Eur J Appl Physiol (2000) 82: 206–214

ORIGINAL ARTICLE

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Recovery of skeletal muscle contractility after high- and moderate-intensity strength exercise

Accepted: 14 February 2000

Abstract To examine neuromuscular fatigue and recovery, ten male strength athletes [mean (SE) 27.5 (1.4) years] performed a moderate- and a high-intensity strength exercise protocol. In the high-intensity protocol, the load was 100% of the subject's three-repetition maximum (3-RM) for squats and front squats, and 100% of the subject's 6-RM for knee extensions. In the moderate-intensity protocol, the load was 70% of the high-intensity protocol, and both protocols lasted 90 min. The contractile properties of the leg extensor muscles were tested using isokinetic knee extensions, electrical stimulation, and squat jumps. Tests were done before exercise, 5–20 min after exercise, and frequently for 33 h after exercise. The decrements in knee extension performance were greater after the high-intensity protocol (12–14%), as compared to the moderate-intensity protocol (6–7%, $P < 0.01$). Similar decrements were seen in squat-jumping performance after the high-intensity protocol. Decrements in electrically evoked force were also greatest after the high-intensity protocol ($P < 0.05$), and were more pronounced at 20 Hz stimulation than at 50 Hz stimulation ($P < 0.05$). The recovery of performance showed a biphasic pattern, with a rapid recovery within the first 11 h after exercise, followed by a leveling off or a second drop in performance 11–22 h after exercise. All variables were back to baseline by 3 h after the moderate-intensity protocol, while all variables were back to baseline by 33 h after the 100% protocol. The role of structural changes (excitation-contraction coupling and contractile proteins) in the long-lasting performance decrements seen after the high-intensity protocol is discussed.

Key words Strength exercise · Fatigue · Recovery · Urea · Creatine kinase

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Introduction

A bout of heavy resistance exercise results in acute neuromuscular fatigue. This fatigue is caused by a decrease in the force-production capacity of muscle fibers and/or decreased neural activation of the exercised muscle (Newham et al. 1991; Häkkinen 1993). The most well-documented changes in contractile properties following resistance exercise seem to be reduced maximal voluntary force production (Bigland-Ritchie et al. 1983; Vøllestad et al. 1988; Kroon and Naeije 1991; Newham et al. 1991; Häkkinen 1993), reduced electrically evoked force production (Bigland-Ritchie et al. 1983; Vøllestad et al. 1988; Newham et al. 1991), reduced neural drive (Bigland-Ritchie et al. 1983, 1986; Häkkinen 1993), slowing of the rate of force development (Bigland-Ritchie et al. 1983; Häkkinen et al. 1988; Häkkinen 1993), and slowing of the rate of relaxation (Bigland-Ritchie et al. 1983; Gollnick et al. 1991; Häkkinen 1993, 1994).

Most studies in which neuromuscular fatigue has been investigated have used repeated isometric contractions (e.g., Bigland-Ritchie et al. 1983, 1986; Vøllestad et al. 1988; Newham et al. 1991) or high-force eccentric contractions (e.g., Newham et al. 1987; Tiidus and Shoemacher 1995; Brown et al. 1997). The metabolic changes and the mechanical stress conferred upon the contractile apparatus differ between these protocols; it is not clear if similar mechanisms are responsible for the neuromuscular fatigue seen after normal dynamic strength exercise in athletes. The different strains placed upon the neuromuscular system may be illustrated by studies showing that the recovery of maximal voluntary isometric contractions (MVC) may take less than 2 h after isometric contractions (Kroon and Naeije 1991), but may take more than 2 weeks after high-force eccentric contractions (Newham et al. 1987; Howell et al. 1993). The time of recovery seems to be dependent upon the degree of eccentric force production involved, the muscle groups involved, fiber-type distribution,

gender, and the training status of the subjects. Häkkinen (1993) found that recovery of MVC of the quadriceps after a bout of 20 maximal squats took approximately 2 days in well-trained male and female athletes. However, tests were separated by 22–24 h, and the authors gave no information about changes that may have occurred between these tests.

Creatine kinase (CK) has been used as a marker of skeletal muscle damage after eccentric exercise (Clarkson et al. 1992; Nosaka and Clarkson 1995). However, CK elevations in serum do not seem to reflect changes in contractile function or muscle soreness when trained athletes are compared with untrained subjects after strength exercise (Vincent and Vincent 1997). It is not clear if this relationship is better within a group of well-trained athletes. Morning serum urea concentration $[urea]_{pl}$ has been suggested to be a marker of the previous day's training stress in endurance athletes (Bacharach et al. 1996). If this is true for strength training, $[urea]_{pl}$ should be different the day after a heavy and a light workout for strength athletes.

Studies describing recovery from neuromuscular fatigue have either focused on the 1st h after exercise (Wiles and Edwards 1982; Gollnick et al. 1991; Vøllestad et al. 1997) or, if recovery is studied over several days, each test is usually separated by 20–24 h (Häkkinen 1994; Gibala et al. 1995; Vincent and Vincent 1997). We believe that further insight into the mechanisms involved in the recovery process could be gained by combining different tests of contractile properties repeated frequently after exercise. The purpose of this study was to investigate the recovery of contractile properties of the knee extensors after strength exercise of high- and moderate-intensity in well-trained athletes. A preliminary report of this work has been presented in abstract form (Raastad and Hallén 1998).

Methods

Subjects

Eight male power-lifters, one javelin-thrower and one speed-skater, mean (SE) age 27.5 (1.4) years, were recruited to participate in this study. The exercise protocols in the present study are typical for power-lifters, and both the javelin-thrower and the speed-skater were performing similar training in the months before the study. Their body mass ranged from 67 to 110 kg [84.5 (4.2) kg]. The study was approved by the Regional Ethics Committee of the Norwegian Research Council for Science and the Humanities.

Experimental design

The subjects participated in two strength exercise trials of different intensities. In addition, six of the subjects participated in a rest-control trial. At each trial, the first test of contractile properties was performed at 0800 hours. The strength exercises lasted from 0830 hours to 1000 hours. The second test started approximately 5 min after the end of the resistance exercises. Tests were also performed 3, 7, 11, 22, 26, 30, and 33 h after exercise. Blood samples were collected just before the tests of contractile properties, except for the tests performed 26 and 30 h after exercise. Blood samples were also collected 30 min into the strength exercises, and every 15 min during the 1st hour after exercise. The rest-control trial was similar to day 1 between 0800 hours and 1700 hours, except that the subjects rested between 0830 hours and 1000 hours. Meals were served at the same time of the day during all trials. The subjects ate the same sort and the same amount of food at each trial. The order of the trials was counterbalanced between the subjects. The time between each trial was 1–2 weeks, except for two subjects who performed the rest-control trial 8 weeks after the last exercise trial.

Exercise protocols

The strength exercise protocols consisted of squats, front squats, and bilateral knee extensions. The subjects did exactly the same number of repetitions at both trials, but the intensity was different. During warm-up, the subjects increased their loads gradually during five sets of squats, four sets of front squats, and one set of knee extensions. After the warm-up, the subjects did three sets of three repetitions with a load that could be lifted for three repetitions maximum (100% of 3-RM) in squats and front squats. The subjects continued with three series of six repetitions at a load of 100% of 6-RM for the knee extensions in the high-intensity strength exercise (hereafter called the 100% protocol). The reason for choosing a different number of repetitions and load for the knee extensions was that for these athletes the 6-RM load is normal in the knee extension exercise. In the moderate-intensity protocol, the subjects did the same number of repetitions and sets, but the load was reduced to 70% during the squats and front squats, and 76% during the knee extensions (hereafter called the 70% protocol). The contractions were performed slowly and in a well-controlled manner. Both training sessions lasted 90 min, and the time between sets was 6 min during squats and front squats, and 4 min during warm-up and knee extensions (Table 1).

Measurements

Contractile properties were tested in three different ways: (1) voluntary isokinetic knee extension at 60 and $240° \cdot s^{-1}$, (2) electrically evoked isometric contractions of the vastus medialis, and (3) squat jumps on a forceplate. The tests were always performed in this order and took 15 min per subject (5 min per test). These measurements were selected because they cause little fatigue, together they represent the general contractile properties of the muscles exercised, and they have a high test-retest reproducibility. All subjects went through at least four familiarization tests before they were qualified for the study.

The isokinetic knee extensions were performed in a Cybex 6000 dynamometer (Lumex, Ronkonkoma, N.Y., USA). The range of motion was set from a knee angle of 90–20° from full extension. The subjects performed four warm-up contractions followed by three maximal contractions at each speed. The test at $60° \cdot s^{-1}$ was performed before the test at $240° \cdot s^{-1}$, with 30 s of rest in between. Peak torque and total work were calculated for data analysis (coefficient of variation, CV < 5%). Only the right leg was tested.

After the isokinetic knee extension, the subjects remained seated in the Cybex 6000 and were prepared for the electrical stimulation. The stimulator was a S11 Stimulator (Grass Instruments, Mass.,

Table 1 Training load for the 100% and 70% protocols. Data are given as the mean (SE). (*RM* Repetition maximum)

Exercise	100%	70%
Squat, 3-RM (kg)	169 (7)	118 (5)
Front squat, 3-RM (kg)	121 (5)	87 (4)
Knee extension, 6-RM (kg)	68 (4)	52 (2)

USA). The leg was fixed at a knee angle of 90°, and the belly of vastus medialis was washed with isopropanol. Electrodes (5 × 10 cm, Polartrode, Medi-Stim, Oslo, Norway) were placed longitudinally on the vastus medialis. The positions of the electrodes were marked on a transparency, together with any birthmarks, to ensure the same positioning in each trial. During each trial electrode positioning was standardized by marking the skin with indelible ink. The stimulation protocol comprised two trains of stimuli at 300 ms duration and 20 Hz frequency, and two trains of stimuli at 300 ms duration and 50 Hz frequency. Each square-wave pulse lasted 0.5 ms, and the voltage was fixed at 120 V. Force was measured by a strain gauge (HBM U2AC2, Darmstadt, Germany) connected to the Cybex 6000 level arm, which was attached to the subject's right wrist. The data sampling frequency was 1 kHz, and the force/time curves were smoothed by taking the average of 20 samples for each time point. The mean values from the two trains at each frequency of maximum force generated (CV < 10%), the 20:50 Hz force ratio (CV < 5%), the maximum rate of force generation (RFG_{max}, CV < 10%) and the maximum rate of relaxation (RR_{max}, CV < 5%) were included in the data analysis. RFG_{max} was calculated as the peak value of dF/dt divided by peak force, and RR_{max} was calculated as the nadir value of dF/dt divided by peak force.

Squat jumps were performed on a forceplate (SG-9, Advanced Mechanical Technologies, Newton, Mass., USA), and signals were filtered through a 1050-Hz low-pass filter. Jumps were performed with no counter-movement, from a knee angle of 90° with hands fixed to the hip. Jump height was calculated from the impulse during take-off position. At each test, the subjects did at least five jumps, and the mean of the three best jumps was included in the data analysis to minimize the influence of unstable jumping techniques (CV < 5%).

Blood samples

Blood was drawn from an antecubital vein into 5-ml heparin Vacutainer tubes on ice and, within 30 min, were centrifuged at 3000 rpm (5000 g) for 10 min at 4 °C. Plasma was stored at −20 °C until analyzed. Lactate was analyzed enzymatically using a YSI 1500 Sport (YSI, Yellow Springs, USA), CV < 5%. CK was analyzed using a Reflotron system (Boheringer Mannheim, Mannheim, Germany), CV < 5%. $[Urea]_{pl}$ was measured using a Kodak Ektachem DT60 analyzer (Eastman Kodak, New York, USA), CV < 5%. All samples from each subject were analyzed on the same day.

Statistical analysis

The area under the curves (AUCs) from tests 0–11 h and from tests 22–33 h after exercise, were calculated and used to identify any differences in performance between the 70% and 100% trials. AUCs were compared using paired *t*-tests. In addition, selected time points within each trial were compared with respective baseline values to describe the time course of the recovery. This was done using paired *t*-tests, and the selected time points were immediately post-exercise, and 3, 11, 22 and 33 h after exercise. Selected bivariate relationships were examined using a Pearson Product moment correlation coefficient. A one-way analysis of variance (1 × 5) was used to identify indications of diurnal variation for tests performed on the rest-control trial. The data are presented as the mean (SE).

Results

There were no significant differences between the baseline values of contractile tests between the 100% and the 70% trials (Table 2).

Table 2 Baseline values in the contractile tests performed during the 100% and 70% protocols. Data are given as the mean (SE). (RFG_{max} Maximum rate of force generation, RR_{max} maximum rate of force relaxation)

Test	70%	100%
Peak torque $60° \cdot s^{-1}$ (Nm)	304 (16)	296 (14)
Peak torque $240° \cdot s^{-1}$ (Nm)	185 (8)	189 (7)
Jump height (cm)	36.8 (2.6)	37.6 (2.7)
Force at 20 Hz (N)	84 (12)	76 (11)
Force at 50 Hz (N)	118 (17)	111 (18)
RFG_{max} ($\%_{peak}$/10 ms)	11.4 (0.3)	10.7 (0.3)
RR_{max} ($\%_{peak}$/10 ms)	10.8 (0.3)	11.0 (0.3)

Isokinetic knee extension

Peak torque and total work at $60° \cdot s^{-1}$ (PT_{60} and TW_{60}, respectively) were significantly lower 0–11 h after the 100% as compared to the 70% protocol ($P < 0.05$), but not the day after exercise. PT_{60} and TW_{60} were reduced by 12–14% and 6–7% ($P < 0.01$) 5 min after the 100% and 70% protocols, respectively. PT_{60} and TW_{60} were not significantly different from baseline 3 h after the 70% protocol, while both were still significantly reduced 11 and 22 h after the 100% protocol. Peak torque and total work at $240° \cdot s^{-1}$ (PT_{240} and TW_{240}, respectively) tended to be lower 0–11 h after the 100% as compared to the 70% protocol ($P < 0.10$), but not the day after the exercise. The decreases in PT_{240} and TW_{240} 5 min after exercise were similar to those at $60° \cdot s^{-1}$ after both protocols (Fig. 1). PT_{240} and TW_{240} were not significantly different from baseline 3 h after either exercise protocol. However, PT_{240} was significantly lower than baseline 22 h after the 100% protocol ($P < 0.05$). PT and TW at both speeds showed biphasic recovery courses with a rapid component within the first 3–7 h, followed by a leveling off or a second drop in performance until the next morning. A slow recovery followed the 2nd day for all of the isokinetic variables. Neither peak torque nor total work, at any speed, were different from baseline 33 h after exercise.

Squat jumps

The reductions in jump height were significantly greater the same day, and the day after the 100% compared to the 70% protocol ($P = 0.01$). The jump height was reduced by 12 (1)% ($P < 0.01$) 15 min after the 100% protocol, while it was not significantly changed after the 70% protocol. Jump height did not reach baseline values during the 1st day, and was still significantly reduced 22 h after the 100% protocol ($P < 0.01$). Jump height was not significantly different from baseline 33 h after the 100% protocol, but it was 5 (1)% higher than baseline 33 h after the 70% protocol ($P < 0.0l$). The recovery of jump height after the 100% protocol followed the same biphasic course as the isokinetic peak torque and total work (Fig. 2).

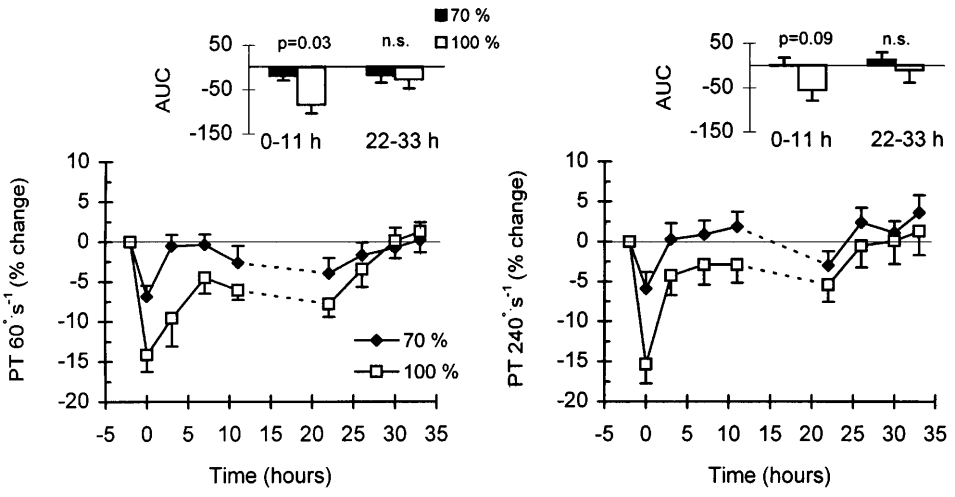

Fig. 1 Changes in knee extension peak torque (*PT*) at $60° \cdot s^{-1}$ (*bottom left panel*) and $240° \cdot s^{-1}$ (*bottom right panel*) after strength exercise at a high intensity (the 100% protocol, *open squares*) and at a moderate intensity (the 70% protocol, *closed diamonds*). The *upper panels* show the areas under the curves (AUC) between 0 and 11 h after exercise, and 22–33 h after exercise, of the graphs shown immediately below them (*closed squares* 70% protocol, *open squares* 100% protocol)

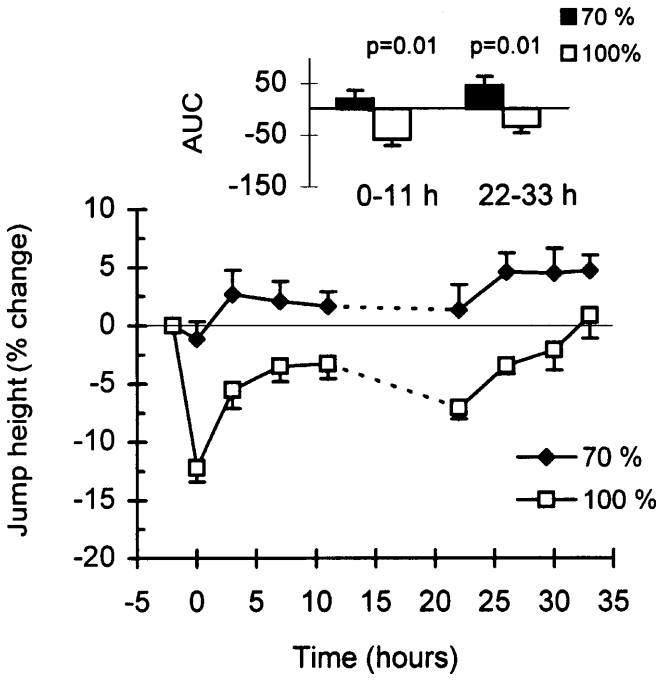

Fig. 2 The *lower graph* shows changes in jump height after strength exercise at high intensity (100% protocol, *open squares*) and moderate intensity (70% protocol, *closed diamonds*). The *upper panel* shows the AUC between 0 and 11 h after exercise, and 22–33 h after exercise, of the lower graph (*closed squares* 70% protocol, *open squares* 100% protocol)

Electrical stimulation

Force

There were no significant differences between changes in electrical-stimulation-induced force after the 70% and 100% protocols at either 20- or 50-Hz stimulation, 0–11 h and 22–33 h after exercise (Fig. 3). However, the 20:50 Hz force ratio was significantly lower 0–11 h after the 100% protocol, and tended to be lower the day after as compared to the 70% protocol (Fig. 4). The force at 20 and 50 Hz stimulation was significantly lower than the baseline values 10 min after both protocols ($P < 0.05$). Both the 20- and 50-Hz evoked force recovered to baseline values during the 1st day, but the force was significantly reduced 22 h after the 100% protocol [21 (5)% at 20 Hz and 14 (3)% at 50 Hz, $P < 0.01$]. Like the isokinetic knee-extension variables, the recovery of electrically evoked force followed a biphasic course. The 20:50 Hz force ratio was reduced 10 min after both the 70% [11 (2)%, $P < 0.01$] and the 100% protocols [21 (5)%, $P < 0.01$]. This ratio was not different from baseline 3 h after the 70% protocol. The tendency of the 20:50 Hz force ratio to be lower than baseline 33 h after exercise, and to be lower the day after the 100% protocol as compared to the 70% protocol ($P < 0.10$), indicates a slow recovery of this ratio.

RFG_{max} and RR_{max}

RFG_{max} was decreased 10 min after the 70% protocol [8 (3)%, $P < 0.05$], but was unchanged after the 100% protocol. RR_{max} increased 10 min after the 100% protocol [6 (1)%, $P < 0.01$], but was unchanged after the 70% protocol. There were no significant differences for either RFG_{max} or RR_{max} at any other time point.

Rest-control trial

The results from the six subjects performing a rest-control trial showed no indications of circadian variation in any of the voluntary variables (Fig. 5). Mean force evoked at 20 and 50 Hz increased 9–11% from the 0800 hours until 1700 hours, but the increases were not

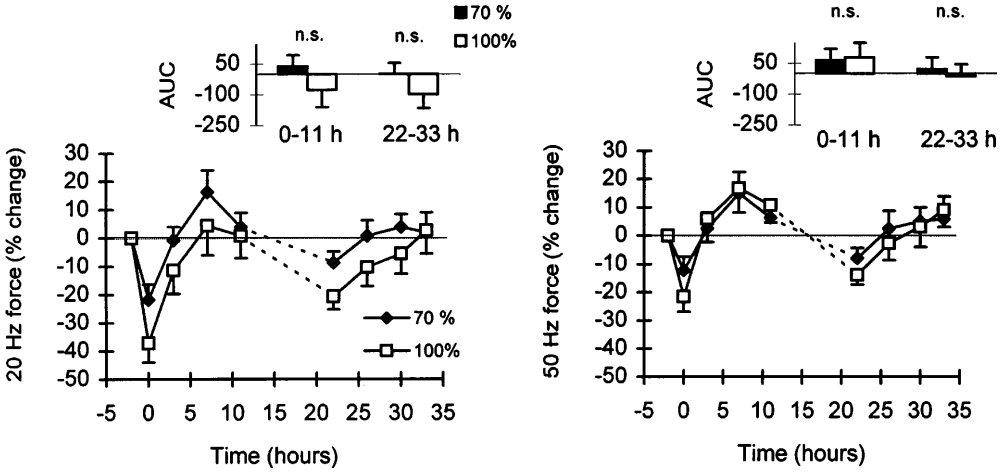

Fig. 3 Change in isometric knee extension force evoked by 20 (*lower left panel*) and 50 Hz (*lower right panel*) electrical stimulation protocols after strength exercise at high intensity (100% protocol, *open squares*) and moderate intensity (70% protocol, *closed diamonds*). The *upper panels* show the AUC between 0 and 11 h after exercise, and 22–33 h after exercise, of the graphs shown immediately below them (*closed squares* 70% protocol, *open squares* 100% protocol)

significant due to individual variation. The 20:50 Hz force ratio was stable throughout the 9 h monitored. RFG_{max} showed indications of circadian variation, with values obtained at 1000 hours being less than those obtained at 0800 hours. RR_{max} was stable throughout the rest-control trial.

Lactate, CK and urea

The 100% protocol elevated the plasma lactate concentration ($[La^-]_{pl}$) to 8.5 (0.9) mmol · l^{-1} just after the squat exercise. The $[La^-]_{pl}$ peaked at the end of the exercise at the 70% trial [2.6 (0.3) mmol · l^{-1}; Fig. 6].

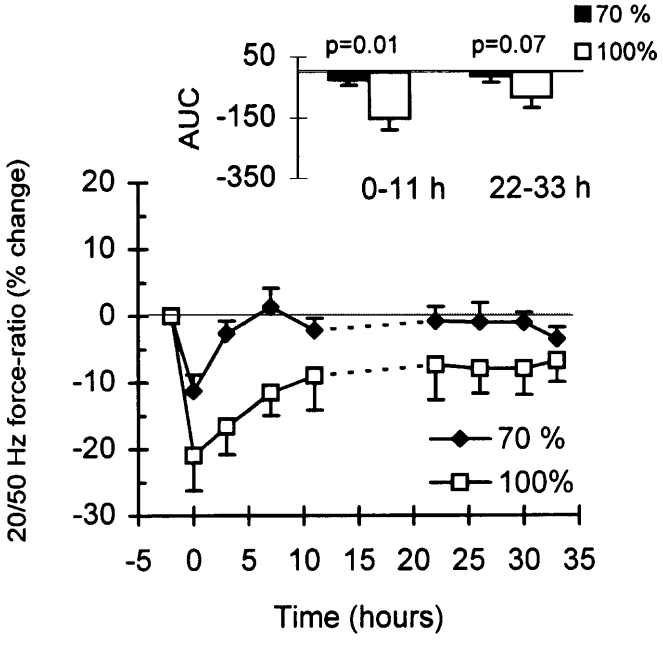

Fig. 4 Changes in 20:50 Hz force ratio after strength exercise at high intensity (100% protocol, *open squares*) and moderate intensity (70% protocol, *closed diamonds*). The *upper panel* shows the AUC between 0 and 11 h after exercise, and 22–33 h after exercise, of the lower graph (*closed squares* 70% protocol, *open squares* 100% protocol)

The plasma CK concentration ($[CK]_{pl}$) peaked 11 h after both the 70% and 100% protocols. The $[CK]_{pl}$ level after the 100% protocol was not back to baseline by the end of the study (33 h after exercise). There were no significant correlations between individual changes in $[CK]_{pl}$ and individual changes in contractile function for either protocol. $[Urea]_{pl}$ was reduced during and for 1 h after both protocols. $[Urea]_{pl}$ tended to increase in the afternoon of both days, but was only significantly higher than baseline 11 h after the 70% protocol.

Discussion

In the present study, strength exercise of moderate or high intensity resulted in skeletal muscle fatigue in well-trained athletes. The reductions in performance were greater and the recovery of performance was slower after the heavy 100% protocol as compared to the 70% protocol. All performance measures showed the same pattern of recovery after the 100% protocol. There was a drop in performance of 12–22% post-exercise. Recovery was biphasic, with rapid recovery occurring during the first 11 h, followed by a leveling off or a second drop in performance until the next morning, 22 h after exercise. All variables returned to baseline levels 33 h after exercise.

Diurnal variation

The results from the six subjects on a rest-control day showed no signs of diurnal variation in the voluntary tests. It should be kept in mind, however, that the rest-control trial only lasted for 9 h (0800–1700 hours). Reilly and Down (1992) have shown a circadian rhythm in jumping and sprinting performance, with an increase of 2–4% from morning until afternoon. In accordance with our results, however, Cabri et al. (1988) did not find any diurnal variation in isokinetic knee-extension performance. We can not rule out diurnal variation in electrically evoked force, because

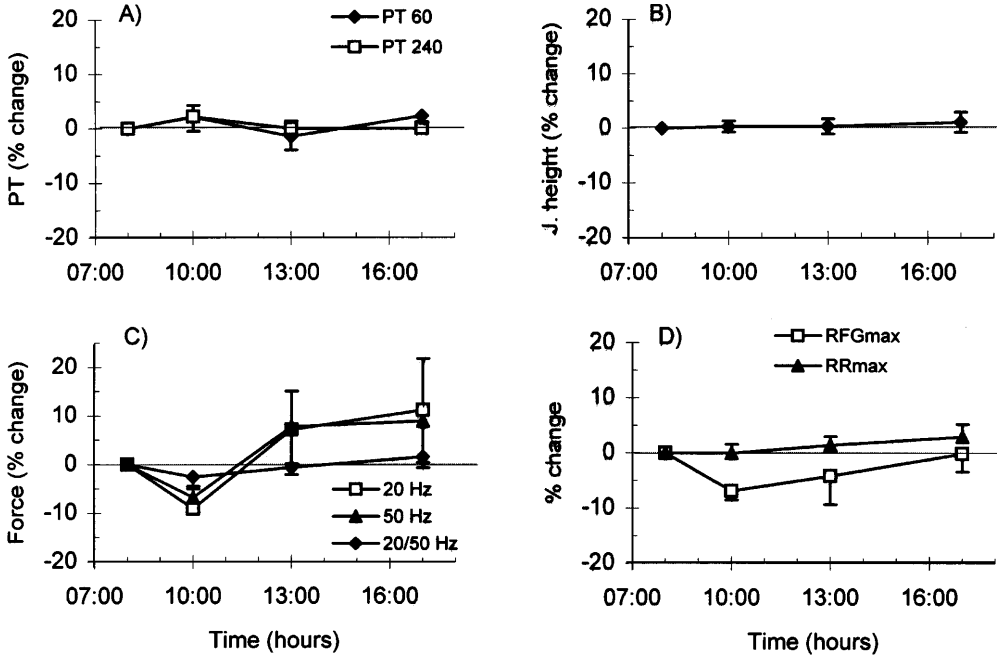

Fig. 5A–D Variation in performance from 0800–1700 hours in the rest-control trial for $n = 6$ subjects. **A** PT at the speed of $60° \cdot s^{-1}$ and $240° \cdot s^{-1}$. **B** Jump height (*J. height*). **C** Electrically evoked force at 20 Hz (*open squares*) and 50 Hz (*closed triangles*) stimulation, and the 20: 50 Hz force ratio (*closed diamonds*). **D** Maximum rate of force generation (RFG_{max}, *open squares*) and maximum rate of relaxation (RR_{max}, *closed triangles*) at 50 Hz stimulation

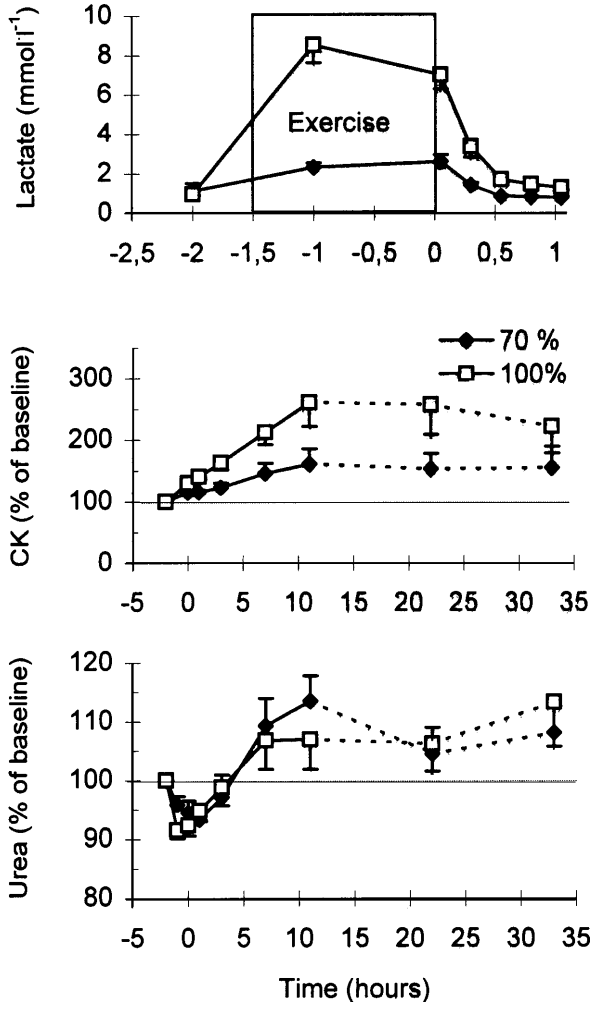

Fig. 6 The lactate response (*upper panel*), creatine kinase (*CK*) response (*middle panel*), and the urea response (*lower panel*) in plasma during the 70% (*closed diamonds*) and 100% (*open squares*) trial

greater inter-individual differences may have masked any significant variation. The force evoked by 20- and 50-Hz stimulation seems to co-variate so that the 20:50 Hz force ratio was stable through the rest-control trial. The RR_{max} was also stable throughout the rest control trial, but RFG_{max} showed some variation.

Fatigue

The 12–14% drop in maximal voluntary performance observed 5–20 min after the end of the 100% strength protocol, is difficult to compare with earlier observations on well-trained athletes. Elite weight-lifters exhibit a 7–8% drop in isometric knee extension force after exercises and intensities related to, but not directly comparable with, those used in the present study (Häkkinen et al. 1988). In a study where male and female strength athletes performed 20×1-RM squats with 3 min rest between sets, the 1-RM decreased by 10% and maximal isometric knee extension torque decreased by 22–24% after exercise (Häkkinen 1993). Squat exercise with high volume (10×10 repetitions) and moderate intensity (70% of 1-RM) reduced maximal isometric force by 47% in male, and by 29% in female strength athletes (Häkkinen 1994). In all three of these studies, contractile properties were measured immediately after exercise, while in the present study 5 min elapsed from the cessation of exercise to the first test.

The 100% protocol in the present study is a mixture of what is called "neural" loading (3-RM) and "hypertrophic" loading (6-RM). It is likely that these loadings separately and differently influence acute fatigue and recovery (Häkkinen 1993, 1994). However, in the present study the intention was to describe fatigue and recovery after a normal heavy (100% protocol) and light

(70% protocol) workout for these athletes. In the following discussion, the contribution from all three exercises will be considered as a whole, but it should be kept in mind that the mechanisms and magnitude of the acute fatigue might have been different if the tests had been performed right after the squat and front squat exercises.

The causes of fatigue after this kind of strength exercise may be both central (reduced motor drive) and peripheral (within the muscle) in origin. The 22% decrease in electrically evoked force following 50 Hz stimulation observed in the present study, indicates that the reduction in the voluntary performance is, at least partly, of peripheral origin. However, the stimulation was restricted to the vastus medialis muscle, and the force produced by 50-Hz stimulation was 5–10% of the maximal voluntary force production during an isometric knee extension. Therefore, these results can not exclude central fatigue. Superimposed brief electrical shocks during maximal contractions have indicated both reduced neural activation of quadriceps (Newham et al. 1991) and no change in neural activation (Bigland-Ritchie et al. 1986) immediately after different protocols of fatiguing resistance exercise. However, reduced neural activation seems to recover within 5 min after exercise (Behm 1995). Consequently, we conclude that some of the fatigue induced in this study 5 min after the cessation of exercise is located within the muscles.

The mechanisms underlying peripheral fatigue can be one of many, and both metabolic and structural changes have been suggested. Reduced creatine phosphate (CrP) concentration and increased inorganic phosphate and hydrogen ion concentrations ($[P_i]$ and pH, respectively) are possible metabolic causes of fatigue (Sahlin et al. 1998; Westerblad et al. 1998), and these changes have been observed in the quadriceps muscles after strength exercise (Tesch et al. 1986). However, [CrP], $[P_i]$ and pH seem to recover to near-control values within 5 min after repeated submaximal and maximal isometric contractions (Baker et al. 1993; Saugen et al. 1997). The fact that 5 min elapsed from the end of exercise until the first test in the present study, suggests that recovery of force due to metabolic changes was almost complete. Therefore, we suggest that metabolic changes not are the major causes of fatigue observed 5–20 min after exercise.

One mechanism that reduces force-generating capacity, as recently reviewed by Bruton et al. (1998), is a change in the excitation-contraction coupling depressing the release of Ca^{2+} from the sarcoplasmic reticulum (SR). However, in the studies reviewed, the force reduction was 60% during in vitro fatigue protocols. This raises the question of whether similar mechanisms are present in vivo when force is depressed by 10–20%. Even if it is unclear in this study, it could explain some of the fatigue seen at maximal voluntary and electrically induced contractions 5–20 min after exercise. Reduced Ca^{2+} released from the SR could also explain the depressed 20:50 Hz force ratio, due to the sigmoidal relationship between cytosolic Ca^{2+} concentration and force.

Gibala et al. (1995) observed disruptions in 33% of fibers from the biceps brachii immediately after pure concentric strength exercise, and in 82% of fibers after pure eccentric strength exercise in untrained men. Even though our subjects were well adapted to this kind of strength exercise, it seems reasonable to assume that the 100% strength protocol led to some mechanical disruption of the contractile apparatus because of the high-intensity loading.

Possible force-reducing metabolic changes seem to recover rapidly after exercise; consequently, we suggest that reduced Ca^{2+} release due to changes in the excitation-contraction coupling, and disruptions of contractile proteins, are the most likely explanations for the peripheral fatigue observed 5–20 min after exercise.

Contractile speed

Contradictory to our observations, reduced speed of relaxation has been observed after normal-strength exercise (Häkkinen 1993, 1994). The use of voluntary contractions, instead of electrically evoked contractions, makes it difficult to compare these results directly with ours. However, repeated isometric knee extensions at 60% of MVC has been observed to reduce the half-relaxation time ($RT_{0.5}$) 10 min after the cessation of exercise (Vøllestad et al. 1997). The time after exercise required for the determination of $RT_{0.5}$ and RR_{max} may explain some of the differences found after resistance exercise. This is supported by the fact that Vøllestad et al. (1997) observed a reduced speed of relaxation immediately after exercise, but an increased speed of relaxation 10 min after exercise. Differences in the rate of recovery of factors affecting speed of relaxation in different directions (e.g., temperature, pH and [CrP]) might explain such changes in the 1st min after exercise.

Recovery of force

After the 70% strength protocol, all tests showed that performance was back to baseline 3 h after exercise. After the 100% strength protocol, there was a recovery during the first 11 h, but only PT_{240} and electrically evoked force reached baseline values. Isokinetic knee extension torque at $60° \cdot s^{-1}$ and jump height did not return to baseline during the 1st day after the 100% strength protocol. This supports our suggestion that the fatigue is caused by mechanisms other than metabolic. Changes in the excitation-contraction coupling may take more than 10 h to recover after repeated isometric contractions in combination with mechanical strain in vitro (Bruton et al. 1995). This could also explain the long-lasting depression in the 20:50 Hz force ratio seen after the 100% protocol. This ratio has been shown to be depressed for several days after more severe eccentric exercise (Newham et al. 1987; Brown et al. 1997).

Damage of the contractile apparatus may also cause long-lasting reductions in maximal force-generating

capacity. Disrupted proteins in the sarcomeres must be replaced by new proteins. This is a slow process which, after more severe exercise-induced damage, may take more than 1 week (Lowe et al. 1995). Elevations in the rate of muscle protein synthesis after normal-strength exercise in humans can last for 2 days (MacDougall et al. 1995; Phillips et al. 1997). Replacement of damaged contractile proteins may partly explain the recovery of performance observed during the 1st and 2nd day.

The second drop in performance in the jump test, and electrical stimulation between 11 and 22 h after the 100% exercise, can be explained by normal diurnal variations in the force-generating capacity or by a delayed exercise-induced mechanism. The voluntary tests show no sign of diurnal variation, and the mean variation seen in electrically evoked force only explains 50% of the reduction seen between tests carried out 11 and 22 h after exercise. Therefore, we suggest that the second drop in performance is due mainly to a delayed exercise-induced mechanism.

Infiltration of macrophages and neutrophil granulocytes may be a delayed exercise-induced mechanism underlying the second drop in performance. After eccentric exercise of the quadriceps muscle, an infiltration of leukocytes has been shown to gradually increase and peak in concentration 20–25 h after exercise (MacIntyre et al. 1996). A second drop in eccentric torque from a test performed 4 h after exercise to the next tests performed 20 and 24 h after exercise, was also observed. It has been shown that phagocytic infiltration 24 h after exercise is associated with increased protein degradation in mice (Lowe et al. 1995). As far as we know, the present study is the first to show this biphasic recovery pattern of maximum force-generating capacity after normal, high-force, concentric-eccentric strength exercise in well-trained athletes. We postulate that the same inflammatory response is initiated after this kind of exercise as seen after eccentric exercise, but the magnitude of the response may differ. Earlier reports may have failed to show biphasic recovery courses after normal concentric-eccentric strength exercise because of less frequent testing during the first 24 h after exercise (e.g., Häkkinen 1993, 1994).

We found reductions in $[urea]_{pl}$ during, and for 1 h after both resistance exercise protocols. The reductions in $[urea]_{pl}$ observed during the 100% strength protocol were significant, despite a 6% reduction in plasma volume, and might be due to reduced hepatic blood flow during exercise. Kraemer et al. (1993) found no change in $[urea]_{pl}$ in women during and after resistance exercise of different intensities. Our subjects were exercising in a fasted condition and were not allowed to eat until 1.5 h after exercise. Oral intake of protein might explain the tendency to increased $[urea]_{pl}$ throughout the rest of the day (Young et al. 1998). Another explanation would be the delayed exercised-induced skeletal muscle protein degradation discussed earlier. If this was the case, the $[urea]_{pl}$ increases observed after the 100% strength protocol would be expected to be greater than after the 70% strength protocol; however, this was not the case.

The accumulation of CK in plasma was significantly greater after the 100% protocol than after the 70% protocol, but even the 100% protocol raised $[CK]_{pl}$ only moderately. This was expected for well-trained athletes (Vincent and Vincent 1997). Despite the relatively homogenous group of subjects, there was a great intra-individual difference in the CK response at both trials. There were no correlations between accumulation of CK in plasma and decreases in performance at any time. Vincent and Vincent (1997) observed that even though experienced weight-lifters accumulated less CK in their plasma than untrained subjects after the same relative strength exercise, the reduction in maximal force and the sensation of soreness was not different between the two groups. This might indicate that the release of CK from exercised muscles after strength exercise is related to changes in permeability for CK through the plasma membrane, which does not seem to reflect damage of the contractile apparatus when different individuals are compared.

Conclusion

Our study has demonstrated similar courses of recovery for force-generating capacity, determined by different tests. Recovery of force-generating capacity followed a biphasic pattern that was characterized by a rapid recovery within the first 3–7 h after exercise, a leveling off or a further decline between 11 and 22 h after exercise, followed by the second phase of recovery for the next 11 h. We suggest that changes in the excitation-contraction coupling and disruptions in contractile proteins are responsible for the long-lasting depression in the force-generating capacity, and that phagocytic activity might be involved in in the performance decrements observed 11–22 h after exercise.

Acknowledgements Financial support for this study was obtained from Olympiatoppen. The authors would like to acknowledge the support of Lise Roald, who performed the analyses of blood variables, Sigbjørn Inselset for technical assistance, and Ingar Holme for statistical advice. The experiments comply with the current national laws.

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