ISSN 0362-1197, Human Physiology, 2015, Vol. 41, No. 1, pp. 75–82. © Pleiades Publishing, Inc., 2015.
Original Russian Text © A.D. Minigalin, A.R. Shumakov, A.V. Novozhilov, A.V. Samsonova, E.A. Kosmina, M.I. Kalinski, T.I. Baranova, I.V. Kubasov, V.I. Morozov, 2015, published in Fiziologiya Cheloveka, 2015, Vol. 41, No. 1, pp. 89–98.

Effect of Exhaustive Weightlifting Exercise on the Maximal Isometric Force, Electromyogram Parameters, Muscle Pain, and Biochemical Markers of Muscle Damage[1]

A. D. Minigalin[a], A. R. Shumakov[b], A. V. Novozhilov[b], A. V. Samsonova[c], E. A. Kosmina[c], M. I. Kalinski[d], T. I. Baranova[a], I. V. Kubasov[b], and V. I. Morozov[b]

[a] *St. Petersburg State University, St. Petersburg, Russia*
[b] *Sechenov Institute of Evolutionary Physiology and Biochemistry, Russian Academy of Sciences, St. Petersburg, Russia*
[c] *Lesgaft National University of Physical Training, Sports, and Health, St. Petersburg, Russia*
[d] *Murray State University, Murray KY, USA*
e-mail: wolverine.surgut@mail.ru

Received January 14, 2013

Abstract—The effect of exhaustive weightlifting exercise (WE) on the time-related changes in performance capacity was studied along with measuring several physiological and biochemical variables during exercise. The work performed decreased soon after the start of exercise and stabilized after reducing the amount of weight used (40–10% 1RM). The maximal amplitude of the surface electromyogram (EMG) of m. rectus femori strongly tended to increase in the first half of WE and stabilized at the end of WE. WE substantially increased the blood plasma lactate level, the myoglobin concentration grew twice as high, while creatine kinase (CK) activity remained unchanged. It was assumed to explain the observed decrease in performance capacity that fast motor units (MUs) progressively refuse to work, while weaker intermediate and slow MUs continue working. Unchanged CK activity and an insignificant increase in plasma myoglobin suggested only minor, if any, WE-induced damage to myocyte membranes in the subjects.

Keywords: weightlifting exercise, performance capacity, m. rectus femoris, electromyogram, blood plasma, creatine kinase, myoglobin

DOI: 10.1134/S0362119714060073

INTRODUCTION

Fatigue developing during exercise changes the physiological and biochemical characteristics of muscle tissue and is seen as an alteration of the movement kinematics, pattern, and efficiency and a decrease in maximal voluntary contraction [1, 2]. When working intensely, a muscle becomes hypoxic because of insufficient blood supply, and hypoxia activates glycolysis and facilitates an accumulation of lactic acid. Acidification of the sarcoplasm leads to a less efficient function of glycolysis enzymes, reduces the ATP production, and impairs the contractile function [2–4]. Delayed-onset muscle soreness (DOMS) may develop during the recovery from highly intense, mostly eccentric, exercise, being similarly associated with a decrease in the strength of muscle contraction [5–7]. DOMS is presumably determined by a set of responses to damage, including edema and the inflammatory response, which develops in the muscle and involves leukocytes [8–11]. Studies in humans [2, 12–16] and animals [17] have shown that electrical parameters of muscles deteriorate in DOMS, reflecting a poorer functional state and a decrease in the maximal strength of contraction. Along with DOMS, muscle proteins leak into circulation because of damage to the myocyte membrane [18]. At the same time, DOMS poorly reflects the extent of damage to the muscles from eccentric exercise, and changes in indirect markers of this damage are not necessarily accompanied by DOMS [19].

Thus, there is a discrepancy in the available data on the relationships of the strength of muscle contraction, DOMS, and biochemical markers of exercise-associated muscle damage. The discrepancy possibly arises because different models of exercise and different durations of the observation period are used in relevant studies. We have previously described how exhausting weightlifting exercise (WE) affects the recovery dynamics of physiological (the amplitude and frequency on the interference electromyogram (iEMG), muscle tone, and DOMS severity) and biochemical (lactate and creatine kinase (CK)) parameters characterizing the state of muscles [20].

[1] This article is devoted to V.I. Morozov, Dr. Sci., who have made invaluable contribution to the formation of this field of research.

A decrease in iEMG frequency has been observed immediately and 3 days after WE with DOMS developing and CK leaking into circulation during a postexercise recovery period. It remained unclear how WE affects the maximal strength of contraction as an integral parameter of the functional state of muscles.

The objectives of this work were to study the acute and delayed effects of exhausting WE involving the knee extensor muscles on the maximal isometric force (MIF) in correlation with iEMG characteristics of the functional state of the muscles, DOMS, and biochemical markers of muscle damage.

METHODS

We examined nine healthy males aged 19–23 years 20.1 ± 1.45 years, $X \pm SD$), who were informed about the nature of the study and voluntarily gave their consent to participate. The study procedures were approved by the Ethics Committee at the St. Petersburg State University and were in agreement with the Declaration of Helsinki. Prior to the study, the subjects were examined by a therapist and found to have no contraindications to taking part in the study.

We used a leg extension device (Technogym, Italy) for knee extensor muscle training in the sitting position. To evaluate the exercise load, the one repetition maximum (1RM) was estimated by trial and error, understanding 1RM as the maximal weight that the subject can lift only once on a given device. The subjects were evaluated for their 1RM 14 days before the test WE session, and the weights corresponding to 80, 70, 60, 50, 40, 30, 20, and 10% of 1RM were calculated. The session was preceded by a 5-min warm-up, which included squatting and bending. During the session, a subject sat on the device (in the initial position, the legs were relaxed, the knee joint was bent at 90°, and the roller was over the ankle joints) and straightened the legs until the angle at the knee joint was 170°–180°. The session consisted in working with each weight until failure. The exercise was stopped when the subject failed to straighten the legs to 170°–180° at the knee joint three times. The exercise was continued after 1-min rest, the weight being changed during this period. The subjects did the exercise at an average rate of 25–30 extensions per minute. The weight was decreased in a stepwise manner from 80 to 10% 1RM. The exercise was designed to completely fatigue the working muscle groups.

Finger-prick blood samples were collected prior to and immediately after the WE session and then 1 h and 1, 2, 3, 5, 7, and 9 days after the session in Microvette 500 EDTA tubes (Sarstedt, Germany), which contained an anticoagulant. The samples were centrifuged, and the plasma and erythrocytes were divided into aliquots and stored at –80°C.

MIF of the knee extensor muscles was measured using a DOR-3 dynamometer (PetVes, St. Petersburg) prior to and immediately after the WE session and then 1 h and 1, 2, 3, 5, 7, and 9 days after the session. A baseline MIF measurement was carried out 1 h before the WE session. A subject sat on the device in the initial position and tensed his knee extensor muscles to apply a maximal force to a constant-length steel bar, which was fixed rigidly with one end to the frame of the device and connected with the other end to a dynamometer and a tensiometer with an electronic display showing the isometric force developed. Prior to a measurement, an angle of 90° at the knee joint was checked using a laser level gage. Because the change in length was no more than 2 mm for the tensile element and was negligible for the steel bar, the muscle contraction involved was considered to be isometric (without any appreciable change in muscle length or any movement at the knee joint).

A surface iEMG was acquired using a MIOKOM electromyograph (RITM, Taganrog) with adhesive concentric skin silver/chloride electrodes ($d = 10$ mM). Prior to applying the electrodes, hairs were shaved, and the skin wiped with an ethanol swab. To record an iEMG in a monopolar configuration, an active electrode was placed over the motor point of m. vastus lateralis, and a reference electrode was attached over the tibial bone prominence of the same leg. Technical parameters of the electromyograph were as follows: maximal input voltage, 2 mV; input signal (3 dB) frequency range, 20–500 Hz; internal noise referred to input, no more than 0.3 μV effective value; EMG envelope averaging time, 10 ms; sampling rate, 200 Hz for each channel; analog-to-digital conversion, 16 bit. The iEMGs were processed using StabMed 2 software to calculate the iEMG maximal amplitude (MA).

To estimate the muscle pain severity, the subjects were asked to report their sensations during the total study. Pain in the knee extensor muscles was evaluated using a set of motor tests (a single squat, walking down stairs (10 steps), and muscle palpation). The pain severity was estimated using the modified Borg CR10 scale for each test, and the average score was calculated for each of the subjects.

The lactate concentration and CK activity were measured using Olvex Diagnosticum kits (St. Petersburg), and the myoglobin concentration was assayed using a DRG Instruments kit (Marburg, Germany) as recommended by the manufacturers.

A statistical analysis was carried out using Statistica 6.0 and IMB SPSS 19 software. The median, mean, and standard deviation were calculated. Differences were tested for significance by the t and Mann–Whitney tests and were considered significant at $p < 0.05$.

RESULTS

The mean amount of work performed by the subjects was 23964 ± 5819 (15 675–32 482) J, and its duration was 10.46 ± 2.53 min (7.16–15.22 min) ($X \pm SD$).

On average, the amount of work performed reached its maximum at 80% 1RM, then decreased significantly ($p < 0.05$) to a minimum at 50–30%

1RM, and then increased again at 20–10% 1RM (Fig. 1a). The greatest variation of the amount of work was observed at 80 and 10% 1RM. A more detailed analysis allowed us to divide the group into two subgroups by the amount of work performed. Subgroup 1 subjects ($n = 4$) performed a greater amount of work with lower weights, while subgroup 2 subjects ($n = 5$) displayed a decrease in the amount of work by the middle of the working cycle and a stabilization at 50–10% 1RM (Fig. 1b). The total amount of work performed was 29 018 J in subgroup 1 and 18 968 J in subgroup 2.

On average, MIF measured for the knee extensor muscles immediately after the WE session was 53% of its baseline value (Fig. 2).

The parameter was restored quickly during the first day after WE, reaching 71% of its baseline value. Then the restoration slowed down so that MIF remained virtually the same from day 2 to day 5. MIF started to increase again afterwards, but was still 18% below the baseline level 7 days after WE. Subgroups 1 and 2 did not significantly differ in MIF.

An EMG analysis showed a substantial variation of m. vastus lateralis iEMG MA measured during maximal voluntary contraction (MIF measurement) or exercise at high (80–70% 1RM) loads (Fig. 3). The finding suggested heterogeneity for the subject group (the response of the subjects' neuromuscular system to loading). The parameter stabilized only at 50% 1RM (with a distinct decrease in the amount of work performed).

As averaged over the subject group, the iEMG MA did not significantly differ in a range of 80–30% 1RM. A significant ($p < 0.05$) decrease in iEMG MA was observed only at 20 and 10% 1RM as compared with exercise at high loads. The median iEMG MAs were 0.80 mV at 80% 1RM, 0.76 mV at 70 1RM, 0.68 mV at 60% 1RM, 0.65 mV at 50% 1RM, 0.67 mV at 40% 1RM, 0.61 mV at 30% 1RM, 0.56 mV at 20% 1RM, and 0.62 mV at 10% 1RM. The iEMG MA measured after the WE session was significantly lower ($p < 0.03$) only than the iEMG MAs recorded at 80–60% 1RM. Only minor changes in iEMG MA were observed during postexercise recovery (Fig. 3).

The blood lactate concentration showed a threefold increase after the WE session (from 3.7 to 10.6 mmol/L, $p < 0.01$). CK activity was 3.6 times higher than the baseline 1 day after the WE session and 7.1 times higher than the baseline 3 days after the session (Fig. 4a).

Maximal CK activity was detected 5 days after the WE session, increasing over the baseline by a factor of 8.3. CK activity remained substantially higher than the baseline even 9 days after the WE session. The myoglobin concentration in the blood plasma changed quicker than CK activity, being significantly higher than the baseline immediately after the WE session (Fig. 4b). Then the myoglobin concentration gradually increased and reached its maximum (23 times that at the baseline) 3 days after the session. On day 9, the myoglobin concentration was still significantly higher

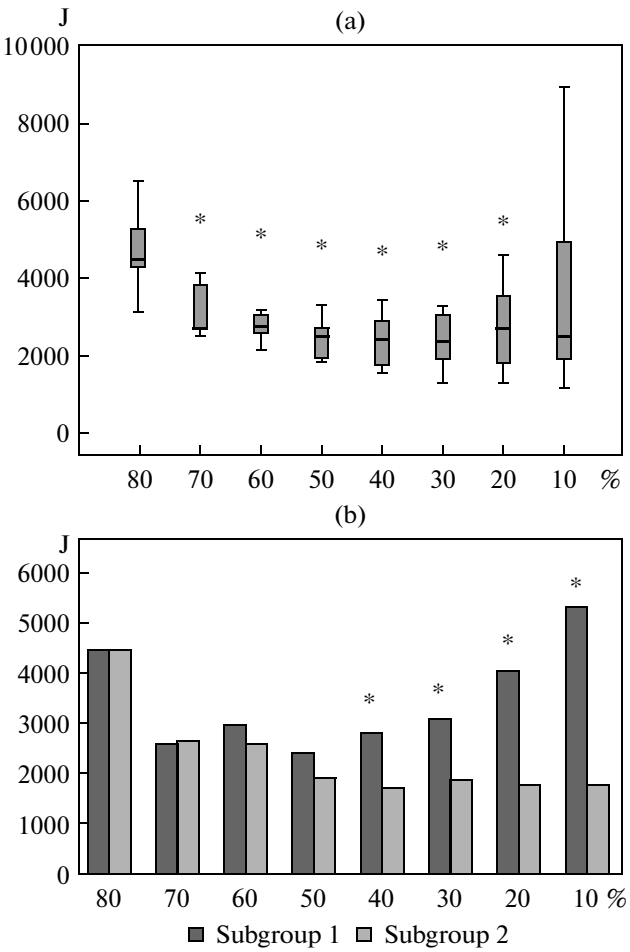

Fig. 1. (a) Amount of work (J) performed by the subjects and (b) its distribution between the subgroups. Differences were significant at (*) $p < 0.05$. Abscissa, load, % 1RM. Ordinate, work, J.

than the baseline. Subgroups 1 and 2 did not significantly differ in lactate or myoglobin concentrations.

The restoration period was characterized by pain development in the knee extension muscles from day 1 after the WE session. The highest pain severity was recorded 2 days after the WE session. On day 9, the subjects did not complain of muscle pain. Significant differences were not observed between subgroups 1 and 2.

DISCUSSION

Performance capacity. The amount of work performed by the subjects was maximal at the greatest load (80% 1RM), decreased significantly at lower loads, and stabilized at 50% 1RM to remain the same to the end of the WE session. The greatest and lowest variations of the parameter were observed at 80% and 10% 1RM, respectively. We analyzed the causes of the variation and observed that the subject group distinctly tended to segregate into two subgroups. Subgroup 1 subjects performed a greater amount of work (55%) at

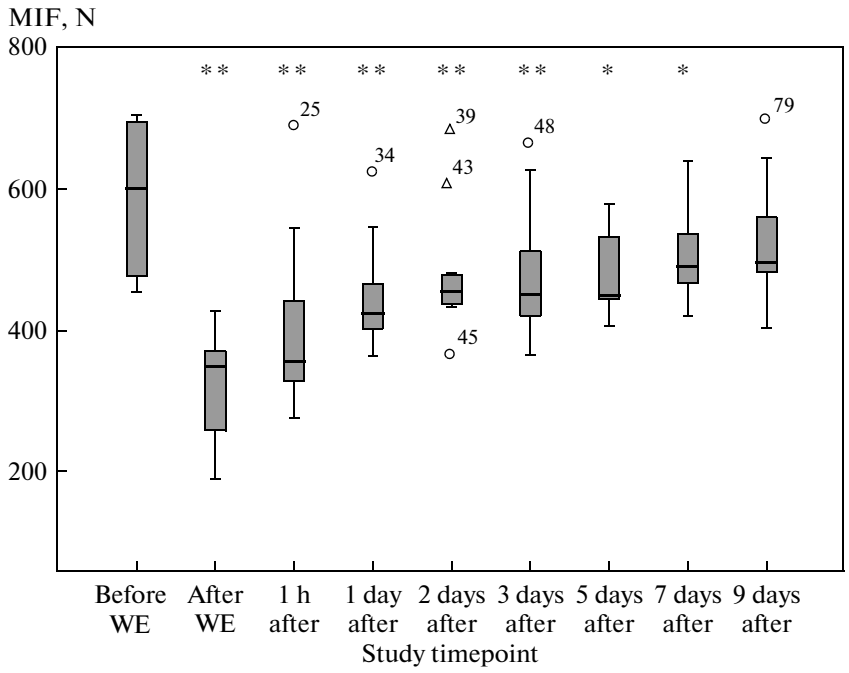

Fig. 2. Changes in maximal isometric force (MIF, N) during recovery. Differences were significant at (*) $p < 0.05$ or (**) $p < 0.01$. $\circ^{45}$, abnormal value, which is 1.5–3 times the interquartile range (Q_1–Q_3) below quartile 1 or above quartile 3; $\triangle^{43}$, extreme abnormal value, which is more than 3 times the interquartile range (Q_1–Q_3) below quartile 1 or above quartile 3. Abscissa, time. Ordinate, MIF. WE, weightlifting exercise session.

40–10% 1RM, while subgroup 2 subjects performed a major portion of work (65%) at 80–50% 1RM. The total amount of work performed by the subgroup 1 subjects was 1.5 times higher than that in subgroup 2. The difference in performance capacity between subgroups 1 and 2 might be due to a difference in muscle fiber composition.

According to current views, numerous muscle fibers of both type I and type II are recruited while working at maximal loads (80–70%), when strength generation is of importance, while the duration and power of exercise at lower loads (20–10% 1RM) depend on the amount of type I and type IIa muscle fibers [21]. Hence, a greater content of slow type I and type IIa fibers can be assumed for the subgroup 1 subjects compared with the subgroup 2 subjects. Type II muscle fibers are especially important for producing force by a muscle. In particular, this is true for m. vastus lateralis, which is one of the anterior surface muscles of the thigh [22]. Human type IIa muscle fibers produce a far greater force as compared with type I fibers [23]. Studies with animals (rats) have shown that a decrease in force generation in vitro is grater for fast muscle fibers and lower for slow fibers [24]. At the same time, we did not observe any significant difference between subgroups 1 and 2 apart from the difference in performance capacity. This circumstance was possibly related to the exhaustive character of the WE session.

Acute decrease in muscle force. MIF dropped to 53% of its baseline value at the end of the WE session. At least a threefold increase in blood plasma lactate was observed after the session. A decrease in pH due to lactate accumulation in muscles affects the muscle function in several ways. A H^+ accumulation with increasing lactate concentration can accelerate fatigue by changing the calcium–troponin association (inhibition of the excitation–contraction coupling); altering the function of the Na^+/K^+ pump (a decrease in excitability and development of neuromuscular fatigue); and suppressing glycolysis via inhibiting phosphofructokinase, which is one of the main glycolysis enzymes [25–27]. In addition, a lactate accumulation can prevent actin–myosin crossbridging as a result of actomyosin ATPase inhibition, thus reducing the force generation [28, 29].

Lack of significant differences in iEMG MA between the tests at MIF measurement and exercise at 80–30% 1RM could be explained by the fact that the subjects worked up to failure at each load; i.e., all possible reserves were involved to produce the maximal muscle force. Accordingly, the iEMG MA increased to a maximal extent. In contrast, individual motor units (MUs) were recruited in an alternating manner at lower loads (20–10% 1RM), and the iEMG MA consequently decreased. A drop in iEMG MA at 50% 1RM was probably related to fatigue and exclusion of type IIb MUs from working. The assumption is supported by the finding that the amount of work performed decreased relative to exercise at higher loads (80–70% 1RM). The same factor could similarly determine the drop in iEMG MA seen immediately

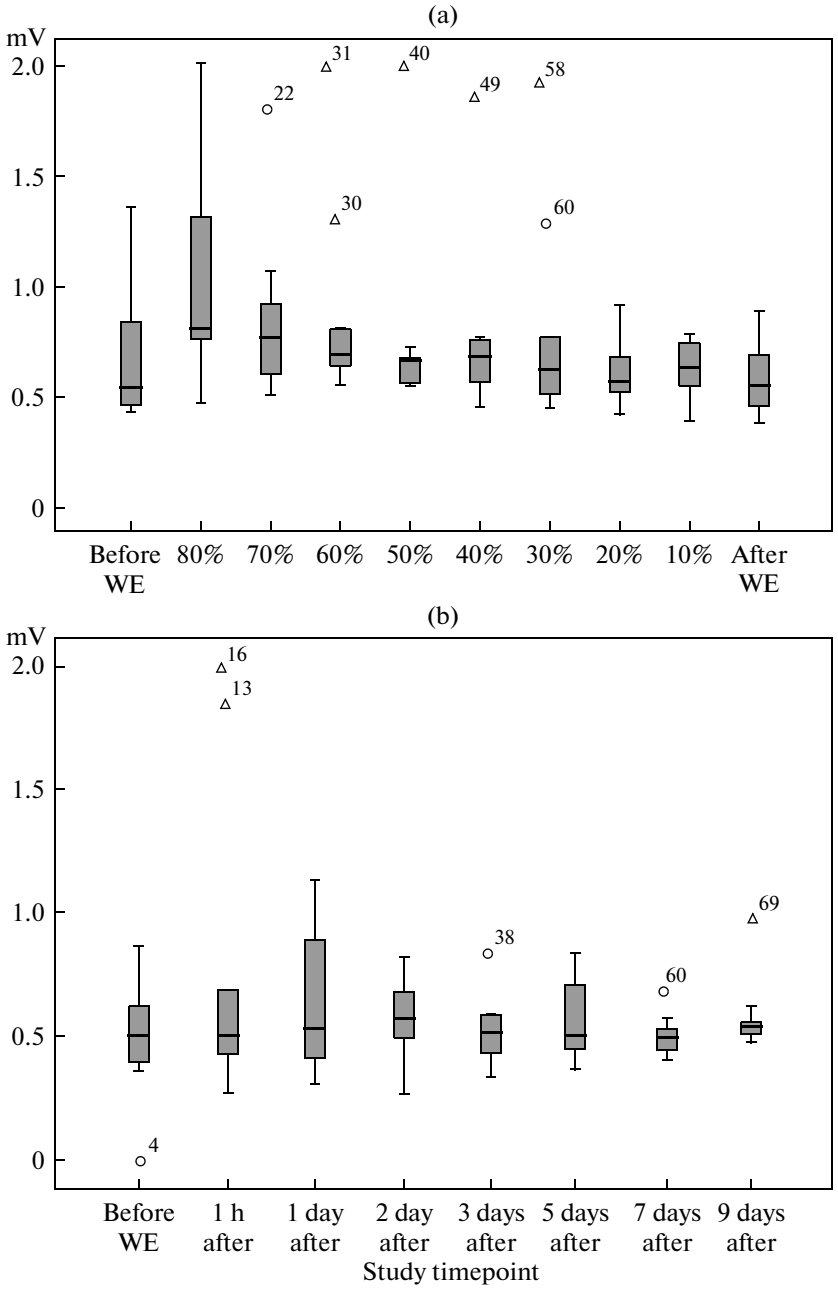

Fig. 3. iEMG maximal amplitude (mV) during (a) exercise and (b) recovery. Abscissa, (a) load, % 1RM or (b) study timepoint. Other designations are as in Fig. 2.

after the end of the WE session relative to WE at higher loads. Thus, the EMG results showed that the muscle under study was substantially fatigued during the WE session, as was necessary for our experimental model.

A decrease in EMG frequency, which is another indicator of substantial muscle fatigue, has also been observed after the end of exercise in a similar WE model [20].

Restoration of muscle force after loading. A significant increase in MIF, up to 71% of its baseline value, was observed 1 day after the WE session. It is possible to assume that the rapid partial restoration of the contractile function was due to metabolite elimination and normalization of pH and the levels of macroergic compounds and neuromediators in intact fibers. The MIF restoration rate decreased afterwards, and MIF remained almost unchanged (75–76%) from day 2 to day 5 days after the WE session. An increase in MIF (+7%) was detected only on day 7. The beginning of the period when MIF restoration decelerated was characterized by the highest pain severity, which was observed 2 days after the WE session. Then, muscle pain gradually decreased and virtually disappeared by the end of the observation period. Deceleration of

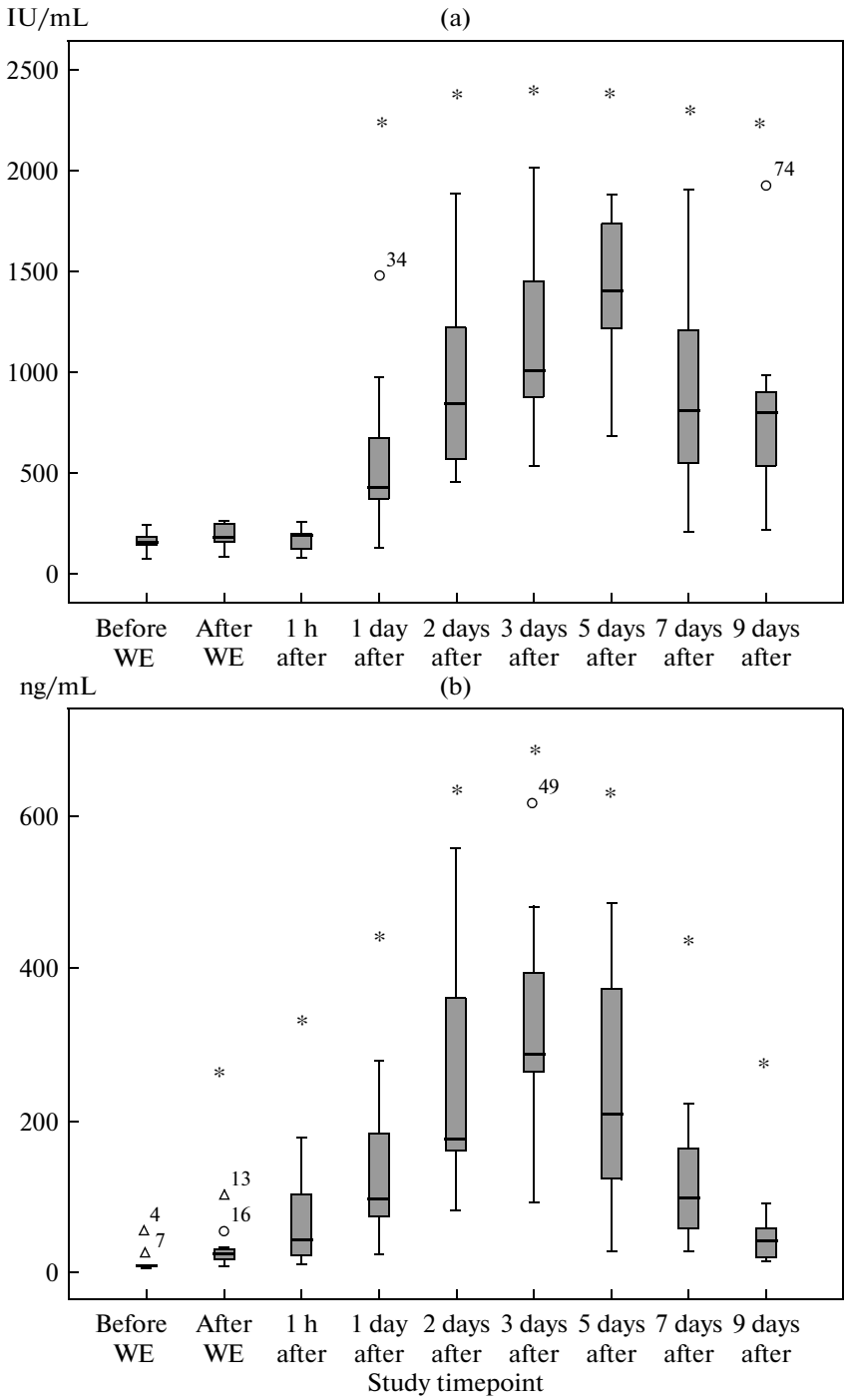

Fig. 4. Changes in (a) creatine kinase activity (CK, IU/mL) and (b) myoglobin concentration (ng/mL) during recovery. Differences were significant at (*) $p < 0.05$. Abscissa, study timepoint. Other designations are as in Fig. 2.

MIF restoration partly coincided with an increase in plasma myoglobin and CK, which are markers of muscle damage. The changes in muscle pain were seen earlier than the changes in damage markers, indicating that DOMS-initiating events occur earlier than certain biochemical mechanisms are triggered to damage muscle fibers. It is possible to assume that DOMS is underlain by (1) edema, which activates the peripheral pain receptors and causes muscular soreness, and (2) subsequent inflammation, which involves leukocytes [7, 11, 30]. There is evidence that an increase in muscle tone after WE is associated with edema developing in fatigued muscles [31]. An increase in muscle tone with substantial fatigue has similarly been observed in our previous studies [20]. Progressive damage to muscle fibers (development of a catabolic phase) as a result

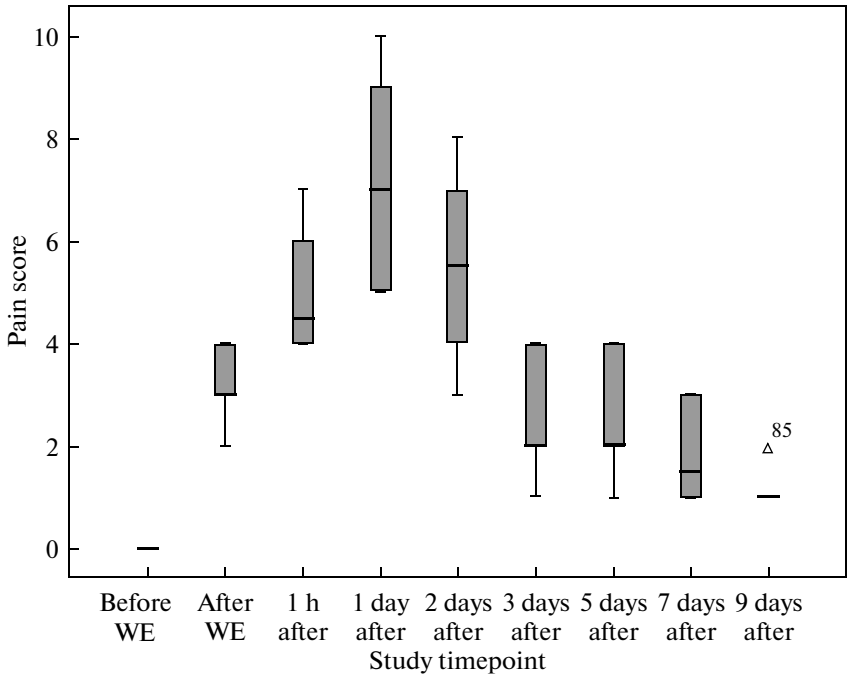

Fig. 5. Changes in muscle pain during recovery. Differences were significant at (*) $p < 0.05$. Abscissa, study timepoint. Ordinate, pain score. Other designations are as in Fig. 2.

of triggering certain biochemical mechanisms (activation of calpains, cyclooxygenase 2, and lipid peroxidation) is accompanied by an increase in damage markers in the blood and further decelerates the restoration of MIF. A mass leakage of muscle proteins is indicative of substantial damage to the myocyte membrane [32–36]. Deterioration of the muscle functional state is evident from a drop in iEMG frequency (with iEMG amplitude and MIF being unchanged) [20] and a decrease in EMG power spectrum frequency during the same period [2]. A further contribution to delaying the MIF restoration can be made by the fact that neuromuscular transmission remains less efficient as long as the myocyte membrane is not completely restored.

The finding that MIF starts increasing again afterwards can be mediated by a partial restoration of contractile structures during the anabolic restoration phase (supercompensation) [37–39]. The blood level of muscle proteins still remains elevated, indicating that restoration of the myocyte membrane is incomplete.

CONCLUSIONS

Our findings demonstrate that the contractile capacity of muscles is restored in a nonlinear manner after exhausting WE. The dynamic changes in MIS can be related to several factors, which act consecutively.

(1) Acidification of the sarcoplasm, depletion of macroergic compound and neuromediator pools, and mechanical damage to muscle fibers are responsible for an acute decrease in MIF, which is observed immediately after the end of exercise. It can be assumed that a partial restoration of MIF is due to a restored balance of macroergic compounds and neuromediators (a rapid increase in MIF) and a restored pH balance due to elimination of lactic acid.

(2) Edema and inflammation, which underlie DOMS and damage muscle fibers, are another factor (a decrease in MIF restoration on days 2–5 after exercise).

(3) Restoration of contractile structures occurs next (MIF starts to increase again). The observed individual variation in performance capacity was possibly determined by the typological features of our subjects and the resistance of muscle fibers to a damaging effect of exercise.

ACKNOWLEDGMENTS

We are grateful to G.P. Vinogradov (head of the Department of the Theory and Methodology of Weight Training, Lesgaft National University of Physical Training) for the possibility to use the experimental facilities of the department and to A.V. Lozyanko and S.N. Zabavin (engineers of the same department) for technical assistance in our study.

This work was supported by the Russian Foundation for Basic Research (project no. 13-04-00509).

REFERENCES

1. Finni, T., Kyröläinen, H., Avela, J., and Komi, P.V., Maximal but not submaximal performance is reduced by constant-speed 10-km run, *J. Sports Med. Phys. Fitness*, 2003, vol. 43, p. 411.
2. Hedayatpour, N., Falla, D., Arendt-Nielsen, L., and Farina, D., Effect of delayed-onset muscle soreness on

muscle recovery after a fatiguing isometric contraction, *Scand. J. Med. Sci. Sports*, 2008, vol. 20, p. 145.
3. Maughan, R. and Gleeson, M., *The Biochemical Basis of Sports Performance*, New York: Oxford Univ. Press, 2004.
4. Proske, U., Gregory, J.E., Morgan, D.L., et al., Force matching errors following eccentric exercise, *Hum. Mov. Sci*, 2004, vol. 23, p. 365.
5. Cheung, K., Hume, P., and Maxwell, L., Delayed onset muscle soreness: Treatment strategies and performance factors, *Sports Med.*, 2003, vol. 33, p. 145.
6. Connolly, D.A.J., Sayers, S.P., and McHugh, M.P., Treatment and prevention of delayed onset muscle soreness, *J. Strength Cond. Res.*, 2003, vol. 17, p. 97.
7. Coudreuse, J.M., Dupont, P., and Nicol, C., Delayed post effort muscle soreness, *Ann. Readapt. Med. Phys.*, 2004, vol. 47, p. 290.
8. Pyne, D.B., Exercise-induced muscle damage and inflammation: A review, *Aust. J. Sci. Med. Sport*, 1994, vol. 26, p. 49.
9. MacIntyre, D.L., Reid, W.D., Lyster, D.M., et al., Presence of WBC, decreased strength, and delayed soreness in muscle after eccentric exercise, *J. Appl. Physiol.*, 1996, vol. 80, no. 3, p. 1006.
10. Morozov, V.I., Usenko, T.N., and Rogozkin, V.A., Neutrophil antiserum response to decrease in proteolytic activity in loaded rat muscle, *Eur. J. Appl. Physiol.*, 2001, vol. 84, p. 195.
11. Morozov, V.I., in *Exercise and Cellular Mechanisms of Muscle Injury*, Kalinski, M.I. and Peake, J., Eds., New York: Nova Science Publishers, 2011, p. 217.
12. Linnamo, V., Bottas, R., and Komi, P.V., Force and EMG power spectrum during and after eccentric and concentric fatigue, *J. Electromyogr. Kinesiol.*, 2000, vol. 10, p. 293.
13. Howatson, G., Van Someren, K., and Hortobgyi, T., Repeated bout effect after maximal eccentric exercise, *Int. J. Sports Med.*, 2007, vol. 28, p. 557.
14. Hedayatpour, N., Falla, D., Arendt-Nielsen, L., et al., Motor unit conduction velocity during sustained contraction after eccentric exercise, *Med. Sci. Sports Exerc.*, 2009, vol. 41, no. 10, p. 1927.
15. Bottas, R., Miettunen, K., Komi, P., and Linnamo, V., Acute (0–2h) and delayed (2–8D) effects of exercise-induced muscle damage and soreness on elbow target movements, *Motor Control*, 2011, vol. 15, no. 4, p. 525.
16. Vila-Chã, C., Hassanlouei, H., Farina, D., and Falla, D., Eccentric exercise and delayed onset muscle soreness of the quadriceps induce adjustments in agonist-antagonist activity, which are dependent on the motor task, *Exp. Brain Res.*, 2012, vol. 216, no. 3, p. 385.
17. Kano, Y., Masuda, K., Furukawa, H., et al., Histological skeletal muscle damage and surface EMG relationships following eccentric contractions, *J. Physiol. Sci.*, 2008, vol. 58, no. 5, p. 349.
18. Widrick, J.J. and Barker, T., Peak power of muscles injured by lengthening contractions, *Muscle Nerve*, 2006, vol. 34, no. 4, p. 470.
19. Nosaka, K., Newton, M., and Sacco, P., Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage, *Scand. J. Med. Sci. Sports*, 2002, vol. 12, no. 6, p. 337.
20. Minigalin, A.D., Shumakov, A.R., Baranova, T.I., et al., Acute and delayed biochemical and physiological effects of exhaustive weightlifting exercise, *Hum. Physiol.*, 2011, vol. 37, no. 2, p. 200.
21. Wilmore, J.H. and Costill, D.L., Functional anatomy of exercising muscle, *Physiology of Sport and Exercise*, Champaign, Illinois: Human Kinetics, 2012, 5th ed., p. 29.
22. Terzis, G., Spengos, K., Karampatso, G., et al., Acute effect of drop jumping on throwing performance, *J. Strength Cond. Res.*, 2009, vol. 23, p. 2592.
23. Gilliver, S.F., Degens, H., Rittweger, J., et al., Variation in the determinants of power of chemically skinned human muscle fibres, *Exp. Physiol.*, 2009, vol. 94, p. 1070.
24. Macpherson, P.C., Schork, M.A., and Faulkner, J.A., Contraction-induced injury to single fiber segments from fast and slow muscles of rats by single stretches, *Am. J. Physiol.*, 1996, vol. 271, no. 5, p. 1438.
25. Sahlin, K., Metabolic factors in fatigue, *Sports Med.*, 1992, vol. 13, p. 99.
26. Fowles, J.R., Green, H.J., Tupling, R., et al., Human neuromuscular fatigue is associated with altered Na+-K+-ATPase activity following isometric exercise, *J. Appl. Physiol.*, 2002, vol. 92, p. 1585.
27. Plowman, S.A. and Smith, D.L., *Exercise physiology*, Philadelphia, PA: Lippincott Williams and Wilkins, 2011, 3rd ed.
28. Hogan, M.C., Gladden, L.B., Kurdak, S.S., and Poole, D.C., Increased lactate in working dog muscle reduces tension development independent of pH, *Med. Sci. Sports Exercise*, 1995, vol. 27, p. 371.
29. Gladden, L.B., Lactate metabolism: A new paradigm for the third millennium, *J. Physiol.*, 2004, vol. 558, p. 5.
30. Peake, J., Nosaka, K., and Suzuki, K., Characterization of inflammatory responses to eccentric exercise in humans, *Exerc. Immunol. Rev.*, 2005, vol. 11, p. 64.
31. Crenshaw, A.G., Thornell, L.E., and Fridn, J., Intramuscular pressure, torque and swelling for the exercise-induced sore vastus lateralis muscle, *Acta. Physiol. Scand.*, 1994, vol. 152, no. 3, p. 265.
32. Gibala, M.J., MacDougall, J.D., Tarnopolsky, M.A., et al., Changes in human skeletal muscle ultrastructure and force production after acute resistance exercise, *J. Appl. Physiol.*, 1995, vol. 78, no. 2, p. 702.
33. Fridén, J. and Lieber, R.L., Eccentric exercise-induced injuries to contractile and cytoskeletal muscle fibre components, *Acta. Physiol. Scand.*, 2001, vol. 171, no. 3, p. 321.
34. Fuglevand, A.J., Zackowski, K.M., Huey, K.A., and Enoka, R.M., Impairment of neuromuscular propagation during human fatiguing contractions at submaximal forces, *J. Physiol.*, 1993, vol. 460, p. 549.
35. Fuller, D., Sullivan, J., and Fregosi, R.F., Expiratory muscle endurance performance after exhaustive submaximal exercise, *J. Appl. Physiol.*, 1996, vol. 80, p. 1495.
36. Ross, E.Z., Goodall, S., Stevens, A., and Harris, I., Time course of neuromuscular changes during running in well-trained subjects, *Med. Sci. Sports Exerc.*, 2010, vol. 42, p. 1184.
37. Yakovlev, N.N., *Biokhimiya sporta* (Biochemistry of Sports), Moscow: Fizkul'tura Sport, 1974.
38. Volkov, N.I., *Biokhimiya myshechnoi deyatel'nosti* (Biochemistry of Muscle Activity), Moscow: Olimpiiskii sport, 2001.
39. Myakinchenko, E.B. and Seluyanov, V.N., *Razvitie lokal'noi myshechnoi vynoslivosti v tsiklicheskikh vidakh sporta* (Training for Local Muscle Endurance in Cyclic Sports), Moscow: SportAkademPress, 2005.

Translated by T. Tkacheva