (VAS) (Lee et al. 1991; Bijur et al. 2001; Nosaka et al. 2002). No pain or soreness was recorded as 0 and the worst possible soreness or pain as 100. Pain and soreness intensity were evaluated at BL, P-30 min, P-24 h, P-48 h, and P-72 h.

Dietary logs

Participants were instructed to record as accurately as possible everything they consumed during each 4-day trial. For the following experimental trial, participants were required

to duplicate the content, quantity, and timing of their daily diet during the 24 h prior. Participants were instructed not to eat or drink (except water) within 10 h of reporting to the laboratory for each experimental trial. The USDA Nutritional Database (US Department of Agriculture, Beltsville, MD, USA) was used to analyze total calories, carbohydrates, protein, and fat.

Statistical analysis

A Shapiro–Wilk test was used to assess the normal distribution of the data. If the assumption of sphericity was violated, a Greenhouse–Geisser correction was applied. Performance and biochemical data were analyzed using a two-factor (trial \times time) analysis of variance (ANOVA) with repeated measures. In the event of a significant F ratio, dependent *t* tests with a Bonferroni adjustment were used to examine pairwise comparison between trials for each time point. In the event of a significant trial \times time interactions each group was analyzed separately by a one-factor ANOVA with repeated measures on time. For effect size (ES), the partial eta squared was reported, and according to Stevens (2009), 0.01, 0.06, and 0.14 represent small, medium, and large effect sizes, respectively. Where appropriate, percent change was calculated as follows: [(post-exercise mean – pre-exercise mean)/pre-exercise mean] \times 100. Pearson product moment correlations were used to examine selected bivariate relationships. Significance was accepted at an alpha level of $p\leq 0.05$, and all data are reported as mean \pm SD.

Results

Performance Assessments

A significant trial \times time interaction was found for CMJP ($F=9.281$; $p<0.001$; $\eta^2 = 0.458$). A significant trial difference in CMJP (see Fig. 2) was noted at P-30 min ($p=0.001$) and P-48 h ($p=0.002$). During HV, CMJP performance was significantly reduced from baseline (BL) at P-30 min (-15.9% ; $p<0.001$), P-24 h (-9.6% ; $p=0.002$), and P-48 h (-7.8% ; $p=0.009$), while during HI, CMJP was reduced from BL at P-30 min only (-5.5% ; $p<0.01$). Results for isokinetic and isometric performance measures are depicted in Table 1.

Significant interactions were also noted for MVIC ($F=5.477$; $p=0.003$; $\eta^2 = 0.473$) and ISOK60 ($F=4.763$; $p=0.008$; $\eta^2 = 0.302$). Pairwise comparisons indicated significant differences between HI and HV at and at P-30 min ($p=0.002$) and P-24 h ($p=0.003$) for ISOK60 and at P-48 h ($p=0.009$) and P-72 h ($p=0.006$) for MVIC. Decrements in performance from BL during HV were significant

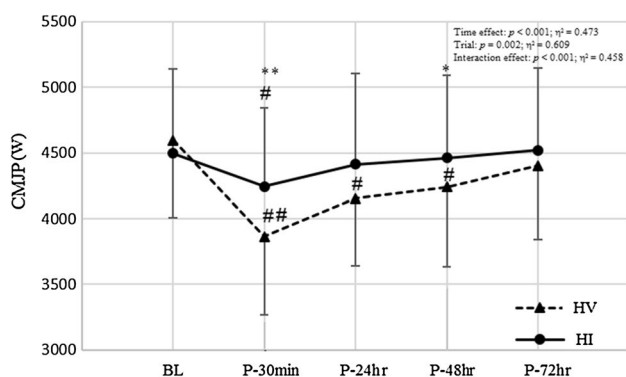


Fig. 2 Changes in counter movement jump peak power (CMJP) 30-min (P-30 min), 24-h (P-24 h), 48-h (P-48 h), and 72-h (P-72 h) post-exercise. HV high-volume protocol, HI high-intensity protocol. * Indicates a significant ($p \leq 0.01$) difference between trials; ** indicates a significant ($p \leq 0.001$) difference between the two trials; # indicates a significant ($p \leq 0.01$) difference from BL. All data are reported as mean \pm SD

at P-30 min for MVIC (-23.6% ; $p \leq 0.01$) and ISOK60 (-26.0% ; $p < 0.01$). MVIC still remained below BL values in HV at P-72 h (-11% ; $p = 0.010$). MVIC and ISOK60 performances in HI were not significantly different from BL at any time point ($p > 0.05$).

No significant interactions were observed for ISOK180 ($F = 0.19$; $p = 0.944$; $\eta^2 = 0.187$). However, a significant main effect for time was noted ($F = 12.4$, $p < 0.001$; $\eta^2 = 0.530$). In addition, no significant interactions were observed for IMTP, ISQ, and pRFD20 at IMTP and pRFD20 at ISQ. Furthermore, when collapsed across trials, no main effects for time were seen for maximal isometric strength expressed at IMTP ($F = 1.76$; $p = 0.229$; $\eta^2 = 0.469$), nor were any main effects for time noted for pRFD20 in both IMTP and ISQ ($F = 0.79$; $p = 0.559$; $\eta^2 = 0.285$ and $F = 2.19$; $p = 0.182$; $\eta^2 = 0.548$, respectively). A significant main effect of time was detected for ISQ ($F = 21.82$; $p < 0.001$; $\eta^2 = 0.686$). Examples of ISOK60 torque-time curve collected on a typical participant at BL and at P-30 min following both HI and HV exercise protocols are reported in Fig. 3a.

Ultrasound measurements

Changes in all ultrasound measures can be observed in Table 2. Significant trial \times time interactions were found for CSA ($F = 4.58$; $p = 0.032$; $\eta^2 = 0.294$) and EI ($F_{1-11} = 4.46$; $p = 0.024$; $\eta^2 = 0.289$). Pairwise comparisons revealed significant trial differences for CSA at P-30 min ($p < 0.001$), P-24 h ($p = 0.007$), and at P-48 h ($p = 0.005$). Significant increases from BL were observed for VL CSA in HV at P-30 min ($+11.3\%$; $p < 0.001$), P-24 h ($+4.19\%$; $p = 0.003$), and P-48 h ($+4.28\%$; $p = 0.001$). No changes from BL were

noted during HI at any time point. EI was significantly elevated from BL during HV at P-30 min ($+18.2\%$; $p < 0.001$), while no significant changes ($p > 0.1$) were observed after the HI session. No significant interaction ($F = 3.15$; $p = 0.052$; $\eta^2 = 0.223$) was noted for MT. However, a significant time effect ($F = 10.41$; $p < 0.001$; $\eta^2 = 0.486$) was observed for MT. With trials combined, a significant ($p = 0.001$) increase from BL was noted at P-30 min. Examples of thigh ultrasound images collected on a typical participant at BL and at P-30 min following both HI and HV exercise protocols are reported in Fig. 3b.

Biochemical measures

Changes in cortisol and testosterone concentrations are shown in Figs. 4 and 5, respectively. A significant interaction was noted for cortisol ($F = 15.77$; $p < 0.001$; $\eta^2 = 0.589$), but not testosterone ($F = 3.14$; $p = 0.089$; $\eta^2 = 0.643$) or the testosterone-to-cortisol ratio (T/C ratio) ($F = 1.54$; $p = 0.228$; $\eta^2 = 0.134$). Cortisol concentrations during HV were significantly greater ($p < 0.001$) than HI at P-30 min. No other significant differences were noted in changes in cortisol concentrations. A significant main effect for time ($F = 9.05$; $p = 0.001$; $\eta^2 = 0.475$) was observed in testosterone and in T/C ratio ($F = 8.06$; $p = 0.009$; $\eta^2 = 0.446$). Testosterone concentrations and T/C ratio were significantly reduced at P-30 min ($p = 0.003$ and $p = 0.001$, respectively). No other differences were noted.

A significant trial \times time interaction was observed for IL-6 ($F = 5.02$; $p = 0.020$; $\eta^2 = 0.334$). Pairwise comparison revealed that IL-6 concentrations were significantly higher ($p = 0.005$) at P-30 min in HV compared to HI. No significant trial \times time interaction ($F = 1.64$; $p = 0.232$; $\eta^2 = 0.154$) was noted in CRP concentrations, and no main effect for time ($F = 1.07$; $p = 0.356$; $\eta^2 = 0.107$) was observed as well. Changes in IL-6 and CRP concentrations during HI and HV are depicted in Figs. 6 and 7, respectively.

Changes in the muscle damage markers CK, Mb, and LDH can be observed in Table 3. No significant trial \times time interactions were observed for CK ($F = 1.86$; $p = 0.185$; $\eta^2 = 0.145$), Mb ($F = 1.10$; $p = 0.367$; $\eta^2 = 0.091$) or LDH ($F = 0.861$; $p = 0.463$; $\eta^2 = 0.073$). However, significant time effects were observed for CK ($F = 8.00$; $p = 0.004$; $\eta^2 = 0.421$), Mb ($F = 19.73$; $p = 0.000$; $\eta^2 = 0.642$), and LDH ($F = 3.846$; $p = 0.021$; $\eta^2 = 0.259$). CK concentrations were significantly increased in both HI and HV from BL at P-30 min ($p = 0.003$) and P-24 h ($p = 0.034$), while Mb levels were significantly elevated from BL at P-30 min ($p < 0.001$). LDH concentrations were significantly increased from BL at both P-30 min ($p = 0.001$) and P-24 h ($p = 0.031$). No other significant changes were observed.

A significant interaction ($F = 53.7$; $p < 0.001$; $\eta^2 = 0.830$) was observed in blood lactate concentrations.

Table 1 Changes in performance measures

| Assessment | Trial | BL | P-30 min | P-24 h | P-48 h | P-72 h | Time Effect | | Trial | | Interaction effects | | | |
|----------------------------------|-------|-----------------|-----------------------------|-----------------------------|---------------------------|---------------------------|-------------|----------|-------|----------|---------------------|----------|---|----------|
| | | | | | | | p | η^2 | p | η^2 | p | η^2 | p | η^2 |
| | | | | | | | | | | | | | | |
| ISOK60 (Nm) | HV | 253.0 ± 56.1 | 187.9 ± 53.5 [†] # | 201.8 ± 56.1 [†] # | 210.0 ± 61.9 | 219.0 ± 60.2 | 0.000 | 0.566 | 0.022 | 0.394 | 0.008 | 0.302 | | |
| | HI | 243.1 ± 45.3 | 223.8 ± 58.0 | 232.6 ± 60.9 | 240.8 ± 56.5 | 237.0 ± 61.0 | 0.000 | 0.530 | 0.040 | 0.330 | 0.944 | 0.187 | | |
| ISOK180 (Nm) | HV | 202.8 ± 44.5 | 161.3 ± 42.1 | 171.0 ± 42.0 | 169.9 ± 52.7 | 180.0 ± 50.7 | 0.000 | 0.530 | 0.040 | 0.330 | 0.944 | 0.187 | | |
| | HI | 213.2 ± 37.3 | 178.7 ± 40.5 | 181.7 ± 38.9 | 185.3 ± 41.59 | 194.4 ± 43.0 | 0.000 | 0.473 | 0.017 | 0.418 | 0.003 | 0.473 | | |
| MIVC* (Nm) | HV | 343.5 ± 60.6 | 262.5 ± 71.4 [#] | 293.1 ± 73.3 | 295.8 ± 74.4 [†] | 305.8 ± 67.9 [#] | 0.000 | 0.473 | 0.017 | 0.418 | 0.003 | 0.473 | | |
| | HI | 338.7 ± 61.9 | 310.0 ± 64.8 | 332.1 ± 77.1 | 346.1 ± 78.4 | 353.2 ± 85.9 | 0.057 | 0.194 | 0.171 | 0.163 | 0.192 | 0.133 | | |
| IMTP (N) | HV | 3670.3 ± 602.8 | 3379.7 ± 675.2 | 3489.9 ± 635.8 | 3498.8 ± 648.8 | 3580.1 ± 625.9 | 0.057 | 0.194 | 0.171 | 0.163 | 0.192 | 0.133 | | |
| | HI | 3695.2 ± 713.1 | 3656.1 ± 671.5 | 3705.9 ± 754.6 | 3620.7 ± 607.9 | 3676.5 ± 639 | 0.296 | 0.105 | 0.808 | 0.006 | 0.268 | 0.113 | | |
| IMTP pRFD20 (N s ⁻¹) | HV | 8718.8 ± 2021.5 | 7232.5 ± 1756.5 | 7780.4 ± 1492.7 | 7727.9 ± 2017.8 | 7504.6 ± 1695.7 | 0.296 | 0.105 | 0.808 | 0.006 | 0.268 | 0.113 | | |
| | HI | 7938.3 ± 2084.7 | 7821.2 ± 1995.3 | 7702.1 ± 2182.1 | 7985.7 ± 3060.0 | 7955.0 ± 1820.7 | 0.000 | 0.686 | 0.479 | 0.051 | 0.073 | 0.216 | | |
| ISQ (N) | HV | 2749.2 ± 403.2 | 2342.2 ± 428.4 | 2532.9 ± 381.2 | 2619.6 ± 349.6 | 2726.2 ± 388.7 | 0.000 | 0.686 | 0.479 | 0.051 | 0.073 | 0.216 | | |
| | HI | 2737.8 ± 417.3 | 2539.9 ± 388.9 | 2614.2 ± 402.9 | 2649.4 ± 404.8 | 2719.3 ± 395.2 | 0.051 | 0.231 | 0.565 | 0.034 | 0.602 | 0.057 | | |
| ISQ pRFD20 (N s ⁻¹) | HV | 5195.0 ± 1249.7 | 4180.0 ± 1581.4 | 4451.4 ± 1611.2 | 4665.8 ± 1482.8 | 5084.2 ± 1807.6 | 0.051 | 0.231 | 0.565 | 0.034 | 0.602 | 0.057 | | |
| | HI | 5004.6 ± 1507.7 | 4783.3 ± 1344.7 | 4476.3 ± 1179.8 | 4885.0 ± 1556.2 | 5117.5 ± 1270.0 | | | | | | | | |

ISOK60 isokinetic peak torque at 60°/s; ISOK180 isokinetic peak torque at 180°/s; MIVC maximum voluntary contraction; IMTP isometric mid-thigh pull; IMTP pRFD20 peak rate of force development at IMTP; ISQ isometric squat; pRFD20 peak rate of force development at ISQ

[†]Indicates a significant ($p < 0.05$) difference between the two protocols at different time points (pairwise comparison)

[#]Indicates a significant ($p \leq 0.01$) difference from BL. All data are reported as mean ± SD

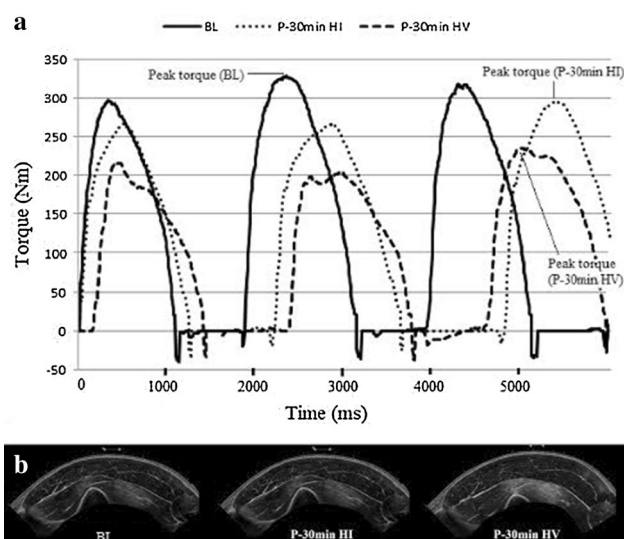


Fig. 3 Example of torque/time curve collected on a typical participant using the isokinetic dynamometer (a) and of high ultrasound image (b) at BL and at P-30 min following both HI and HV. All the data belong from the same participant. The figure shows the greater reduction in ISOK60 peak torque following HV compared to HI exercise protocol. VL ultrasound images show the increase in CSA following the HV protocol only

Elevations of blood lactate concentrations from BL were seen at P-30 min in both HV (1.64 ± 1.24 to 9.31 ± 3.14 mmol L⁻¹, respectively) and HI (1.65 ± 0.81 to 3.29 ± 1.82 mmol L⁻¹, respectively), but were significantly greater ($p < 0.001$) for HV. Relative to BL, plasma volume decreased by $-4.60 \pm 2.62\%$ at P-30 min ($p < 0.001$) for HV and increased $4.22 \pm 5.31\%$ for HI ($p < 0.001$). Blood variables were not corrected for plasma volume shifts due to the importance of molar exposure at the tissue receptor level.

Pain and soreness

The results of the VAS for both pain and soreness can be observed in Table 4. A significant trial \times time interaction ($F = 27.02$; $p < 0.001$; $\eta^2 = 0.711$) was observed for soreness intensity. Pairwise comparisons revealed that participant soreness intensity was elevated from BL following both trials, the soreness intensity following HV was significantly greater than HI at P-30 min ($p = 0.001$), P-24 h ($p < 0.001$), P-48 h ($p < 0.001$) and P-72 h ($p < 0.001$). No significant interaction ($F = 1.71$; $p = 0.240$; $\eta^2 = 0.461$) or time effect ($F = 1.98$; $p < 0.190$; $\eta^2 = 0.498$) was seen in pain scores.

Correlation between variables

The change in CSA from BL to P-30 min in HV was negatively correlated to changes in CMJP ($r = -0.68$; $p = 0.01$), MIVC ($r = -0.58$; $p = 0.05$), and ISOK180 ($r = -0.80$; $p = 0.001$). Significant correlations were also observed for changes from BL and P-24 h in CSA and both ISOK60 ($r = -0.787$; $p = 0.002$) and ISOK180 ($r = -0.678$; $p = 0.015$) during HV.

Significant correlations were observed between circulating IL-6 concentrations for HV and the magnitude of reduction in CMJP at both P-30 min ($r = 0.76$; $p = 0.004$) and P-48 h ($r = 0.66$; $p = 0.798$). In addition, a significant correlation ($r = 0.660$; $p = 0.019$) was also noted between IL-6 concentrations and the increase in CSA at P-24 h for HV. A significant correlation was also observed between CK concentrations at P-72 h and the change from BL for CSA at P-72 h in HV ($r = 0.60$; $p = 0.037$). No other significant correlations were noted.

Table 2 Changes in ultrasound measures

| Assessment | Trial | BL | P-30 min | P-24 h | P-48 h | P-72 h | Time effect | | Trial | | Interaction effects | |
|------------------------|-------|-----------------|--------------------------------|-------------------------------|-------------------------------|-----------------|-------------|----------|----------|----------|---------------------|----------|
| | | | | | | | <i>p</i> | η^2 | <i>p</i> | η^2 | <i>p</i> | η^2 |
| CSA (cm ²) | HV | 36.91 \pm 8.2 | 41.09 \pm 9.0 ^{†##} | 38.47 \pm 9.3 ^{†#} | 38.49 \pm 9.0 ^{†#} | 39.39 \pm 7.8 | 0.001 | 0.469 | 0.000 | 0.803 | 0.032 | 0.294 |
| | HI | 36.34 \pm 8.4 | 36.92 \pm 8.4 | 36.66 \pm 7.9 | 39.94 \pm 8.1 | 36.8 \pm 8.3 | | | | | | |
| MT (cm) | HV | 2.21 \pm 0.4 | 2.50 \pm 0.4 | 2.36 \pm 0.4 | 2.38 \pm 0.4 | 2.39 \pm 0.4 | 0.000 | 0.486 | 0.048 | 0.310 | 0.052 | 0.223 |
| | HI | 2.16 \pm 0.4 | 2.27 \pm 0.4 | 2.23 \pm 38.9 | 2.26 \pm 0.4 | 2.24 \pm 0.3 | | | | | | |
| EI (a.u.) | HV | 40.53 \pm 8.6 | 47.91 \pm 11.6 ^{##} | 39.62 \pm 9.7 | 39.23 \pm 9.6 | 40.51 \pm 9.5 | 0.000 | 0.698 | 0.267 | 0.111 | 0.024 | 0.289 |
| | HI | 41.27 \pm 8.8 | 43.32 \pm 9.7 | 39.86 \pm 8.4 | 39.96 \pm 9.8 | 39.21 \pm 8.2 | | | | | | |

CSA cross-sectional area, MT muscle thickness, EI echo intensity

[†]Indicates a significant ($p < 0.05$) difference between the two protocols (pairwise comparison)

[#]Indicates a significant ($p \leq 0.01$) difference from BL

^{##}Indicates a significant ($p \leq 0.001$) difference from BL. All data are reported as mean \pm SD

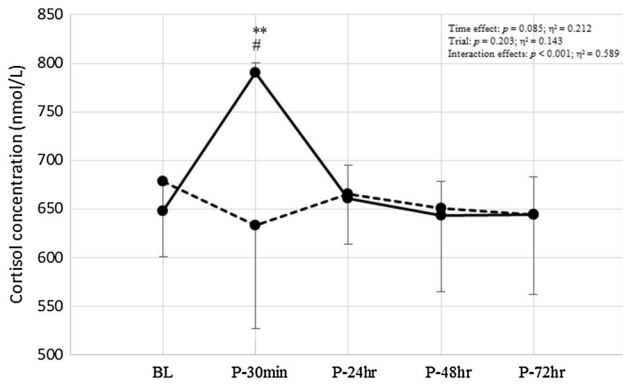


Fig. 4 Changes in cortisol concentrations 30-min (P-30 min), 24-h (P-24 h), 48-h (P-48 h), and 72-h post (P-72 h) the training session. *HV* high-volume protocol, *HI* high-intensity protocol. *Indicates a significant ($p \leq 0.01$) difference between trials; **indicates a significant ($p \leq 0.001$) difference between trials; # indicates a significant ($p \leq 0.01$) difference from BL. All data are reported as mean \pm SD

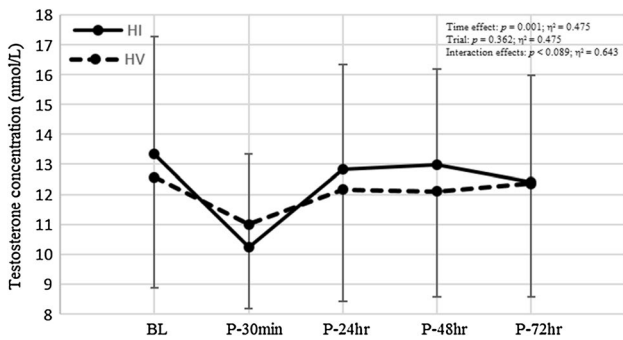


Fig. 5 Changes in testosterone concentrations 30-min (P-30 min), 24-h (P-24 h), 48-h (P-48 h), and 72-h post (P-72 h) training session. *HV* high-volume protocol, *HI* high-intensity protocol. *Indicates a significant ($p \leq 0.01$) difference between trials; # indicates a significant ($p \leq 0.01$) difference from BL. All data are reported as mean \pm SD

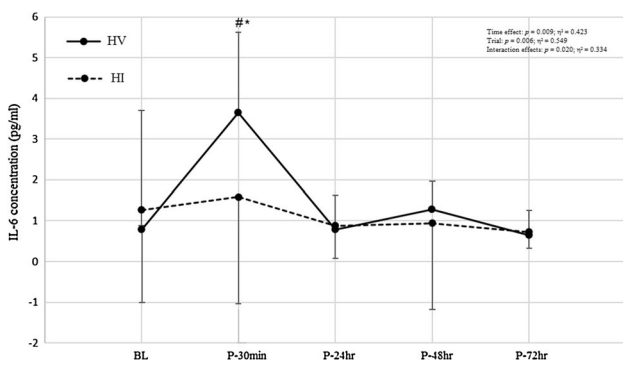


Fig. 6 Changes in IL-6 concentrations 30-min (P-30 min), 24-h (P-24 h), 48-h (P-48 h), and 72-h post (P-72 h) training session. *HV* high volume protocol, *HI* high intensity protocol. *Indicates a significant ($p \leq 0.01$) difference between trials; # indicates a significant ($p \leq 0.01$) difference from BL. All data are reported as mean \pm SD

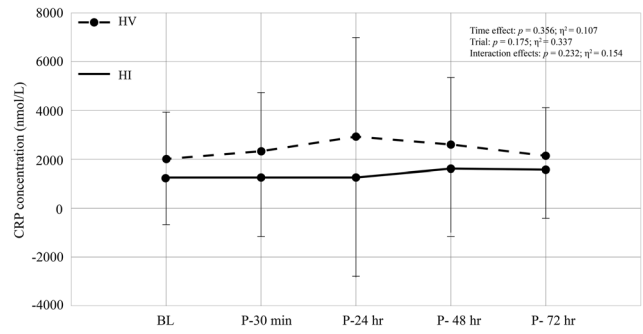


Fig. 7 Changes in CRP concentrations 30-min (P-30 min), 24-h (P-24 h), 48-h (P-48 h), and 72-h post (P-72 h) training session. *HV* high-volume protocol, *HI* high-intensity protocol. *Indicates a significant ($p \leq 0.01$) difference between trials; # indicates a significant ($p \leq 0.01$) difference from BL. All data are reported as mean \pm SD

Discussion

HV, moderate intensity resistance exercise with short rest intervals, consistent with hypertrophic-based training programs, resulted in greater strength and power performance decrement compared to an exercise session focused on strength development, characterized by *HI* workloads and longer rest intervals. The traditional hypertrophic-based exercise protocol resulted in a pronounced fatigue condition still persisting 72 h post-exercise. In contrast, strength and power performances were completely restored 24 h following the *HI* session. These results were consistent with the previous research reporting a similar reduction in vertical jump performance lasting 72 h following an *HV*-lower body training session (Byrne and Eston 2002).

The different time course of performance decrements occurring between *HV* and *HI* likely reflects the differences observed in the inflammatory response between these two exercise protocols. Although the increase in the CSA of the VL reached its peak at P-30 min following the *HV* workout, it was still significantly greater than BL at P-72 h. The initial increase in CSA at P-30 min following the *HV* protocol may be a consequence of the hyperemia involving knee extensor muscles. This physiological reaction has been associated with an increase in vasodilation (Wunsch et al. 2000) and to greater muscle fiber recruitment (Shoemaker and Hughson 1999). The continued increase in CSA at P-24 h, P-48 h, and P-72 h may be related to the reactive hyperemia and to a delayed onset of muscle swelling (Clarkson et al. 1992) that was not detected following the *HI* protocol.

Significant elevations in blood lactate and cortisol concentrations at P-30 min reflect the metabolic stress associated with the *HV* protocol. No significant changes in

Table 3 Changes in muscle damage parameters

| Assessment | Trial | BL | P-30 min | P-24 h | P-48 h | P-72 h | Time effect | | Trial | | Interaction effects | |
|------------|-------|----------------|----------------|----------------|----------------|----------------|-------------|----------|-------|----------|---------------------|----------|
| | | | | | | | p | η^2 | p | η^2 | p | η^2 |
| | | | | | | | | | | | | |
| CK (U/L) | HV | 208.10 ± 132.9 | 259.86 ± 165.1 | 442.10 ± 297.8 | 304.24 ± 209.9 | 305.80 ± 337.0 | 0.002 | 0.254 | 0.079 | 0.254 | 0.185 | 0.145 |
| | HI | 169.17 ± 89.1 | 205.10 ± 102.4 | 281.28 ± 133.2 | 219.16 ± 91.6 | 199.77 ± 50.2 | | | | | | |
| Mb (ng/mL) | HV | 26.25 ± 7.5 | 59.97 ± 20.8 | 38.03 ± 24.7 | 33.65 ± 20.7 | 38.71 ± 37.1 | 0.001 | 0.872 | 0.383 | 0.070 | 0.367 | 0.091 |
| | HI | 26.75 ± 7.1 | 62.88 ± 18.3 | 28.34 ± 6.2 | 27.48 ± 5.6 | 29.1 ± 6.2 | | | | | | |
| LDH (mU/L) | HV | 704.72 ± 166.7 | 841.71 ± 257.7 | 773.24 ± 236.1 | 757.48 ± 235.8 | 739.04 ± 237.4 | 0.012 | 0.390 | 0.022 | 0.390 | 0.463 | 0.073 |
| | HI | 692.51 ± 185.4 | 746.50 ± 160.4 | 728.81 ± 205.7 | 703.61 ± 198.3 | 704.11 ± 136.8 | | | | | | |

CK creatine kinase, MB myoglobin, LDH lactate dehydrogenase, SOR soreness

#Indicates a significant ($p \leq 0.01$) difference from BL. All data are reported as mean ± SD

cortisol concentrations were detected following HI. These results are consistent with other investigations comparing HV and HI lower limb resistance exercise protocols (Gonzalez et al. 2015; Wells et al. 2016). Testosterone concentrations showed a significant decrease after both protocols but returned to BL at P-24 h. Similar reductions in circulatory testosterone concentrations following an HV-lower body resistance exercise were reported by Hakkinen and Pakarinen (1993) and more recently by Gonzalez et al. (2015). Differences in acute hormonal responses following the two training protocols, with particular reference to cortisol, may have influenced the recovery of muscle contractility after the workout, acting on the rate of skeletal muscle protein turnover (Phillips et al. 1997). Changes in T/C ratio following a resistance training program have been associated with overall training stress (Fry et al. 2000). However, the decrease in T/C ratio was noted following both HV and HI at P-30 min only. Although the reduction in T/C ratio at P-30 min was consistent with the performance decrements seen in both trials, it returned to baseline levels at P-24 h. However, performance continued to be attenuated for HV only indicating that acute changes in T/C ratio do not appear to be a sensitive marker for performance decrements during recovery from resistance exercise.

Changes in plasma concentrations of LDH, CK, and Mb are often used as a peripheral marker of exercise-induced muscle damage (Brancaccio et al. 2006; Clarkson et al. 2006). Although CK was increased for both trials, no significant between group differences were noted indicating that both HV and HI workouts produced similar degrees of muscle damage. These results appear to contrast with Wells et al. (2016), who reported greater increases in muscle damage markers following HI compared to HV workouts. However, these authors used forced repetitions to complete the required number of repetitions per set, while the present study reduced the load on the bar if the participant was unable to complete the required number of repetitions. Forced repetitions may have induced greater damage to the muscles involved in the workout. In the present study, however, physiological parameters suggest that differences in performance recovery observed between HI and HV may be more related to the metabolic stress of the workout, rather than differences in muscle damage.

Changes in IL-6 and CRP are often used as markers of an inflammatory response (Cheyne et al. 2010; Ebbeling and Clarkson 1989). Although significant elevations were observed in IL-6 concentrations following the HV protocol, no differences were noted in CRP at any time point for either HV or HI. These results appear to support others studies (Febbraio and Pederson 2005; Serrano et al. 2008) suggesting that elevations in IL-6 concentrations may be related to an exercise-induced metabolic stress.

Table 4 Changes in muscle pain and soreness

| Assessment | Trial | BL | P-30 min | P-24 h | P-48 h | P-72 h | Time effect | | Trial | | Interaction effects | |
|------------|-------|------|----------------------------|----------------------------|----------------------------|----------------------------|-------------|----------|----------|----------|---------------------|----------|
| | | | | | | | <i>p</i> | η^2 | <i>p</i> | η^2 | <i>p</i> | η^2 |
| PAIN (mm) | HV | 0.00 | 16.17 ± 21.3 | 26.92 ± 30.3 | 26.42 ± 0.7 | 16.33 ± 20.7 | 0.190 | 0.498 | 0.012 | 0.452 | 0.240 | 0.461 |
| | HI | 0.00 | 4.67 ± 8.42 | 2.58 ± 7.4 | 1.42 ± 3.6 | 1.25 ± 3.6 | | | | | | |
| SOR (mm) | HV | 0.00 | 49.25 ± 24.6 ^{##} | 61.42 ± 23.4 ^{##} | 57.92 ± 21.5 ^{##} | 35.92 ± 17.1 ^{##} | 0.000 | 0.902 | 0.000 | 0.850 | 0.001 | 0.711 |
| | HI | 0.00 | 13.25 ± 11.1 | 23.75 ± 19.6 | 17.00 ± 14.25 | 8.50 ± 13.4 | | | | | | |

All data are reported as mean ± SD

SOR soreness intensity

[#]Indicates a significant ($p \leq 0.01$) difference from BL

^{##}Indicates a significant ($p \leq 0.001$) difference from BL

Mechanical stress is thought to represent the primary factor in muscle adaptive response by inducing disarrangements in the structure of sarcomeres (Sjogaard et al. 1985) and stimulate inflammatory responses in the muscle. There is evidence, however, that metabolic stress, including heavy demands on glycolysis promotes muscle post-exercise swelling (Sjogaard et al. 1985), which may have a substantial role in stimulating muscle hypertrophy (Goto et al. 2005). Results of the present study indicate that a greater metabolic stress was experienced during HV compared to HI, while the mechanical stress appeared to be similar for both exercise protocols. The greater number of lengthening contractions performed in HV may represent an important factor for muscle damage (Talbot and Morgan 1998) and may compensate for the lower workloads used compared to the HI protocol by increasing the mechanical stress induced by this protocol.

Fatigue resulting from the HV protocol induced elevations in muscle soreness that was still present 72 h following the workout. Soreness intensity was not related to changes in the performance decrements. This is consistent with Nosaka et al. (2002), who suggested that delayed-onset muscle soreness (DOMS) cannot be considered as a good indicator of muscle damage, since subjective and individual perceptions influence the sensation of muscle pain (Ohnhaus and Adler 1975).

Strong correlations were observed between the exercise-induced decrease in CMJP and increases in IL-6 concentrations at P-30 min, P-24 h, and P-48 h following the HV protocol. In addition, changes in CMJP were also related to the increase in CSA. Our results suggest that, in our participants' sample, changes in CMJP appeared to be a more sensitive measure than the isokinetic and isometric measures. Changes in MVIC and ISOK60 were still attenuated 72 and 24 h after the HV and the HI protocol, respectively, and were not associated with any marker of inflammation or muscle damage.

Maximal force and rate of force development expressed in IMTP and ISQ were not affected by either HV or HI at any time point. The IMTP and ISQ are compound, closed-chain isometric tests involving multiple, large muscle groups (Beckam et al. 2013). The back extensor muscles are an important contributor to performance in these isometric assessments (Nuzzo et al. 2008), and the squat protocols used in the present study may not have caused the level of fatigue in back extensors to impair IMTP and ISQ performances. Performance assessments are often used to monitor the recovery process (Byrne and Eston 2002; Clarkson and Sayers 1999). The quick recovery observed following the HI workout suggests that the same muscle group can be trained more frequently by experienced individuals using this protocol compared to HV protocols. Results of this study indicate that CMJP appears to be the most sensitive assessment of lower body recovery.

In conclusion, results of this investigation indicated that recovery from high-volume resistance exercise is slower than following exercise protocols of higher intensity. Differences in recovery between the training protocols appeared to be related to the greater metabolic stress associated with the high-volume exercise protocol. The higher metabolic stress was also reflected by a greater inflammatory response, which was associated with changes in muscle cross-sectional area, and subsequently with performance changes.

Compliance with ethical standards

Conflict of interest The authors have no conflict of interest to declare.

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