Neuromuscular Fatigue after Resistance Training

Authors	M. Izquierdo[1], J. Ibañez[1], J. A. L. Calbet[2], M. González-Izal[1], I. Navarro-Amézqueta[1], C. Granados[1], A. Malanda[3], F. Idoate[4], J. J. González-Badillo[5], K. Häkkinen[6], W. J. Kraemer[7], I. Tirapu[1], E. M. Gorostiaga[1]
Affiliations	Affiliation addresses are listed at the end of the article

Abstract
▼

This study examined the effects of heavy resistance training on dynamic exercise-induced fatigue task (5×10RM leg-press) after two loading protocols with the same relative intensity (%) (5×10RM$_{Rel}$) and the same absolute load (kg) (5×10RM$_{Abs}$) as in pretraining in men (n=12). Maximal strength and muscle power, surface EMG changes [amplitude and spectral indices of muscle fatigue], and metabolic responses (i.e. blood lactate and ammonia concentrations) were measured before and after exercise. After training, when the relative intensity of the fatiguing dynamic protocol was kept the same, the magnitude of exercise-induced loss in maximal strength was greater than that observed before training. The peak power lost after 5×10RM$_{Rel}$ (58–62%, pre-post training) was greater than the corresponding exercise-induced decline observed in isometric strength (12–17%). Similar neural adjustments, but higher accumulated fatigue and metabolic demand were observed after 5×10RM$_{Rel}$. This study therefore supports the notion that similar changes are observable in the EMG signal pre- and post-training at fatigue when exercising with the same relative load. However, after training the muscle is relatively able to work more and accumulate more metabolites before task failure. This result may indicate that rate of fatigue development (i.e. power and MVC) was faster and more profound after training despite using the same relative intensity.

Correspondence
Dr. M. Izquierdo
Gobierno de Navarra
Centro de Estudios,
Investigación y Medicina
del Deporte
C/Sangüesa 34
31005 Pamplona
Spain
Tel.: +34/94/829 26 23
Fax: +34/94/829 26 36
mikel.izquierdo@ceimd.org

Introduction
▼

Muscle hypertrophy [1,15], increases in maximal strength and power output [15,19] and enhanced agonist EMG activity [2,15,16] are typical adaptations during short-term heavy resistance training (i.e. 7–10 weeks). Only a limited number of studies, however, have examined the effects of strength training on dynamic exercise-induced fatigue of the leg extensor muscles with the same absolute and relative loading as in pretraining. Delayed development of exercise-induced leg fatigue as a result of isometric strength training has been reported through the assessment of maximal isometric voluntary contraction (MVC) before and immediately after isometric fatiguing exercise [10,29]. In contrast, similar acute decreases in maximal isometric strength have been observed when the relative intensity of the loading was kept the same before and after a long-term dynamic strength training period (21 weeks) [3]. In other studies the magnitude of exercise-induced loss (before and after training) in maximal strength has not been reported [18,25,28]. Despite the dynamic nature of most motor tasks in athletic and voluntary physical activities, many studies have frequently used isometric rather than dynamic fatiguing tasks to examine muscle fatigue, thus hindering any assessment of the effects of fatigue on power. Moreover, the effects of short-term heavy-resistance training on absolute and relative exercise-induced neuromuscular fatigue (e.g. 5 sets of 10 repetitions at a relative or absolute load) following high-power dynamic tasks with a submaximal load remain to be elucidated.

Thus, although enhanced maximal strength and power after a heavy resistance training program may give advantages to maintain power and/or enhance the recovery after a fatiguing task with the same submaximal absolute loading (i.e. same kg) as in pretraining, the question arises at to whether similar or even worse ability would be observed in maintaining muscle power after a dynamic fatiguing task with the same relative loading (i.e. same % of MVC) as in pretraining.

Fatigue-related decreases in muscle voluntary activation to maintain a given muscle power output (i.e. dynamic task failure) have been exclusively assessed by the measurement of the EMG signal during maximal voluntary isometric contractions to avoid the limitations imposed by dynamic contractions on the validity of surface EMG recordings [6–8, 11, 23, 26]. However, since the pattern of neural activation is likely to be different during dynamic and static contractions, extracting information from EMG signals obtained during a static contraction to infer fatigue-elicited changes during dynamic contractions may be questionable [8]. To extract valid information from the EMG activity recorded during dynamic muscle actions non-stationary EMG analysis techniques should be used [9, 12].

Failure to maintain a given level of peak force and muscle power under conditions of exercise-induced fatigue has been also associated with accumulation of lactate, H^+ inorganic phosphate (Pi) and ammonia and a decreased number and force of cross bridges in fast-twitch fibres, decreased myofibrillar Ca^{2+} sensitivity, and also a decline in muscle adenine nucleotide stores, mainly through a pronounced reduction in muscle ATP content [13, 17, 22, 30, 33]. To our knowledge, the effects of strength training on exercise-induced changes in blood ammonia and lactate concentrations, maximal strength, muscle power and the surface EMG signal with the same absolute and the same relative loading as in pretraining have not been studied. Indeed, we hypothesize that fatigue will elicit similar neural adjustments and, therefore EMG changes before and after training when the fatiguing task is carried out at the same relative intensity. However, we also expect that training will allow greater metabolic demand with a higher accumulation of metabolites (i.e. blood lactate and ammonia) before task failure. Therefore, the purpose of the present study was to examine during short-term heavy resistance training (first 7 weeks) neuromuscular and metabolic responses after two loading protocols with the same relative intensity [% of one repetition maximum (1RM)] and the same absolute load (kg) as in pretraining.

Methods

Subjects

Twelve physically active men volunteered to participate in the study. The subjects' mean (±SD) age, height, body mass and percentage of body fat were 33 (±4.4) years, 1.77 (±0.06) m; 72.4 (±6.9) kg and 9.2 (±2.5)% respectively. The subjects had experience with recreational training, although none had been involved in any regular strength training program at the beginning of the study. Each subject gave his written informed consent to participate after the risks of the investigation were carefully explained. The experimental procedures were approved by the Institutional Review Committee of the Instituto Navarro del Deporte and were in accordance with the Declaration of Helsinki. Before inclusion in the study, all subjects were medically screened by a physician and were free from any orthopaedic, electrocardiographic, endocrine or medical problems that would contraindicate their participation or influence the results of the investigation. None of them was taking exogenous anabolic-androgenic steroids, drugs, medications or dietary supplements with potential effects on physical performance.

Experimental design

A longitudinal randomized research design was used to compare the neuromuscular responses and recovery profile elicited by two loading protocols with the same relative load (%) and the same absolute load (kg) before and after short-term heavy resistance training (7 weeks). The experimental design comprised three acute heavy-resistance exercise protocols (AHREP). One of them was performed before training (5×10 RM leg press) and the other two after the 7-week experimental strength training period. Baseline testing was completed during the first three weeks of the study before the start of the training program. The two AHREP sessions performed after training were separated by 7 days and were performed with the same relative load (%) and the same absolute load (kg) as in pretraining, respectively (see ○ **Fig. 1**). The volunteers were familiarized with the experimental testing procedures about 2 weeks before the AHREP session. After a thorough familiarization period one week before the
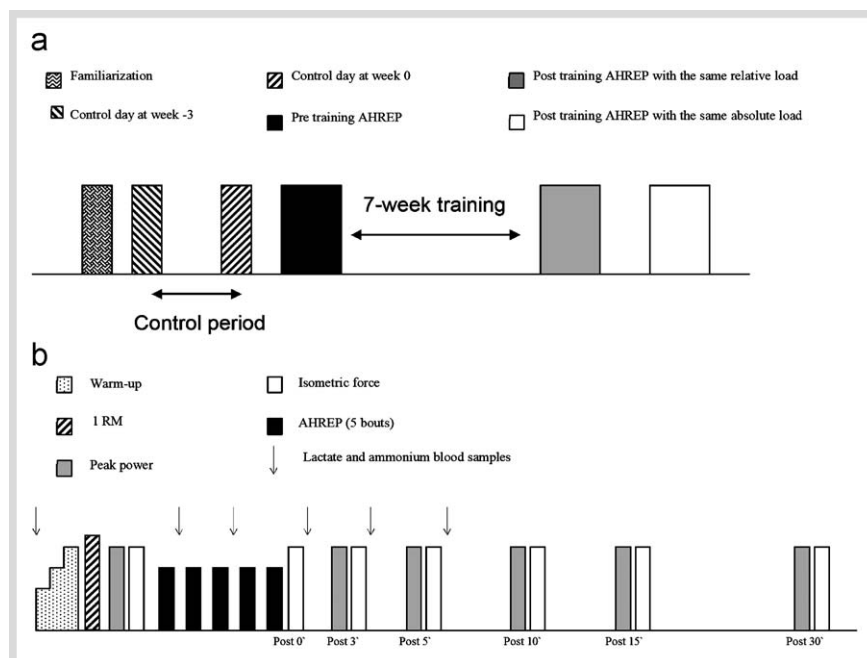

Fig. 1 Longitudinal experimental design (**a**) and acute heavy resistance protocol design (**b**).

AHREP session the subjects participated in a control testing day to determine one repetition maximum (1RM), maximal voluntary contraction (MVC), muscle power, and the maximum load possible to achieve 10 repetitions (10RM) (○ **Fig. 1**). Before training, the measurement of muscle function was performed twice in order to assess reproducibility.

Immediately before each AHREP (pre-exercise) session each subject's 1RM and MVC were determined. Muscle power was assessed with the load corresponding to pre-exercise 10RM (i.e. control). After each AHREP, i.e. in the fatigued state, the MVC and muscle power with the actual load corresponding to 10RM were performed immediately post-exercise (post 0), and 3, 5, 10, 15 and 30 min into the recovery period (○ **Fig. 1**).

Acute heavy resistance loading protocols

The experimental design comprised the examination of strength training-induced adaptations on the acute neuromuscular responses with the same absolute (kg) and relative load (% of 1RM). Before training, the AHREP consisted of 5 sets with the load corresponding to 10RM in leg press with 120 s of rest between the sets. After training each subject performed a randomized AHREP (separated by 7 days) with the same relative load ($5 \times 10RM_{Rel}$) and the same absolute load ($5 \times 10_{Abs}$) as in the pretraining testing protocols. If the subject failed to perform 10 repetitions (before and after training in the $5 \times 10RM_{Rel}$ loading protocol) due to fatigue on any given set, the load was subsequently adjusted (i.e. lightened) to allow the completion of 10 repetitions on the same set. For comparison purposes, the $5 \times 10RM_{Abs}$ loading protocol after the strength training period was performed with the same absolute loads used before training.

A bilateral leg extension exercise machine (i.e. leg press action in a sitting position) (Technogym, Gambettola, Italy) was used for all trials. The seat was individually adjusted to minimize displacement between the lower back and the backrest during muscular force exertion. Strong verbal encouragement was given to all the subjects to motivate them to perform each test action as maximally and as rapidly as possible. The subjects were asked to eat similar diets before the loading sessions. Subjects did not perform any strenuous exercise for 48 h before the experimental exercise session.

Maximal strength and muscle power output

One repetition maximum (i.e. the heaviest load that could be correctly pressed only once using the correct technique) was determined for the leg press exercise machine (Technogym, Gambettola, Italy). The subject was in a seated position so that the knee angle was 90°. Warm-up consisted of a set of 5 repetitions at 50% of the estimated maximum. Thereafter, three to four repetitions at 75% of perceived maximum and 1 repetition at 90% of perceived maximum. Three to four subsequent attempts were made to determine the 1RM. The rest between maximal attempts was always 2 min.

Maximal isometric force was also measured on a modified leg press exercise machine (Technogym, Gambettola, Italy) at knee and hip angles of 90° and 45° respectively. In this test, the subjects were instructed to exert their maximal force as fast as possible during a period from 2.5 to 4 s.

Muscle power output of the leg extensor muscles was measured during the concentric phase of leg press action using the individual maximum load corresponding to what the subject could perform 10 times (10RM). The subject was instructed to move the weight as fast as possible. Two testing trials were recorded and the best trial was taken for further analysis. The exercise machine incorporated several force transducers on a foot platform located below the subject's feet. The strain gauges recorded the applied force (N) to an accuracy of 1 Newton at 1 000 HZ. In addition, an optical encoder (Computer Optical Products Inc, California, USA) was attached to weight plates to record the position and direction of the displacement to an accuracy of 0.2 mm at 1 000 Hz. Customized software was used to calculate range of motion, peak power output and average velocity for each repetition. The test-retest intraclass correlation coefficients for all strength and power variables were greater than 0.95 and the coefficients of variation (CV) ranged from 0.9% to 2.1%.

Muscle Cross-Sectional Area (CSA) and anthropometry

The muscle CSA of the left quadriceps femoris (QF) was assessed before and after the 7-wk resistance training period using MRI (SIEMENS Magnetom Impact Expert, 1 T). Once the subject was positioned inside the magnet, the thighs of both legs were kept parallel to the MRI table and the feet were strapped together to prevent rotation. The length of the femur (Lf), taken as the distance from the intercondilar notch of the femur to the superior boundary of the femoral head, was measured on a coronal plane. Subsequently, 15 axial scans of the thigh interspaced by a distance of 1/15 Lf were obtained from the level of 1/15 Lf to 15/15 Lf (slice 3 being closer to the knee joint). Great care was taken to reproduce the same individual Lf each time by using the appropriate anatomical landmarks. In addition, one scan was taken at the site (marked on the skin by the ink tattoo) of the muscle biopsy of the vastus lateralis (VL) muscle. For each axial scan, CSA computation was carried out on the QF as a whole and, individually, on the VL, vastus medialis (VM) vastus intermedius (VI), and rectus femoris (RF). For the final calculation of the CSA, slices 5/15–12/15 were used for all muscles examined except for the RF, which was analyzed only for slices 5/15–12/15. CSA (cm^2) was determined by hand tracing of the border of each muscle. Body mass and percent body fat (estimated from the thickness of seven skin-fold sites) were taken before and after each training period [20].

Muscle biopsies

Needle muscle biopsies were obtained from the middle section of the vastus lateralis muscle under local anaesthesia without suction, but with mild pressure on the lateral aspect of the thigh. Biopsies before and after the 7-week period were obtained from six of the subjects. The muscle samples were immediately mounted with Tissue-Tek and frozen in isopentane cooled with liquid nitrogen, and stored at −80°C. MHC analyses were performed on the muscle biopsies using sodium dodecylsulfate polyacrylamide gel electrophoresis (SDS-PAGE) [31]. From each biopsy 20–40 serial cross-sections (10 μm) were cut and placed in 200–500 μL of lysing buffer and heated for 3 min at 90°C. Between 2 and 12 μl of the myosin-containing samples were loaded on a SDS-PAGE. Gels were run at 70 V for 43 hours at 4°C. Subsequently, the gels were Coomassie stained and MHC isoform bands (I, IIa, IIx) were determined based on known migration patterns and quantified with Un-scan-it gel software (Orem, UT).

Electromyography

EMG activity of the VL and VM muscles during leg extension was recorded from the right leg by using bipolar surface electrodes

(Blue Sensor N-00-S, Medicotest) with a 20 mm inter-electrode distance during dynamic muscle actions. A detailed description of the EMG procedures can be found elsewhere [2, 16]. Briefly, after careful preparation of the skin (shaving, abrasion, and cleaning with alcohol), electrode pairs were placed longitudinally on the medial portion of the muscles. The positions of the electrodes were marked on the skin by small ink tattoos [16]. The dots ensured the same electrode positioning in all tests. EMG signals were recorded at a 1 kHz sampling rate, with Muscle Tester ME3000 (Mega Electronics Ltd) (bandwidth of 8–500 Hz/3 dB and a common mode rejection ratio >100 dB). EMG signals were full-wave rectified and filtered by a moving root-mean-square filter with a time constant of 50 ms. The movement was divided into four intervals of 22.5° of the knee displacement. Spectral indices were calculated over time at the same joint angle to assess relative, rather than absolute, myoelectric manifestations of fatigue [9, 14]. The following parameters were identified in the first interval of the movement (from 90° to 112.5° of the knee displacement) for the two muscles because it was the part of the movement where the muscles were more activated during the concentric phase of the leg press actions: MAV (mean average voltage) calculated as the integrated EMG/integration time [2] normalized by the MAV value of the first quarter of the 1RM, and median frequency (Fmed).

$$\int_{f_1}^{Fmed} PS(f) \cdot df = \int_{Fmed}^{f_2} PS(f) \cdot df$$

where PS(f) was the spectral power for the currency frequency f and f1 = 8 Hz and f2 = 500 Hz were the high and low-pass frequencies of the amplifier filter.

A new highly sensitive spectral index proposed by Dimitrov and co-workers [9] was also calculated in the same intervals. This spectral index (FI_{nsm5}) has been proposed to overcome the relatively low sensitivity of the median frequency and the mean frequency. The index is calculated as the ratio between the signal spectral moment of order (-1), which emphasizes the increase in low and ultra-low frequencies in the EMG spectrum attributable to increased negative after-potentials during muscular fatigue, and normalizing spectral moment of order 5, which emphasizes the effect of decreases in the high frequencies attributable to the increased duration of the intracellular action potentials and the decreased action potential propagation velocity [9].

$$FI_{nsm5} = \frac{\int_{f_1}^{f_2} f^{-1} \cdot PS(f) \cdot df}{\int_{f_1}^{f_2} f^5 \cdot PS(f) \cdot df}$$

where PS(f) was the spectral power for the currency frequency f and f1 = 8 Hz and f2 = 500 Hz were the high and low-pass frequencies of the amplifier filter.

After calculating all of these parameters, an arithmetical mean was calculated between the parameters of VM and VL to obtain a value which included the effects of the fatigue of the two muscles.

Blood lactate and ammonia analysis

Capillary blood samples for the determination of lactate and ammonia concentrations were obtained from a hyperemized earlobe *pre-exercise, after the 1st set, after the 3rd set (mid-exercise), and peak value post-exercise*. Samples for whole blood lactate determination (100 µl) were deproteinized, placed in a preservative tube (YSI 2315 Blood Lactate Preservative Kit) stored at 4°C, and analyzed (YSI 1500) within 5 days of completing the test. According to the manufacturer's instructions, placing the capillary samples in these preservative tubes allows the blood samples to be stored for 3–5 days with stable blood lactate concentration values (Pooled Estimate of Standard Deviation: $0.15 \, mmol \cdot l^{-1}$ for a concentration range of 0 to $10 \, mmol \cdot l^{-1}$). The blood lactate analyzer was calibrated after every fifth blood sample dosage with three known controls (5, 15 and $30 \, mmol \cdot l^{-1}$). After cleaning and puncturing, a single 20 µmol of whole blood sample was taken from hyperemized earlobe with an Eppendorf varipipette and immediately analyzed with an ammonia checker (BAC) II (model AA-4120, Kyoto Daiichi, Kayaku Co., Ltd. Japan, Menarini Diagnostic, Italy) with a simple volume of 20 µl blood. This analyzer uses a reflectometer to optically measure the reflection intensity (45°) of reagent color reaction in biochromatic mode and was calibrated before and after every test with a known control ($58.7 \, \mu mol \cdot l^{-1}$).

Resistance training program

A trained researcher supervised each workout session carefully so that exercise prescriptions were properly administered during each training session (e.g. number of repetitions, rest and velocity of movement). Compliance with the study was 100% of the programmed sessions.

Subjects were asked to train two times per week for 7 weeks to perform dynamic resistance exercises from 45 to 60 min per session. A minimum of 2 days elapsed between 2 consecutive training sessions. During the training period, the core exercises performed were parallel-squat and bench press, in addition to supplementary strengthening exercises for selected muscle groups (leg press, leg extension, shoulder press, lateral pull-down, abdominal crunch, trunk extension, and standing leg curl). The resistance training consisted of a nonlinear, multi-set, multi-exercise, progressive program performed 2 times per week. The daily workouts were alternated by varying the resistance (intensity), and the volume (sets × repetitions × load) over the week. On Tuesday the sets were performed at 12–15RM with 2 min rest between sets. Finally, on Thursday the sets were performed at the 10RM intensity. Three to five sets were performed during the training program. The assigned training intensities were gradually increased during the course of the 7-week training period using a repetition maximum approach.

Statistical analyses

Standard statistical methods were used for the calculation of the means and standard deviations (SD). The training-related effects on acute AHREP-induced responses in maximal isometric, muscle power and several EMG indices were assessed using a two-way ANOVA with repeated measures. When a significant F-value was achieved, Bonferroni po*st hoc* procedures were performed to locate the pairwise differences between the means. Statistical comparison during the control period (from week –3 to week 0) was performed by Student's paired *t* -test. Selected absolute and relative changes in maximal isometric, muscle power and several EMG indices were analyzed via one-way analysis of variance. Statistical power calculations for this study ranged from 0.75 to 0.80. The $p < 0.05$ criterion was used to establish statistical significance.

Results

▼

Anthropometry and muscle CSA

After the training period, significant changes took place in body mass and body fat percentage, from 72.4 ± 6.9 to 73.4 ± 6.2 kg and from 9.2 ± 2.5 to 9.7 ± 1.5 % respectively. No significant change was observed in free fatty mass.

The Quadriceps CSA from MRI scans increased significantly during the training period (P<0.001) along with the length of the thigh from 6/15 to 12/15 Lf. The mean increase of the thigh muscles at different Lt was 4.4% (from 130.1 ± 10.6 to 135.7 ± 12.2 cm^2 after 7 wks of training) ranging from 2.9 to 9.6 %, but these did not differ significantly from each other (○ **Fig. 2**).

Myosin heavy chain isoform distribution

Strength training resulted in an increased amount of MHC type IIa (from 21.7 ± 0.7 to 38.6 ± 2.3 %; $p \leq 0.05$) and a reduction in the amount of MHC type I (from 54.1 ± 1.8 to 49.5 ± 2.0 %; $p \leq 0.05$) and MHC type IIx (from 18.2 ± 0.7 to 7.9 ± 0.6 %; $p \leq 0.05$).

Maximal bilateral isometric, 1RM dynamic strength and muscle power output

Maximal strength and muscle power output remained unaltered during the 3-week control period (from week -3 to week 0). During the 7-week training period significant increases of 10.8 ± 1.3 % (P<0.05) and 19.7 ± 4.7 % (P<0.01) were recorded in maximal isometric force (from 1557 ± 211 N to 1772.7 ± 304 N) and 1RM (from 190.6 ± 30.2 to 237.9 ± 38.8 kg).

After the 7-week training period, peak power output with the same absolute load as pretraining increased by 16.2 ± 12.8 % (from 1163.2 ± 239.9 to 1414.4 ± 372.0 W, P<0.001), whereas no significant changes were observed in peak power output with the same relative load used in pretraining (from 1163 ± 239 to 1159 ± 320 W).

After the 7-week training period, peak velocity with the same absolute load as pretraining significantly increased by 15% (from 0.67 ± 0.1 m·s^{-1} to 0.79 ± 0.1 m·s^{-1}; P<0.001), whereas a similar

peak power output with the same relative load as pretraining was related to a higher absolute load [i.e. similar relative (10RM) as in pretraining], but with a decrease in peak velocity (from 0.67 ± 0.1 m·s^{-1} to 0.56 ± 0.1 m·s^{-1}; P<0.001).

Acute heavy 10RM resistance loading

After the 7-week training period the initial load of the 5×10$_{Rel}$ loading protocol was increased from 160.2 ± 26.3 to 198.9 ± 33.9 kg (P<0.001). Before training, the % level of the 1RM used during the 10RM loading was 84.1 ± 4.8% of 1RM (ranging between 75.1% and 90.5%) which was similar to that of 83.7 ± 4.8% of 1RM (ranging between 74.9 and 91.1) used after training. After training total work (sets × reps × load) performed with the same relative load (5×10$_{Rel}$ loading) was increased by 15.5 ± 6.6 % compared to that used before training (from 7515.7 ± 1040.1 to 8939.6 ± 1340.6 kg, P<0.001) (○ **Table 1**).

After training, the load used during the 5×10RM$_{Abs}$ represented 67.6 ± 5.7 % of the post-training 1RM. Total work (sets × reps × load) performed with the same absolute load (5×10RM$_{Abs}$ loading) was similar before and after training.

After training, the relative decrease (%) of the load during the 5×10$_{Rel}$ protocol was higher (P<0.05) for the 2nd, 4th and 5th bouts compared with that recorded before training. (○ **Table 1**). For comparison purposes, after training the absolute decrease of the load during the 5×10$_{Abs}$ loading protocol was matched to that recorded before training.

AHREP-induced changes in maximal isometric and muscle power output before and after training

Maximal isometric force immediately after the 5×10RM$_{Rel}$ loading was decreased by 23.4 ± 11.7 % before and 34.2 ± 15.8% after training (both P<0.05), but with a significantly greater exercise-induced loss of MVC after training (before vs. after training, P<0.01). MVC remained low during the first 30 min of the recovery period (○ **Fig. 3A**) under both conditions.

Training attenuated the losses in MVC elicited by the 5×10RM$_{Abs}$ loading protocol (11.4%) compared to those observed immediately after those elicited by the 5×10RM$_{Rel}$ loading protocol, either before or after the training period. After the 5×10RM$_{Abs}$ loading protocol MVC remained low during the first 30 min of recovery (○ **Fig. 3A**).

Peak power output decreased similarly (P<0.05) immediately after the 5×10RM$_{Rel}$ loading protocols both before and after training (58.4% and 62.3% respectively) and remained diminished until 10 min and 15 min of recovery respectively (○ **Fig. 3B**). After training, significant decreases (P<0.05) were observed in peak power output immediately post-exercise (0 min post) in the 5×10RM$_{Abs}$ loading protocol (20.3%), but pre-exercise peak power was re-established at 3 min post-exercise (○ **Fig. 3B**). The 5×10RM$_{Abs}$ loading protocol elicited lower reductions in peak power output after training than 5×10RM protocol before training and 5×10RM$_{Rel}$ after training (P<0.05).
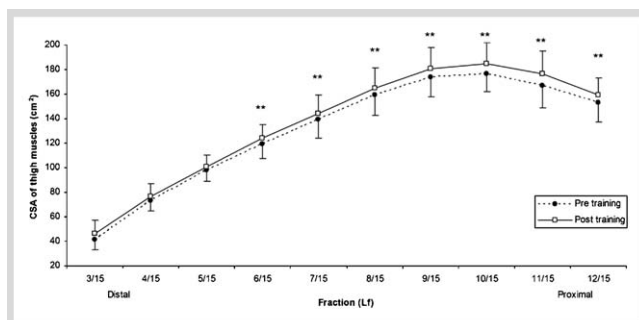

Fig. 2 Mean ± SD cross-sectional areas (CSAs) of the quadriceps femoris (CF) at the lengths of the femur (Lf) from 3/15 to 12/15 in subjects before and after strength training period (**P<0.01).

Table 1 Initial load (kg) and load decrease (expressed in % of initial load) for each bout during the acute heavy resistance protocols (Five sets of ten repetition maximum), pre and post training with the same relative load.

	Initial load (10RM, kg)	1st bout (Δ%)	2nd bout (Δ%)	3rd bout (Δ%)	4th bout (Δ%)	5th bout (Δ%)
5×10$_{Rel}$ pretraining	160.2 ± 26*	0.62 ± 1.6	2.1 ± 3.7 §	5.3 ± 6.1	8.9 ± 6.6[abc §]	11.9 ± .8.3[abc §]
5×10$_{Rel}$ post-training	198.9 ± 33.9	0.9 ± 1.6	5.0 ± 3.7[ab]	9.4 ± 7.3 [a]	15.5 ± 7.9[abc]	17.3 ± 8.7[abc]

*Significant difference (P<0.001) between pre and post training initial 10RM load (kg). [a] Significant difference (P<0.05) compared to the corresponding 1st bout value for each protocol. [b] Significant differences (P<0.05) from the corresponding 2nd bout value, for each protocol. [c] Significant differences (P<0.05) from the corresponding 3rd bout value, for each protocol. §P<0.05 significant differences between 5×10 pre training and 5×10 post training with the same relative load
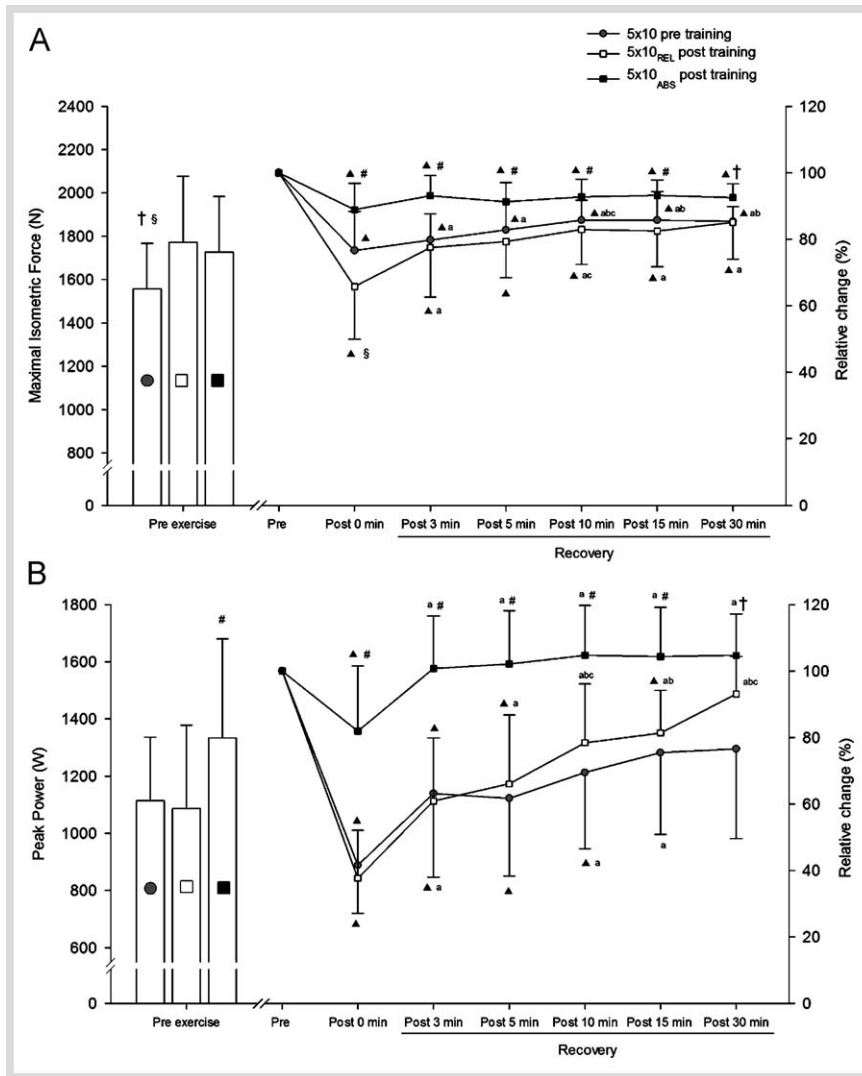
Fig. 3 Relative changes in maximal isometric force (**A**) and peak power output (**B**), before, post-exercise and during recovery, for the three acute heavy resistance protocols performed at pretraining (5 × 10 pretraining) and with both the same relative (5 × 10RM$_{Rel}$) and the same absolute load (5 × 10$_{Abs}$) as pretraining. ▲ Significant difference (P<0.05) compared to the corresponding pre-exercise value, for each protocol. [a] Significant differences (P<0.05) from the corresponding post 0 value, for each protocol. [b] Significant differences (P<0.05) from the corresponding post 3 min value, for each protocol. [c] Significant differences (P<0.05) from the corresponding post 5 min value, for each protocol. # P<0.05 significant differences between 5 × 10$_{Abs}$ post training and the other two protocols. †P<0.05 significant differences between 5 × 10 pre training and 5 × 10$_{Abs}$ post training. §P<0.05 significant differences between 5 × 10 pretraining and 5 × 10$_{Rel}$ post-training.

Peak velocity declined to the same extent after the loading in the 5 × 10RM$_{rel}$ protocol regardless of the training status (42.1% and 42.2% of the baseline value, before and after, respectively, P<0.05), remaining diminished until 10 min and 15 min of recovery respectively. After training, no significant decreases were observed in peak velocity inmediately post-exercise (0 min post) in the 5 × 10$_{Abs}$ loading protocol.

Acute EMG responses to training
EMG activity of the VL and VM muscles remained unaltered during the 3-week control period (from week -3 to week 0).
Compared to pre-loading, MAV was significantly increased after the 5 × 10$_{rel}$ loading protocols in both before and after training, and remained unchanged after the 5 × 10RM$_{abs}$ protocol. During the recovery period, the MAV after the 5 × 10RM$_{rel}$ protocol before training reached initial values at 3 min, whereas the MAV after the 5 × 10RM$_{rel}$ protocol after training did not reach them until 5 min post-exercise. Pre-loading MAV was comparable at the beginning of the three protocols (○ **Fig. 4A**)
The median frequency decreased significantly (P<0.05) in the 5 × 10RM$_{rel}$ loading protocols in both pretraining and post-training and regained its initial value 3 min and 5 min post-exercise respectively. No significant changes were observed in the median frequency of the 5 × 10RM$_{abs}$ (○ **Fig. 4B**).

The FI$_{nsm5}$ increased significantly (P<0.05) after loading in the 5 × 10RM$_{rel}$ loading protocols and decreased to initial values 3 min and 5 min after the 5 × 10$_{rel}$ loading protocol before and after training, respectively. No significant changes were observed in the FInsm5 of the 5 × 10RM$_{abs}$ loading protocol. The FInsm5 values of both 5 × 10RM$_{rel}$ loading protocols were significantly higher than the values obtained in the 5 × 10RM$_{abs}$ loading protocol at 0 and 3 min post-exercise respectively (○ **Fig. 4C**).

Blood lactate and ammonia concentrations
Blood lactate concentrations increased (P<0.05) after the first bout, mid-exercise and peak after loading in all protocols examined before and after the strength training period. After training, the mean of the blood lactate was greater (P<0.05 after the first bout, mid exercise and peak post exercise) in the 5 × 10RM$_{rel}$ compared with that observed in the 5 × 10RM protocol performed before training. The blood lactate response was lower (p<0.05) during the entire loading and the recovery period in the 5 × 10RM$_{Abs}$ compared with the response observed in the 5 × 10RM$_{Rel}$ (○ **Fig. 5A**).
There were no differences between the protocols in blood ammonium concentrations at rest (○ **Fig. 5B**). Blood ammonium concentrations increased (P<0.05) after the first bout, in mid-exercise and after loading in all protocols examined, before and after the strength training period. After training, the mean of the
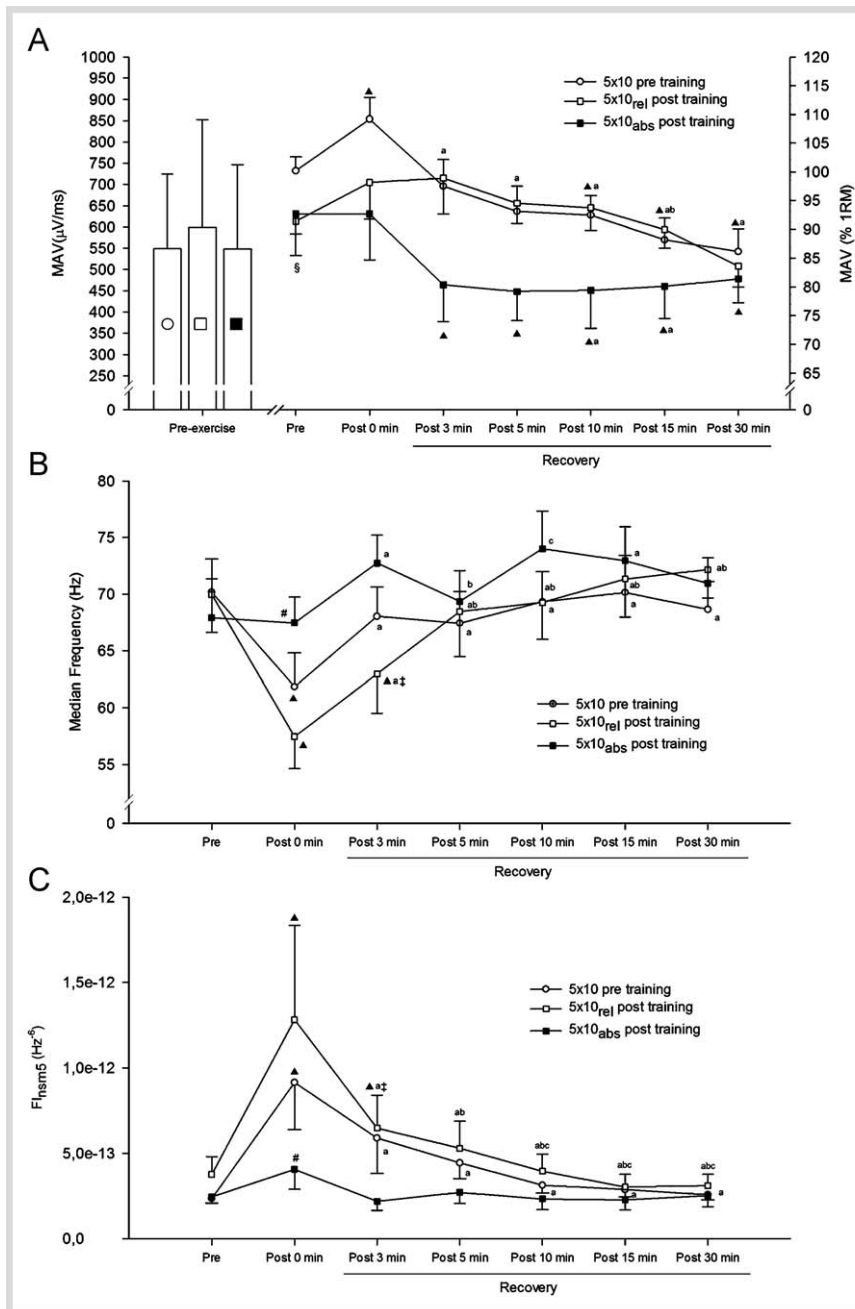
Fig. 4 EMG parameters between 90° and 112.5° of the knee extension movement before, post-exercise and during recovery, for the three acute heavy resistance protocols performed at pretraining (5×10 pretraining) and with both the same relative (5×10RM$_{Rel}$) and the same absolute load (5×10$_{Abs}$) as pretraining. Mean average voltage (**A**), median frequency (**B**) and FI$_{nsm5}$(**C**). ‡Significant differences (p<0.05) between 5×10$_{Abs}$ and 5×10$_{Rel}$ post training protocols. Significances are as described in ○ **Fig. 3**.

blood ammonium was lower (P<0.05 at mid-exercise and peak post exercise) in the 5×10RM$_{Abs}$ compared with the levels observed in both the 5×10RM$_{rel}$ protocols performed before and after training. After training, the peak ammonium value reached at the end of the exercise was higher (P<0.05) in the 5×10RM$_{rel}$ compared with that observed in the other two protocols.

Discussion

▼

The main findings of this study were that after short-term heavy resistance training, when the relative intensity of the fatiguing dynamic protocol was kept the same 1) the magnitude of exercise-induced loss in maximal strength was greater than that observed before training, 2) the peak power lost after AHREP (58–62%, before and after training) was greater than the corresponding exercise-induced decline observed in maximal

dynamic (23–34%) and isometric strength (12–17%), but this was followed by a more rapid and complete recovery, 3) the magnitude of the exercise-induced neuromuscular changes were similar than before training, as well 4) higher accumulation of blood lactate and ammonia concentration after training were observed. As expected, after training the signs of acute exercise-induced fatigue in the protocol with the same absolute load as in pretraining were much less than those observed in post-training. After a short-term strength training period, the main mechanisms responsible for the increased capacity to work with the same relative intensity are mainly of a peripheral nature, since similar neural adjustments but higher accumulated fatigue and metabolic demand (i.e. blood lactate and ammonia accumulation) were observed after multiple sets of dynamic fatiguing high-power contractions with the same relative load as in pretraining. This result may indicate that rate of fatigue devel-
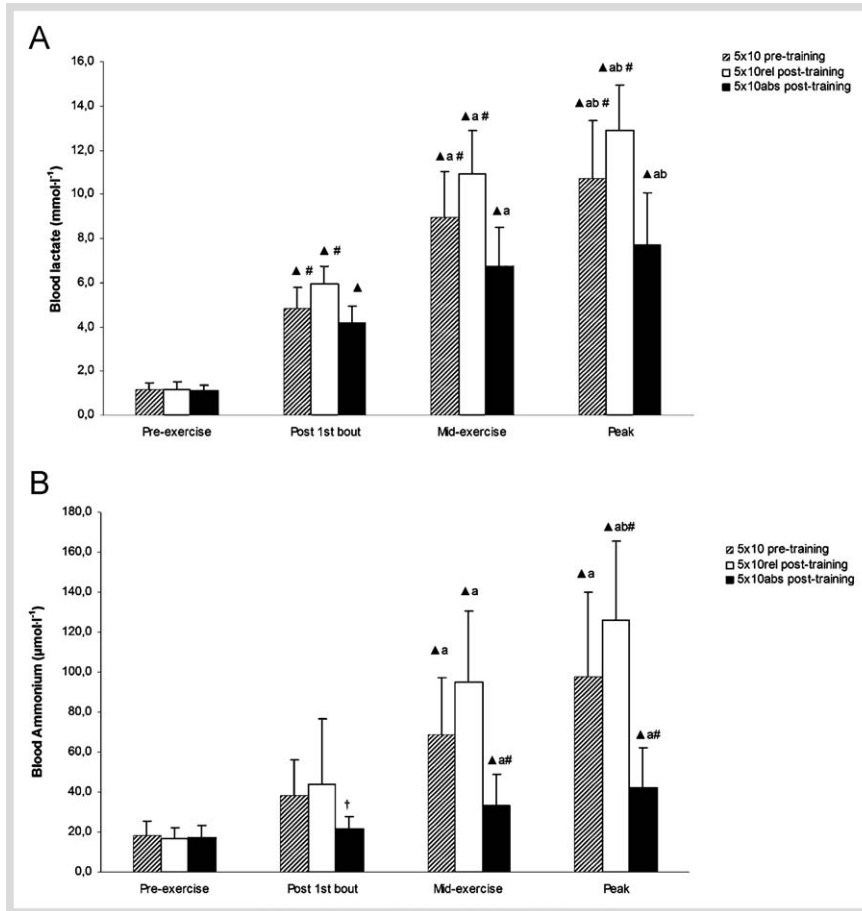
Fig. 5 Blood lactate (**A**) and ammonium (**B**) concentrations ($\mu mol \cdot l^{-1}$) before and after the first bout, after the middle of exercise (3rd bout) and peak value after the exercise, or the three acute heavy resistance protocols performed at pretraining (5×10 pretraining) and with both the same relative (5×10RM$_{Rel}$) and the same absolute load (5×10$_{Abs}$) as pretraining. Significances are as described in ◎ **Fig. 3**.

opment was faster and more profound after training despite using the same relative intensity.

AHREP-induced changes in maximal isometric and muscle power output before and after training

A comparison of our results with other studies is difficult because no study has examined the early-phase effects of heavy-resistance training on dynamic exercise-induced fatigue task and recovery profile in muscle power output and maximal strength (dynamic and isometric) lost in terms of both absolute and relative loading terms. Our results partially support previous cross-sectional studies using moderate-load isotonic dynamic muscle actions (i.e. 50% of the MVC) [8,21,23]. In general, these studies report greater losses in isotonic power than in MVC followed by a faster and more complete recovery in power output than in MVC [8,21,23]. Furthermore, similarly to previous studies the loss of power after dynamic task failure was mainly related to a reduction in muscle shortening velocity [8,35], rather than to a decline in force generating capacity. In the present study the velocity of load displacement was equally reduced (42%) before and after the strength training period.

The maximal strength (dynamic and isometric) lost after AHREP was less than the corresponding exercise-induced decline observed in power, but it was incompletely recovered after 30 min post exercise, before and after the strength training intervention. Therefore, it is likely that muscle power (velocity) and force loss and recovery after AHREP may be influenced by the involvement of different limiting factors. In contrast to the limiting factors to sustain and recover muscle power output and velocity during an isoinertial (i.e. fixed mass) dynamic task to

failure the force-generating capacity seems to be more dependent on the number of active cross-bridges than on the kinetics of the cross-bridge [5,8]. Maximal isometric force loss and short-term recovery (i.e. after 5–10 min) after dynamic fatiguing contractions of a short duration, induced by moderate loads (i.e. 50% of MVC), has been related to excitation-contraction (E-C) coupling failure [8,23] rather than to altered neuromuscular transmission and/or sarcolemmal excitation [8,23]. An incomplete recovery of isometric strength and iEMG-activity after 2 days has also been also reported after dynamic task failure using higher loading (i.e. 10 RM or 80% of 1RM), partly related to preceding fatigue of mainly central origin [26]. These findings indicate that different physiological mechanisms account for fatigue during isometric and dynamic muscle contractions. Since the metabolic component that supposedly alters muscle contraction is similar before the power and MVC assessment, the observed difference in the rate of fatigue accumulation and recovery between MVC and peak power likely reflects the greater influence of fatigue-eliciting metabolites on muscle shortening velocity than on force-producing capacity [8,13].

Furthermore, a unique finding of the present study was that after a short-term training period, and despite the significant training-induced increases observed in maximal strength, muscle mass and muscle power output in absolute terms, the loading protocol with the same relative load as in pretraining led to a similar reduction in muscle power output but greater decreases in MVC and 1RM. Strength training resulted in a remarkable increase in exercise performance (i.e. reduced fatigability) as shown by the attenuation of the degree of fatigue elicited by the same absolute load after training. Power and maximal strength

losses were about 40 and 18 % lower respectively. Moreover, not only the magnitude of fatigue was reduced but the recovery process was also faster. In contrast to the present study, similar decreases in maximal isometric strength were observed in physically active and strength-trained male athletes [3] when the relative intensity of the loading was kept the same before and after a long-term strength training period (21 weeks), whereas in other studies the magnitude of exercise-induced loss in maximal strength or muscle power was not reported [19, 25, 28].

AHREP-induced changes in blood lactate and ammonia concentration before and after training

Strikingly, the subjects endured more rate of fatigue development after training when exercising at the same relative intensity. This finding indicates that the subjects were able to perform each set more intensively after the training period, with an increasing degree of failure and ATP turnover. This is supported by the higher accumulation of blood lactate and ammonia concentration after training and coincides with certain sprint training studies that show increased capacity to use muscle glycogen after training [14, 22, 27, 36]. Our finding could also indicate that fatigue eliciting sensory feedback from the fatigued muscles after training is either reduced or processed in a different way, such that the muscle is pushed further and the relative loss of performance is higher [4]. It cannot be ruled out, however, that other factors at the molecular level could also have contributed to the present findings, such as the observed change in myosin heavy chain composition, with shift towards faster MHC phenotypes [5, 31, 34]. Our results are in agreement with previous studies [31, 34] which reported an increase of MHC IIa with a subsequent decrease in type IIX fiber percent and a reduction of MHC I after short-term heavy resistance training. Glycolytic fibres are able to produce higher power output than type I. In addition, type II fibres have greater glycolytic power. Thus, the higher lactate accumulated in blood after training would also be compatible with this kind of adaptation.

The fact that the same relative loading leads to increased rate of fatigue development after training has important practical implications. This may suggest that despite the enhanced ability to produce maximal strength and muscle power after a strength training program, it is important to note that when prescribing training programs a similar relative load as in pretraining could lead to a greater rate of fatigue development and, therefore, may induce different training effects.

AHREP-induced changes in EMG responses before and after training

In the present study, in addition to the classical spectral and amplitude analyses of surface EMG, the potential contribution of peripheral muscle fatigue during dynamic contractions was also quantified based on the analysis of new spectral indices of the EMG signal [9]. In doing so, we overcame the problem of the relatively low sensitivity of median frequency analysis and used a more valid EMG analysis technique method to assess peripheral fatigue during dynamic contractions [9, 12]. Both loading protocols with the same relative loading performed before and after training also led to major neuromuscular fatigue of similar magnitude observed with the acute increase in the surface EMG amplitude, with a shift of the EMG power spectrum towards lower frequencies, as well as with a sevenfold increase in the magnitude of the spectral fatigue index analyzed. As expected, after training the signs of acute exercise-induced fatigue in the

protocol with the same absolute load as in pretraining were much smaller than those observed in post-training. In the present study, increased EMG amplitude and decrement in the median frequency in the presence of fatigue in the $5 \times 10_{Rel}$ protocols in both pretraining and post-training may be primarily attributed to additional motor unit recruitment and/or increased spatial or temporal motor unit synchronization, presumably to compensate muscle fibre fatigue [32]. Moreover, the new spectral parameter proposed by Dimitrov and coworkers [9], showed a greater increase in the presence of fatigue than that reported by the median frequency. These changes in the frequency spectrum showed by the median frequency and the new spectral parameter towards the low frequencies may be partly related to an increase in the duration of the motor unit action potential waveform and a subsequent decrease in muscle fibre conduction velocities [6]. These results would suggest that neural adjustments such as the subjects´ capacity to generate neural drive and EMG power spectrum, and their modifications over time to attain a certain level of muscle power output may be compromised after dynamic fatiguing task when fatigue is examined after dynamic contractions with submaximal loads (i.e. 83–84 % of 1RM) but with maximal effort (i.e. maximal velocity). Similar findings have also been reported from EMG fatigue quantification based on isometric contractions before and after other repetitive isokinetic [24] and isotonic dynamic exercises [8, 23] suggesting that peripheral impairments may be also the primary factor involved in the loss of performance after moderately loaded (i.e. 50 % or less of MVC) repetitive dynamic contractions.

In addition, an interesting finding of the present study was that the magnitude of the acute neuromuscular fatigue produced by the loading was similar before and after the short-strength training period, when the relative intensity of the loading was kept the same. Thus we may assume a similar pattern of motor unit recruitment at fatigue in both pre- and post-training. This also indicates that the neural mechanisms eliciting fatigue were also similar pre- and post-training when exercising with the same relative load, suggesting that an increased capacity to perform more exercise and endure a greater loss of strength during the AHREP$_{Rel}$ protocols was mainly due to peripheral adaptations. Furthermore, as expected, when the acute heavy resistance protocol was performed with the same absolute loading as in pretraining, no significant neuromuscular muscle fatigue impairments were observed after the short-strength training period.

Taken together our findings indicate that after a short-term strength training period the main mechanisms responsible for the increased capacity to work with the same relative intensity are mainly of a peripheral nature, since similar neural adjustments but higher accumulated fatigue and metabolic demand (i.e. blood lactate and ammonia accumulation) were observed after multiple sets of dynamic fatiguing high-power contractions with the same relative load as in pretraining. This study therefore supports the notion that similar changes are observed in the EMG signal pre- and post-training at fatigue when exercising with the same relative load. However, after training the muscle is relatively able to work more and can accumulate more metabolites before task failure. This result may explain that task failure occurred with a greater loss of functional capacity (power and MVC) in the trained state despite using the same relative intensity.

Acknowledgements

▼

Affiliations
[1] Studies, Research and Sport Medicine Center, Government of Navarre, Spain
[2] Department of Physical Education, University of Las Palmas of Gran Canaria, Spain
[3] Department of Electric and Electronic Engineering, Public University of Navarre, Spain
[4] Department of Radiology, Clinic of San Miguel, Navarre, Spain
[5] Department of Physical Education and Computer Sciences, University Pablo of Olavide, Sevilla, Spain
[6] Department of Biology of Physical Activity, University of Jyväskylä, Finland
[7] Department of Kinesiology, Human Performance Laboratory, University of Connecticut, Storrs, CT, USA

References

1 *Aagaard P, Andersen JL, Dyhre-Poulsen P, Leffers AM, Wagner A, Magnusson SP, Halkjaer-Kristensen J, Simonsen EB.* A mechanism for increased contractile strength of human pennate muscle in response to strength training: changes in muscle architecture. J Physiol 2001; 534: 613–623

2 *Aagaard P, Simonsen EB, Andersen JL, Magnusson P, Dyhre-Poulsen P.* Increased rate of force development and neural drive of human skeletal muscle following resistance training. J Appl Physiol 2002; 93: 1318–1326

3 *Ahtiainen JP, Pakarinen A, Alen M, Kraemer WJ, Hakkinen K.* Muscle hypertrophy, hormonal adaptations and strength development during strength training in strength-trained and untrained men. Eur J Appl Physiol 2003; 89: 555–563

4 *Amann M, Proctor LT, Sebranek JJ, Eldridge MW, Pegelow DF, Dempsey JA.* Somatosensory feedback from the limbs exerts inhibitory influences on central neural drive during whole body endurance exercise. J Appl Physiol 2008; 105: 1714–1724

5 *Andersen JL, Aagaard P.* Myosin heavy chain IIX overshoot in human skeletal muscle. Muscle Nerve 2000; 23: 1095–1104

6 *Bigland-Ritchie B, Donovan EF, Roussos CS.* Conduction velocity and EMG power spectrum changes in fatigue of sustained maximal efforts. J Appl Physiol 1981; 51: 1300–1305

7 *Bonato P.* Recent advancements in the analysis of dynamic EMG data. IEEE Eng Med Biol Mag 2001; 20: 29–32

8 *Cheng AJ, Rice CL.* Fatigue and recovery of power and isometric torque following isotonic knee extensions. J Appl Physiol 2005; 99: 1446–1452

9 *Dimitrov GV, Arabadzhiev TI, Mileva KN, Bowtell JL, Crichton N, Dimitrova NA.* Muscle fatigue during dynamic contractions assessed by new spectral indices. Med Sci Sports Exerc 2006; 38: 1971–1979

10 *Duchateau J, Hainaut K.* Isometric or dynamic training: differential effects on mechanical properties of a human muscle. J Appl Physiol 1984; 56: 296–301

11 *Enoka RM, Duchateau J.* Muscle fatigue: what, why and how it influences muscle function. J Physiol 2008; 586: 11–23

12 *Farina D.* Interpretation of the surface electromyogram in dynamic contractions. Exerc Sport Sci Rev 2006; 34: 121–127

13 *Fitts RH.* Cellular mechanisms of muscle fatigue. Physiol Rev 1994; 74: 49–94

14 *Green HJ, Daub BD, Painter DC, Thomson JA.* Glycogen depletion patterns during ice hockey performance. Med Sci Sports 1978; 10: 289–293

15 *Hakkinen K, Alen M, Komi PV.* Changes in isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of human skeletal muscle during strength training and detraining. Acta Physiol Scand 1985; 125: 573–585

16 *Hakkinen K, Kallinen M, Izquierdo M, Jokelainen K, Lassila H, Malkia E, Kraemer WJ, Newton RU, Alen M.* Changes in agonist-antagonist EMG, muscle CSA, and force during strength training in middle-aged and older people. J Appl Physiol 1998; 84: 1341–1349

17 *Hellsten Y, Apple FS, Sjodin B.* Effect of sprint cycle training on activities of antioxidant enzymes in human skeletal muscle. J Appl Physiol 1996; 81: 1484–1487

18 *Hickson RC, Hidaka K, Foster C, Falduto MT, Chatterton RT Jr.* Successive time courses of strength development and steroid hormone responses to heavy-resistance training. J Appl Physiol 1994; 76: 663–670

19 *Izquierdo M, Ibanez J, Gonzalez-Badillo JJ, Hakkinen K, Ratamess NA, Kraemer WJ, French DN, Eslava J, Altadill A, Asiain X, Gorostiaga EM.* Differential effects of strength training leading to failure versus not to failure on hormonal responses, strength, and muscle power gains. J Appl Physiol 2006; 100: 1647–1656

20 *Jackson AS, Pollock ML.* Generalized equations for predicting body density of men. Br J Nutr 1978; 40: 497–504

21 *James C, Sacco P, Jones DA.* Loss of power during fatigue of human leg muscles. J Physiol 1995; 484 (Pt 1): 237–246

22 *Karlsson J, Saltin B.* Oxygen deficit and muscle metabolites in intermittent exercise. Acta Physiol Scand 1971; 82: 115–122

23 *Klass M, Guissard N, Duchateau J.* Limiting mechanisms of force production after repetitive dynamic contractions in human triceps surae. J Appl Physiol 2004; 96: 1516–1521

24 *Komi PV, Tesch P.* EMG frequency spectrum, muscle structure, and fatigue during dynamic contractions in man. Eur J Appl Physiol 1979; 42: 41–50

25 *Kraemer WJ, Staron RS, Hagerman FC, Hikida RS, Fry AC, Gordon SE, Nindl BC, Gothshalk LA, Volek JS, Marx JO, Newton RU, Hakkinen K.* The effects of short-term resistance training on endocrine function in men and women. Eur J Appl Physiol 1998; 78: 69–76

26 *Linnamo V, Hakkinen K, Komi PV.* Neuromuscular fatigue and recovery in maximal compared to explosive strength loading. Eur J Appl Physiol 1998; 77: 176–181

27 *MacDougall JD, Hicks AL, MacDonald JR, McKelvie RS, Green HJ, Smith KM.* Muscle performance and enzymatic adaptations to sprint interval training. J Appl Physiol 1998; 84: 2138–2142

28 *McCall GE, Byrnes WC, Fleck SJ, Dickinson A, Kraemer WJ.* Acute and chronic hormonal responses to resistance training designed to promote muscle hypertrophy. Can J Appl Physiol 1999; 24: 96–107

29 *Mitchell JH, Reeves DR Jr, Rogers HB, Secher NH.* Epidural anaesthesia and cardiovascular responses to static exercise in man. J Physiol 1989; 417: 13–24

30 *Mohr M, Krustrup P, Nielsen JJ, Nybo L, Rasmussen MK, Juel C, Bangsbo J.* Effect of two different intense training regimens on skeletal muscle ion transport proteins and fatigue development. Am J Physiol 2007; 292: R1594–R1602

31 *Perez-Gomez J, Olmedillas H, Delgado-Guerra S, Ara I, Vicente-Rodriguez G, Ortiz RA, Chavarren J, Calbet JA.* Effects of weight lifting training combined with plyometric exercises on physical fitness, body composition, and knee extension velocity during kicking in football. Appl Physiol Nutr Metab 2008; 33: 501–510

32 *Potvin JR.* Effects of muscle kinematics on surface EMG amplitude and frequency during fatiguing dynamic contractions. J Appl Physiol 1997; 82: 144–151

33 *Sahlin K, Henriksson J.* Buffer capacity and lactate accumulation in skeletal muscle of trained and untrained men. Acta Physiol Scand 1984; 122: 331–339

34 *Staron RS, Karapondo DL, Kraemer WJ, Fry AC, Gordon SE, Falkel JE, Hagerman FC, Hikida RS.* Skeletal muscle adaptations during early phase of heavy-resistance training in men and women. J Appl Physiol 1994; 76: 1247–1255

35 *Stauber WT, Barill ER, Stauber RE, Miller GR.* Isotonic dynamometry for the assessment of power and fatigue in the knee extensor muscles of females. Clin Physiol 2000; 20: 225–233

36 *Thorstensson A, Sjodin B, Karlsson J.* Enzyme activities and muscle strength after "sprint training" in man. Acta Physiol Scand 1975; 94: 313–318