

Research article

SPRINT-INTERVAL TRAINING INDUCES HEAT SHOCK PROTEIN 72 IN RAT SKELETAL MUSCLES

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ABSTRACT

Previous studies have demonstrated that endurance exercise training increases the level of heat shock proteins (HSPs) in skeletal muscles. However, little attention has been drawn to the effects of high intensity-short duration exercise, or sprint-interval training (SIT) on HSP72 level in rat skeletal muscles. This study performed to test the hypothesis that the SIT would induce the HSP72 in fast and slow skeletal muscles of rats. Young male Wistar rats (8 weeks old) were randomly assigned to a control (CON) or a SIT group (n = 8/group). Animals in the SIT group were trained (1 min/sprint, 6~10 sets/day and 5~6 days/week) on a treadmill for 9 weeks. After the training period, HSP72 levels in the plantaris (fast) and soleus (slow) muscles were analyzed by Western blotting method. Enzyme activities (hexokinase, phosphofructokinase and citrate synthase) and histochemical properties (muscle fiber type compositions and cross sectional area) in both muscles were also determined. The SIT resulted in significantly ($p < 0.05$) higher levels of HSP72 in both the plantaris and soleus muscles compared to the CON group, with the plantaris producing a greater HSP72 increase than the soleus (plantaris; $550 \pm 116\%$, soleus; $26 \pm 8\%$, $p < 0.05$). Further, there were bioenergetic improvements, fast-to-slow shift of muscle fiber composition and hypertrophy in the type IIA fiber only in the plantaris muscle. These findings indicate that the SIT program increases HSP72 level of the rat hindlimb muscles, and the SIT-induced accumulation of HSP72 differs between fast and slow muscles.

KEY WORDS: Hindlimb, treadmill running, enzyme activity, fiber type shift, hypertrophy.

INTRODUCTION

Numerous studies have shown that heat shock proteins (HSPs) play important physiological roles in both the normal and stressed cell. Indeed, it has been thought that HSPs provide the ability of cellular protection against a variety of stresses (Knowlton, 1997; Locke, 2002). HSPs can be divided into several groups based upon their molecular masses. Of particular interest is the inducible form of the 70 kilo Dalton family of HSPs

(HSP72). The production of the HSP72 in cells is dependent upon both the duration and the intensity of the stressor (Liu et al., 2000; Locke et al., 1990; Milne and Noble, 2002; Skidmore et al., 1995).

In skeletal muscle cells, it has been postulated that the production of HSP72 is necessary to maintain myofibrillar integrity and assist in the assembly of cellular proteins (Liu and Steinacker, 2001; Locke, 1997; Neuffer et al., 1996). Previously, hyperthermia, decreased pH, glycogen depression, changes of calcium ion concentration, oxidative

that the blood lactate concentration from a tail vein was over 6 mM, and an elevation of rat rectal temperature was less than 1°C after completing a single session of the final week program (data are not shown).

Tissue preparation

Forty-eight hours after the training period, animals were anesthetized with pentobarbital sodium (50 mg·kg⁻¹). After reaching a surgical plane of anesthesia, the plantaris and soleus muscles were quickly removed, weighted and frozen with liquid nitrogen and then stored at -85°C until analysis.

Biochemical analysis

Homogenizing procedures: Frozen muscle samples from the right leg were minced and homogenized in ice-cold homogenization buffer (for hexokinase and HSP72, 1 mM EDTA, 50 mM KCl, 50 mM Tris-HCl, pH 7.6; for phosphofructokinase and citrate synthase, 100 mM potassium phosphate, 5 mM EDTA, pH 7.4). Homogenates were centrifuged at 400 g for 15 min and the supernatants were used for analyses of each enzyme activity and HSP72. Total protein concentrations of supernatants were determined using the Bradford method (Bradford, 1976).

Enzyme activities: To determine the enzymatic changes, hexokinase (HK: EC 2.7.1.1) and phosphofructokinase (PFK: EC 2.7.1.11) activities were assayed as an index of glycolytic capacity, and citrate synthase (CS: EC 4.1.3.7) activity was measured for the oxidative capacity. All enzyme activities were measured by spectrophotometer (U-2000, Hitachi, Tokyo, Japan) at 25°C. The assays of HK, PFK and CS activities were based on the methods by Uyeda and Racker (1965), Shonk and Boxer (1963) and Srere (1969), respectively. All assays were duplicated and the mean values were used to evaluate the activities of these enzymes.

HSP72 levels: To determine the levels of HSP72 in the muscles we performed one-dimensional sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) and immunoblotting with the technique described by Naito et al. (2001). Samples containing 20 µg of total protein were loaded into sample wells of 10% precast SDS-PAGE Ready Gel (BioRad, Hercules, CA) and then electrophoresed in running buffer (pH 8.3) using a Bio-Rad Ready Gel Cell at a constant voltage of 200V for 35 min. After the separation, proteins were transferred to nitrocellulose membranes (pore size 0.45 µm, Bio-Rad) using a Bio-Rad Mini Trans-Blot Cell at a constant voltage of 100 V for 60 min. After the

transfer, membranes were blocked for 30 min using blocking buffer (3% gelatin, Tris Buffered Saline, pH 7.5) and then incubated for 2 h with an alkaline phosphatase conjugated monoclonal antibody, which is specific to HSP72 (SPA-810AP, diluted 1:1000, StressGen, BC, Canada). The membranes were subsequently reacted with bromochloroindolyl phosphate-nitro blue tetrazolium substrate (Bio-Rad, CA). All incubations were carried out at room temperature. Quantification of the bands from the immunoblots was performed by computerized densitometry (NIH image ver. 1.63).

Histochemical analysis

For histochemical analysis, a belly of frozen muscle samples from the left leg was sectioned (10 µm) in a cryostat (Minotome, Damon, MA) at -20°C. The sections were stained with the pH sensitivity of myofibrillar adenosintriphosphatase (pH 4.6) (Guth and Samaha, 1969). After the staining, the images of the muscle cross sections were magnified using a light microscope (Olympus BHB, Tokyo, Japan) and stored into a personal computer. Muscle fibers were classified into three major types of Type I, IIA and IIB, and the composition of these fiber types was determined. The fiber cross sectional area of each fiber type was expressed as the average of around 50 muscle fibers. These analyses were performed using NIH image (ver. 1.63).

Statistics

The data are presented as means ± SD. The group differences were analyzed by an unpaired Student's t-test. Statistical significance was set at $p < 0.05$.

RESULTS

Body weight

Body weight at the end of training period was 328 ± 14 g in the CON group and 293 ± 16 g in the SIT group with the SIT group being lower than the CON group ($p < 0.05$).

Muscle weight

Muscle weights in the CON and the SIT groups at the end of training period were 288 ± 28 mg, and 282 ± 18 mg for the plantaris muscle, and 111 ± 9 mg and 125 ± 16 mg for the soleus muscle, respectively. No differences were observed between the two groups in both muscles. The ratio of muscle weight to body weight in the CON and the SIT groups at the end of training period were 0.88 ± 0.07 mg·g⁻¹ and 0.96 ± 0.03 mg·g⁻¹ for the plantaris muscle, and 0.34 ± 0.03 mg·g⁻¹ and 0.43 ± 0.04 mg·g⁻¹ for the soleus muscle, respectively. The ratios

Table 2. Enzyme activities of the plantaris and soleus muscles (n = 8 for each muscle) in the control (CON) and the sprint-interval training (SIT) groups. Values are means (\pm SD).

Muscle	Group	Enzyme activity (nmol·min ⁻¹ ·mg protein ⁻¹)		
		Hexokinase	Phosphofructokinase	Citrate synthase
Plantaris	CON	20.3 (1.7)	303 (43)	191 (8)
	SIT	26.1 (3.5) *	382 (38) *	276 (24) *
Soleus	CON	35.8 (3.6)	118 (25)	323 (23)
	SIT	35.7 (2.5)	135 (22)	342 (24)

* p < 0.05 significantly different from the CON group.

of the SIT group were significantly higher than those of the CON group in both muscles (p < 0.05)

Enzyme activities

The enzyme activities were summarized in Table 2. HK, PFK and CS activities in the SIT group were significantly higher than the CON group in the plantaris muscle (p < 0.05). In the soleus, on the other hand, no differences were observed between the two groups in any of the three enzyme activities.

Histochemical properties

Histochemical properties were shown in Table 3. As for the fiber type composition after the training period, type IIA fiber was significantly higher (p < 0.05) and type IIB fiber was significantly lower (p < 0.05) for the SIT group than the CON group in the plantaris muscle. On the other hand, no significant differences in fiber type composition were observed in the soleus muscle. In fiber cross sectional area, the SIT resulted in a larger cross sectional area only in the plantaris type IIA (p < 0.05).

HSP72 levels

The levels of HSP72 were shown in Figure 1. HSP72 levels were standardized relative to the plantaris muscle of the CON animals. HSP72 level in the SIT group was significantly higher in both the plantaris (p < 0.05) and the soleus muscles (p < 0.05) than the CON group. It should be noted that

the increase in HSP72 after SIT was much greater for the plantaris than for the soleus muscle (the plantaris; 550 \pm 116% vs. the soleus; 26 \pm 8%, p < 0.05, Figure 2).

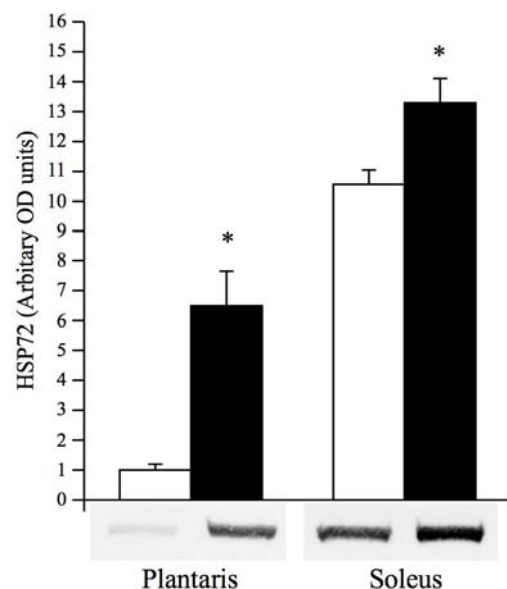


Figure 1. Relative HSP72 levels of the control (open bar) and the sprint-interval training (black bar) groups in the plantaris and soleus muscles. The data of HSP72 in the control (CON) group of the plantaris muscle was used as a standard value. Values are means \pm SD (n = 8 for each group). * p < 0.05 significantly different from the CON group of the same muscle.

Table 3. Histochemical properties of the plantaris and soleus muscles (n = 8 for each muscle except for the SIT in the plantaris muscle [n = 7]) in the control (CON) and the sprint-interval training (SIT) groups. The number of samples of the SIT group in the plantaris was seven since one sample was not in good condition of freezing. Values are means (\pm SD).

Muscle	Group	Fiber type composition (%)			Fiber cross sectional area (μm^2)		
		I	IIA	IIB	I	IIA	IIB
Plantaris	CON	11.2 (1.3)	22.6 (3.6)	66.2 (3.8)	1484 (138)	1363 (114)	2623 (229)
	SIT	11.2 (2.3)	30.2 (4.3) *	58.6 (3.3) *	1488 (191)	1537 (152) *	2694 (265)
Soleus	CON	85.1 (5.7)	14.9 (5.7)	—	2248 (245)	2002 (272)	—
	SIT	83.3 (3.9)	16.6 (3.9)	—	2238 (215)	1891 (271)	—

* p < 0.05 significantly different from the CON group.

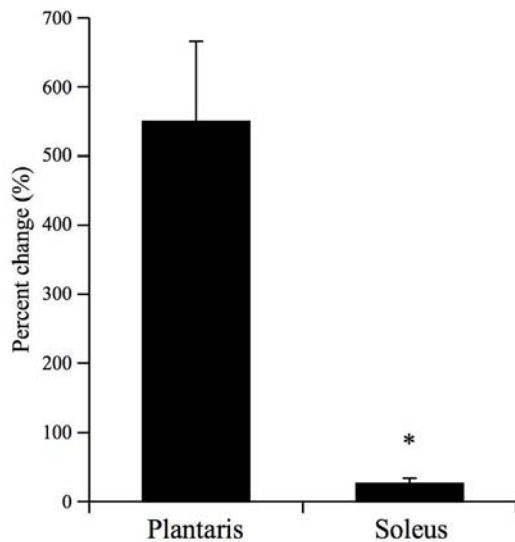


Figure 2. Percent change of HSP72 levels above each control group in the plantaris and soleus muscles after the sprint-interval training. Values are means \pm SD ($n = 8$ for each muscle). * $p < 0.05$ significantly different from the plantaris muscle.

DISCUSSION

All animals in the SIT group ran successfully throughout the SIT program. As the results, the SIT group demonstrated a decrease in the body weight and an increase in the ratio of muscle weight to the body weight in the plantaris and the soleus muscles as compared to the CON group. Furthermore, bioenergetic improvements in both anaerobic and aerobic enzyme activities, fast-to-slow shift of muscle fiber type composition, and hypertrophy in the type IIA fiber were also observed only in the plantaris muscle (Table 2 and 3). These training responses are similar to the those reported in a previous study with similar training protocol (Takekura and Yoshioka, 1990).

It is well known that a prolonged period of physical exercise or endurance training can induce an accumulation of HSP72 in skeletal muscles of rat and human (Desplanches et al., 2004; Liu et al., 2004; Liu et al., 1999; Milne and Noble, 2002; Naito et al., 2001; Skidmore et al., 1995). On the other hand, little attention has been given to the effects of sprint-type training on the accumulation of HSP72 in the skeletal muscles. Therefore, the purpose of this study was to clarify whether sprint interval training (SIT) could induce the accumulation of HSP72 in the plantaris and soleus muscles of rats. Our data indicated that the high intensity-short duration sprint interval training ($\sim 80 \text{ m}\cdot\text{min}^{-1}$) was sufficient to increase the HSP72 in rat skeletal muscles.

In an endurance training program, it is postulated that the primary factor to increase HSP72 in skeletal muscles is a prolonged elevation of the

body temperature (over 3°C above the resting temperature) (Hamilton et al., 2001; Skidmore et al., 1995). In the SIT program, however, we observed that the elevation of rat rectal temperature was less than 1°C although many muscles other than the plantaris and the soleus muscles must be recruited for the sprint running. It was impossible that we could exactly know the rise in working muscles temperature in the rats because we didn't directly measure the temperature in the working muscle. However, it has been reported that the temperature in the working human muscle during prolonged high intensity exercise was slightly higher (about 0.5°C) than the rectal temperature (Saltin and Hermansen, 1966). Furthermore, in the current study, each SIT training session consisted of 6~10 sets of 1 min sprinting, and each set was separated by enough recovery period (2~5 min). Since the temperature of the working muscles with a plenty of blood flow is gradually increasing, it is inconceivable that the temperature of the working muscles would locally elevate and keep over $2\text{--}3^\circ\text{C}$ higher than the rectal temperature during only the 1 min sprint running. In addition, the total exercise duration was no more than 10 min a day and the time exposed for the plantaris and the soleus muscles to hyperthermia would not be so long. Taken together, the impact of SIT-induced changes in muscle temperature would not be so large to induce HSP72 accumulation; therefore, hyperthermia would not be the main contributing factor for the increased HSP72 level in the present study. Instead, it has been reported that sprint exercise requires a higher anaerobic energy supply compared to the endurance exercise (Ross and Leveritt, 2001), and results in a decreased muscle glycogen and a greater accumulation of lactate within the muscle and blood which leads to a decreased pH (McLellan et al., 1990). Indeed, our preliminary data demonstrated that the blood lactate level during the SIT program reached 6 mM, which was over the lactate threshold. Further, the anaerobic enzyme activities were also improved in the plantaris muscle, which was, however not seen in the soleus. Therefore, anaerobic metabolism-related stresses by the SIT, e.g. acidosis and/or a decreased glycogen level in the skeletal muscle, could be the main factors to accumulate HSP72 for the plantaris and soleus muscles.

What is interesting is that the adaptation of HSP72 was clearly different between the plantaris and soleus muscles (Figure 2). Although the levels of HSP72 were significantly increased by the SIT program in both the plantaris and the soleus muscles, the magnitude of the increase in the plantaris was 550% whereas that in the soleus was only 26%. Endurance training studies also showed that the

accumulation of HSP72 was greater in the fast muscle (plantaris) than the slow muscle (soleus) (Desplanches et al., 2004; Naito et al., 2001). It has also been shown that the accumulations of HSP72 after heating the hindlimb ($\sim 42^{\circ}\text{C}$ for 1 h) were 6.8 and 2.2 times higher than the control condition in the plantaris and the soleus muscles, respectively (Oishi et al., 2003). Therefore, the capability of accumulating HSP72 with a certain level of stress seems to be fiber type specific, and it is higher in the fast muscle than the slow muscle. In the present study, the basal level of HSP72 tended to be smaller in the plantaris (fast) muscle (Figure 1). It could be speculated that, following stressful stimuli the magnitude of the increase in the HSP72 levels is greater in the muscles which have low basal level of HSP72. If there is a certain required level of HSP72 in response to training stimuli, there is no doubt that HSP72 levels need to be increased at a greater rate to maintain the cellular homeostasis in the plantaris muscle. In that respect, the soleus with high basal levels of HSP72 would require less amount of increase after SIT.

While the magnitude of the increase in SIT-induced HSP72 levels in the plantaris muscle (550%) seemed much greater than the result of our previous endurance training study ($\sim 94\%$) (Naito et al., 2001), the HSP72 levels in the soleus muscle were very similar between the two studies (26% vs. $\sim 22\%$). One possible explanation for different levels of HSP expression following SIT and endurance training could be the recruitment pattern of muscles during different types of running exercises. Since, the plantaris muscle is predominantly recruited in the SIT type of exercises (Sullivan and Armstrong, 1978), SIT may result in high level of physiological changes specifically in the plantaris muscle. Indeed, our data demonstrates that an improvement of anaerobic enzyme activities and hypertrophy in the type IIA fiber occurred only in the plantaris muscle. Therefore, fast-twitch fibers in the plantaris muscle would be anaerobically recruited during the sprint running, as a result, conditions associated with the induction of HSP as glycogen depletion, hypoxia, lower pH by lactic acid formation, and so on in the muscle cells might be largely changed (Locke, 1997).

The skeletal muscle has a great adaptability to exercise training such as bioenergetic improvements and transformation of contractile proteins (Booth and Thomason, 1991; Booth et al., 1998). The enzymatic improvements, fast-to-slow shift of fiber type proportion and muscle fiber hypertrophy were in fact observed in the plantaris muscle in our current study (Table 1 and 2). These exercise-induced adaptations in skeletal muscle are

accomplished by the cascade of gene activation, transcription and translation associated with each functional protein, and such cascade is followed by the synthesis of newly proteins in trained muscle cells. This synthesis of newly protein is accomplished by the assist of molecular chaperon that is HSP72 (Liu and Steinacker, 2001; Locke, 1997; 2002). The accumulation of HSP72 was, therefore, a fundamental phenomenon for the training adaptations including the enzymes or contractile proteins to take place.

Physical exercise training causes various stress conditions on muscle cells, including thermal, metabolic, mechanical and oxidative stress (Liu and Steinacker, 2001; Locke, 1997). Our data suggested that without having major hyperthermia, the anaerobic stresses induced by SIT might result in HSP72 accumulations in rat skeletal muscles. The intensity of mechanical stress may also affect the alteration in the level of HSP72 because the magnitude of HSP72 increase in the plantaris is much higher following SIT than that of the endurance training. However, it is not clear how these associated stresses with the SIT interacted to yield our findings. Therefore, further studies may be warranted to evaluate the influence of each factor to accumulate HSP72 with monitoring exactly the changes of temperature in the working muscles during the SIT.

CONCLUSION

The SIT program ($\leq 10 \text{ min}\cdot\text{day}^{-1}$) induced an accumulation of HSP72 in rat skeletal muscles. The SIT-induced accumulation of HSP72 in the fast muscle was greater than that of the slow muscle.

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KEY POINTS

- There is no study about the effects of high intensity but short duration exercise, or sprint-interval training (SIT) on heat shock protein 72 (HSP72) level in skeletal muscles.
- The SIT program ($\leq 10 \text{ min} \cdot \text{day}^{-1}$) accumulated HSP72 in rat skeletal muscles.
- The SIT-induced accumulation of HSP72 in the plantaris (fast) muscle was drastic compared to the soleus (slow) muscle and accompanied with the improvements of enzyme activities, fast-to-slow shift within fast muscle fiber type and muscle hypertrophy.

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