

The Effect of Static Stretching on Delayed Onset Muscle Soreness Before Eccentric Concentration

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Abstract: The main purpose of this study was to determine the effect of static stretching before eccentric contractions on DOMS. Twenty one active female volunteers were selected. All subjects were right handed and healthy. Subjects were assigned in one of two experimental group (n=10) (age: 21.6±1.71 years, height: 161.45±2.71 cm weight: 57.25±6.99 kg. and $V_{O_{2,max}}$: 34.18±2. ml. kg⁻¹. min⁻¹) and control group (n=11), (age: 24.25 ±4.30 years, height: 159.81±4.86 cm, weight: 54.69±3.82 kg. and maximal oxygen consumption $V_{O_{2,max}}$: 36.1±3.79 ml.kg⁻¹. min⁻¹). The experimental protocol was 80 incremental resistance eccentric contraction and approved by the ethics committee and all subjects were informed of the risks and purposes of the study before their written consent was obtained. Blood samples (n = 21) were drawn from the anticubital vein before, immediately after, 24 and 48 hours after an exercise. Ambient temperature during running was 17°C. Then Experimental group, first performed static stretching on shoulder, elbow for 20 minutes and then they performed incremental resistance eccentric contraction. Control group performed, only incremental resistance eccentric contraction. Data was analyzed using ANOVA test. There were an increasing CK and LDH enzymes levels in subjects blood. The results showed warm up by static stretching did not prevent not only the muscular damage cellular damage, but also induced increasing CK and LDH. Muscle sourness are acute (immediately after exercise) or delayed after, 24-48 hours after exercise. That is better static stretching use after general warm up, because muscle fibers and member cells do not damage and destroy and deplete enzymes.

Key words: Muscle soreness · CK · LDH · Contraction

INTRODUCTION

Muscular adaptation to physical exercise has been explained by the classical damage-inflammation-repair pathway [1-4]. By definition, this process involves, exercise-induced muscle damage, release of chemo-attractive factors, vasodilatation, leukocyte adhesion, neutrophils and macrophages migration and activation of satellite cells [5, 6]. Adaptation of skeletal muscle to physical exercise has also been compared with muscle development and regeneration following damage in the sense that satellite cell activation is believed to be inevitable [3, 7]. In a recent study in several pieces of evidence indicated that muscle adaptation to physical exercise might not occur only via this classical pathway because similar inflammatory changes in skeletal muscle occurred in both the exercise and control groups [2, 7]. Gibala *et al.* and Stupka *et al.* have demonstrated a greater degree of ultra-structural damage in eccentrically versus concentrically exercised muscle [8-10], while Yu *et al.*

could not detect any alteration in desmin structure after different modes of eccentric exercise [11]. Stupka *et al.* also found an increased number of leukocyte common antigen positive cells in skeletal muscle 48 h after eccentric exercise. The total number of inflammatory cells in each muscle section could not be evaluated in their study [10, 12]. Thus, it can be argued that exercise-induced disruption of the cytoskeleton is a sign of remodeling [11, 13] and that the evidence for a consequent muscle inflammation should be viewed with caution.

The present study investigated the effect of warm up on CK and LDH. The main purpose was to determine the effect of static stretching before contractions [14, 8]. Eccentric contractions that use in exercise, it is possible induced releasing enzymes. Many studies showed that DOMS (an event in the epimysium), CK and LDH enzymes increased in recovery period. It was hypothesized that muscle injury would be greater with eccentric than with all-out or prolonged exercise and that immune changes

might provide an indication that supplements the information provided by traditional markers such as creatine kinase (CK) or delayed-onset muscle soreness, creatine kinase (CK) were also measured before and 24 h after exercise as markers of the "acute-phase response" and muscle damage [1]. Brenner *et al.* examined eight healthy males were each assigned to four experimental conditions. 5 min of cycle ergometer exercise at 90% VO_{2max}, a standard circuit-training routine, 2 h cycle ergometer exercise at 60% VO_{2max}, or remained seated for 5h. CK levels were only elevated significantly 72 h following circuit-training. Results showed correlation between CK and LDH concentrations and muscle soreness and immune markers of the inflammatory response [7].

MATERIALS AND METHODS

Twenty one volunteers female university student (P.E. students) were selected. Who were normal healthy, with no positive clinical finding. After having the procedures fully explained to them written information consent. Subjects were randomly assigned in one of two experimental group (n=10) (age: 21.6±1.71 years, height: 161.45±2.71 cm weight: 57.25±6.99 kg. and Vo₂ max: 34.18±2. ml. kg⁻¹. min⁻¹) and control group (n=11), (age: 24.25±4.30 years, height: 159.81±4.86 cm, weigh: 54.69±3.82 kg. and Vo₂ max 36.1± 3.79 ml.kg⁻¹. min⁻¹) groups, All subjects were right handed. As pre test venues blood sampling of all subjects were taken and after that experimental group performed static stretching on shoulder, elbow and arm for 15 minutes, then they performed eccentric contraction. Control group performed, only eccentric contraction. Blood sampling were obtained before, immediately after, 24 and 48 hours after exercise and, were drawn from an anticubital vein with subjects in the seated position. After, 24 h and 48 h after exercise, muscle soreness at rest was estimated by the subjects on a 0 -10 subject rating scale (0 = no soreness and 10 = very, very sore). Pain was estimated by the subjects on the 0-10 subject rating scale delayed onset muscle soreness (DOMS) was estimated in the subjects. Serum CK and LDH activity was measured using a standard laboratory kit. Data was analyzed using ANOVA test, α was set at 0/05. Data are presented as mean ± ME of the mean.

RESULTS

There were an increasing CK and LDH enzymes levels in subjects blood. So subjects had pain and inflammatory in their hands. Changes were higher in experimental group (Table 1). The results showed that eccentric contraction induced DOMS, CK and LDH

Table 1: Changes CK, LDH in 2 groups

Time	M ± S.d			
	CK		LDH	
	Con.g	Exp.g	Con.g	Exp.g
Pre test	72.72±33.11	78.50±17.03	373.18±125.63	372.50±125.63
Post test	107.72±56.14	94.50±20.46	434.09±74.72	470±117.61
24h. after	157.27±41.64	177.50±31.91	531.81±79.35	570±112.96
48h. after	148.18±35.46	261.66±23.42	273.18±26.57	272.77±55.46

Table 2: Result ANOVA

	F			
	Pre	Pos	24 h.a.	48 h.a.
Ck	0.07	0.27	0.08	0.5*
LDH	0.27	0.71	0.82	0.001*

U/L

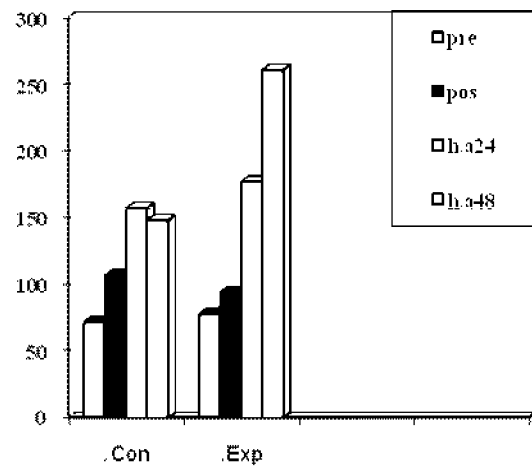


Fig. 1: Changes of CK

U/L

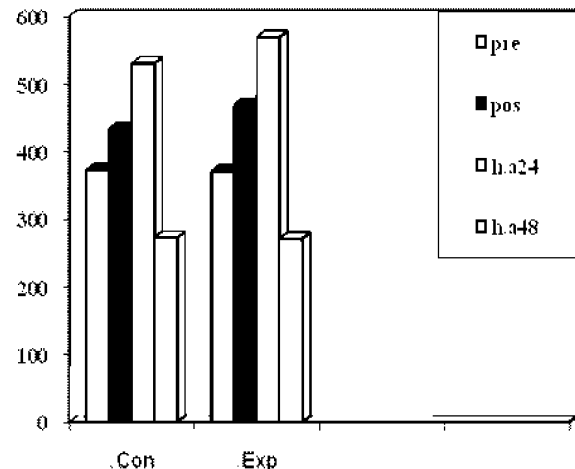


Fig. 2: Changes of LDH

enzymes levels increased in two groups (especially in experimental group) (Figure 1 and 2), post test and continuously 24 hours after that the but the effects of static stretching of CK, LDH enzymes and DOMS are not significant (Table 2).

DISCUSSION

It show the static stretching has a effect on CK and LDH. The results showed warm up not only did not prevent the muscular damage and did not decrease the pain of cellular damage, but also the warm up by eccentric stretching increased CK and LDH [15, 16]. MS (muscle sourness) are acute (Immediately after exercise) or delayed after, 24 - 48 hours after exercise. That is better static stretching use after general warm up, because muscle fibers and member cells do not damage and deplete enzymes. There were significant correlation between CK in both groups and LDH in experimental group and CK and LDH in recovery period on experimental group. It show the warm up has a significant effect on CK and LDH correlation in recovery period [17]. The results showed warm up prevented the muscular cramp and decrease the pain of cellular damage, also the warm up changes the depletion of CK and LDH after strenuous activities [15]. It is suggested that exercise can induce DOMS by activating inflammatory factors present in the epimysium before exercise. Repeated physical training may alter the content of inflammatory factors in the epimysium and thus reduce DOMS and types and intensities of exercise is important [1]. The exercise protocol used in this study resulted in decreased muscle function and inflicted severe muscle discomfort that hindered normal every day activities. DOMS and muscle pain were related to markers of inflammation in the epimysium and significant increases in muscle soreness, serum CK and LDH activity. Of course the correlations between immunological variables in human blood and skeletal muscle found in the previous study. Studies interpret that muscular adaptation to physical exercise is fundamentally different from the muscle cell repair mechanisms activated by experimentally inflicted damage, the sensation of DOMS and pain after unaccustomed physical exercise is confined to immunological reactions in muscle epimysium and individual differences between subjects result in larger deviations when investigating muscle adaptation to

physical exercise than when tissue repair after trauma is studied [7].

REFERENCE

1. Lieber, R.L., L.E. Thornell and J. Friden, 1996. Muscle cytoskeletal disruption occurs within the first 15 min of cyclic eccentric contraction. *J. Appl. Physiol.*, 80: 278-84.
2. Malm, C., *et al.*, XXXX. Leukocytes, Cytokines, Growth Factors and Hormones in Human Skeletal Muscle and Blood after Uphill or Downhill Running, pp: 11.
3. Chambers, R.L. and J.C. McDermott, 1996. Molecular basis of skeletal muscle regeneration. *Can. J. Appl. Physiol.*, 21: 155-184.
4. Clarkson, P.M. and S.P. Sayers, 1999. Etiology of exercise-induced muscle damage, *Can. J. Appl. Physiol.*, 24: 234-248.
5. Tidball, J.G., 1995. Inflammatory cell response to acute muscle injury, *Med. Sci. Sports Exerc.*, 27: 1022-1032.
6. Peter, M., XXXX. Skeletal muscle damage and repair. *Human Kinetics*, pp: 59-76.
7. MacIntyre, D.L., W.D. Reid and D.C. McKenzie, 1995. Delayed muscle soreness. The inflammatory response to muscle injury and its clinical implications, *Sports Med.*, 20: 24-40.
8. Lund, H., P. Vestergaard-Poulsen and I.L. Kanstrup Sejrsen, 1998. The effect of passive stretching on delayed onset muscle soreness and other detrimental effects following eccentric exercise. *J. Med. Sci. Sports*, 8: 216-221.
9. Gibala, M.J., S.A. Interisano, M.A. Tarnopolsky, B.D. Roy, J.R. Macdonald, K.E. Yarasheski and J.D. Macdougall, 2000. Myofibrillar disruption following acute concentric and eccentric resistance exercise in strength-trained men. *Can. J. Physiol. Pharmacol.*, 78: 656-661.
10. Stupka, N., S. Lowther, K. Chorneyko, J.M. Bourgeois, C. Hogben and M.A. Tarnopolsky, 2000. Gender differences in muscle inflammation after eccentric exercise. *J. Appl. Physiol.*, 89: 2325-2332.
11. Yu, J.G., D.O. Furst and L.E. Thornell, 2003. The mode of myofibril remodelling in human skeletal muscle affected by DOMS induced by eccentric contractions. *Histochem. Cell. Biol.*, 119: 383-393.

12. Malm, C., P. Nyberg, M. Engstrom, B. Sjodin, R. Lenkei, B. Ekblom and I. Lundberg, 2000. Immunological changes in human skeletal muscle and blood after eccentric exercise and multiple biopsies, *J. Physiol.*, 529: 243-262.
13. Nosaka, K., 2008. Muscle Soreness and Damage and the Repeated-Bout Effect. In Tiidus, Peter M. Skeletal muscle damage and repair. *Human Kinetics*, pp: 59-76.
14. High, D.M., E.T. Howley and B.D. Franks, 1989. The effects of static stretching and warm-up on prevention of delayed-onset muscle soreness, *Res. Q. Exerc. Sport*, 60(4): 357-61.
15. Craig, R. Denegar and H. Perrin, 1992. Effect of Transcutaneous Electrical Nerve Stimulation, Cold and a Combination Treatment on Pain, Decreased Range of Motion and Strength Loss Associated with Delayed Onset Muscle Soreness, *J. Athl. Train.*, 27(3): 200-202, 204-206.
16. Herbert, R.D. and M. Noronha, 2007. Stretching to prevent or reduce muscle soreness after exercise. *Cochrane Database of Systematic Reviews*, Issue 4. Art. No.: CD004577. DOI: 10.1002/14651858.
17. Kokkinos, P., 2009. *Physical Activity and Cardiovascular Disease Prevention*. Jones and Bartlett Learning, pp: 111-112.