Cooling leg muscles affects dynamics of indirect indicators of skeletal muscle damage

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Abstract. Objectives: To determine the effect of leg immersion in cold water after stretch-shortening exercise (SSE) on the time-course of indirect indicators of exercise-induced muscle damage (EIMD).

Methods: Twenty healthy untrained men twice performed SSE consisting of 100 drop jumps (DJs) from 0.75 m height performed with maximal intensity with an interval of 20 s between the jumps. DJs were performed with counter-movement to 90 degrees angle in the knee and with immediate maximal rebounds. After SSE the subject’s legs immediately, as well as after 4 h, 8 h and 24 h, were immersed into a bath filled with water at 15 ± 1°C. Quadriceps muscle voluntary contraction force (MVCF) and force evoked by electrostimulation (ESF) at different frequencies and at different muscle length, jump height (H), muscle soreness and creatine kinase (CK) activity in the blood were measured before SSE, immediately after SSE and within 72 h of recovery.

Results: After stretch-shortening exercise MVCF, ESF and H were significantly (P<0.001) decreased and were not restored even after 72 h in the group that did not receive the cooling. Within 24–48 h after SSE the subjects felt great muscle pain and CK activity in their blood was increased (P<0.001). The cooling significantly accelerated the disappearance of all these indicators, except for low-frequency fatigue, but only within 24–72 h after SSE.

Conclusion: Cold water immersion after SSE accelerates the disappearance of the majority of indirect indicators of EIMD.

Keywords: Exercise, electrostimulation, muscle recovery, cryotherapy

1. Introduction

Exercise-induced muscle damage in humans frequently occurs after unaccustomed exercise, particularly if the exercise involves a large amount of eccentric contractions [1,2,28,33,35]. The well documented indicators of muscle damage include disruption of intracellular muscle structure, sarcolemma and extracellular matrix [2,7,41], prolonged impairment of muscle function measured during both voluntary and electrically stimulated contractions [4,41], protein leakage from injured muscle fibres, acute inflammation reaction and delayed-onset muscle soreness, stiffness and swelling [2,7,13,24,29].

Several studies have been undertaken to find ways to alleviate and treat the indicators of exercise-induced muscle damage [8,9,15,17,20,23].

One modality of such a treatment involves cryotherapy. The effectiveness of this treatment, however, is not clear due to limited research and the variations in treatment modality, frequency of applications, duration of treatment, as well as due to structural and functional muscle indices that have effect on this treatment. When cryotherapy is applied at an appropriate frequency, duration and temperature to the injured muscle, it reduces the inflammatory response and alleviates spasm and pain after muscle injury [5]. Numerous studies have been conducted examining the effect of cryotherapy on tissue temperature, blood flow, pain and swelling [16, 23,40]. Retarding secondary injury is an important theoretic benefit of cryotherapy [20,27].

There is no unanimous opinion as to the efficacy of applying cryotherapy in eliminating indirect indicators
of exercise-induced muscle damage. As observed by Prentice [32] the combination of cold treatment and stretching was most effective, whereas the positive effect of this has not been observed by others [5,17,31,43]. It is possible that the lack of agreement between studies may be attributable to the nature of cryotherapy procedure and the timing of the application [11,19,23,26]. Besides, it may depend on the indirect indicators of skeletal muscle damage chosen for the study, as well as on the level of muscle adaptiveness of the subjects.

Notwithstanding the results of the research presented above that indicate in what way and why the exercise-induced skeletal muscle damage manifests itself, as well as results of the first research (with the subjects to whom muscle cooling had not been applied) showing a positive effect on the disappearance of indirect indicators of muscle damage a few questions remain to be answered: 1) which of the indirect indicators of muscle damage is more sensitive to the effect of cooling – muscle contraction force, neuromuscular performance, muscle soreness or CK? 2) does cryotherapy exert the same influence on the maximal voluntary contraction force and jump height at stretch-shortening cycle as well as on the force evoked by electrostimulation of the muscle at different frequencies and at different length? 3) when does the effect of cooling manifest itself: immediately after cooling or within a certain period of time? 4) does the disappearance of low frequency fatigue (LFF) depend on the effect of muscle cooling after exercise? The primary aim of the present study was to answer these questions.

2. Methods

2.1. Rationale of experimental approach

The aim of the research was to establish the effect of leg immersion in cold water after stretch-shortening exercise (SSE) on the time-course of indirect indicators of exercise-induced muscle damage. To examine whether cold water immersion accelerates the disappearance of indirect indicators of exercise-induced muscle damage two experiments have been carried out: the first – without muscle cooling (the control one) and the second (the experimental one) – with muscle cooling after SSE that, undoubtedly, brings about muscle damage. In order to avoid individual differences of muscle adaptation and to establish more accurately the effect of cooling on the indirect indicators of muscle damage the same subjects participated in both experiments. The choice of their participation in the first and second experiments was made at random. The time interval between experiments done was 9–10 months, i.e. enough for the exercise-induced muscle damage not to have any residual effect [30,38].

2.2. Subjects

Healthy untrained men (age 20.4 ± 1.7 years, body weight 76.2 ± 4.7 kg, height 180.7 ± 6.5 cm, BMI body mass index (BMI) 22.8 ± 2.7 kg/m², n = 20) gave their informed consent to take part in two experiments within the study. The untrained subjects were physically active but did not take part in any formal physical exercise or sport program. Each subject read and signed the written informed consent form consistent with the principles outlined in the Declaration of Helsinki.

2.3. Muscle function measurements

The equipment and technique for measuring muscle force was the same as has been used in a previous study [34,37,38]. Subjects were placed in an experimental chair. They sat upright in the experimental chair with a vertical back support. A strap secured the hips and thighs to minimise uncontrolled movements. The right leg was clamped in a force-measuring device with the knee kept at an angle of 90 degrees (long muscle length, LL) or 135 degrees (short muscle length, SL).

Equipment and procedure for electrical stimulation were essentially the same as has been described previously [34,37,38]. A high voltage stimulator (MG 440, Medicor, Budapest, Hungary) was used. Electrical stimuli to the quadriceps muscle were delivered through surface electrodes (9 × 18 cm) padded with cotton cloth and soaked in saline solution. The electrical stimulation was always delivered in trains of square wave pulses of 1 ms duration (voltage 150 V, which induces 60–75 per cent of maximal voluntary contraction force; MVCF).

The following variables were measured: the force of the quadriceps muscle, aroused by electrical stimulation at 20 Hz (P20) and 50 Hz (P50) frequencies (the duration of each electrical stimulation series was 1 s) and MVCF. The rest interval between muscle electrostimulations was 10 s and between MVCF it was 1 min. The ratio of P20/P50 kinetics after exercise was used for the evaluation of LFF [10,18,25,39]. The contractile force induced by electrostimulation was measured at SL and LL in a randomised manner for each subject. MVCF was measured only at knee-joint angle of 90 degrees.
2.4. Vertical jump performance

Before jumping fitness was tested, each subject performed warming-up exercises. After warming-up each subject performed maximal drop jumps (DJs) from the height of 0.75 m on a Kistler force plate (type 9286A, USA) (3 jumps performed with an interval of 20 s were allowed per person and the best jump was counted). Heights of the DJs were calculated making use of an earlier described technique by applying the following formula: \( H = 122.625 \times T_f^2 \) (m) where: \( H \) = jump height and \( T_f \) = flight time (s) [3]. Jumps were performed in this way: DJs from the height of 0.75 m with immediate maximal rebounds, with counter-movement to 90 degrees angle in the knee. When performing the jumps the subject held his hands on the waist in all cases. Techniques for DJs were the same as described previously [37].

2.5. Leg immersion in cold water

The subjects’ legs immediately after the SSE and at 4 h, 8 h and 24 h after it were immersed in a water-bath at the temperature of 15 ± 1°C (this was done twice for 15 min with an interval of 10 min). The water was maintained at the recommended temperature of 15 ± 1°C by adding cold or hot water. Our muscle cooling procedure coincided with the cryotherapy methods recommended [11,26]. During the procedures applied muscle temperature decreases approximately by 7–10°C [26].

2.6. Plasma creatine kinase activity

Approximately 5 ml of blood was drawn from the arm vein at each measurement time point (before exercise, 24 h and 48 h after exercise). Plasma creatine kinase (CK, IU/L) activity was determined by using automatic biochemical analyzer “Monarch” (Instrumentation Laboratory SpA, USA-Italy).

2.7. Muscle soreness

Muscle soreness was evaluated subjectively (from 0 to 10 points) at 24 h, 48 h and 72 h after the SSE. These muscle soreness evaluation methods have also been used in our previous studies [37,38].

2.8. Stretch-shortening exercise (SSE)

The subjects in both experiments performed 100 intermittent (every 20 s) DJs from the height of 0.75 m with counter-movement to 90 degrees angle in the knee and immediate maximal rebound (stretch-shortening exercise, SSE). Jumps were performed using the Kistler force plate. After each jump the subjects were informed of the height of the jump and were motivated to perform each jump as high as possible. A similar research protocol was applied in previous studies [37,38] too, except for the fact that in preceding researches drop jumps were performed from the height of 0.4 m.

2.9. Experimental protocol

The subject was seated in the experimental chair and blood was drawn from the arm vein for the first time, and after 5 min, muscle contractile properties were recorded in the following sequence: P20, P50 and MVCF. Then the subjects undertook light exercise as warm-up. Afterwards the height of DJs was established. Then the SSE was undertaken: 100 DJs of maximal intensity from a 0.75 platform every 20 s. The subject stepped on 0.75 m high platform with his left leg, i.e. the leg in which muscle contraction force was not tested. After 100 dropping jumps the height of DJs of the subjects was established. Then the subjects were seated in the experimental chair once again and both voluntary and electrostimulation-induced muscle contraction properties were registered (they were registered 2–3 min after the end of the jumping exercise) (A0). MVCF was also registered, but only twice. At 4 h, 8 h, 24 h, 48 h and 72 h the testing procedure was repeated in the sequence, as prior to the load. Besides, at 24 h, 48 h and 72 h muscle soreness was determined. Similarly, at 24 h and 48 h CK activity was determined too. The subjects’ legs immediately after the SSE and at 4 h, 8 h and 24 h after it were immersed in cold water, the immersion was done after the evaluation of MVCF, P20 and P50.

2.10. Data analysis

The two-way analysis of variance (two way ANOVA) for repeated measures was used to determine differences between experiments 1 and 2. When the ANOVA was significant, a paired Student’s test was used to determine differences between the groups. Statistical significance was set at \( P < 0.05 \) and \( P < 0.001 \). To evaluate the effect of muscle cooling the percentage
difference between the indices without cooling (experiment 1) and with cooling (experiment 2) at different time intervals (4 h, 8 h, 24 h, 48 h and 72 h) was determined.

3. Results

3.1. Testing the muscle function prior to the SSE

There was no difference \( (P > 0.05) \) in the initial values of the MVCF and muscle contraction force evoked at different electrostimulation frequencies (Table 1) as well as height of DJs between the experiments 1 and 2. Still in both cases muscle force evoked by electrostimulation at 20 Hz and 50 Hz frequencies at SL was greater than stimulating the muscle at LL (Table 1).

3.2. SSE loading

The height of DJs before the SSE during experiments 1 and 2 was 36.7 ± 2.1 cm and 36.2 ± 4.6 cm compared to 33.5 ± 3.5 cm and 34.1 ± 5.8 cm after the SSE respectively \( (P > 0.05) \) in the case of baseline values of both experiments being compared; \( P < 0.001 \), as compared to pre-exercise values). The results of the research have shown that time-courses of the height of DJs during SSE in experiments 1 and 2 was alike (Fig. 1). It is worth noting that the average jump height of 100 jumps during SSE in experiments 1 and 2 was 36.6 ± 5.7 cm and 37.2 ± 3.9 cm accordingly \( (P > 0.05) \) between experiments 1 and 2; \( P > 0.05 \) as compared to pre-exercise values).

After SSE during experiment 1 there was a significant decrease in the height of DJs \( (P < 0.001) \), (Fig. 2A), MVCF \( (P < 0.001) \), (Fig. 2B), as well as the force evoked by electrostimulation at 20 Hz and 50 Hz frequencies (by electrostimulation at 20 Hz and at SL in particular), \( (P < 0.001) \) and had not recovered within 72 h (Fig. 4A, B, C, D). Within 24-72 h after the SSE the subjects felt an acute muscle pain (5–7 points approximately) (Fig. 3A). Besides, the CK activity in the blood within 24–48 h after the SSE had increased roughly up to 1200 IU/L \( (P < 0.001) \), as compared to the pre-exercise value (Fig. 3B).

3.3. Effect of cooling on time–course of recovery of MVCF and height of DJs

Leg muscles cold water immersion after SSE significantly \( (P < 0.001) \) increased the recovery speed of DJs height (Fig. 2A) and MVCF (Fig. 2B). In the case of experiment 1 height \( (H) \) of DJ and MVCF had not recovered to their initial level even within 72 h after the SSE \( (P < 0.001) \), as compared to the initial pre-exercise values), whereas due to legs cooling during experiment 2 MVCF had recovered within 24 h and there was no significant difference in \( H \) of DJs from its initial value within 48 h after the SSE \( (P > 0.05) \). There was a significant difference \( (P < 0.05) \) in the values of both \( H \) of DJs and MVCF registered during experiments 1 and 2 at 24 h, 48 h and 72 h after the SSE. It is interesting to note that during experiment 1 the values of \( H \) of DJs and MVCF during recovery after the end of SSE until 48 h following it had even decreased \( (P < 0.05) \), as compared to the values of 0 h and 48 h), whereas due to muscle cooling the recovery had not been impaired in the least.

3.4. Effect of cooling on time-course of recovery of muscle force induced by different stimulation frequencies at different muscle length

The results of research have shown that due to the cooling of leg muscles there was a significant acceleration in the recovery of muscle force evoked by electrostimulation at 20 Hz and 50 Hz frequencies both at LL (Fig. 4A, C) and SL (Fig. 4B, D). If in the case of experiment 1 P20 and P50 registered both at SL and LL had not recovered to their initial state as long as 72 h after the SSE \( (P < 0.001) \), during experiment 2 within 72 h after the SSE the values of above mentioned indicators did not differ from their initial state \( (P > 0.05) \). Besides, when comparing the recovery of P20 and P50 during experiments 1 and 2 we can see a significant difference \( (P < 0.05) \) between P20 and P50 (both at SL and LL) registered in the course of experiments 1 and 2 within 72 h after SSE. Still LFF both at SL and LL estimated according to changes in P20/P50 was not dependent on muscle cooling (Fig. 5). It is of interest to note that LFF was greater at SL than at LL only immediately after SSE.
### Table 1
Control values of indices of men’s electrostimulation-induced contractions of quadriceps muscle, MVCF (mean ± SD)

<table>
<thead>
<tr>
<th>Knee angle (degree)</th>
<th>P20, N</th>
<th>P50, N</th>
<th>P20/P50</th>
<th>MVCF, N</th>
</tr>
</thead>
<tbody>
<tr>
<td>First experiment (without cooling)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>90</td>
<td>331.1 ± 41.2</td>
<td>437.9 ± 61.5</td>
<td>0.76 ± 0.06</td>
<td>739.1 ± 71.2</td>
</tr>
<tr>
<td>135</td>
<td>442.8 ± 60.1</td>
<td>614.1 ± 79.2</td>
<td>0.65 ± 0.06</td>
<td></td>
</tr>
<tr>
<td>Difference (P)</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td>Second experiment (with cooling)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>90</td>
<td>343.4 ± 56.1</td>
<td>452.7 ± 78.9</td>
<td>0.77 ± 0.06</td>
<td>719.8 ± 99.2</td>
</tr>
<tr>
<td>135</td>
<td>462.1 ± 69.1</td>
<td>644.8 ± 99.1</td>
<td>0.66 ± 0.06</td>
<td></td>
</tr>
<tr>
<td>Difference (P)</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td></td>
</tr>
</tbody>
</table>

P20 and P50 – muscle contraction force evoked by stimulating quadriceps muscle at 20 Hz and 50 Hz frequencies. MVCF – maximum voluntary contraction force.

### 3.5. Does muscle cooling after SSE have the same effect on time-course of recovery of electrostimulation induced muscle force and neuromuscular performance?

The results presented in Fig. 6 show that the effect of muscle cooling on the recovery of muscle force electrostimulation induced by and neuromuscular performance does not manifest itself immediately after cooling: only at 24 h after SSE a significant effect of cooling on the recovery of H of DJ, MVCF, P20 and P50 at SL is observed (\(P < 0.05\), with these indicators’ values between experiments 1 and 2 being compared).

### 3.6. Effect of cooling after SSE on dynamics of CK and muscle soreness

The results of the research indicate that CK activity in blood registered at 24 h and 48 h after SSE was considerably smaller in the case of experiment 2 than after SSE in experiment 1 (\(P < 0.05\) and \(P < 0.001\)) (Fig. 3B). Besides, a similar effect of cold water immersion on muscle soreness was observed, i.e. at 24 h, 48 h and 72 h after SSE it was significantly smaller in the case of experiment 2 than during experiment 1 (\(P < 0.001\)) (Fig. 3A).

### 4. Discussion

The main findings in this study are as follows: 1) cold water immersion after SSE accelerated the disap-
Fig. 2. Time course of changes in the height of drop jumps (A) and maximal voluntary contraction force (MVCF) (B) (mean±SD) after SSE. SSE – stretch-shortening exercise (100 drop jumps performed from 0.75 m with counter-movement to 90 degrees in the knee with immediate maximal rebound, with 20 s interval between the jumps). * - $P<0.05$ and ** - $P<0.001$ – compared to initial level; † - $P<0.05$ and †† - $P<0.001$, between experiments I and II.

4.1. Main causes of fatigue origin during and after SSE

Neuromuscular fatigue induced during the experiment is likely not to be attributed to an increase in the myoplasm of metabolites like phosphate and hydrogen ions, since the duration of the jump (0.3–0.4 s) was too small for ATP and phosphocreatine (PCr) to be decreased significantly while the resting period of 20 s was sufficient for ATP and PCr to be restored. The causes of muscle fatigue when performing exercises of this type (stretch-shortening exercise, SSE) are therefore associated with non-metabolic factors, most likely having something to do with muscle damage [14, 21,22,37]. Exercise-induced skeletal muscle damage after performing SSE in the case of our experiments shows the manifestation of indirect indicators of muscle damage within 24–72 h after load; the rise of muscle soreness, the increased CK activity (Fig. 3A, B), prolonged impairment of neuromuscular performance (H and MVCF) (Fig. 2A, B), as well as the muscle force evoked by low and high electrostimulation frequencies (Fig. 4A, B, C, D). The non-metabolic cause of the rise of muscle fatigue is supported by the fact that LFF manifested itself in the muscle immediately after SSE and it remained about 24 h after the load (Fig. 5). Besides, immediately after SSE, LFF was greater at SL than at LL ($P<0.05$).
Fig. 3. Muscle soreness (A) and CK (B) (mean ± SD) before and after SSE in the first (I) and second (II) experiments. SSE – stretch-shortening exercise (100 drop jumps performed from 0.75 m with counter-movement to 90 degrees in the knee with immediate maximal rebound, with 20 s interval between the jumps). **– *P* < 0.001 – compared to initial level; † – *P* < 0.05 and †† – *P* < 0.001, between experiments I and II.

Exercise-induced muscle damage in our case is supported by the findings of other authors showing that well documented indicators of muscle damage include prolonged impairment of muscle function measured during both voluntary and electrically stimulated contractions [4,41], protein leakage from injured muscle fibres, acute inflammation reaction and delayed-onset of muscle soreness, stiffness and swelling [2,7,24,29]. According to Warren et al. [41], one of the most informative criteria of muscle damage is decrease in muscle force. The extent of isometric strength loss after exercise induced muscle damage, however, is dependent on the muscle length or joint angle at which it is measured, being more pronounced at short muscle lengths [4,6,33,36]. Therefore a greater decrease in muscle contraction force at SL than at LL might be one of the indicators of muscle damage. This has been observed by us immediately after SSE too (Fig. 6).

Low-frequency fatigue 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 [10,18,34,35,42]. In our case there was a decrease in the force evoked not only by low stimulation frequencies (20 Hz) (Fig. 4A, B) but by high stimulation frequencies (50 Hz) as well (Fig. 4 C, D). It is but rarely that one can find physical load capable of bringing about a decrease in the force evoked by low stimulation frequencies since in nearly all cases there is a greater or smaller decrease in the force evoked by high stimulation frequencies too [25,28,34,38].

It has been shown 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 [12,14,21,38]. This is in accord with our findings since there was significant decrease (*P* < 0.05) in H and MVCF (Fig. 2A, B) from the end of the SSE until 48 h in the case of I experiment.

Notwithstanding the obvious fact that 100 drop jumps performed every 20 s with maximum intensity bring about muscle damage not only the damage mechanism itself but also its effect on the time-course of muscle function and neuromuscular performance remains to be cleared up. The clarification of this phenomenon is complicated by the fact that decrease in muscle and neuromuscular function during and after SSE is a complex process that depends on a great number of interrelated factors that often are very difficult to consider separately. There is no doubt that not only muscles but also mechanisms of central motor control participate in this process.

4.2. Why muscle cooling decreased the manifestation of indirect indicators of skeletal muscle damage?

This is the first study devoted to the analysis of the effect of leg immersion in cold water both on CK and muscle soreness and on the force evoked by different stimulation frequencies at different muscle length, as well as neuromuscular performance. There exists a considerably great number of publications devoted to the study of muscle cooling effect on CK, muscle pain and swelling, muscle stiffness and voluntary generated muscle force [9,11,17,23,26,31,40].
Fig. 4. Time course of changes in P20 and P50 at muscle short length (SL) and long length (LL) (mean ± SD) after SSE. SSE – stretch-shortening exercise (100 drop jumps performed from 0.75 m with counter-movement to 90 degrees in the knee with immediate maximal rebound, with 20 s interval between the jumps). * – $P < 0.05$ and ** – $P < 0.001$ – compared to initial level; † – $P < 0.05$ and †† – $P < 0.001$, between experiments I and II. P20 and P50 – muscle contraction force evoked by stimulating quadriceps muscle at 20 Hz and 50 Hz frequencies.

We did not come across any publications, however, devoted to the study of the effect of muscle cooling on the muscle force evoked by electrostimulation and on the drop jump performance. Besides, most of the research has been done applying methods of ice cooling, whereas method of the leg immersion in cold water has been used but by very few authors [11,31].

The results obtained by us that the cooling of the muscles decreases the manifestation of indirect indicators of exercise-induced muscle damage coincide with research done by other scientists who have also reported a positive effect of cooling on the manifestation of certain indicators [9,11,15,23,40].

It has been shown that various models of applying ice are used routinely in clinical practice to provide pain relief, to diminish inflammatory responses, and reduce swelling for numerous types of injuries [11,23,40]. It was concluded that although cold water immersion may reduce muscle stiffness and the amount of post-exercise damage after strenuous eccentric exercise, it had no effect, however, on changes in muscle force [11]. A similar conclusion has been made by Howatson and Van Someren [15], namely, that ice massage reduces the appearance of CK and it has no other effect on indicators associated with exercise-induced muscle damage (muscle soreness and muscle force). It is possible that the lack of agreement between studies may be attributable to the nature of cryotherapy procedure and the timing of its application.

Contrary to the results of our research, however, Eston and Peters [11] have not observed any faster recovery (they have observed but positive tendency) in the voluntary isometric muscle force due to cold water immersion. These differences might be accounted for by the fact that in our case the same subjects participated in both experiments, as compared to different groups in
their case; our protocol of cooling was concentrated on the first 24 h after exercise, whereas they have spread out a similar dose of cooling through 72 h. Besides, the load that brought about muscle damage in our case was greater than in theirs; we studied leg muscles and they – elbow flexors muscles.

Our findings contradict previous observations [5, 17, 43]. This disagreement may be due to variation in applying the methods used. The form of cryotherapy used in this study was essentially different from that in previous studies, which used direct application, an ice-massage on the skin. We think that immersion in cold water allows for the possibility of greater temperature control. Although our findings generally contradict previous observations it is possible that the repeated treatments applied in this study might provide a valuable insight into the effects that cryotherapy may have on some of the functional consequences of exercise-induced muscle damage.

We have not come across any publications devoted to the study of the effect of cooling on the disappearance of muscle LFF. At first we thought that cooling should accelerate the disappearance of LFF since the origin of LFF is associated with processes similar to exercise-induced muscle damage. It is due to muscle damage that there occurs calcium removal from sarcoplasmic reticulum as well as disruption of sarcomeres and these processes do not recover for a long time [2, 7, 33]. Besides, muscle force evoked by low stimulation frequencies is greatly dependent on these processes [1, 18, 34, 38, 42]. We thought that in case cooling inhibited, for example, secondary damage (during recovery period) of muscle membrane and sarcoplasmic reticulum, as well as secondary decrease of muscle protein degradation, consequently, there should be a faster disappearance of LFF. The results of our research, however, did not show this (Fig. 5). Since the effect of cooling was significant for the force evoked by both low and high stimulation frequencies, thus it did not affect the P20/P50. This indicates that muscle cooling effect on the recovery of electrostimulation induced muscle force does not depend either on stimulation frequency or on muscle length. It remains for scientists to clear up what mechanisms were of the greatest importance in this process.

It is suggested that the main cryotherapy mechanisms are aimed at reducing secondary tissue injury [10, 16, 20]. Retarding secondary injury is an important theoretical benefit of cryotherapy [27]. This theory is supported by the results of our research too since due to cold water immersion there occurred decrease not only in CK and muscle soreness but also in P20 and P50 registered within 24–72 h after terminating the SSE (Figs 4A, B, C, D and 6). Besides, there was no manifestation of secondary decrease in H of DJ and MVCF either (Fig. 2A, B).

5. Conclusions

In conclusion, the main findings in this study are as follows: 1) cold water immersion after SSE accelerated the disappearance of indirect indicators of muscle damage (CK in blood, muscle soreness, decrease in muscle force and neuromuscular performance); 2) cooling did not bring about any changes in the dynamics of LFF disappearance; 3) cooling effect did not manifest itself immediately but within 24–72 h following the SSE. All this is indicative of the fact that notwithstanding the positive effect of leg cold water immersion on the disappearance of indirect indicators of stretch-shortening exercise-induced muscle damage it still remains for scientists to clear up the mechanisms of the effect on the different indicators of exercise-induced muscle damage.

References

Fig. 5. Mean values in per cent compared to pre-exercise values of P20/P50 immediately (0), 4, 8, 24, 48 and 72 h after SSE exercise (mean ± SD). * – P < 0.05 – compared to initial (average P20/P50) level. P20 and P50 – muscle contraction force evoked by stimulating quadriceps muscle at 20 Hz and 50 Hz frequencies. SSE – stretch-shortening exercise (100 drop jumps performed from 0.75 m with counter-movement to 90 degrees in the knee with immediate maximal rebound, with 20 s interval between the jumps). 135 I, 135 II, 90 I and 90 II – P20/P50 registered at knee angle 135 during the first and second experiment and at 90 knee angle during the first and second experiment respectively.

Fig. 6. Mean values (%) in influence of leg muscle cooling on time course of recovery height of drop jumps (H), maximal voluntary contraction force (MVCF), muscle force induced by electrostimulation at low (20 Hz) and high (50 Hz) frequencies at knee angle 90 and 135 degrees (P20 90, P20 135, P50 90 and P50 135 respectively). * – P < 0.05 compared to values at 4 h after SSE. SSE – stretch-shortening exercise (100 drop jumps performed from 0.75 m with counter-movement to 90 degrees in the knee with immediate maximal rebound, with 20 s interval between the jumps). According to the difference in per cent between non cooling values and cooling values the cooling effect was calculated.


