Fatigue after submaximal intensive stretch-shortening cycle exercise

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ABSTRACT

STROJNIK, V., and P. V. KOMI. Fatigue after submaximal intensive stretch-shortening cycle exercise. *Med. Sci. Sports Exerc.*, Vol. 32, No. 7, pp. 1314–1319, 2000. **Objective:** The aim of the present study was to examine some sites of neuromuscular fatigue after submaximal intensity stretch-shortening cycle exercise. **Methods:** Twelve male subjects performed consecutive sledge jumps at 60% of maximal height until exhaustion (mean duration 443.7 s ± 304.9 s, mean ± SD). **Results:** During the exercise, the blood lactate increased from 1.8 ± 0.6 mmol·L^{-1} (before exercise) to 6.1 ± 1.7 mmol·L^{-1} ($P < 0.001$) and serum creatin-kinase from 248 ± 142 IU·L^{-1} to 584 ± 344 IU·L^{-1} ($P < 0.001$). Electrical stimulation of the vastus lateralis and quadriceps femoris muscles to induce isometric knee extension resulted in decreased peak torque during single and double twitch after workout (from 22.1 ± 6.3 Nm to 17.3 ± 8.0 Nm, $P < 0.05$, and from 96.6 ± 15.4 Nm to 76.2 · 19.8 Nm, $P < 0.001$, respectively), whereas there were no significant changes in contraction and relaxation times. Torque during 20-Hz stimulation decreased significantly (from 23.7 ± 9.2 to 16.1 ± 7.8 Nm, $P < 0.01$) but not at 100-Hz stimulation. During maximal voluntary isometric knee extensions, the rate of torque development was significantly ($P < 0.01$) more impaired than maximal torque (from 1619 ± 390 Nm·s^{-1} to 1004 ± 360 Nm·s^{-1} and from 185 ± 30.7 Nm to 151 ± 32.3 Nm, respectively, both $P < 0.001$). At the same time, the muscle activation level increased by 15.8 ± 24.1% ($P < 0.05$). The mean EMG amplitude of vastus lateralis during MVC increased by 34.9 ± 39.2% ($P > 0.05$). **Conclusion:** It was concluded that after submaximal stretch-shortening exercise, the low-frequency fatigue occurred, very likely caused by lower Ca^{2+} release per single action potential. **Key Words:** ELECTRICAL STIMULATION, FATIGUE SITES, MUSCLE DAMAGE, SLEDGE JUMPS

Muscle fatigue involves multiple factors, which may be acting at numerous sites in a neuromuscular system (3). The actual fatigue mechanisms depend on the exercise conditions as well on the subject's level of physical fitness. During maximal intensive short-lasting exercises, fatigue may originate from ionic shifts, e.g., high extracellular K^+, acting as a conduction block for an action potential propagation (28,30). At sufficient workloads, the duration of exercise may be limited by increased muscle acidosis, which inhibits the myosin ATPase activity and thus decreases the muscle force production capability. With the method of single supramaximal electrical shock, it has been demonstrated that the muscle's contractile characteristics show a clear fatigue induced force decline and prolongation of the relaxation and contraction times (2). Muscle damage may also cause a reduction in the maximal muscle force and peak power similar to the effects of fatigue. In particular, repeated eccentric contractions may cause muscle damage (1).

Stretch-shortening cycle (SSC) is a natural form of muscle action, which utilizes active prestretch (eccentric action) followed immediately by active shortening (concentric action) (17). Gollhofer et al. (13) found a reduced EMG activation during the eccentric phase of SSC exercise fatigue. This was later confirmed by Nicol et al. (25) showing a smaller monosynaptic EMG response of calf muscles to passive stretch after submaximal SSC exercise. Interaction between joint stiffness, reflex responsiveness, and muscular mechanical performance in drop jumps has been established after exhaustive SSC exercise performed at 70% of maximal jumping height (14). High blood lactate and creatin-kinase values, the latter considered as an indirect indicator of muscle cell membrane disruption (26), have been associated with exhaustive exercise (14,25).

It was expected that numerous mechanisms may contribute to fatigue after SSC exercise at submaximal intensity as stated above. Because not much is known about changes in muscle contractile characteristics, high- and low-frequency fatigue, and maximal voluntary activation during such exercises, the aim of the present study was to examine these mechanisms after submaximal SSC exercise.

MATERIALS AND METHODS

Subjects. Twelve healthy male subjects volunteered for this study (age: 28.1 ± 5.8 yr, height: 179.8 ± 5.3 cm, body mass: 78.9 ± 12.3 kg). They were not involved in any severe training but were regularly physically active (sport

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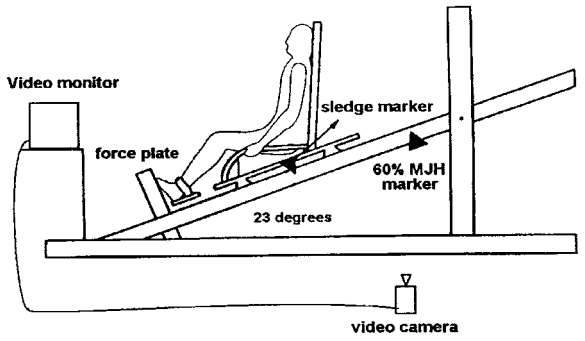

Figure 1—Fatigue workout setup. Subjects performed jumps on a sledge apparatus with a build-in force plate (17). On-line visual feedback was provided via a video monitor positioned in front of the subject. A video camera filmed the movement of the sledge according to the marker for a 60% of the maximal jumping height (MJH), enabling subjects to control the intensity of jumping by aligning both markers.

recreation). The subjects were informed about possible risks associated with the experiment and gave their informed consent before participating in the experiment. The study was approved by the university ethical committee.

Experimental design. After the warm-up, which consisted of 6 min of stepping on a 20-cm bench with a frequency of 0.5 Hz with a leg exchange each minute, the tests for assessment of initial status were performed in the following sequence: blood sample, response of the relaxed vastus lateralis muscle to single electrical impulse (twitch), response of the relaxed quadriceps femoris muscle (QF) to double electrical impulse (double twitch), response of the vastus lateralis muscle to electrical stimulation (ES) with 20 Hz and 100 Hz, explosive maximum voluntary knee extension, and steady maximum voluntary extension with superimposed double electrical impulse (for a muscle activation level assessment). Thereafter, the maximum jumping height was determined on the sledge ergometer, which was used for the fatigue exercise. The same test sequence and timing was used after the fatigue; the last test ended 4 min after the workout. Additionally, blood was collected 5 min and 2 d (after the same warm-up) after the fatigue test was completed. Surface EMG activity from the vastus lateralis (VL) muscle was recorded by bipolar silver chloride miniature skin electrodes (Beckman, Schiller Park, IL) with an interelectrode distance of 20 mm and transmitted telemetrically (Glonner Biomes 2000, Munich, Germany). The electrodes were placed longitudinally on the muscle belly. The interelectrode resistance did not exceed 5 kOhm. All signals from all tests were digitized with a sampling frequency of 1 kHz and led to computer for further processing. Simultaneously, they were stored also on the magnetic tape recorder (Racal, V-Store, Southampton, England).

Fatigue workout. The fatigue workout was performed on a special sledge apparatus (Fig. 1), which consisted of a 33-kg sledge, gliding on the track inclined at 23° from the horizontal. The apparatus has been described in detail elsewhere (17). The maximum jumping height was measured by releasing the subjects from 80 cm above the position when the legs were maximally extended (zero position). Once the maximum jumping height was established, 60% of the distance between the zero position and the maximum height was marked as an indicator for the fatigue workout intensity. The subjects were asked to perform consecutive jumps without pause to the 60% marker until told to stop. When they were no longer able to maintain the defined jumping height (exhaustion), the workout was terminated. The subjects had on-line visual feedback control of the attained jumping height. During jumping, the subjects achieved 90° knee angle at the lowest sledge position. Verbal feedback was provided to ensure correct angular position of the knee. To prevent trunk movements, the subjects were fixed well onto the sledge with straps around torso. Their arms were placed on the seat's sides next to the thighs and were not moved during jumping. Verbal encouragement was used to motivate the subjects throughout the workout.

Electrical stimulation. In all measurements with ES, as well as during explosive voluntary knee extension, the subjects sat in a knee extension measuring device and were fixed to the apparatus at the pelvis and over the distal part of the thigh to prevent trunk and thigh movements. The distal part of the shank was fixed to the force transducer, which had a constant lever arm to the knee joint axis. The knee joint angle was fixed at 45°. The self-adhering neurostimulation electrodes (5 × 5 cm, Axelgaard Manufacturing Co, Fallbrook, CA) were placed over VL, vastus medialis, and rectus femoris (RF) muscles. The distal electrodes were placed over the distal part of the muscle belly and the proximal ones over the middle part of the muscle belly. Each pair of electrodes was connected to its own stimulation channel that was galvanically separated from the others. In all occasions, the constant current square biphasic impulses of 0.3 ms duration were employed. A custom-made computer controlled electrical stimulator was used.

Single twitch test. Three supramaximal stimuli were delivered consecutively (one per second) to the relaxed VL. The torque signals from the twitch responses were smoothed (moving average, $N = 5$ points) and averaged with a trigger point at stimulus delivery. Maximum twitch torque (Ttw), electromechanical delay (EMDtw), time to peak torque (TPtw), half relaxation time (HRTtw), and peak rate of torque development (TRtw) were calculated.

Double pulse test. Two supramaximal stimuli with a 10-ms delay were delivered to the relaxed QF simultaneously via all three pairs of electrodes. The torque signal of the double pulse response was smoothed (moving average, $N = 5$ points) and analyzed for a maximum torque (Tdp) and maximum torque rise (TRdp). Tdp was used for the QF activation level assessment as well (described later). The rate of torque rise for single twitch and double pulse were normalized to a corresponding peak torque.

Low-high frequency torque test. Relaxed VL was stimulated with two consecutive trains of impulses with a frequency of 20 Hz (1-s duration) and 100 Hz (0.8-s duration). The stimulation intensity was set to three times the motor threshold intensity (30–54 mA), and it was kept the same for the both frequencies. The motor threshold intensity was determined as intensity when the first torque increase

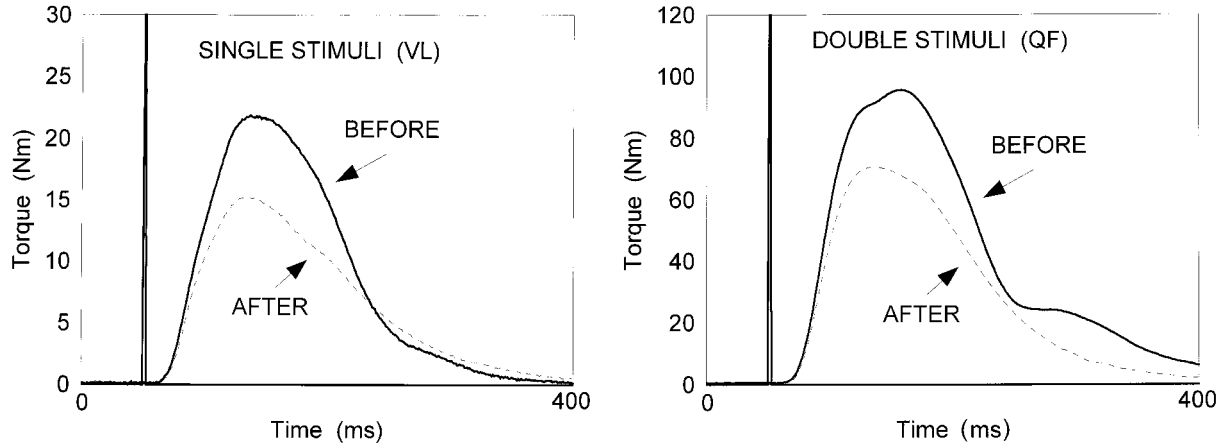

Figure 2—Typical sample responses of the relaxed muscle of one subject as measured after the warm-up and 2.5 min after the end of workout. In both cases, smaller peak torque with no temporal changes appeared after the end of the workout when the subject was no longer able to jump more than 60% of his maximum jumping height. Left panel: response of VL to single supramaximal stimulus; right panel: response of QF to double supra-maximal stimulus.

during 1-s long 100-Hz stimulation was observed. The mean torque during the last 50 ms of stimulation for each frequency (F20 and F100) was obtained. The ratio HLT was calculated according to equation HLT = F100/F20 · 100.

Voluntary explosive knee extension test. The subjects were placed onto the same chair under the same conditions as for the electrically evoked measurements. On command, the subjects tried to achieve their maximum isometric knee extension torque as fast as possible. They maintained the maximum knee torque for 2 s. The torque signal was smoothed (moving average, $N = 5$ points) and analyzed for a maximum torque (Texp) and maximum slope of the torque rise (TRexp).

Activation level. The activation level (AL) was assessed by a superimposed double pulse technique, derived from the twitch interpolation method, described by Merton (21). Torque produced by a double pulse at rest was compared with torque produced during a superimposed double pulse on the maximum voluntary knee extension. On the command signal, the subjects started to exert force. Two seconds after the start of voluntary contraction, when the maximum torque (T_{max}) was attained, the subjects received two stimuli in the same way as in the double pulse test. The difference (D) between torque level just before the double pulse (Tb) and the maximum torque during superimposed double pulse was compared with Tdp according to equation

$$AL = 100 - D\cdot(Tb/Tmax)/Tdp \cdot 100$$

A correction of D was included into the original equation, because double twitch was not always applied during the maximum torque level (T_{max}) but later, when the torque might already have declined slightly. Because at the lower torque than maximal, D tends to grow and because this relation was shown to be linear (21), the expression Tb/T_{max} can be applied to correct D. Maximal voluntary contractions for the voluntary explosive knee extension test and activation level were performed separately.

EMG activity during isometric MVC. EMG signal from VL was rectified and integrated (EMGvl) for the steady part of the maximum knee extension, appearing before the superimposed double twitch during a 0.5-s long interval.

Blood analysis. The blood samples for a blood lactate concentration (LA) analysis (20 μL) were taken from the finger tip, whereas the blood samples for determination of serum creatin-kinase activity (CK) were taken from the antecubital vein of the right arm. The LA was measured using a lactate analyzer (Roche, model 640). The CK activity was determined using a CK ultraviolet test kit (Boehringen, Mannheim, Germany).

Statistics. *T*-tests for paired samples were used to calculate the statistical significance of differences between initial measurement and after workout. Additionally, the Pearson correlation coefficients were calculated between relative changes in parameters after workout according to the initial state. Statistical significance was accepted at $P < 0.05$ (two-tailed).

RESULTS

Average exercise duration was 443.7 ± 304.9 (mean and SD) s. Blood lactate concentration changed from 1.80 ± 0.61 mmol·L^{-1} after the warm-up to 6.16 ± 1.66 mmol·L^{-1} ($P < 0.001$) 5 min after the end of the workout. CK, a marker for muscle damage, increased from 248.9 ± 142.4 IU·L^{-1} before exercise to 584 ± 344.0 IU·L^{-1} 2 d after the exercise.

Mean muscle responses to the single and double supramaximal electrical stimulus after exercise were similar (Figs. 2 and 3). The maximal single twitch torque declined from 22.1 ± 6.3 Nm to 17.3 ± 8.0 Nm ($P < 0.05$) and the maximal double pulse torque from 96.6 ± 15.4 Nm to 76.2 ± 19.8 Nm ($P < 0.001$). There were no statistically significant changes in any of observed time parameters (EMDtw, TPtw, and HRTtw) (Table 1). Accordingly, the maximal slopes of the torque rise decreased in the same manner as peak torques. When the slopes were normalized

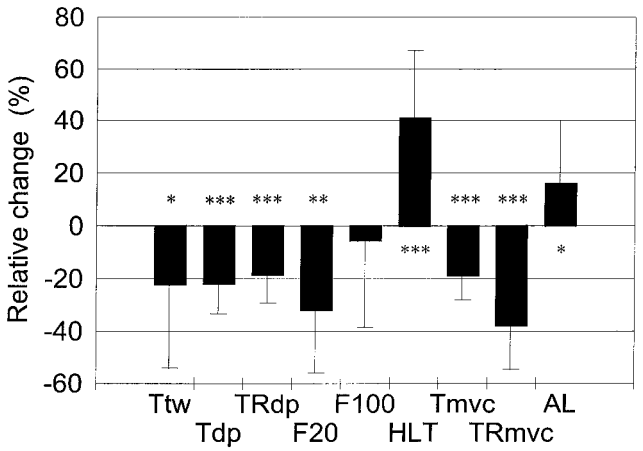

Figure 3—Relative changes in mechanical MVC and ES parameters. Ttw, single twitch peak torque; Tdp, double pulse peak torque; TRdp, peak torque slope of double twitch; F20, torque during 20 Hz ES; F100, torque during 100 Hz ES; HLT –F100/F20 torque ratio (see Methods); Tmvc, maximal torque during explosive MVC; TRmvc, peak torque slope during explosive MVC; AL, activation level. *Vertical lines* denote SD (* $P < 0.05$), ** $P < 0.01$, *** $P < 0.001$).

for corresponding peak torques, the differences before and after exercise became insignificant.

As presented in Figure 3, the torque at 20-Hz electrical stimulation decreased from 23.7 ± 9.2 Nm to 16.1 ± 7.8 Nm ($P < 0.01$), whereas the 100-Hz torque reduction was nonsignificant (from 39.6 ± 15.5 Nm to 36.1 ± 15.1 Nm). Consequently, the HLT ratio, used here as a measure of a frequency dependent fatigue (7), increased from 1.67 ± 0.23 to 2.31 ± 0.31 ($P < 0.001$). In subjects with the greater HLT after exercise, the maximal rate of torque rise in double twitch decreased more ($r = -.75$, $P < 0.01$).

In the isometric explosive knee extension (Fig. 3), both the maximal torque and the rate of torque rise decreased after exercise from 185.2 ± 30.7 Nm to 151.1 ± 32.3 Nm ($P < 0.001$) and from 1619.4 ± 389.8 Nm·s^{-1} to 1004.6 ± 359.8 Nm·s^{-1} ($P < 0.001$), respectively. The mean relative torque rise changed significantly more than the mean relative maximal torque ($P < 0.01$). The comparison of changes after exercise between the double twitch and the explosive MVC knee extension in the maximum torque and the rate of torque development showed no significant differences in the maximum torques but revealed more decreased voluntary rate of torque rise as compared with the electrically induced one. A higher activation level (from 69.0 ± 9.6% to 78.5 ± 12.1%, $P < 0.05$) and a higher mean EMG amplitude of VL during the sustained isometric MVC knee extension (from 100 ± 40 μV to 138 ± 84 μV, $P < 0.05$) were observed after the exercise as well.

TABLE 1. Changes in time parameters of the single twitch test.

	Before	After	*P*
EMDtw	21.1 ± 1.7	20.2 ± 2.3	0.102
TPtw	68.3 ± 9.4	63.2 ± 10.2	0.215
HRTtw	70.2 ± 9.6	68.2 ± 18.6	0.745

Times (in ms) in columns Before and After present mean ± SD of listed parameters, which were measured before and after the exercise. *P* denotes a level of statistical significance of *t*-test for paired samples (two way). EMDtw, electro-mechanical delay; TPtw, time to peak torque; HRTtw, half relaxation time.

DISCUSSION

The most important response to submaximal exhaustive SSC exercise was seen in the contractile characteristics of the quadriceps femoris muscle. The peak torques during single and double twitch declined without significant changes in temporal parameters. The torque during 20-Hz ES declined as well, but remained during 100-Hz ES almost unchanged. Further changes included a reduced maximal voluntary torque at isometric knee extension and an even more pronounced decrease in the maximal rate of torque development.

These phenomena can be partly explained through changes occurring in the muscle metabolism including pH-changes as well as structural impairments. As expected, the blood lactate level increased after the high-intensity exercise, involving large muscle mass. However, the duration of the exercise was too long to be classified as typical anaerobic activity, especially because the longest exercise lasted for 14 min. Even though the blood lactate level didn't reach high values, it was possible to conclude on a decreased pH in the working muscles. Decreased pH would increase the requirement for Ca^{2+} in the contraction process and decrease maximal tension (8).

Muscle damage is also among factors influencing muscle force production. The increased serum CK level strongly implied its presence. The finding of increased serum creatinkinase suggests that some form of muscle damage may have occurred. The presence of CK cannot, however, be used to quantify precisely the damage (19). In pure eccentric work, the initial local damage results from mechanical strain rather than from metabolic mechanisms (23). Changes in SR structure after high-intensity exercise may influence Ca^{2+} release and uptake (6). Therefore, it is possible to expect the effects of muscle damage already during the exercise and the tests performed immediately after the exercise should reveal this phenomenon.

Results obtained with 20- and 100-Hz electrical stimulation showed the presence of a low frequency fatigue (LFF), which has been connected to the excitation-contraction coupling failure (9). Low-frequency fatigue implies smaller Ca^{2+} release from sarcoplasmatic reticulum (SR) (27) and/or inhibition of Ca^{2+} binding to troponin (5), resulting in fewer active cross-bridges. Jones (15) suggested that LFF may result from exercise induced mechanical damage of SR. Increased Pi may also contribute to LFF reducing the Ca^{2+} sensitivity of the myofibrils (16). The twitch changes may further illuminate a dynamics of Ca^{2+} release and uptake from SR during the exercise. It is possible to speculate that Ca^{2+} release from SR was impaired whereas Ca^{2+} uptake might not change significantly. Reduced peak twitch torque without changes in contraction and relaxation times implied that a reduced number of active cross-bridges (22) may be more important for the reduced peak torque than smaller force per single cross-bridge. In that view, a reduced number of cross-bridges may be connected to lower pH and reduced Ca^{2+} release from SR.

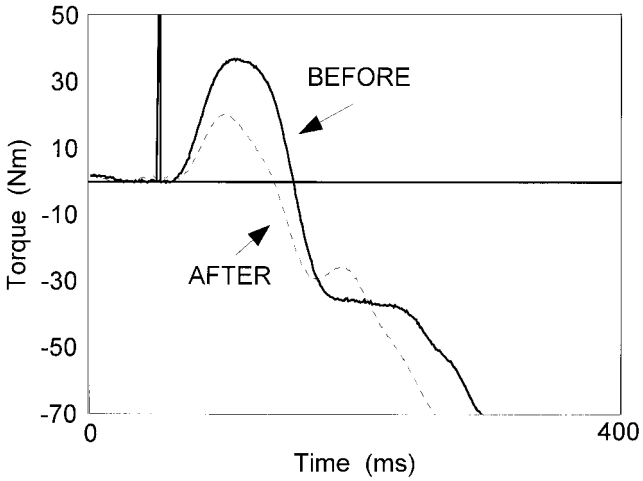

Figure 4—Responses of one subject to double stimuli superimposed over MVC before and after workout. Torques at the instant of stimulus delivery were aligned and set to zero to enable better comparison. Torque rise due to double stimuli was much smaller after the workout, denoting that less of the unused muscle contractile capacity was left to be activated with ES.

The well-preserved torque during 100-Hz stimulation suggests that the muscle action potential propagation was not impaired significantly and that the SR was still capable to maintain sufficient concentration of Ca^{2+} when the firing frequency was high enough. Similar results in LFF as in the present study were observed also after interval and continuous running at the level of anaerobic threshold (29) and after maximal eccentric contractions of the elbow flexors (23). These observations may support a view that the muscles were mostly affected by impaired excitation-contraction coupling, whereas the contractile part itself and the muscle action potential propagation may have not been changed critically.

While MVC isometric knee torque decreased, the activation level was increased (Fig. 4) implying recruitment of additional motor units. Increased iEMG, obtained also in the present study, may provide additional support for such occurrence (9). Results of this study are not similar to those during sustained MVC, but in agreement with those during submaximal intermittent contraction (2). One of the possible explanations is that in the present study the MVC torque did not correspond to maximal muscle capacity to produce force but was submaximal instead. On the other hand, the maximal rate of torque rise decreased even more than the maximal torque. More pronounced decrease in the maximal torque development capability than the maximal torque level in voluntary contraction was not reflected in the twitch contractions. Because a higher firing frequency is needed during force rise than during maximal force maintenance (12), the reason for more decreased maximal torque development may be related to an inability to deliver corresponding firing frequencies. Because the muscle action potential propagation seemed not to be impaired, the site to control the firing frequency could lie proximal to the neuromuscular junction. There are more possible mechanisms of interest: 1) an inhibition of alpha motoneuron via group III and IV afferents due to biochemical changes (11) 2), a disfacilitation of alpha motoneuron due to reduced muscle spindle activity (25), and/or 3) a recurrent inhibition of alpha motoneuron (18). It is not possible to name any of above mechanisms as an exclusive one for the resulted fatigue, although it is most likely that the main processes occurred at the alpha motoneuron level. Nevertheless, a central fatigue mechanism cannot be completely ruled out.

In addition to the recruitment of new motor units, as suggested by increased activation level, increased mean EMG amplitude might be influenced also by motor unit synchronization, higher firing frequency, and slower conduction velocity. The alternative mechanism would be a smaller Ca^{2+} release or Ca^{2+} utilization per single action potential and thus demanding more neural activity (action potentials) for the same force. It is also possible to assume that some muscle fibers were activated but did not respond because of muscle damage or contractile failure.

In contrast to maximal short-lasting exercise (30), the motor unit action potential propagation may not have been interfered into fatigue processes after the submaximally intensive SSC workout. The fatigue appearance after the submaximal SSC exercise may be mainly attributed to increased blood lactate concentration, increased Pi, appearance of muscle damage, and smaller Ca^{2+} release influencing the alterations in muscle contractile characteristics.

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