Laser homeostatics on delayed onset muscle soreness

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Abstract. Delayed onset muscle soreness (DOMS) and its photobiomodulation were reviewed from the viewpoint of function-specific homeostasis (FSH) in this paper. FSH is a negative-feedback response of a biosystem to maintain the function-specific fluctuations inside the biosystem so that the function is perfectly performed. A stressor may destroy a FSH. A stress is a response of a biosystem to a stressor and may also be in stress-specific homeostasis (StSH). A low level light (LLL) is so defined that it has no effects on a function in its FSH or a stress in its StSH, but it modulate a function far from its FSH or a stress far from its StSH. For DOMS recovery, protein metabolism in the Z-line streaming muscular cell is the essential process, but the inflammation, pain and soreness are non-essential processes. For many DOMS phenomena, protein metabolism in the Z-line streaming muscular cell is in protein metabolism-specific homeostasis (PmSH) so that there are no effects of LLL although the inflammation can be inhibited and the pain can be relieved. An athlete or animal in the dysfunctional conditions such as blood flow restriction and exercise exhaustion is far from PmSH and the protein metabolism can be improved with LLL.

1. Introduction
Skeletal muscle is one of the most vulnerable tissue in the extremities. Exercise-induced muscle damage (EIMD) is commonly experienced following either a bout of unaccustomed physical activity or following physical activity of greater than normal duration or intensity, which manifests as prolonged muscle dysfunction, delayed onset muscle soreness (DOMS), and leakage of muscle proteins into circulation. The mechanistic factor responsible for the initiation of EIMD is not known; however, it is hypothesised to be either mechanical or metabolic in nature. This model states that

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damage initiation may be either metabolic or mechanical, or a combination of both, depending on the mode, intensity and duration of exercise and the training status of the individual[1]. EIMD/DOMS is a well-known phenomenon in exercise training and physical activities. Effective therapy to reduce skeletal muscle injury associated with severe or eccentric exercise is needed. Many therapeutic approaches have been used to speed recovery in view of previous theories proposed for its mechanism, such as lactic acid, muscle spasm, connective tissue damage, muscle damage, inflammation, the enzyme efflux theories, and an integration of two or more theories, but few of them were effective [2]. In this paper, EIMD/DOMS phenomena and its photobiomodulation (PBM) was reviewed from the viewpoint of function-specific homeostasis (FSH).

2. Function-specific homeostasis

Homeostasis is a classic concept in physiology [3]. However, it is too obscure to be studied so that it has been developed as FSH[4-5]. FSH is a negative-feedback response of a biosystem to maintain the function-specific fluctuations inside the biosystem so that the function is perfectly performed.

The quality of a FSH includes function complexity and performance stability. An elite athlete has a sport-specific homeostasis (SpSH) to maintain sport performance. For a 100 m sprint runner, the 100 m sprint time represents the function complexity. The less the running time, the more complicated the run. The coefficient of variation (CV) of the sprint time represents the performance stability. The higher the CV, the lower the performance stability.

There are many subsystems to maintain a SpSH, SpSH-essential subsystems (SESSs) and SpSH-non-essential subsystems (SNSs), which are in their respective SES-specific homeostasis (SESH) and SNS-specific homeostasis (SNSH), respectively [4]. In order to realize the transition from low quality SpSH1 into high quality SpSH2, there also are two kinds of training, extraordinary training (ET) and ordinary training (OT). ET establishes the SESHs of SpSH2 by destroying SpSH1. OT maintaining the SESHs has two phases, OTA during which the SNSHs of SpSH2 and then SpSH2 are established, and OTB during which SpSH2 is maintained.

A stress is a function of a biosystem[6]. A function-specific stressor (FSSR) is so defined that it disrupts the FSH of the function. A function-specific stress (FSS) of a biosystem is defined to be a response of the biosystem to a FSSR and may also be in FSS-specific homeostasis (FSSH) in which a FSS disrupt FSH1 but establish FSH2. A factor is called low level factor (LLF) if there is no effects of the factor on a function in its FSH or a FSS in its FSSH, but modulate a function far from its FSH or a FSS far from its FSSH. Obviously, novel eccentric (lengthening contraction) exercise (ECC) is a sport-specific stressor, and ET-OTA is in sport-specific stress-specific homeostasis because it realized the transition from SpSH1 into SpSH2. ECC can increase the optimum lengths of both the knee extensors and knee extensors flexors during the preseason in professional soccer[7]. Only 30 min of eccentric exercise per week for eight weeks was sufficient to improve health risk factors [8].

ET results in EIMD/DOMS. The physiological stress associated with ECC induced muscle damage impairs insulin stimulation of Insulin-stimulated insulin receptor substrate (IRS)-1, phosphatidylinositol (PI) 3-kinase, and Akt (protein kinase B)-kinase, presumably leading to decreased insulin-mediated glucose uptake[9]. Using high density oligonucleotide based microarrays, MacNeil et al. [10] screened for differences in mRNA expression caused by 17beta-estradiol (E2) and 150 single-leg
ECC, and found that the signaling and regulation of early Ras homologue gene family, member A (RhoA) and nuclear factor of activated T-cells (NFAT) are altered following the ECC for muscle remodeling and repair.

Increases in the mRNA expression of interleukin (IL)-6, IL-8, and cyclooxygenase 2 (COX2) occur in the vastus lateralis as a result of damaging ECC in young, recreationally trained males [11].

Inflammation induced by high-force ECC in skeletal muscle is greater when a high carbohydrate (CHO) compared to a low CHO diet is consumed during recovery [12]. Even grounding the body to the earth alters measures of immune system activity and pain [13].

Unaccustomed strenuous exercise that includes lengthening contraction (LC) often causes DOMS, a kind of muscular mechanical hyperalgesia. Augmentation of the mechanical response in muscle thin-fiber sensory receptors might be related to the muscle tenderness in DOMS after ECC [14]. Nerve growth factor (NGF) upregulation through activation of B(2) bradykinin receptors is essential (though not satisfactory) to mechanical hyperalgesia after exercise [15].

DOMS has three subsequent phases: Z-line streaming, proteolysis of damaged proteins and protein synthesis, among which the last two phases is just protein metabolism that is the essential process of DOMS recovery [16]. The DOMS normally recovers in about seven days if the protein metabolism is in its protein metabolism-specific homeostasis (PmSH). For EIMD/DOMS, there are oxidative stress, inflammation, pain/soreness and protein metabolism. All the other processes might be far from their respective FSH, oxidative-process-specific homeostasis (OSH), inflammation-specific homeostasis (ISH) and pain/soreness-specific homeostasis (PsSH), but protein metabolism might be in protein-metabolism-specific homeostasis (PmSH). The factor effects on EIMD/DOMS would then be discussed.

3. DOMS in PmSH

Fu [17] has studied the effects of niacin supplement at low level on the inflammation of skeletal muscle, level of serum CK and lactate dehydrogenase (LDH), protein level of sirtuin 1 (SIRT1), p65 and 20S protease of skeletal muscle in rats following a bout of ECC. She found niacin supplement could reduce inflammatory of EIMD/DOMS, which indicated the process of inflammatory may be far from ISH. Niacin supplement increased SIRT1 protein level, reduced p65 protein level and then inhibited inflammation for a bout of ECC induced EIMD / DOMS. Niacin supplement did not affect the expression of 20S protease in soleus and the levels of serum CK and LDH, respectively, which indicated that protein metabolism may be in PmSH, and the niacin supplements did not affect the metabolic processes.

Any LLFs can not affect normal DOMS recovery. DOMS, creatine kinase (CK) activity, and isometric and concentric torque all changed over the 72-hour period after ECC; however, oral beta-hydroxy-beta-methylbutyrate (HMB) and alpha-ketoisocaproic acid (KIC) had no significant effect on any of the indices of muscle damage [18]. Early RhoA and NFAT signaling and regulation are altered following the ECC for muscle remodeling and repair, but are not affected by E2 [10].

Eight healthy male volunteers performed 200 maximal eccentric contractions with each leg. Although Non-steroidal anti-inflammatory drugs (NSAIDs) inhibit the exercise-induced satellite cell proliferation, Mikkelsen et al. [19] observed myofibrillar and collagen protein synthesis were unaffected.
by the local NSAID infusion. Five hours post-exercise, the mRNA expression of COX2 was sixfold higher in the NSAID leg compared with the unblocked leg, but the expression of growth factors and matrix-related genes were unaffected by NSAID. All subjects completed 4 sets of 12 repetitions of eccentric elbow flexion with their non-dominant arm. Goldfarb et al. [20] reported that 4 weeks of pretreatment with a fruit/berry/vegetable concentrate (FVC) can attenuate blood oxidative stress markers induced by ECC but had no significant impact on the functional changes related to pain and muscle damage. These phenomena indicated that oxidative process is far from oxidative-process-specific homeostasis (OSH), but protein metabolism is in PmSH. As McGinley et al. [21] have reviewed, there is little evidence to support a role for vitamin C and/or vitamin E in protecting against muscle damage although there is some evidence to show that both antioxidants can reduce indices of oxidative stress, and antioxidant supplementation may actually interfere with the cellular signalling functions of reactive oxygen species (ROS), thereby adversely affecting muscle performance. Since the potential for long-term harm does exist, the casual use of high doses of antioxidants by athletes and others should perhaps be curtailed[21].

Forty-five repetitions of eccentric elbow flexion were performed with 90% of one maximum repetition by each subject. The exercises were done as three sets of 15 repetitions with 3 minutes rest between sets. Afroudeh et al. [22] found carbohydrate increased the inflammatory (IL-6) response following resistance exercise, but had no effects on C-reactive protein (CRP) and CK. Consumption of theaflavin-enriched black tea extract led to improved recovery and a reduction in oxidative stress and DOMS responses to acute anaerobic intervals, but it had no significant effects on either peak power or mean power during the 30 s Wingate Anaerobic Test (WAnT). These phenomena indicated that inflammation is far from inflammation-specific homeostasis (ISH), but protein metabolism is in PmSH.

Branched-chain amino acid (BCAA) supplementation may attenuate muscle soreness, but it does not ameliorate eccentric exercise-induced decrements in muscle function or increases in reputed blood markers of muscle damage, when consumed before exercise and for 3 d after an ECC bout[23]. Prophylactic supplementation of N-acetyl-cysteine (NAC) and epigallocatechin gallate (EGCG) blunted soreness ratings, but significant increases in muscle levels of bax and bcl-2 were observed in all groups with no significant differences between groups [24]. These phenomena indicated that soreness is far from pain/soreness-specific homeostasis (PsSH), but protein metabolism is in PmSH. The PmSH can be upgraded if there is a beneficial stress in stress-specific homeostasis so that the normal DOMS recovery can be promoted. Protease supplementation seems to attenuate muscle strength losses after eccentric exercise[25]. Subjects performed two sets of 20 maximal eccentric elbow flexion exercises with one arm. Supplementation with ellagitannins from pomegranate extract significantly improves recovery of isometric strength 2-3 d after the damaging eccentric exercise [26].

The initial downhill run of mice at 8 wk of age consisted of 150 min of running on a motorized treadmill. The pretreatment with a food pellet containing 10 mg of curcumin powder blunted the increase in plasma creatine kinase 24 h after the EE[27]. Adenosine A3 receptor stimulation induces protection of skeletal muscle from the EIMD induced by the EE [28]. Calcium entry blocker nifedipine by gavage (2 mg/kg/day in two equal doses) attenuated EE induced muscle damage caused due to its beneficial effect on EE impaired muscle microcirculation[29]. Consuming milk-based
carbohydrate-protein (CHO-P) after muscle-damaging exercise is more beneficial in attenuating decreases in muscle performance and increases in active DOMS at 48 h than ingestion prior to exercise [30], but post-exercise milk-based CHO-P supplementation at lower dose has no influences of on muscle glycogen replacement, inflammation, or muscle function [31], and the addition of whey protein isolate to a dietary carbohydrate supplement did not attenuate systemic indices of muscle damage or inflammation, nor did it restore muscle function more rapidly than when the carbohydrate fraction was ingested alone [32-33]. BCAA supplementation (isoleucine:leucine:valine = 1:2.3:1.2) at about 100 mg/kg body weight may suppress muscle damage [34-35], but BCAA supplementation at lower dose does not ameliorate eccentric exercise-induced decrements in muscle function or increases in reputed blood markers of muscle damage [23], and adding leucine to carbohydrate beverages did not affect acute muscle recovery and squat performance during both initial testing and during a subsequent exercise bout 72 hours later in resistance trained subjects [36].

4. DOMS far from PmSH

The recovery of DOMS in PmSH is normal recovery. The normal recovery can be impaired by many factors so that DOMS is far from PmSH. After exhaustive endurance exercise, muscle damage can be produced by metabolic disturbances associated with ischaemia [37]. The blood flow restriction (BFR) resulted in more soreness than exercise without BFR and greater reductions in pain-pressure threshold and maximal voluntary contraction [38].

DOMS is far from PmSH due to ageing. A reparative capability of muscle was reduced in response to a single exhaustive bout of heavy resistance weight lifting in old (>120 wk of age) compared with young (14-20 wk of age) rats [39]. Older individuals (age 66±2 yr) did not exhibit the compensatory increase in beta-cell secretion observed among young individuals (age 22±1 yr) after ECC [40]. Thus, with increasing age, the pancreatic beta-cell may be less responsive to the physiological stress associated with ECC.

DOMS is far from PmSH due to insulin resistance. Increased phosphorylation of the 70-kDa ribosomal S6 kinase (p70S6k) signaling is strongly correlated with the degree of muscle adaptation following exercise. Katta et al. [41] have compared the phosphorylation of p70S6k, Akt, and mammalian target of rapamycin (mTOR) in the tibialis anterior (TA) muscles of lean and obese Zucker rats following a bout of eccentric exercise (EE). EE increased p70S6k (Thr389) phosphorylation at 0, 1 and 3 h after the EE in the lean TA and at 3 h in the obese TA Zucker rats. mTOR (Ser2448) phosphorylation was elevated in the lean TA immediately after EE but remained unaltered in the obese TA. EE increased Akt (Thr308) and Akt (Ser473) phosphorylation in the lean but not the obese TA. The subjects completed exhaustive downhill running protocol on a motorized treadmill. Allicin supplementation had significantly lower plasma levels of CK, muscle-specific CK and IL-6, and reduced perceived muscle soreness after exercise [42].

5. Photobiomodulation

PBM is a modulation of laser irradiation or monochromatic light (LI) on biosystems, which stimulates or inhibits biological functions but does not result in irreducible damage [4-5]. The LI used in PBM is always low intensity LI (LIL), ~10 mW/cm². However, moderate intensity LI (MIL), 10²-3 mW/cm², is
of PBM if the radiation time is not so long that it damages organelles or cells. LIL is a kind of LLF, and MIL is also a kind of LLF if the radiation time is short enough. Both the two kind of LI are called low level LI (LLL). LLL has been used to treat DOMS after EIMD. LLL can promote the recovery of DOMS far from PmSH. Exhaustive downhill running was used to induce muscle injury in rat gastrocnemius muscle. LIL significantly reduced serum CK activity at 48 h after exercise\(^{[43]}\). The load-resistance swimming test forced adult male Wistar rats to swim until exhaustion. 40 s MIL treatment lowered CK activity and muscular apoptosis\(^{[44]}\).

LLL can not promote the recovery of DOMS in PmSH. The experimenter raised the subjects elbow position to 135° of flexion (upright position) and the subject was asked to lower this one-repetition maximum (1-RM) weight eccentrically for 3 sec. 80 s MIL treatment significantly decreased pain, but had no effects on girth and resting angle\(^{[45]}\). Isokinetic exercise was performed to induce pain related to DOMS. LIL treatment significantly decreased pain, but had no effects on isokinetic peak torque\(^{[46]}\).

6. Discussion
PBM can be also used to treat DOMS before EIMD. After MIL treatment with 810 nm diodes at 1724 mW/cm\(^2\) for 30 s or placebo treatment, subjects performed 75 maximal knee extensors eccentric contractions (five sets of 15 repetitions; velocity = 60 degrees seg\(^{-1}\); range of motion = 60 degrees ). Baroni et al.\(^{[47]}\) found that MIL treatment before ECC was effective in terms of attenuating the increase of muscle proteins in the blood serum and the decrease in muscle force. The DOMS is in PmSH, but MIL pretreatment can promote its recovery. In this case, MIL might be a beneficial stress in stress-specific homeostasis, but it needs to be further studied.

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8. References
[1] Tee J C, Bosch A N and Lambert M I 2007 Metabolic consequences of exercise-induced muscle damage. *Sports Med.* **37** 827

[2] Cheung K, Hume P and Maxwell L 2003 Delayed onset muscle soreness : treatment strategies and performance factors. *Sports Med.* **33** 145

[3] Cannon W B 1932 The Wisdom of the Body. *WW Norton, New York.*

[4] Liu T C Y, Liu R, Zhu L,Yuan J Q, Hu M and Liu S H 2009 Homeostatic photobiomodulation. *Front Optoelectron China.* **2** 1

[5] Liu C Y and Zhu P (Eds) 2009 Intranasal Low Intensity Laser Therapy. Beijing: *People’s Military Medical Press.* **50** 413.

[6] Liu T C Y, Li F H, Zhu L and Liu S H 2010 Photobiomodulation on stress. *Chin. J. Laser.* *(to be published)*.
[7] Brughelli M, Mendiguchia J, Nosaka K, Idoate F, Arcos A L and Cronin J 2010 Effects of eccentric exercise on optimum length of the knee flexors and extensors during the preseason in professional soccer players. Phys. Ther. Sport. 11 50

[8] Paschalis V, Nikolaidis M G, Theodorou A A, Panayiotou G, Fatouros I G, Koutedakis Y and Jamurtas A Z 2010 A Weekly Bout of Eccentric Exercise Is Sufficient To Induce Health-Promoting Effects. Med. Sci. Sports Exerc.

[9] Del Aguila L F, Krishnan R K, Ulbrecht J S, Farrell P A, Correll P H, Lang C H, Zierath J R and Kirwan J P 2000 Muscle damage impairs insulin stimulation of IRS-1, PI 3-kinase, and Akt-kinase in human skeletal muscle. Am. J. Physiol. Endocrinol Metab. 279 E206

[10] MacNeil L G, Melov S, Hubbard A E, Baker S K and Tarnopolsky M A 2010 Eccentric exercise activates novel transcriptional regulation of hypertrophic signaling pathways not affected by hormone changes. PloS. One.18;5 e10695.

[11] Buford T W, Cooke M B, Shelmadine B D, Hudson G M, Redd L and Willoughby D S 2009 Effects of eccentric treadmill exercise on inflammatory gene expression in human skeletal muscle. Appl. Physiol. Nutr. Metab. 34 745

[12] Depner C M, Kirwan R D, Frederickson S J and Miles M P 2010 Enhanced inflammation with high carbohydrate intake during recovery from eccentric exercise. Eur. J. Appl. Physiol. 109 1067

[13] Brown D, Chevalier G and Hill M 2010 Pilot study on the effect of grounding on delayed-onset muscle soreness. J. Altern. Complement Med. 16 265.

[14] Taguchi T, Sato J and Mizumura K 2005 Augmented mechanical response of muscle thin-fiber sensory receptors recorded from rat muscle-nerve preparations in vitro after eccentric contraction. J. Neurophysiol. 94 2822

[15] Murase S, Terazawa E, Queme F, Ota H, Matsuda T, Hirate K, Kozaki Y, Katanosaka K, Taguchi T, Urai H and Mizumura K 2010 Bradykinin and nerve growth factor play pivotal roles in muscular mechanical hyperalgesia after exercise (delayed-onset muscle soreness). J. Neurosci. 30 3752

[16] Liu T C Y, Huang P, Liu X G, Chen X Y, Liu J, Wang S X, Cui L P, Xu X Y Guo H, Jin H, Deng S X and Ji L L 2006 Delayed Onset Muscle Soreness: Three-Phase Hypothesis and Its Clinical Applications. Med. Sci. Sport Exer. 38 S124

[17] Fu D R 2010 The molecular mechanism of exercise induced skeletal muscle damage and its niacin effects. Ph. D. thesis, South China Normal University.

[18] Nunan D, Howatson G and van Someren K A 2010 Exercise-induced muscle damage is not attenuated by beta-hydroxy-beta-methylbutyrate and alpha-ketoisocaproic acid supplementation. J. Strength Cond. Res. 24 531
[19] Mikkelsen U R, Schjerling P, Helmark I C, Reitelseder S, Holm L, Skovgaard D, Langberg H, Kjaer M and Heinemeier K M 2010 Local NSAID infusion does not affect protein synthesis and gene expression in human muscle after eccentric exercise. *Scand. J. Med. Sci. Sports.*

[20] Goldfarb A H, Garten R S, Cho C, Chee P D and Chambers L A 2010 Effects of a Fruit/Berry/Vegetable Supplement on Muscle Function and Oxidative Stress. *Med. Sci. Sports Exerc.*

[21] McGinley C, Shafat A and Donnelly A E 2009 Does antioxidant vitamin supplementation protect against muscle damage? *Sports Med.* 39 1011

[22] Aaroundeh R, Siahkouhian M and Khalili A 2010 The effect of post-exercise carbohydrate ingestion on inflammatory responses to short time, high-force eccentric exercise. *J. Sports Med. Phys. Fitness.* 50 182

[23] Jackman S R, Witard O C, Jeukendrup A E and Tipton K D 2010 Branched-chain amino acid ingestion can ameliorate soreness from eccentric exercise. *Med. Sci. Sports Exerc.* 42 962

[24] Kerksick C M, Kreider R B and Willoughby D S 2010 Intramuscular adaptations to eccentric exercise and antioxidant supplementation. *Amino. Acids.* 39 219

[25] Buford T W, Cooke M B, Redd L L, Hudson G M, Shelmadine B D and Willoughby D S 2009 Protease Supplementation Improves Muscle Function after Eccentric Exercise. *Med. Sci. Sports Exerc.* 41 1908

[26] Trombold J R, Barnes J N, Critchley L and Coyle E F 2010 Ellagitannin consumption improves strength recovery 2-3 d after eccentric exercise. *Med. Sci. Sports Exerc.* 42 493

[27] Davis J M, Murphy E A, Carmichael M D, Zielinski M R, Groschwitz C M, Brown A S, Gangemi J D, Ghaffar A and Mayer E P 2007 Curcumin effects on inflammation and performance recovery following eccentric exercise-induced muscle damage. *Am. J. Physiol. Regul. Integr. Comp. Physiol.* 292 R2168

[28] Wang R, Urso M L, Zambraski E J, Rader E P, Campbell K Pand Liang B T 2010 Adenosine A(3) receptor stimulation induces protection of skeletal muscle from eccentric exercise-mediated injury. *Am. J. Physiol. Regul. Integr. Comp. Physiol.* 299 R259

[29] Heap S J, Fulgenzi G L and Hudlicka O 2006 Microcirculation in rat soleus muscle after eccentric exercise: the effect of nifedipine. *Eur. J. Appl. Physiol.* 97 687

[30] Cockburn E, Stevenson E, Hayes P R, Robson-Ansley P and Howatson G 2010 Effect of milk-based carbohydrate-protein supplement timing on the attenuation of exercise-induced muscle damage. *Appl. Physiol. Nutr. Metab.* 35 270.
[31] Wojcik J R, Walber-Rankin J, Smith L L and Gwazdauskas F C 2001 Comparison of carbohydrate and milk-based beverages on muscle damage and glycogen following exercise. Int. J. Sport Nutr. Exerc. Metab. 11 406.

[32] White J P, Wilson J M, Austin K G, Greer B K, St John N and Panton L B 2008 Effect of carbohydrate-protein supplement timing on acute exercise-induced muscle damage. J. Int. Soc. Sports Nutr. 5 5

[33] Betts J A, Toone R J, Stokes K A and Thompson D 2009 Systemic indices of skeletal muscle damage and recovery of muscle function after exercise: effect of combined carbohydrate-protein ingestion. Appl. Physiol. Nutr. Metab. 34 773

[34] Negro M, Giardina S, Marzani B and Marzatico F 2008 Branched-chain amino acid supplementation does not enhance athletic performance but affects muscle recovery and the immune system. J. Sports Med. Phys. Fitness. 48 347

[35] Shimomura Y, Inaguma A, Watanabe S, Yamamoto Y, Muramatsu Y, Bajotto G, Sato J, Shimomura N, Kobayashi H and Mawatari K 2010 Branched-chain amino acid supplementation before squat exercise and delayed-onset muscle soreness. Int. J. Sport Nutr. Exerc. Metab. 20 236

[36] Stock M S, Young J C, Golding L A, Kruskall L J, Tandy R D, Conway-Klaassen J M and Beck T W 2010 The effects of adding leucine to pre and postexercise carbohydrate beverages on acute muscle recovery from resistance training. J. Strength Cond. Res. 24 2211

[37] Ebbeling C B and Clarkson P M 1989 Exercise-induced muscle damage and adaptation. Sports Med. 7 207

[38] Umbel J D, Hoffman R L, Dearth D J, Chleboun G S, Manini T M and Clark B C 2009 Delayed-onset muscle soreness induced by low-load blood flow-restricted exercise. Eur. J. Appl. Physiol. 107 687

[39] Tamaki T, Uchiyama S, Uchiyama Y, Akatsuka A, Yoshimura S, Roy R R and Edgerton V R 2000 Limited myogenic response to a single bout of weight-lifting exercise in old rats. Am. J. Physiol. Cell Physiol. 278 C1143

[40] Krishnan R K, Hernandez J M, Williamson D L, O’Gorman D J, Evans W J and Kirwan J P 1998 Age-related differences in the pancreatic beta-cell response to hyperglycemia after eccentric exercise. Am. J. Physiol. 275 E463.

[41] Katta A, Karkala S K, Wu M, Meduru S, Desai D H, Rice K M and Blough E R 2009 Lean and obese Zucker rats exhibit different patterns of p70s6 kinase regulation in the tibialis anterior muscle in response to high-force muscle contraction. Muscle Nerve. 39 503
[42] Su Q S, Tian Y, Zhang J G and Zhang H 2008 Effects of allicin supplementation on plasma markers of exercise-induced muscle damage, IL-6 and antioxidant capacity. *Eur. J. Appl. Physiol.* **103** 275

[43] Liu X G, Zhou Y J, Liu T C and Yuan J Q 2009 Effects of low-level laser irradiation on rat skeletal muscle injury after eccentric exercise. *Photomed. Laser Surg.* **27** 863

[44] Sussai D A, Carvalho Pde T, Dourado D M, Belchior A C, dos Reis F A and Pereira D M 2010 Low-level laser therapy attenuates creatine kinase levels and apoptosis during forced swimming in rats. *Lasers Med. Sci.* **25** 115

[45] Douris P, Southard V, Ferrigi R, Grauer J, Katz D, Nascimento C and Podbielski P 2006 Effect of phototherapy on delayed onset muscle soreness. *Photomed. Laser Surg.* **24** 377

[46] Vinck E, Cagnie B, Coorevits P, Vanderstraeten G and Cambier D 2006 Pain reduction by infrared light-emitting diode irradiation: a pilot study on experimentally induced delayed-onset muscle soreness in humans. *Lasers Med. Sci.* **21** 11

[47] Baroni B M, Leal Junior E C, De Marchi T, Lopes A L, Salvador M and Vaz M A 2010 Low level laser therapy before eccentric exercise reduces muscle damage markers in humans. *Eur. J. Appl. Physiol.*