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The Effect of Resistance and Progressive Training on HSP 70 and Glucose

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ABSTRACT

Skeletal muscle may develop adaptive chaperone and enhancementdefense system through daily exercise stimulation. The present study investigated resistance and exhaustion training alters the expression of chaper one proteins. These proteins function to maintain homeostasis, facilitate repair from injury and provide protection. Exercise-induced production of HSPs in skeletal muscle and peripheral leukocytes and, it may provide insight into the mechanisms by which exercise can provide increased protection against stressors. The aim of this study was to examine the effect of 2 types exercise trainingon HSP70 expression. Nineteen training female in 2 groups taking part in the intervention volunteered to give blood samples. Levels of chaperone proteins weremeasured in response to resistance and exhaustion training. HSP70 levels were increased, immediately and 2 h after Progressive training but decreased after resistance training. The data showed that human skeletal muscle responds to the stress of a single period of Progressive trainingby up regulating and resistance training by down regulating expression of HSP70. Physical exercise can elevate core temperature and muscle temperatures and the expression pattern of HSP70 due to training status may be attributed to adaptive mechanisms.

Keyword: Heat shock proteins; Exercise training; Immune system; Stressor; Inflammation; Injuries cell.

1. INTRODUCTION

Living cells are continually challenged by conditions which cause acute and chronic stress. Heat shock proteins (HSP) are a class of functionally related proteins whose expression is increased when cells are exposed to elevated temperatures or other stress. Molecular chaperones such as heat shock proteins (HSPs) are known to contribute to reducing cellular damage. HSPs havemultiple functions in maintaining intracellular integrity viaprotection, repair and even control of cell death signaling [1-3]. They play an important role in proteins interactions such as folding and assisting in the establishment of proper protein conformation and pre-

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vention of unwanted protein aggregation. Heat shock proteins (HSP) are increasingly seen as important players in the response of our biochemistry to stresses and damage. Few data have been reported concerning expression of HSPs in human tissue after exercise [4].

HSP70 is associated with the protection of striated muscle from injury and the attenuation of skeletal muscle atrophy. Therefore enhancementof potentially contributes to the protection of myofibers and the maintenance of muscle mass. The expression of HSP70 is reported to be enhanced by thermalstress and exercise [5,6]. Inhuman leukocyteand skeletal muscle, exhaustive endurance exercise [7] and resistance exercise [8] markedly increase HSP70 expression. Togetherthese studies suggest that the expression of molecular chaperone proteins may play important roles in protection and repair of skeletal muscle from exercise induced stress [2]. Puntschart examined the effect of an acute bout of exercise on HSP70 expression and found, the content of HSP70 mRNA increased, but there was no change in the HSP70 protein content. HSP70 was selected because this protein typically demonstrates very large increases after various forms of cellular stress [4]. Detectable levels of HSP70 are present in the systemic circulation of healthy individuals. Previous studies have shown that the systemic concentration of HSP70 is elevated after exercise [9].

Several investigators have demonstrated the induction of HSP72 synthesis in specific regions in response to hyperthermia [10], ischemia, and hypoxia and energy depletion. HSPs are quintessentially viewed as intracellular proteins with a vital role in maintaining cellular homeostasis;important extracellular roles for HSPs have been identified.The depletion of energy stores, hypoxia and ischemia has been shown to induce the synthesis of HSP70 [11]. Prior exercise trainingattenuates contraction-induced injury in skeletal muscle followingan acute single bout of exercise. The acquisition of muscle tolerance to contraction-induced muscledamage through exercise training appears to be partially associatedwith molecular mechanisms including chaperone functions in additionto neuromuscular and morphological adaptations. Several studieshave reported that prolonged exercise training increases several molecular chaperone proteins in skeletal muscle, such as HSP25, HSP70 and glucose-regulated protein (GRP) [2, 5 and 12]. HSPs have protectivefunctions (antioxidation,antiapoptosis) and helping protein formation [3]. These functions of HSPs may potentially contribute to acquisition of a muscledefense system with training [2]. HSPs are increasingly seen as important players in the response of our biochemistry to stresses and damage.

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2. MATERIAL AND METHODS

Nineteen healthy females participated in the study (9 on Progressive training group with mean age 23 ± 2 years [±SEM], body mass 54 ± 3 kg, and maximal oxygen uptake [VO_{2max}] 4.9 ± 0.2 L/ min and 10 on resistance training group with age 20.28 ± 3.18 years [±SEM], weight 57.3 ± 6.53, height 161.1± 3.21 cm, body mass 22.12 ± 1.53 kg, and maximal oxygen uptake[VO_{2max}] 5.1 ± 0.4 L /min). Subjects were informed as to the potential risks associated with participation in the study before obtaining their written informed consent to participate.The study was carried out in accordance with the IAU and approved by the Ethical Committee. The influence of exhaustion training on HSP70 and glucose in peripheral leukocytes evaluated, before and at 0 and 2 h after by flow cytometry and RT/PCR, respectively and glucocard 01.

Progressive training subjects performed Bruce Protocol on a treadmill (T 9700 HRT, USA). The laboratory temperature during all trials was 21 ± 1°C. Resistance training Subjects performed 30 minute resistance training. The laboratory temperature during all trials was 21 ± 1°C. Paired samples of anticubital venous blood were collected in heparinized syringes and kept on ice until analysis for hematocrit on ABL 700 apparatus.

To determine the serum HSP70 protein concentra-

tion were obtained and placed in a tube containing a clot-inducing plug. This tube spun in a centrifuge at 1200 × g at 4°C. A highly sensitive, enzyme-linked immune sorbent assay method was used to determine the concentration of HSP70 protein in serum. All samples were tested in duplicate.All data are expressed as means ± SEM. Statistical analysis was conducted by using analysis of variance (ANOVA) repeated measures and LSD test was used for post hoc multiple comparisons among means. A P value < 0.05 was considered statistically significant.

3. RESULTS

After Progressive, the anticubital serum HSP70 concentration was elevated but after resistance training serum HSP70 concentration was decreased (Table and Figure 1). Repeated Measurers test were significantly in two groups (Table 2) (P>0.05). The changes serum glucose concentration (Figure 2) were significantly in two groups (Table 2) (P>0.05). But LSD test were not significantly. Analysis of variance (ANOVA) were not significantly in two groups (Table3) (P>0.05).

4. DISCUSSION

Exercise is an important intervention in maintaining health. However exhaustion training results in an elevation in constitutive levels of this protective protein in leukocyte [13]. Data suggest that Heat shock proteins (HSP) possibly have a systemic function, including mediation of the effects of exercise on immune function [14]. Increased Hsp70 indicated that the exercise stress caused sufficient intracellular disruption to trigger an HSP response [9]. An increased content of HSPs will facilitate any cellular remodeling, that occurs after unaccustomed exercise [4]. Oxidative and other stresses are an important component of the cellular protective response. These proteins facilitate successful repair from injury and to aid adaptation and remodeling of the cell to prevent the damage [4].Ac-

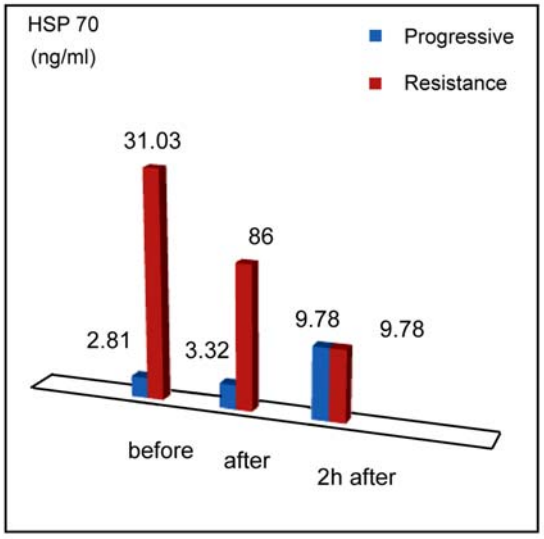

Figure 1: *Mean HSP70 before exercise and in recovery in 2 groups.*

Table 1: *Mean±Std. deviation HSP70 and glucose before exercise and in recovery.*

Variables	Time	N	Mean±Std. Deviation	
			HSP70 (Ng/ml)	Glucose (Mg/dl)
Progressive training	before	9	2.81±1.32	88.63±12.77
	after		3.32±1.74	119.25±26.21
	2h after		9.78±4.25	91.43±5.88
Resistance training	before	10	31.03±18.21	89.77±18.05
	after		19.62±16.05	86.00±7.94
	2h after		9.78±7.26	88.5±11.33

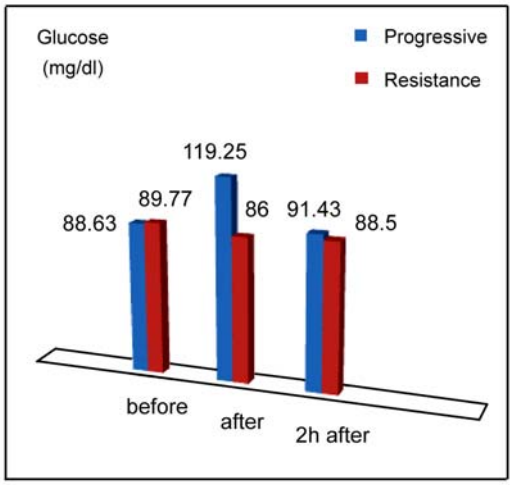

Figure 2: *Mean Glucose before exercise and recovery in 2 groups.*

cumulating evidence strongly suggests that extracellular Hsp72 has potent immune regulatory effects [15]. Temperature increase,oxidative stress and inflammatory reactions after exhaustion exercise were expected to stimulate synthesis of HSPs in peripheral blood leukocytes. Strenuous exercise increased HSP expression in blood immediately at the end of running, which shows a positive function of HSP in leukocytes of athletes to maintain function after heavy exercise [16]. HSP expression is also altered during glucose depletion and oxidative stress. Cells that are starved for glucose over produce a set of proteins called glucose-regulated proteins (GRP). The functions of HSP, GRP and OSP are incompletely understood, but evidence suggests that many stress proteins are enzymes that either provide immediate stress protection or conduct cellular repair processes. Sustained physical activity results in the progressive depletion of glucose and glycogen stores, a phenomenon that is highly correlated with fatigue. In cellsdeprived of glucose or oxygen or treated with agents that perturb calcium homeostasis, synthesis GRP and HSP70 [17].

The elevation of body temperature and depletion of glycogen [11] are all regarded as factors that induce HSP70 expression in skeletal muscle during exercise [18]. When body temperature was maintained during exhaustion exercise, HSP accumulated in peripheral leukocytes. These findings suggest the possibility that the treadmill running, as used in the present study, may stimulate stress response to induce HSP70. We

Table 2: *The results of (ANOVA) repeated measures HSP70 and glucose in 2 groups.*

Time	Sig.	F
before	0.55	4.326
after	0.099	3.096
2h after	0.459	0.59

Table 3: *Table 3: The results of ANOVA HSP70 and glucose in 2 groups.*

Group	Factor	Sig.	F
Progressive training	HSP70	0.04*	7.03
	glucose	.000*	733.470
Resistance training	HSP70	0.002*	23.75
	glucose	.000*	658.91

concluded that exhaustion exercise elevates the resting level of peripheral leukocytes HSP70 and that the resultant accumulation of HSP70 helps to protect stress-loaded cells from injury due to the elevation of chaperone activity [18].

However,comparison amongst studies is complicated by variations in exercise protocol (mode, intensity, durationand damage), muscle group, and differences in subject characteristics (training and nutritional status, age, sex). In resistance training group the down regulation of HSP-positive cells seems to be a result of adaptation mechanisms to training [19].

So the reduction of HSP70 as an indication that exercise training reduces inflammation. The mechanism are involved in systemic low-grade inflammation is not known. It is possible HSPs are leaked into the extracellular compartment due to necrotic cell death [19] HSP70 can be released independently of necrotic cell death in response to a number of stressful conditions including exhaustive exercise [19]. Future investigations will explore the physiological significance of extracellular HSPs.

5. CONCLUSIONS

The data presented show that human leukocyte re-

sponds to the stress of a single period of exhaustion training by up regulating and resistance training by down regulating expression of HSP70. It is possible the HSP 70 response to exercise in relation to the tissue assayed (skeletal muscle, lymphocyte, venous and arterial serum). The differences observed when HSP70 in the present study may be related to the mode of exercise and the amount of protein damage associated with the exercise.

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