

# Effects of Strength Training on Muscle Fatigue Mapping from Surface EMG and Blood Metabolites

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## ABSTRACT

IZQUIERDO, M., M. GONZÁLES-IZAL, I. NAVARRO-AMEZQUETA, J. A. L. CALBET, J. IBAÑEZ, A. MALANDA, F. MALLOR, K. HÄKKINEN, W. J. KRAEMER, and E. M. GOROSTIAGA. Effects of Strength Training on Muscle Fatigue Mapping from Surface EMG and Blood Metabolites. *Med. Sci. Sports Exerc.*, Vol. 43, No. 2, pp. 303–311, 2011. **Purpose:** This study examined the effects of heavy resistance training on the relationships between power loss and surface EMG (sEMG) indices and blood metabolite concentrations on dynamic exercise-induced fatigue with the same relative load as in pretraining. **Methods:** Twelve trained subjects performed five sets consisting of 10 repetitions in the leg press, with 2 min of rest between sets before and after a strength training period. sEMG variables (the mean average voltage, the median spectral frequency, and the Dimitrov spectral index of muscle fatigue) from vastus medialis and lateralis muscles and metabolic responses (i.e., blood lactate, uric acid, and ammonia concentrations) were measured. **Results:** The peak power loss after the posttraining protocol was greater (61%) than the decline observed in the pretraining protocol (46%). Similar sEMG changes were found for both protocols, whereas higher metabolic demand was observed during the posttraining exercise. The linear models on the basis of the relations found between power loss and changes in sEMG variables were significantly different between pretraining and posttraining, whereas the linear models on the basis of the relations between power loss and changes in blood metabolite concentrations were similar. **Conclusions:** Linear models that use blood metabolites to map acute exercise-induced peak power changes were more accurate in detecting these changes before and after a short-term training period, whereas an attempt to track peak power loss using sEMG variables may fail after a strength training period. **Key Words:** SURFACE ELECTROMYOGRAPHY, MEAN AVERAGE VOLTAGE, POWER OUTPUT, NEUROMUSCULAR ADAPTATIONS, DYNAMIC CONTRACTION

Associations between EMG variables and manifestations of muscle fatigue during exercises at maximal level of voluntary contraction have been extensively related with increases in surface EMG (sEMG) amplitude (30) or shifts in sEMG power spectrum to lower frequencies (5,10). Moreover, failure to maintain a given level of peak force and muscle power has been related to intramuscular bioenergetic changes (20,26). Therefore, rela-

tionships between decline in muscle tension and changes in muscle phosphocreatine stores and a concomitant cytoplasmic inorganic phosphate accumulation (9,19), changes in muscle lactate or pH (24), and adenylate depletion and subsequent increase in the concentration of muscle IMP (27) and blood ammonia (16) have been observed in many studies. Indeed, electrophysiological and biochemical changes associated with manifestations of muscle fatigue have been used to indirectly estimate the functional state of the muscle contractile machinery and thus the power output ability of the contracting skeletal muscles. Only a limited number of studies, however, have examined the effects of strength training on the relationship between EMG variables, blood metabolite concentrations, and dynamic exercise-induced fatigue of the leg extensor muscles.

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 before and immediately after isometric fatiguing

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exercise (12,28). 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 wk) (3). In previous studies (21,22), we found 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 because 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. Therefore, because of these metabolic and neuromuscular adaptations caused by resistance training, it is not clear whether the relationships between mechanical manifestation of muscle fatigue, EMG variables, and blood metabolite concentrations were similar before and after the training sessions. Several attempts have been made to develop models that associate changes in sEMG and metabolic variables to power/torque loss (as a direct measurement of muscle fatigue) during maximal dynamic contractions (13–15). We hypothesized that because of neuromuscular adaptations after resistance training, the linear models using sEMG and metabolic variables to map changes in force/power loss would differ from that reported after 7 wk of resistance training. Thus, the aim of this study was to compare the accuracy to track acute exercise-induced changes in muscle power output using sEMG variables and blood metabolite concentrations before and after 7 wk of resistance training.

## MATERIALS AND METHODS

**Subjects.** Twelve physically active men (mean  $\pm$  SD: age = 33.0  $\pm$  4.4 yr, height = 1.77  $\pm$  0.06 m, body mass = 72.4  $\pm$  6.9 kg) volunteered to participate in the study. After being informed about the experimental procedure and the risks of the investigation, the subjects gave their written informed consent to participate. The experimental procedures were approved by the institutional review committee of the Instituto Navarro del Deporte 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 medical problems that would contraindicate their participation or influence the results of the study. None of them was taking drugs, medications, or supplements with potential effects on physical performance.

**Experimental design.** A longitudinal research design was used to compare the relationships between changes in neuromuscular responses and changes in muscle power output elicited by two acute heavy resistance loading protocols (AHREP) with the same relative load (%) before and after short-term heavy resistance training (7 wk). The volunteers were familiarized with the loading exercise procedures about 3 wk before the AHREP session. One week before the AHREP session, the subjects participated in a control

testing day to determine one-repetition maximum (1RM) and to estimate the maximum load to perform a 10RM in the leg press.

The experimental design comprised two AHREP (one before and another after the short-term heavy resistance training). Before training, the AHREP consisted of five sets, with the load corresponding to 10RM in leg press with 120 s of rest between the sets. After training, each subject performed an AHREP with the same relative load (10RM) as in the pretraining testing protocols. The subjects seated on a modified bilateral leg press exercise machine (Technogym, Gambettola, Italy) were asked to perform each leg press maximally and as rapidly as possible. The exercise machine recorded the applied force (N), with an accuracy of 1 N at 1 kHz, from several force transducers placed on a foot platform below the subject's feet. An optical encoder was used to record the position and the direction of the displacement of the weight using these measurements. Customized software was used to calculate the range of motion and the peak power output during the concentric phase of leg press action. The muscle peak power output of the leg extensor muscles (calculated as the maximum of the product between the exerted force and the velocity of movement) was measured during the concentric phase of leg press. The test-retest intraclass correlation coefficients for all power variables were greater than 0.96, and the coefficients of variation ranged from 0.9% to 1.1%. This investigation was part of a large project concerning the examination of power output, neural activation, and muscle and blood metabolites during leg press exercise and recovery and after a high-intensity resistance training period.

**Resistance training program.** Subjects were asked to train two times per week for 7 wk, with 45–60 min per session. A minimum of 2 d elapsed between two consecutive training sessions. Each training session consisted of several dynamic resistance exercises, such as parallel squat and bench press, and supplementary strengthening exercises for selected muscle groups (leg press, leg extension, shoulder press, lateral pulldown, abdominal crunch, trunk extension, and standing leg curl). The daily workouts were alternated by varying the intensity and the volume (sets  $\times$  repetitions  $\times$  load) over the week. In one of the sessions, the sets were performed at 12–15RM with 2 min of rest between sets. During the other session, the sets were performed at the 10RM intensity. The training intensities were gradually increased during the 7-wk training period using a repetition maximum approach.

To ensure that the exercise prescriptions (number of repetitions, time of rest, and velocity of execution) were properly administered, each training session was supervised by a trained researcher.

**Muscle cross-sectional area and anthropometry.** The muscle cross-sectional area (CSA) of the left quadriceps femoris was assessed before and after the 7-wk resistance training period using magnetic resonance imaging (MRI; 1 T; Siemens Magnetom Impact Expert). 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 intercondylar 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 an ink tattoo) of the muscle biopsy of the vastus lateralis (VL) muscle. For each axial scan, CSA computation was carried out on the quadriceps femoris as a whole and individually on the VL, vastus medialis (VM), vastus intermedius, and rectus femoris. For the final calculation of the CSA, slices 5/15 to 12/15 were used for all muscles examined except for the rectus femoris, which was analyzed only for slices 5/15 to 12/15. CSA (cm<sup>2</sup>) was determined by hand tracing of the border of each muscle. Body mass and percent body fat (estimated from the thickness of seven skinfold sites) were measured before and after each training period (23).

**Muscle biopsies.** Needle muscle biopsies were obtained from the middle section of the VL muscle under local anesthesia without suction, but with mild pressure on the lateral aspect of the thigh. Biopsies before and after the 7-wk 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  $-80^{\circ}\text{C}$ . MHC analyses were performed on the muscle biopsies using sodium dodecyl sulfate–polyacrylamide gel electrophoresis (SDS–PAGE). From each biopsy, 20–40 serial cross-sections (10  $\mu\text{m}$ ) were cut and placed in 200–500  $\mu\text{L}$  of lysing buffer and heated for 3 min at  $90^{\circ}\text{C}$ . Between 2 and 12  $\mu\text{L}$  of the myosin-containing samples were loaded on an SDS–PAGE. Gels were run at 70 V for 43 h at  $4^{\circ}\text{C}$ . Subsequently, the gels were Coomassie stained, and MHC isoform bands (I, IIa, and IIx) were determined on the basis of known migration patterns and quantified with the Un-scan-it gel software (Orem, UT).

**EMG.** Surface EMG (sEMG) of the VM and VL muscles was measured during the extension of the leg muscles on a modified bilateral leg press exercise machine (Technogym). The sEMG signals were recorded from the right leg using pairs of bipolar surface electrodes (Blue Sensor N-00-S, Medicotest, Ambu A/S, Ballerup, Denmark), with an inter-electrode distance of 20 mm between them. After careful preparation of the skin (shaving, abrasion, and cleaning with alcohol), the electrode pairs were placed longitudinally on the medial portion of the muscles, following the recommendations of the Surface Electromyography for the Noninvasive Assessment of Muscles Project. The positions of the electrodes were marked on the skin by small ink tattoos (17) to ensure the same electrode positions in both tests. sEMG were recorded at a sampling rate of 1 kHz, using a Muscle Tester ME3000 (Mega Electronics Ltd.,

Kuopio, Finland) (bandwidth of 8–500 Hz/3 dB and a common mode rejection ratio  $> 100$  dB).

The movement was divided into four intervals of  $22.5^{\circ}$  of the knee displacement. The following parameters were identified in the first interval of the movement (from  $90^{\circ}$  to  $112.5^{\circ}$  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: the mean average voltage (MAV), calculated as the integrated EMG/integration time (2); the median frequency ( $F_{\text{med}}$ ), and the Dimitrov spectral index of muscle fatigue (FInsm5) (10).

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

#### **Blood lactate, ammonia, and uric acid analysis.**

Capillary blood samples for the determination of lactate and ammonia concentrations were obtained from a hyperemized earlobe at the beginning of the exercise (preexercise), after the first set, after the third set (midexercise), and after the exercise (peak value postexercise). Samples for whole blood lactate determination (100  $\mu\text{L}$ ) were deproteinized, placed in a preservative tube (YSI 2315 Blood Lactate Preservative Kit, Yellow Springs, OH), stored at  $4^{\circ}\text{C}$ , and analyzed (YSI 1500) within 5 d 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 d with stable blood lactate concentration values (pooled estimate of  $\text{SD} = 0.15$   $\text{mmol}\cdot\text{L}^{-1}$  for a concentration range of 0–10  $\text{mmol}\cdot\text{L}^{-1}$ ). The blood lactate analyzer was calibrated after every fifth blood sample dosage with three known controls (5, 15, and 30  $\text{mmol}\cdot\text{L}^{-1}$ ). In terms of reliability, the manufacturers report a coefficient of variation of 3.2% and 2.6% with lactate standards of 2 and 11  $\text{mmol}\cdot\text{L}^{-1}$ , respectively.

After cleaning and puncturing, a single 20  $\mu\text{mol}$  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  $\mu\text{L}$  of blood. This analyzer uses a reflectometer to optically measure the reflection intensity ( $45^{\circ}$ ) of reagent color reaction in biochromatic mode and was calibrated before and after every test with a known control (58.7  $\mu\text{mol}\cdot\text{L}^{-1}$ ).

Capillary blood samples for the determination of uric acid concentrations were obtained from a hyperemized earlobe at the beginning of the exercise (preexercise) and 16 and 45 min after the end of the exercise protocol. According to the manufacturer's instructions, after cleaning and puncturing, a single 28.5- to 31.5- $\mu\text{L}$  capillary sample was taken and placed over the strip (Reflotron uric acid) for an automatic reflectance photometry analysis (Reflotron; Boehringer Mannheim, Mannheim, Germany) within the first 2–3 min after obtaining the sample. The analyzer was calibrated

(Reflotron Check) before every subject's capillary samples analysis.

**Statistical analysis.** Results in the figures are given as mean and SE values. The changes in percentage between each sEMG-based parameter and muscle power output and the average of the values of the first two contractions were calculated. The changes in percentage between blood metabolite values and their preexercise values were calculated. The percentage changes that did not follow a normal distribution were log transformed (FInsm5 and ammonium concentrations).

Changes in muscle power output, blood metabolites, and sEMG-based parameters were compared in relative terms via a one-way ANOVA with repeated measures. When a significant  $F$  value was achieved, Sheffé *post hoc* procedures were performed to locate the pairwise differences between the means. For comparison purposes, power output and EMG changes of the last five repetitions were compared with the first five repetitions. Pearson product-movement correlation coefficients ( $r$ ) were used to determine the association between changes in sEMG-based parameters and blood metabolite concentrations and changes in muscle power output. Moreover, to quantify the accuracy of tracking changes in muscle power of the different variables (sEMG-based parameters and blood metabolites), a signal-to-noise ratio (SNR) was obtained:

$$\text{SNR} = \frac{R}{\sqrt{\frac{1}{N} \sum_{n=1}^N (I_n - \hat{I}_n)^2}} \quad [1]$$

where  $R$  represents the range of the desired outputs (calculated as the maximum minus the minimum of the percentage change in power output), and the denominator represents the root mean square error between the estimated ( $\hat{I}_n$ ) and the desired outputs ( $I_n$ ) across  $N$  contractions.

To test the similarity of slopes and intercepts of the relationships between the changes in sEMG-based parameters and blood metabolite concentrations and the changes in muscle power outputs obtained before and after the training period, the corresponding  $t$ -test was applied for the model:  $Y_{ij} = \alpha_i + \beta_i X_{ij} + \varepsilon_{ij}$  for  $i = 1, 2$  (1 = pretraining exercise; 2 = posttraining exercise) and  $j = 1, \dots, n_1$  being  $\varepsilon_{ij}$  independent and identically distributed random variables following a distribution  $N(0, \sigma_1)$ . In addition, a stepwise multiple linear regression analysis was used to relate power output changes with the set of all blood metabolites and sEMG-based parameter changes. The independent variables that correlated most significantly with peak power output changes (changes in log-FInsm5,  $F_{\text{med}}$ , MAV, lactate, log-ammonium, and uric) were entered into the stepwise procedure. The probability of  $F$  value was used as the criterion for variable entry to the model and variable removal from the model. Statistical power calculations for this study ranged from 0.76 to 0.82. Statistical significance was set at  $P < 0.05$ .

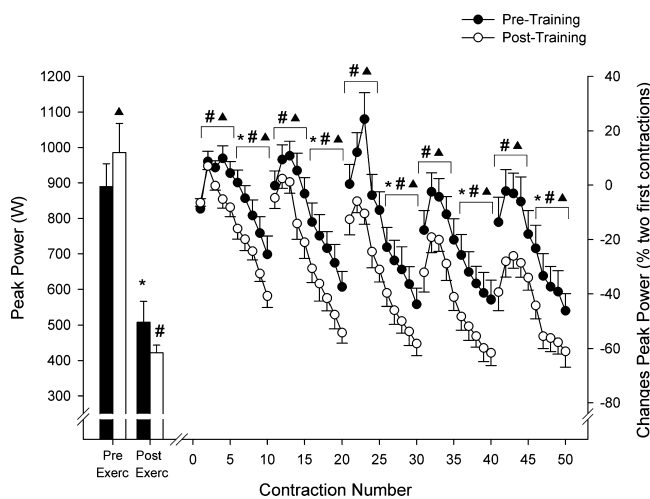
## RESULTS

**Anthropometry and muscle CSA.** After the training period, significant body mass and body fat percentage change significantly from  $72.4 \pm 6.9$  to  $73.4 \pm 6.2$  kg and from  $9.2\% \pm 2.5\%$  to  $9.7\% \pm 1.5\%$ , respectively. The mean increase of the quadriceps CSA from MRI scans was 4.4% (from  $130.1 \pm 10.6$  to  $135.7 \pm 12.2$  cm<sup>2</sup> after 7 wk of training).

**Myosin heavy chain isoform distribution.** Strength training resulted in an increased amount of MHC type IIa (from  $21.7\% \pm 0.7\%$  to  $38.6\% \pm 2.3\%$ ,  $P \leq 0.05$ ) and a reduction in the amount of MHC type I (from  $54.1\% \pm 1.8\%$  to  $49.5\% \pm 2.0\%$ ,  $P \leq 0.05$ ) and MHC type IIx (from  $18.2\% \pm 0.7\%$  to  $7.9\% \pm 0.6\%$ ,  $P \leq 0.05$ ).

**Peak power output.** Muscle power output remained unaltered during the 3-wk control period (from week -3 to week 0). After the 7-wk training period, the absolute value of muscle peak power output attained in the first five repetitions was significantly higher ( $P < 0.05$ ) ( $985.84 \pm 82.50$  W) than that attained during the pretraining loading exercise ( $889.53 \pm 64.57$  W).

At pretraining, the mean peak power output of the last five repetitions of each set significantly decreased compared with that attained during the corresponding first five repetitions of the exercise. However, after the 7-wk training period, the mean peak power output significantly decreased from the last five repetitions of the first set to the last repetitions of the last set. After the pretraining loading exercise, the peak muscle power output attained during the last repetition of the fifth set was 46.08% lower than that attained during the first five repetitions of the first set, whereas the corresponding peak power output value after the posttraining loading exercise was



**FIGURE 1**—Muscle peak power output (mean  $\pm$  SE) absolute values (i.e., average value of repetitions 1–5 and 45–50) and percentage changes with respect to the first two repetitions during the five sets of 10 repetitions of the pretraining and posttraining loading exercise. \*Significant differences ( $P < 0.05$ ) compared with the first five repetitions of the first set during the pretraining loading exercise. #Significant differences ( $P < 0.05$ ) compared with the first five repetitions of the first set during the posttraining loading exercise. ΔSignificant differences ( $P < 0.05$ ) between pretraining and posttraining loading exercises.



the first set. Moreover, no significant differences were found in percentage changes in MAV between the pretraining and the posttraining loading protocols (Fig. 2A).

The averaged median frequency (averaged for VM and VL) during both loading exercises significantly decreased during the last five repetitions of each set compared with the first repetitions of the first set. Moreover, no significant differences were found between loading exercises (Fig. 2B).

The averaged log-FInsm5 significantly increased during the last five repetitions of the first and the second set as well as the whole number of repetitions of third, fourth, and fifth sets in the pretraining and posttraining loading experiments. Moreover, as in the case of median frequency, no significant differences were found between loading experiments (Fig. 2C).

**Blood lactate, ammonia, and uric concentrations.**

After both preloading and postloading exercises, blood lactate concentrations significantly increased after the first bout, midexercise, and peak compared with the preexercise values. Moreover, the blood lactate concentrations were significantly higher during the posttraining exercise compared with the pretraining values (Fig. 3A).

The ammonia concentrations at midexercises and peak values during both preloading and postloading exercises significantly increased compared with the preexercise values. Moreover, the ammonia peak value was significantly higher during the posttraining loading exercise (Fig. 3B).

Peak uric acid concentrations significantly increased after both pretraining and posttraining loading protocols.

**Relationships between average sEMG variables, blood metabolites, and muscle peak power output changes.** Pearson correlation analysis revealed that the log-FInsm5 was the sEMG parameter that most correlated with mechanical peak power output changes for pretraining ( $r = -0.62$ , SNR = 6.47,  $P < 0.01$ ) and posttraining ( $r = -0.67$ , SNR = 5.81,  $P < 0.01$ ) loading protocols. Moreover, a significant relationship ( $r = -0.32$ , SNR = 5.33,  $P < 0.01$ ) was found between MAV and peak power output during pretraining loading exercise, whereas no significant relationships were found at posttraining.

In addition, one of the changes in uric acid concentration was the blood metabolite, which showed the highest correlation with changes in power output during pretraining ( $r = -0.89$ , SNR = 6.47,  $P < 0.01$ ) and posttraining ( $r = -0.70$ , SNR = 5.50,  $P < 0.01$ ) loading exercise (Table 1).

TABLE 1. Correlation coefficients between changes (%) in the average of sEMG variables (averaged VM and VL muscles), lactate, ammonium, and uric, and the corresponding changes (%) in muscle peak power output for each loading exercise (pretraining and posttraining loading exercises).

	Pretraining Power	Posttraining Power
MAV	-0.32 (5.33)**	-0.02 (4.33)
$F_{med}$	0.52 (5.92)**	0.42 (4.79)**
Log-FInsm5	-0.62 (6.47)**	-0.67 (5.81)**
Lactate	-0.35 (4.24)*	-0.44 (5.16)**
Log-ammonium	-0.66 (5.19)**	-0.67 (6.25)**
Uric acid	-0.89 (6.47)**	-0.70 (5.50)*

\*\*  $P < 0.01$ ; \*  $P < 0.05$ .

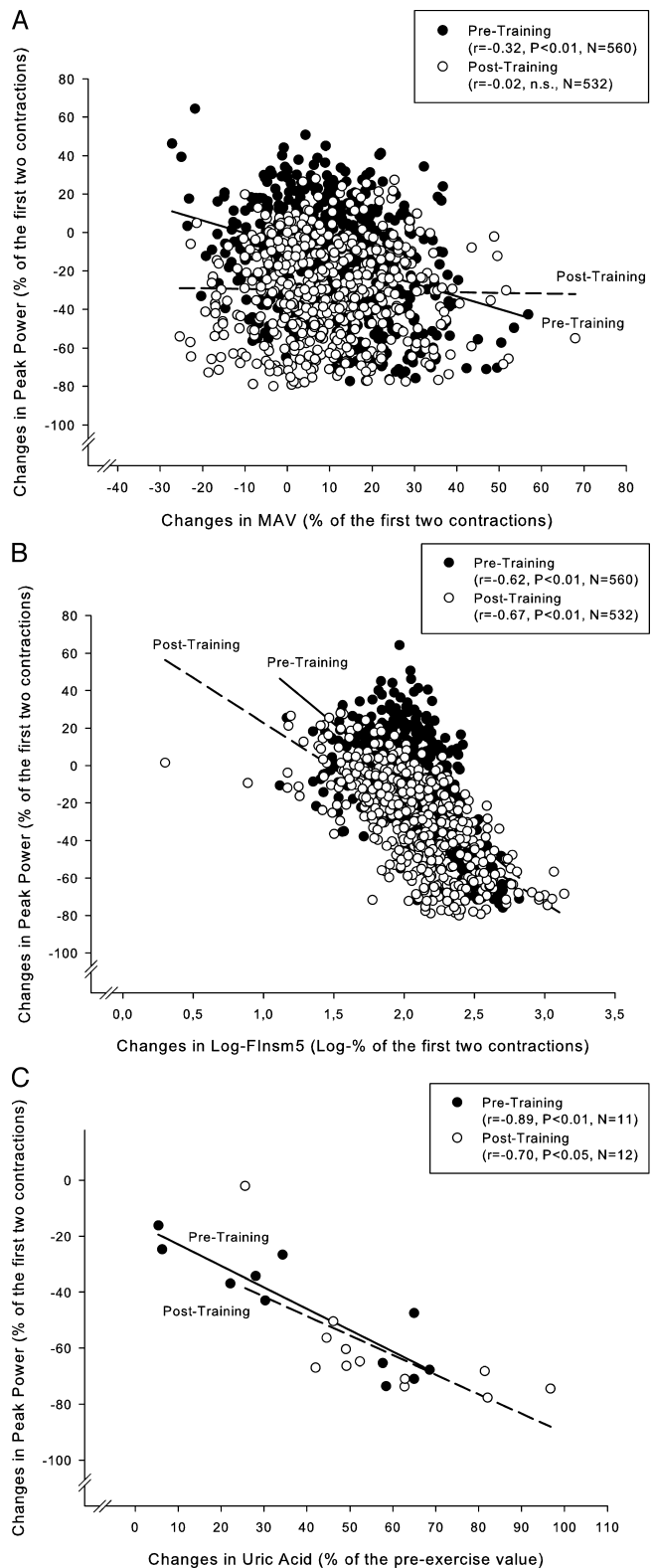


FIGURE 4—Linear representations of changes in peak power output (percentage of the first two repetitions) versus changes in sEMG variables (averaged for VM and VL muscles) (percentage of the first two repetitions) and blood metabolites (percentage of the preexercise value) for all the subjects during pretraining and posttraining loading exercises. A. Changes in power output (%) versus changes in MAV (%). B. Changes in power output (%) versus changes in log-FInsm5 (%). C. Changes in power output (%) versus changes in uric acid (%).

The slope and the intercepts comparison analysis revealed that the linear regressions to estimate muscle peak power output using sEMG parameter obtained during the pretraining loading exercise were significantly different from those obtained during the posttraining loading exercise. Moreover, including the CSA as a covariate in the model to control the possible effects of this variable in the values of sEMG variables and therefore in their relationship with muscle power output changes, the linear regression to estimate muscle peak power output using sEMG parameter obtained during the pretraining loading exercise was also significantly different from that obtained during the posttraining loading exercise. However, the same analysis revealed that the linear regressions to estimate muscle peak power output using blood metabolites were similar during pretraining and posttraining loading exercises (Fig. 4).

Uric acid and log-FInsm5 as a two-factor combination predictor accounted for 83.6% (SNR = 11.61) of the performance variance of changes in power output during the pretraining protocol, whereas only 51.7% (SNR = 5.95) was reached during the posttraining exercise. Moreover, the slope and intercepts comparison analysis revealed that the linear regressions to estimate muscle peak power output using this two-factor combination were not significantly different during pretraining and posttraining loading exercises.

## DISCUSSION

After a short-term strength training period, despite the significant training-induced increases in muscle mass and absolute muscle power output, acute exercise-induced loss in peak muscle power led to a greater reduction in muscle peak power output, similar magnitude of the exercise-induced neuromuscular changes (i.e., MAV,  $F_{med}$ , and log-FInsm5), and higher accumulation of blood lactate and ammonia concentration, when the exercise was performed at the same relative intensity pretraining and posttraining. The log-FInsm5 and the changes in uric acid concentration were the sEMG and blood metabolite variables accounting for 83% and 51% of the performance variance of changes in peak muscle power output before and after short-term heavy resistance training, respectively. The correlation coefficients obtained to estimate changes in power output from sEMG features during repetitive dynamic leg extensions during the posttraining loading exercise were significantly lower from that obtained during the pretraining loading exercise. However, the same analysis revealed that the linear models to track changes in power loss using blood metabolites proved useful in tracking dynamic acute exercise-induced power loss before and after the 7 wk of resistance training. These results may indicate that linear models that use blood metabolites to map acute exercise-induced peak power changes have higher predictive value than changes in sEMG variables, especially after a strength training period. This could be partly related to training status-related differences between the association of exercise-induced muscle power output decrease, sEMG,

and metabolic variables before and after a short-term training period.

As expected, the absolute power value significantly increased as a result of the 7 wk of resistance training (1,21). However, as previously reported, the subjects endured a higher power 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. Other studies (7,32,36) have demonstrated greater susceptibility to fatigue in subjects with higher initial peak power output than individuals with lower initial performance because of a higher proportion of fast twitch fibers (7,36) of the former. In the present study, the percentage of type II fibers increased around 15% after the training period. It is well known that glycolytic activity, power output capacity, and fatigability are more pronounced in type II fibers compared with type I fibers (8,36). Taking these data together, it is therefore reasonable to suggest that a higher percentage of type II fibers of the subjects after training period may contribute to the higher initial power output, the achieved greater fatigue degree at the end of the exercise protocol, and the higher increase in blood markers of metabolic stress observed during the exercise.

The higher accumulation of blood markers of metabolic stress (i.e., lactate and ammonia) observed in posttraining exercise protocol suggests that the energy state of the muscle tissue is lower during exercise than that in pretraining exercise protocol despite of the same relative training load. Moreover, as mentioned above, the subjects endured a higher rate of fatigue development after the training period. This higher fatigue level achieved after the training program could be the result of reductions or changes in the sensory feedback from the fatigue muscles after training (4) and/or the higher activity of AMP deaminase enzyme, which limits the increase in muscular free ADP concentration and thereby maintains the contraction capacity of the muscle (25), which pushed the muscle to higher degree of fatigue and therefore higher energetic disturbance.

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 sEMG amplitude, with a shift of the EMG power spectrum toward lower frequencies and a sevenfold increase in the magnitude of the spectral fatigue index analyzed. However, as previously reported (21), 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 pretraining and posttraining. This also indicates that the neural mechanisms eliciting fatigue were also similar pretraining and posttraining when exercising with the same relative load, suggesting that an increased capacity to perform more exercise and to endure a greater

loss of strength during the acute exercise protocols was mainly due to peripheral adaptations (21). Nevertheless, although there were no changes in the amount of neural drive before and after the resistance training at the beginning of the loading protocol (31,35), the behavior of the sEMG amplitude during the protocol was different before and after the 7 wk of training sessions. Before training, the amplitude increased as the fatigue progressed through several mechanisms, for example, increasing motor unit recruitment (30), firing rate (1,6), and possible greater synchronization of training muscles (i.e., timing and pattern of discharge). However, after the resistance training, there were no significant increases in the percentage changes in sEMG amplitude during the loading protocol as fatigue progressed. Moreover, there was no association between changes in muscle power output and changes in sEMG amplitude after resistance training, whereas before training, a significantly negative association was found ( $r = -0.32$ ,  $P < 0.01$ ). These results could be partly explained by the fact that strength training leads to hypertrophy, which implies that the muscle fibers are capable of providing more tension. Therefore, using the same loading intensity after training, this is related to declines in sEMG amplitude (29) and less amount of activated muscle tissue (33) and consequently a probable similar neural drive when the same relative loading intensity was used. The reasons of the nonexistence of an association, present before training, between changes in muscle power output and changes in sEMG amplitude after the 7 wk of resistance training are not clear. They could be related to motor unit recruitment strategies learned during the training or changes in muscle architecture. It has been found that the sEMG amplitude is strongly affected by the position of the electrodes regarding the active fibers. Dimitrova and Dimitrov (11) suggested that the amplitude of motor unit action potentials may decrease when recorded close to the active fibers, may be unchanged at intermediate distances, and may increase when the electrodes are far from the active fibers, although the amplitude of the intracellular action potential decreases. Although the ink tattoos assured the same position of the electrodes regarding to the subject muscle, changes in the muscle architecture could modify the relative position of the electrodes to the active fibers and therefore the sEMG amplitude behavior as fatigue progresses.

Moreover, greater accuracy in tracking changes in power loss was reported between changes in blood metabolites and changes in muscle power output compared with the association observed with sEMG variables output before and after the training period. Indeed, the linear models that use blood metabolites to track power loss before training were valid

for tracking these changes after 7 wk of resistance training because these relations did not change. However, the linear models that relate changes in sEMG variables to muscle power loss were not valid after a short-term training period because the muscle power loss was greater after the short strength training period than before, and the acute neuromuscular fatigue produced by the loading was similar before and after the training period.

The association between the changes in blood metabolites and the changes in muscle power output paralleled before and after the training period. Therefore, it could be speculated that the training status appeared to have no effect on the association between blood markers of metabolic stress and muscle power output decrease, that is, fatigue. On the other hand, the associations between sEMG variables and fatigue degree were modified after the training period. The strong association observed, before and after the training period, between blood uric acid and changes in power output indicates that those subjects with higher increases in blood uric acid during the exercise presented greater fatigue index than those with lower increases in blood uric acid did, independently of the training status. If we assume that increases in blood uric acid reflects events occurring locally within the muscle (i.e., loss of muscular ATP store) (18,34), it can be concluded that changes in capacity of the leg extensor muscles to generate mechanical power reflect metabolic changes occurring within contractile machinery, independently of training status.

In summary, although the linear models that relate changes in sEMG variables and power loss provide useful information to track muscle fatigue in situations where the power/force is not possible to measure, their use may be limited. Linear models that use blood metabolites to map acute exercise-induced peak power changes were more valid in detecting these changes before and after a short-term training period, whereas attempts to track peak power loss using sEMG variables may fail after a strength training period. This could partly be related to training status-related differences between the association of exercise-induced muscle power output decrease, sEMG, and metabolic variables before and after a short-term training period.

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## REFERENCES

1. Aagaard P. Training-induced changes in neural function. *Exerc Sport Sci Rev.* 2003;31(2):61–7.
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(4):1318–26.
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(6):555–63.
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(6):1714–24.
  5. 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(5):1300–5.
  6. Bigland-Ritchie B, Johansson R, Lippold OC, Smith S, Woods JJ. Changes in motoneuron firing rates during sustained maximal voluntary contractions. *J Physiol*. 1983;340:335–46.
  7. Bosco C, Komi PV, Tihanyi J, Fekete G, Apor P. Mechanical power test and fiber composition of human leg extensor muscles. *Eur J Appl Physiol Occup Physiol*. 1983;51(1):129–35.
  8. Colliander EB, Dudley GA, Tesch PA. Skeletal muscle fiber type composition and performance during repeated bouts of maximal, concentric contractions. *Eur J Appl Physiol Occup Physiol*. 1988; 58(1–2):81–6.
  9. Dawson MJ, Gadian DG, Wilkie DR. Muscular fatigue investigated by phosphorus nuclear magnetic resonance. *Nature*. 1978; 274(5674):861–6.
  10. Dimitrov GV, Arabadzhev 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(11):1971–9.
  11. Dimitrova NA, Dimitrov GV. Amplitude-related characteristics of motor unit and M-wave potentials during fatigue: a simulation study using literature data on intracellular potential changes found in vitro. *J Electromyogr Kinesiol*. 2002;12(5):339–49.
  12. Duchateau J, Hainaut K. Isometric or dynamic training: differential effects on mechanical properties of a human muscle. *J Appl Physiol*. 1984;56(2):296–301.
  13. Gonzalez-Izal M, Falla D, Izquierdo M, Farina D. Predicting force loss during dynamic fatiguing exercises from non-linear mapping of features of the surface electromyogram. *J Neurosci Methods*. 2010;190(2):271–8.
  14. Gonzalez-Izal M, Malanda A, Navarro-Amezqueta I, et al. EMG spectral indices and muscle power fatigue during dynamic contractions. *J Electromyogr Kinesiol*. 2010;20(2):233–40.
  15. Gonzalez-Izal M, Malanda A, Rodriguez-Carreño I, et al. Linear vs non-linear mapping of peak power using surface EMG features during dynamic fatiguing contractions. *J Biomech*. 2010;12(1): 1100–200.
  16. Gorostiaga EM, Asiain X, Izquierdo M, et al. Vertical jump performance and blood ammonia and lactate levels during typical training sessions in elite 400-m runners. *J Strength Cond Res*. 2010; 24(4):1138–49.
  17. Hakkinen K, Kallinen M, Izquierdo M, et al. Changes in agonist-antagonist EMG, muscle CSA, and force during strength training in middle-aged and older people. *J Appl Physiol*. 1998;84(4): 1341–9.
  18. Hellsten-Westing Y, Norman B, Balsom PD, Sjodin B. Decreased resting levels of adenine nucleotides in human skeletal muscle after high-intensity training. *J Appl Physiol*. 1993;74(5):2523–8.
  19. Hogan MC, Richardson RS, Haseler LJ. Human muscle performance and PCr hydrolysis with varied inspired oxygen fractions: a <sup>31</sup>P-MRS study. *J Appl Physiol*. 1999;86(4):1367–73.
  20. Hultman E, Greenhaff PL. Skeletal muscle energy metabolism and fatigue during intense exercise in man. *Sci Prog*. 1991;75 (298 Pt 3–4):361–70.
  21. Izquierdo M, Ibanez J, Calbet JA, et al. Neuromuscular fatigue after resistance training. *Int J Sports Med*. 2009;30(8):614–23.
  22. Izquierdo M, Ibanez J, Calbet JA, et al. Cytokine and hormone responses to resistance training. *Eur J Appl Physiol*. 2009;107(4): 397–409.
  23. Jackson AS, Pollock ML. Generalized equations for predicting body density of men. *Br J Nutr*. 1978;40(3):497–504.
  24. Karlsson J, Saltin B. Oxygen deficit and muscle metabolites in intermittent exercise. *Acta Physiol Scand*. 1971;82(1):115–22.
  25. Lowenstein JM. Ammonia production in muscle and other tissues: the purine nucleotide cycle. *Physiol Rev*. 1972;52(2):382–414.
  26. McKenna MJ, Heigenhauser GJ, McKelvie RS, MacDougall JD, Jones NL. Sprint training enhances ionic regulation during intense exercise in men. *J Physiol*. 1997;501( Pt 3):687–702.
  27. Meyer RA, Terjung RL. Differences in ammonia and adenylate metabolism in contracting fast and slow muscle. *Am J Physiol*. 1979;237(3):111–8.
  28. 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.
  29. Moritani T, deVries HA. Neural factors versus hypertrophy in the time course of muscle strength gain. *Am J Phys Med*. 1979;58(3): 115–30.
  30. Moritani T, Nagata A, Muro M. Electromyographic manifestations of muscular fatigue. *Med Sci Sports Exerc*. 1982;14(3):198–202.
  31. Narici MV, Roi GS, Landoni L, Minetti AE, Cerretelli P. Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. *Eur J Appl Physiol Occup Physiol*. 1989;59(4):310–9.
  32. Paavolainen L, Hakkinen K, Nummela A, Rusko H. Neuromuscular characteristics and fatigue in endurance and sprint athletes during a new anaerobic power test. *Eur J Appl Physiol Occup Physiol*. 1994;69(2):119–26.
  33. Ploutz LL, Tesch PA, Biro RL, Dudley GA. Effect of resistance training on muscle use during exercise. *J Appl Physiol*. 1994;76(4): 1675–81.
  34. Sahlin K, Ekberg K, Cizinsky S. Changes in plasma hypoxanthine and free radical markers during exercise in man. *Acta Physiol Scand*. 1991;142(2):275–81.
  35. Sale DG. Neural adaptations to strength training. In: *Strength and Power in Sport*. Malden (MA): Blackwell Science; 2003. p. 281–314.
  36. Thorstensson A, Karlsson J. Fatiguability and fibre composition of human skeletal muscle. *Acta Physiol Scand*. 1976;98(3):318–22.