The time-course of voluntary and electrically evoked muscle performance during and after stretch-shortening exercise is different

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Abstract

The aim of the study was to establish the dynamics of maximal voluntary contraction force (MVCF), height of drop jump (DJ) and electrically evoked quadriceps muscle force at different stimulation frequencies during and after 100 DJs (stretch-shortening exercise, SSE). Healthy untrained men (n = 11; age = 21.8 ± 1.7 years) participated in the study. DJs were performed with 30 s intervals between jumps from the height of 0.5 m with counter-movement to 90 degrees angle in the knee and immediate maximal rebound. The force of the quadriceps muscle, evoked by electrical stimulation at 1 Hz (Pt), 20 Hz (P20) and 100 Hz (P100) frequencies (electrically evoked performance, EEP), MVCF and height of DJ (voluntary evoked performance, VEP) were established during SSE (after 10, 50, 100 DJ) as well as at 1, 4, 8, 24, 48 and 72 h after SSE. Time-course of P20 and P100 during and after SSE was time (ANOVA: p < 0.001) and frequency dependent (ANOVA: p < 0.001) The Pt, P20 and P100 decreased significantly (p < 0.01) more than MVCF and H of DJ during SSE. At the beginning of SSE (during 1-10 DJs) P20 and P100 decreased significantly (p < 0.001) more than during 11-50 and 51-100 DJs. There was a significant (p < 0.05) increase in Pt, P20 and P100 from 8 h to 48 h, whereas height of DJ and MVCF significantly decreased at that time. In conclusion, the differences in time course of VEP and EEP are most evident at beginning of SSE, where VEP does not change as EEP decreases, and within 8-48 hours after SSE, where VEP decreases as EEP increases.

Key words: Drop jump, muscle damage, electrical stimulation, low frequency fatigue.

Introduction

Exercise-induced muscle damage (EIMD) in humans frequently occurs after unaccustomed exercise, particularly if the exercise involves a large amount of eccentric contractions (Byrne et al., 2004; Clarkson and Hubal, 2002; Nosaka and Clarkson, 1996). The well documented symptoms of EIMD include disruption of intracellular muscle structure, sarcolemma and extracellular matrix (Clarkson and Hubal, 2002; Lieber and Friden, 2002; Nosaka and Clarkson, 1996; Proske and Allen, 2005; Warren et al., 2001). Prolonged impairment of muscle function measured during both voluntary and electrically stimulated contractions (Byrne et al., 2004; Faulkner et al., 1993; Warren et al., 1999), manifestation of low frequency fatigue (LFF) (Rijkelijkhuizen et al., 2005), protein leakage from injured muscle fibres, acute inflammation reaction and delayed-onset muscle soreness, stiffness and swelling (Clarkson and Hubal, 2002; Friden and Lieber, 2001; MacIntyre et al., 2001; Nosaka and Clarkson, 1996).

It is generally agreed that there are two prominent signs of damage in the muscle immediately after it has been subjected to a series of eccentric contractions. These are the presence of disrupted force-bearing structures and damage to excitation-contraction coupling system (Proske and Allen, 2005; Warren et al., 2001, 2002). The initial mechanical damage initiates a cascade of events that produces more severe secondary delayed-onset muscle damage between 1 and 3 days after the initial damage (Clarkson and Hubal, 2002; Faulkner et al., 1993).

It has been shown that strength loss after EIMD was independent of muscle action (isometric, concentric or eccentric) being performed (Byrne and Eston, 2002). The impairment of muscle function, however, was attenuated when the stretch-shortening cycle was used in vertical jumping performance (Byrne and Eston, 2002). Still Horita et al. (2003) concluded that concentric - only muscle activity (squat jump) does not appear to be affected by possible muscle damage, as observed in the case of drop jump. They speculated that these unique responses might be accounted for by the importance of muscle structure in stretch-shortening cycle activities and different motor command and/or control strategies between stretch-shortening cycle and concentric muscle actions.

Notwithstanding the obvious fact concerning the presence of both direct and indirect symptoms of EIMD, still not only the damage mechanism itself but also its effect on the time-course of muscle function and neuromuscular performance remains to be clarified. It has been recently concluded that there is no easy explanation for the strength loss following eccentric contractions, since strength loss is a result of complex interaction of various mechanisms (Butterfield and Herzog, 2005; Prasartwuth et al., 2005; Warren et al., 2002). The present paper was aimed at clarifying the following issues: 1) The difference in dynamics of voluntary evoked performance (VEP) and electrically evoked muscle performance (EEP) not only during stretch-shortening exercise (SSE) but also immediately after SSE, as well as 1, 4, 8, 24, 48 and 72 h after SSE? 2) If the dynamics of voluntary muscle performance during and after SSE dependent on muscle contraction type (isometric vs. stretch-shortening)? 3) If there is any dependence of the dynamics of electrically evoked muscle performance during and after SSE on muscle stimulation frequency (low vs. high frequency stimulation). The primary aim of the present study was to clarify these issues.
Method

Subjects
Healthy untrained men (mean ± SD: age = 21.8 ± 1.7 years, body weight = 74.2 ± 4.7 kg, height = 1.80 ± 0.04 m, n = 11) took part in this study. The untrained subjects were physically active but did not take part in any formal physical exercise or sport program. They had not been involved in any jumping or leg strength training programs during recent years. Each subject read and signed written informed consent form consistent with the principles outlined in the Declaration of Helsinki. This study was approved by the Ethics Committee of Kaunas Medical University.

Stretch-shortening exercise
The subjects performed 100 intermittent (30 s interval between the jumps) drop jumps (DJs) from the height of 0.5 m with counter-movement to 90 degrees angle in the knee and immediate maximal rebound. Time intervals after the 10th and 50th jumps were approximately 4-5 min because of muscle force measurements. During the jumps hands of the subjects were on the waist. The subject stepped on 0.5 m high platform with his left leg, i.e. the leg in which muscle contraction force was not tested. After each jump the subjects were informed of the height of the jump and were motivated to perform each jump as high as possible. Before SSE and during recovery control drop jumps was performed with the same techniques as during SSE. Height of the DJ was calculated by an earlier technique applying the following formula: $h = g \times t^2 / 8$, where $h$ = height of the drop jump, $g$ = acceleration of gravity (9.81 m·s$^{-2}$), $t$ = flight time (s). (Bosco et al., 1982). A similar research protocol was applied in previous researches (Skurvydas et al., 2006). The par- ticipants were required to indicate the severity of soreness in their quadriceps in response to muscle compression, as well as when standing up and walking at the start of each daily session.

Muscle function measurements
The equipment and technique for measuring muscle force was the same as has been used in a previous study (Ratkevicius et al., 1998, Skurvydas et al., 2006). Subjects were placed in an experimental chair. They sat upright in the experimental chair with a vertical back support. A hard disk for subsequent analysis. The output from the force transducer was also displayed on a voltmeter in front of the subject.

Plasma creatine kinase activity
Approximately 5 ml of blood was drawn from the vena cubiti media of the arm at each measurement time point (before exercise as well as 24 h and 48 h after exercise). Plasma samples were pipetted into microcentrifuge tubes and stored in a -20°C freezer until analysis. Plasma creatine kinase (CK, IU/L) activity was determined by using automatic biochemical analyzer “Monarch” (Instrumentation Laboratory SpA, USA-Italy).

Muscle soreness
Muscle soreness was reported subjectively using a visual analogue scale of 0 to 10, where 0 represents “no pain” and 10 represents “intolerably intense pain”. These muscle soreness evaluation methods have also been used in our previous research (Skurvydas et al., 2006). The participants were required to indicate the severity of soreness in their quadriceps in response to muscle compression, as well as when standing up and walking at the start of each daily session.

Experimental protocol
After measuring CK activity in the blood, the subject was seated in the experimental chair and after 5 min, muscle contractile properties were recorded in the following sequence: Pt, P20, P100 and MVCF (MVCF was reached after the 10th and 50th jumps were approximately 4-5 min because of muscle force measurements. The rest interval between muscle electrostimulations was 10 s and between MVCF measurements it was 1 min. The change in ratio of P20/P100 after exercise was used for the evaluation of LFF (Jones, 1996, Skurvydas and Zahovajevas, 1998, Martin et al, 2004). In order to establish the dynamics of muscle fatigue during 100 DJs the decrease in P20, P100 as well as P20/P100 ratio measured in percent per jump after first 10 DJs, from 11 DJs to 50 DJs as well as from 51 DJs to 100 DJs was evaluated. Fatigue index (FI) in percent per jump is calculated according to the formula: (Value before SSE – Value after n jumps)/ Value before SSE/n jumps x 100 (per cent). When FI is measured after 10 DJs, then n is equal to 10, measured from 11 DJs to 50 DJs n is equal to 40 and measured from 51 DJs to 100 DJs n is equal to 50.

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**TABLE 1.** Pre-exercise values of indices of electrostimulation-induced contractions of quadriceps muscle and MVCF in men (n=11). Data are means (±SD).

<table>
<thead>
<tr>
<th>Pt (N)</th>
<th>Ct (ms)</th>
<th>Rt (ms)</th>
<th>P20 (N)</th>
<th>P100 (N)</th>
<th>P20/P100</th>
<th>MVCF (N)</th>
<th>Height of DJ (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>79.7 (27.8)</td>
<td>78.5 (9.7)</td>
<td>58.4 (13.9)</td>
<td>274.7 (21.1)</td>
<td>319.4 (76.2)</td>
<td>.88 (.11)</td>
<td>596.2 (23.4)</td>
<td>40.2 (10.1)</td>
</tr>
</tbody>
</table>

Pt, P20 and P100 – muscle contraction force evoked by stimulating quadriceps muscle at 1 Hz, 20 Hz and 100 Hz frequencies. Ct – Pt contraction time; Rt – relaxation to half Pt time; MVCF – maximum voluntary contraction force; Height of DJ – height of drop jumps.

3 times and the best value was taken for evaluation). Before height of DJ was tested, each subject performed warm-up exercises which consisted of 5 min running on the spot with an intensity that corresponded to heart rate (HR) 130-150 beats per minute (it comprised about 70 percent of maximum HR). Then the subject performed 10 squat-stands. HR was measured with a Polar HR recorder (Polar Electro) (Skurvydas et al., 2006). Afterwards the height of DJs was established. About 1 min later the SSE was undertaken. After 10, 50 and 100 DJs the subjects were seated in the experimental chair once again and both voluntary and electrostimulation-evoked muscle contraction properties were registered (they were registered 2-3 min after the end of the jumping exercise). MVCF was also registered, but only twice. At 1 h, 4 h, 8 h, 24 h, 48 h and 72 h after SSE the testing procedure was repeated in the sequence, as prior to the load. Besides, at 24 h, 48 h and 72 h after SSE muscle soreness as well as CK activity at 24 h and 48 h after SSE was determined.

**Data and statistical analysis**

Descriptive data are presented as means ± SD. The two-way analysis of variance (ANOVA) for repeated measures was used to determine the effects of time (10 levels: before, after 10, 50 and 100 DJ as well as during recovery within 1 h, 4 h, 8 h, 24 h, 48 h and 72 h) and frequencies of stimulation (2 levels: 20 Hz and 100 Hz) on contractile properties of quadriceps muscle. Besides, the effect of time and contraction type (2 levels: isometric vs stretch-shortening) was established. If significant effects were found, post hoc testing was performed applying paired t-tests with a Bonferroni correction for multiple comparisons. The level of significance was set at 0.05. In order to evaluate the relationship between indirect symptoms of EIMD Pearson correlation coefficient was established.

**Results**

VEP and EEP pre-exercise values (mean ± SD) are shown in Table 1. The time-courses of the height of DJs, MVCF, Pt, P20 and P100 during and after SSE are shown in Figure 1 and 2 (the effect of time of all characteristics was p < 0.001). Time-course of MVCF and height of DJ during and after SSE was muscle contraction type dependent (ANOVA between MVCF and height of DJ: p < 0.001). The height of DJ before SSE was 40.2±10.1 cm compared to 36.0 ± 8.5 cm after SSE (p < 0.01). The height of DJ had decreased by 10.3 ± 4.9 % after SSE, whereas MVCF after SSE decreased by 17.4 ± 7.4 %.

Time-course of P20 and P100 during and after SSE was time and frequency dependent (ANOVA: p < 0.001; Figure 1). Pt, Ct and Rt significantly decreased after SSE, and Pt as well as Ct did not recover within 72 h (p < 0.05, compared to pre-exercise value; Figure 2), whereas Rt at
FIGURE 1. Time course of changes in muscle twitch force (Pt), Pt contraction time (Ct), relaxation to half Pt time (Rt) after 10, 50 and 100 DJs and 1, 4, 8, 24, 48 and 72 h after SSE (mean±SD). SSE – stretch-shortening exercise (100 drop jumps performed from 0.5 m with counter-movement to 90 degrees in the knee with immediate maximal rebound, with 30 s interval between the jumps). The dashed line indicates the pre-exercise mean level.

FIGURE 2. Time course of changes in muscle twitch force (Pt), Pt contraction time (Ct), relaxation to half Pt time (Rt) after 10, 50 and 100 DJs and 1, 4, 8, 24, 48 and 72 h after SSE (mean±SD). SSE – stretch-shortening exercise (100 drop jumps performed from 0.5 m with counter-movement to 90 degrees in the knee with immediate maximal rebound, with 30 s interval between the jumps). The dashed line indicates the pre-exercise mean level.

* p < 0.05 compared to pre-exercise values.

1 h after SSE did not differ from pre-exercise level (p > 0.05). After 10 DJs there was a significant decrease in P20 and P100 (both: p < 0.05), height of DJ and MVCF had significantly decreased after 50 DJs (p < 0.01) and all these parameters had not recovered within 72 h (Figure 1). It is of interest to note that even after 10 DJs LFF manifested itself and had not disappeared within 72 h (p < 0.05, compared to the pre-exercise value; Figure 3). The percent per jump decrease in P20, P100 and P20/P100 ratio from 1 to 10 DJs was significantly greater than from 11 to 100 DJs (Figure 4).

There were significant decreases in Pt, P20 and P100, as well as in P20/P100 (p < 0.05, compared with the value immediately after SSE) at 60 min after SSE, but changes in MVCF or height of DJ were not significant (Figure 1, 2 and 3). There was a significant decrease in height of DJ and MVCF from 8 h to 48 h after SSE (p < 0.05), whereas there were no changes in Pt, P20 and P100. Within 24 h - 48 h after the SSE the subjects felt an acute muscle pain (4-5 points approximately; Figure 5). Besides, the CK activity in the blood within 24 h after SSE had increased approximately up to 1300 IU/L (p <
The time-course of muscle performance

FIGURE 4. Fatigue index in percent per jump of P20, P100 and P20/P100 ratio during SSE (mean±SD). P20 and P100 – muscle contraction force evoked by stimulating quadriceps muscle at 20 Hz and 100 Hz frequencies. SSE – stretch-shortening exercise (100 drop jumps performed from 0.5 m with counter-movement to 90 degrees in the knee with immediate maximal rebound, with 30 s interval between jumps).

* p < 0.05 compared to 1-10 drop jumps.

FIGURE 5. Muscle soreness 24, 48 and 72 h after SSE (mean±SD). SSE – stretch-shortening exercise (100 drop jumps performed from 0.5 m with counter-movement to 90 degrees in the knee with immediate maximal rebound, with 30 s interval between jumps).

0.05, as compared to the pre-exercise value; Figure 6).

We have established, that there was a significant relationship between decrease in height of DJs and MVCF at 24 h and 48 h after SSE on the one hand, and muscle soreness (r = 0.72-0.94) and CK (r = -0.6-0.7) on the other hand. There was a negative insignificant correlation (r = -0.31-0.39) between muscle soreness and CK at 24-48 h after SSE.

Discussion

To our knowledge this is the first study that dealt with differences between time-course of human VEP and EEP performance in both during (after 10, 50 and 100 DJs) and after (immediately, 1, 4, 8, 24, 48 and 72 h after exercise) stretch-shortening exercise (drop jumping). In generally the results of the study have shown that time-course of muscle function during and after SSE is significantly dependent on: 1) the specific character of muscle activation (voluntary vs. electrically); 2) muscle voluntary contraction type (isometric vs. stretch-shortening) and 3) muscle stimulation frequencies (low vs. high stimulation frequencies).

Among the more detailed and interesting findings of this study the following might be mentioned: a) electrically evoked muscle force during SSE decreased significantly more than voluntary performance (height of DJs and MVCF); b) at the beginning of exercise (after 10 DJs) Pt, P20 and P100 changed significantly more than during the second part of SSE (at low frequency stimulation in particular); c) both during and after SSE MVCF decreased more significantly than height of DJ; d) there was a significant increase in Pt, P20 and P100 from 8 h to 48 h after SSE, whereas height of DJ, as well as MVCF, decreased significantly; f) the secondary decrease in height of DJ as well as MVCF significantly correlated.
with muscle soreness within 24-48 h. after SSE.

The main causes of changes in VEP and EEP during and after SSE is related with exercise induced muscle damage

Changes in both voluntary and electrically evoked muscle performance during the SSE are likely not to be attributed to an increase in the myoplasm of metabolites, like phosphate and hydrogen ions, since the duration of the jump (0.5-0.55 s) was too small for ATP and PCr to be decreased significantly while the resting period of 30 s was sufficient for ATP and PCr to be restored. The causes of changes in muscle performance when performing SSE are therefore associated with non-metabolic factors, most likely related with muscle damage.

After performing SSE indirect symptoms of muscle damage manifested themselves within 24 h -72 h after the load: the rise of muscle soreness (Figure 5) and the increased CK activity (Figure 6), prolonged impairment of neuromuscular performance (MVCF and height of DJ) (Figure 1), as well as the muscle force evoked by low and high electrostimulation frequencies (Figure 1). There is no doubt whatever that the main reasons for the decrease in VEP and EEP were related to damage force-bearing structures (Clarkson and Hubal, 2002, Warren et al., 2001) and excitation-contraction coupling system (Prasartwuth et al., 2005; Warren et al., 2002). Recent work of Byrne and Eston (2002) has demonstrated that knee extensor strength force recovery was incomplete 7 days after 100 repetitions of eccentric phase of the barbell squat exercise performed with a load 80% of concentric one repetition maximum. They have established that there was a decrease of 5-10 % in height of DJ after the exercise and CK activity after the exercise was similar to the registered by us. It is rather unexpected therefore that within 60 min after SSE significant changes occurred in the muscle force evoked at low and high stimulation frequencies (Figure 1 and 2). If due to muscle damage manifests itself sarcomere disruption (Proske and Allen, 2005), consequently, sarcomere disruption should not regenerate within 4 h - 8 h (Proske and Allen, 2005). It should be supposed, therefore, that within 60 min after SSE an increase in EEP can not be associated with regeneration of sarcomeres.

Secondary decrease manifested itself but in VEP

The results of our research have shown that there is a significant increase in EEP from the end of SSE to 48 h, whereas VEP secondary decreased significantly (Figure 1). This is in accord with research done by other scientists who have found that after exercise that brings about muscle damage instead of the muscle being restored to its initial level after load there even occurs a decrease in neuromuscular performance (Faulkner et al., 1993; Horita et al., 2003; Komi, 2000). Recent data show that most of the early strength loss results from a failure of excitation-contraction coupling processes and that a slow loss of contractile proteins in the days following injury prolongs the time of recovery (Warren et al. 2002). Ingalls et al. (1998) have found a decrease in the mouse muscle force during the first three days after EIMD not to be associated with decreases in the amount of contractile protein, since protein degradation started two days later. Besides, alongside with the onset of protein degradation the recovery of muscle force started. This indicates that the time-course of contractile protein degradation and muscle function does not coincide. It remains for the scientists to clear up if there was a greater decrease in muscle force because of the failure of excitation-contraction coupling or due to muscle sarcomeres damage.

Since after the exercise bringing about muscle damage the muscles are subjected to processes that, undoubtedly, should call forth decrease in MVCF we supposed that, likewise in the case with MVCF and height of DJ, there should also be a decrease in the force evoked by all stimulation frequencies during recovery after SSE. In the case of our experiments, however, there was no manifestation of secondary decrease in the force evoked by stimulation at low and high frequencies (Figure 1). This
clearly show that changes in VEP and EEP is different during recovery after SSE.

The time-course of voluntary muscle performance during and after SSE is dependent on muscle contraction type

The results of our study indicated that SSE affected more significantly stretch-shortening cycle performance (height of DJ) than MVCF (Figure 1). This corresponds to the results of the research done by Byrne and Eston (2002) who have found that the impairment of muscle function was attenuated when the stretch-shortening cycle was used in vertical jumping performance (Byrne and Eston, 2002). It has been shown that the fatigue of the neuromuscular system induced by stretch-shortening exercise cannot be attributed to muscle damage alone since it might also be caused by differences in the modulation of reflex and stiffness interaction as well as compensation by central motor command (Komi, 2000). Horita et al. (2003), however, have concluded that concentric-only muscle activity (squat jump) does not appear to be affected by possible delayed-onset muscle damage, as observed in the case of drop jump. They speculated that these unique responses might be accounted for by the importance of muscle structure in SSC activities and different motor command and/or control strategies between SSC and concentric muscle actions.

The results of Harrison and Gaffney (2004) indicated that SSE significantly affected stretch-shortening cycle performance by causing relatively greater reductions in squat jump performance than DJ. They concluded that the muscle damage intervention also significantly increased leg-spring stiffness, which indicates that the changes in leg stiffness may be an important adaptation resulting from eccentric exercise. Opinions presented above show that there is no generally accepted conclusion in what way EIMD affects voluntary evoked muscle performance depending on muscle contraction type regardless of our research data that show the time-course of voluntary evoked muscle performance to be contraction type dependent. Notwithstanding the generally accepted fact that muscle soreness can influence motor control effectiveness its dependence on the mode of neuromuscular performance remains not clear.

The time-course of electrically evoked muscle performance during and after SSE dependent on muscle stimulation frequency

The results of our research have shown that P20 as well as Pt both during SSE and throughout 72 h after SSE decreased to a greater extent that the P100 (Figure 1 and 2). This indicates that the muscles were subjected to LFF that remained as long as 72 h after the SSE (Figure 3). Other scientists, exactly like us, have observed LFF not to recover after loads for no less than 24 h - 72 h (Westerblad and Allen, 2002). LFF is characterized by a relative loss of force at low frequencies of stimulation and it is important to mention that the force is not impaired or there is but relatively low impairment at high frequencies (Jones, 1996; Ratkevicius et al., 1998; Rijkelijkhuizen et al., 2005; Westerblad and Allen, 2002). In our case there was a decrease in the force evoked not only by low stimulation frequencies (20 Hz) but by high stimulation frequencies (100 Hz) as well (Figure 1). It is but rare that one could find physical load capable of bringing about a decrease in the force evoked by low stimulation frequencies since in nearly all of the cases there is a greater or smaller decrease in the force evoked by high stimulation frequencies too (Martin et al., 2004; Ratkevicius et al., 1998; Strojnik and Komi, 1998).

Although the underlying mechanism is unknown, both metabolite build-up and elevation of intracellular Ca$^{2+}$ concentration, as well as mechanical damage to the muscle, have been suggested to play a role in the development of LFF (Westerblad and Allen, 2002). It might be speculated that in our case the LFF manifestation (Figure 3) is indicative of the excitation-contraction coupling failure since it has been established that one of the mechanisms of LFF depends on the decreased release of Ca$^{2+}$ from the sarcoplasmic reticulum (Westerblad and Allen, 2002). A decrease in Ct and Rt during and after SSE (Fig. 2) also indirectly confirms the assumption that a decreased release of Ca$^{2+}$ from the sarcoplasmic reticulum had taken place.

The result of our research performed with human muscle are in accord with the data of other scientists who maintain that there occurs a significant decrease in muscle function of frogs, mice and rats even after 1-10 eccentric contractions (Brooks et al., 1995; Macpherson et al., 1996; Patel et al., 2004; Yeung et al., 2003). It has been proposed that during a series of eccentric contractions, more and more sarcomeres will become overstretched, beginning with the weakest and including progressively stronger sarcomeres (Proske and Allen, 2005). This is likely to be the main reason why there was a significantly greater decrease in the muscle force evoked by electrostimulation after the first 10 DJs than between 50 DJs and 100 DJs.

Is there correlation among indirect symptoms of EIMD?

We have established, that there is a significant correlation between decrease in height of DJ and MVCF at 24 h and 48 h after SSE on the one hand and muscle soreness and CK on the other hand. In other words, it might be speculated that the greater muscle soreness, the greater decrease in height of DJ and MVCF, the smaller the increase in CK in the blood.

There was a negative nonsignificant correlation between muscle soreness and CK 24-48 h after SSE. This seems to indicate that two indirect symptoms of muscle damage, i.e. CK and muscle soreness are, possibly, not functionally related. We think muscle pain to be more indicative of inflammation, whereas the increase in CK might be more associated with muscle damage. This is confirmed by the finding of MacIntyre et al. (2001) who have found that muscle soreness was associated with inflammation but not with muscle damage.

It has already been observed by other authors earlier that CK activity in blood is a poor predictor of the muscle function after injury (Friden and Lieber, 2001), likewise a delayed onset muscle soreness is a poor indicator of muscle damage (Nosaka et al., 2002). Still there
exist publications asserting that the delayed recovery of the twitch muscle force response is related to changes in CK (Nicol et al., 2003). Therefore, no doubt, scientists will have to find a clearer understanding of inflammation process, damage of muscle contractile and/or activation systems as well as muscle pain. The fact that there exists a strong relationship between muscle soreness within 24-48 h after SSE and decrease in neuromuscular performance (height of DJ and MVCF) can be accounted for by the circumstance that due to muscle soreness transferred by inhibitory signals from III and IV afferents the deterioration of the efficiency of the central motor drive might take place (Le Pera et al., 2001).

Conclusion

In conclusion, the differences in time course of VEP and EEP are most evident at beginning of SSE, where VEP does not change as EEP decreases, and within 8-48 hours after SSE, where VEP decreases as EEP increases. The secondary decrease in VEP within 24-48 h correlated significantly with muscle soreness. Therefore, in estimating muscle function during stretch-shortening exercise it is obligatory that such determinants, as muscle activation and contraction type as well as stimulation frequencies should be taken into account.

References


Key points

• There was no change in voluntary muscle performance while electrically evoked performance decreased significantly during first 10 drop jumps.
• There was a significant increase in electrically evoked muscle performance from 8 h to 48 h after 100 drop jumps, whereas voluntary contraction force, decreased significantly.
• The secondary decrease in the height of drop jump as well as in maximal voluntary contraction force correlated significantly with muscle soreness within 24-48 h after exercise.

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