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Maintenance of myoglobin concentration in human skeletal muscle after heavy resistance training

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Abstract The aim of this study was to determine the effects of 8 weeks of resistance training (RT) on the myoglobin concentration ([Mb]) in human skeletal muscle, and to compare the change in the [Mb] in two different RT protocols. The two types of protocol used were interval RT (IRT) of moderate to low intensity with a high number of repetitions and a short recovery time, and repetition RT (RRT) of high intensity with a low number of repetitions and a long recovery time. A group of 11 healthy male adults voluntarily participated in this study and were divided into IRT ($n = 6$) and RRT ($n = 5$) groups. Both training protocols were carried out twice a week for 8 weeks. At the completion of the training period, the one-repetition maximal force values and isometric force were increased significantly in all the subjects, by about 38.8% and 26.0%, respectively ($P < 0.01$). The muscle fibre composition was unchanged by the 8 weeks of training. The muscle fibre cross-sectional areas were increased significantly by both types of training in all fibre types (I, IIa and IIb, mean +16.1%, $P < 0.05$). The [Mb] showed no significant changes at the completion of the training [IRT from 4.63 (SD 0.63) to 4.48 (SD 0.72), RRT from 4.47 (SD 0.75) to 4.24 (SD 0.80) mg · g^{-1} wet tissue] despite a significant decrease in citrate synthase activity [IRT from 5.27 (SD 1.45) to 4.49 (SD 1.48), RRT from 5.33 (SD 2.09) to 4.85 (SD 1.87) μmol · min^{-1} · g^{-1} wet tissue; $P < 0.05$] observed after both protocols. These results suggested that myoglobin and mitochondria enzymes were regulated by different mechanisms in response to either type of RT. Moreover, the maintained [Mb] in hypertrophied muscle should preserve oxygen transport from capillaries to mitochondria even when diffusion distance is increased.

Key words Citrate synthase activity · Human · Myoglobin concentration · Protocols · Resistance training · Vastus lateralis muscle

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Introduction

Resistance training (RT) has been a commonly used method for increasing the strength, speed and power of muscle effort, often by using free weights, straps, pulleys, springs or oil hydraulic devices. It has been stated that RT protocol can be varied in five ways – choice of exercise, order of exercise, intensity used, number of sets and recovery time between sets (Kraemer et al. 1988). It has been shown that the specific type of RT protocol results in adaptations of the physiological mechanisms that are activated by and stimulated in the exercise (Kraemer and Fleck 1988). Specific physiological adaptations of muscle can be observed typically in bodybuilders and power-lifters, who have been shown to use different protocols of RT (Westcott 1995). The RT protocol used by bodybuilders is of low-to-moderate intensity, has a higher number of repetitions and a short recovery time, features that emphasize muscle hypertrophy. The RT used by power-lifters is of high intensity with a lower number of repetitions and a long recovery time, in which the improvement of motor unit activation is the target rather than muscle hypertrophy.

Tesch et al. (1984) have reported that Olympic power-lifters displayed lower capillary density (CD) than non-athletes, and bodybuilders demonstrated the same CD as non-athletes and a somewhat greater number of capillaries around muscle fibres. They have also

observed that the mitochondorial enzyme activity of the bodybuilders was at levels similar to those of non-athletes, whereas that of the power-lifters was lower than that of the non-athletes) Tesch et al. 1984). Choi et al. (1998a, 1998b) have administered 8-week RT to sedentary individuals and examined the effect of two types of RT protocol (that of bodybuilders and that of power-lifters) on muscle hypertrophy and capillarization. They have observed that CD tended to be decreased by both protocols, but that the capillary-to-fibre ratio (C/F ratio) was increased more by the bodybuilder protocol than by the power-lifter protocol (Choi et al. 1998a). In other experiments, a circuit-type of high intensity interval exercise has brought about an increase in maximal oxygen uptake ($\dot{V}O_{2max}$) and maximal cardiac output in active adults (Petersen et al. 1989) and active young boys (Docherty et al. 1987). These reports have implied that the bodybuilding RT protocol (including circuit interval RT) can stimulate muscle oxidative mechanisms.

The transport of oxygen into muscle is controlled by an extensive network of mechanisms and influences. Myoglobin (Mb) has been thought to relate to oxidative metabolism and to have several roles – facilitated diffusion, oxygen storing and mediation of oxidative phosphorylation (Wittenberg 1970; Kagen 1973; Cole 1982; Gayeski et al. 1985; Wittenberg and Wittenberg 1989), and the Mb concentration ([Mb]) has been found to be associated with mitochondrial enzyme activity (Wittenberg 1970) and with the capillary supply (Reis and Wooten 1970). The adaptation of Mb in response to exercise has been studied solely in conditions of endurance training in animals (Hickson 1981; Hickson and Rosenkoetter 1981; Harms and Hickson 1983; Beyer and Fattore 1984; Hickson et al. 1984) and humans (Jansson and Sylvén 1981; Jansson et al. 1982, 1983; Möller and Brandt 1982; Svedenhag et al. 1983; Sylvén et al. 1984). The [Mb] in rat skeletal muscles has been shown to be increased by endurance training, although that in human skeletal muscle was not. To our knowledge, no previous study has examined the adaptation of [Mb] of human skeletal muscle to RT.

We have observed that the [Mb] of the rat plantaris muscle was maintained at the control level during compensatory hypertrophy induced by ablation of the gastrocnemius muscle (Masuda et al. 1997). This result implied that the increase in Mb protein in the whole plantaris muscle corresponded to the change in muscle cell size. The compensatory activity in the ablation model consisted of high-load and endurance elements, which seemed to have effects similar to those of bodybuilder RT in humans rather than power-lifter RT. Therefore, different protocols of RT may cause specific adaptations of Mb which represent characteristics of these exercise types; the bodybuilder RT may maintain [Mb] at the initial level whereas the power-lifter RT may decrease [Mb], which would be similar to the changes in capillarization that have been produced by these protocols in humans (Tesch et al. 1984; Choi et al. 1998a).

The aim of the present study was to examine the effects of RT on [Mb] in human skeletal muscle and to determine whether different protocols (those of bodybuilders and those of power-lifters) influence the Mb adaptation.

Methods

Subjects

A group of 12 healthy male adults who showed no evidence of neuromuscular diseases participated in this study. They had not been engaging in any regular exercise in their daily lives. They were informed of the content and risk of the experiment in advance and gave written agreement to participate voluntarily in the experiments. These subjects were divided into two groups: those undergoing the bodybuilder protocol (interval RT group, IRT, $n = 6$) and those performing the power-lifter protocol (repetition RT group RRT, $n = 6$). The final number of subjects was 11 (IRT $n = 6$, RRT $n = 5$) because 1 subject of the RRT group dropped out during the training period. The mean age, height and body mass for the IRT group were 28.5 (SD 4.0) years, 173.5 (SD 1.6) cm and 70.7 (SD 5.0) kg, respectively, and for the RRT group were 27.6 (SD 3.7) years, 173.6 (SD 6.1) and 69.8 (SD 5.1) kg, respectively. There were no significant differences in these physical characteristics between the groups.

Performance test

For the assessment of maximal muscle contraction force, each subject performed a test of one-repetition maximal force (1 RM) and isometric force during concentric knee extension before and after RT. In addition, the subjects performed 50 continuous contractions at $180° \cdot s^{-1}$ of angular velocity using an isokinetic machine (Biodex, Biodex Co., U.S.A). The torque obtained during the 50 repetitive contractions was normalized by the values of the first contraction. The relative torque decrease during the 50 contractions was calculated for each subject, and these values were used as an assessment of muscle endurance performance as has been described by Choi et al. (1998b).

Training protocol

Prior to RT, the 1 RM and maximal isometric force were determined in all the subjects. The exercise used for RT was the concentric knee extension of the subject's right leg while seated on a chair. Both the IRT and RRT protocols were performed twice a week for 8 weeks. In the RRT protocol, the exercise intensity and the number of trials were set at 90% of 1 RM and five trials, respectively. In the IRT protocol, the intensity was changed from 80% to 40% of 1 RM over the course of nine trials. The subjects of both groups performed the knee extension until they were too fatigued to continue. Both RT protocols were based on those described by Fleck and Kraemer (1987) and Westcott (1995) and are summarized in Fig. 1.

Preparation of human skeletal muscle

A specimen of the vastus latelaris skeletal muscle of each subject was obtained by the needle biopsy technique (Bergström 1962) before and after the 8-week RT period. The muscle samples were taken from the distal part of the right vastus lateralis. These muscle specimens were frozen in liquid nitrogen immediately after resection, and then stored at −80 °C until the histochemical and biochemical analyses.

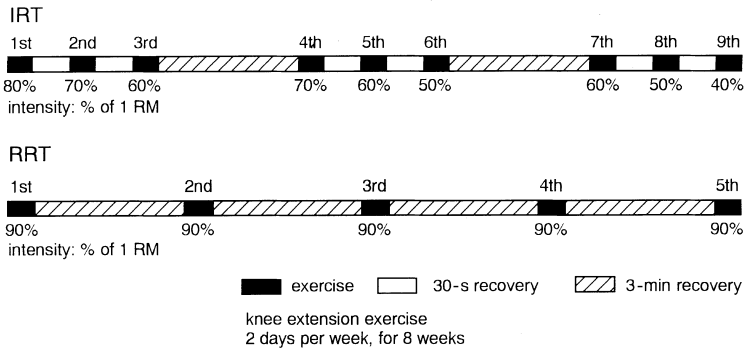

Fig. 1 The experimental interval (*IRT*) and repetition resistance training (*RRT*) protocols. *IRM* one repetition maximal force

Histochemical assay

Tissue samples were mounted and frozen in isopentane pre-cooled with liquid nitrogen. Transverse serial sections (10-μm thick) were cut by a cryostat at −20°C and placed on a cover glass for histochemical staining. Sections were stained for myosin adenosine triphosphatase after pre-incubation at pH 4.6 and 10.3 (Brooke and Kaiser 1970). The muscle fibre types were classified into types I, IIa and IIb. The muscle fibre composition was measured for an average of 580.3 (SD 378.2) fibres per subject. The mean muscle fibre cross-sectional area (CSA) and each fibre type CSA were calculated for (an average of 46.1 (SD 24.8) fibres.

Biochemical assay

The remaining part of the biopsy muscle specimens was used for the biochemical assays. A sample of between approximately 10–20 mg was homogenized with 0.7 ml of pre-cooled 0.04 mol · l^{-1} phosphate buffer (pH 7.2). After centrifugation at 15,000 *g* for 5 min, the supernatant was diluted 1:100 with the same phosphate buffer. The [Mb] was determined by an agglutination method kit with latex fixation, which had been developed for the diagnosis of serum [Mb] (Mb-Latex "SEIKEN", SEIKEN Co., Tokyo, Japan). This method was essentially based on an immunobiochemical reaction using anti-human myoglobin bound with latex.

A spectrophotometric method as has been described by Srere (1969) was used to determine the citrate synthase (CS) activity of oxidative phosphorylation in the mitochondria.

Statistical analysis

To compare the pre-training values with the post-training values in each group, we used the paired Student's *t*-test. For the comparison of changes (%) produced by IRT with those produced by RRT, we used the unpaired Student's *t*-test. The level of significance was set at $P < 0.05$.

Results

The maximal voluntary forces are shown in Table 1. The 1 RM values were increased from 52.9 (SD 6.8) kg to 71.3 (SD 6.3) kg in the IRT group (+34.8%) and from 59.0 (SD 8.2) kg to 84.5 (SD 12.4) kg in the RRT group (+43.2%) by the 8-week RT. The maximal isometric force also increased significantly from 252.9 (SD 49.0) N · m to 301.8 (SD 53.5) N · m in the IRT group (+19.3%) and from 255.7 (SD 60.2) N · m to 348.5 (SD 78.3) N · m in RRT (+36.2%). The gain in maximal force tended to be greater in the RRT group than the IRT group. The muscle endurance performance, evaluated by the 50-times contraction test, was unchanged in the RRT group [68.9 (SD 4.6)% to 67.2 (SD 6.6)%] and was significantly improved in the IRT group [62.4 (SD 6.9)% to 51.2 (SD 6.4)%, $P < 0.05$] after RT.

The [Mb] were observed to decrease from 4.63 (SD 0.63) to 4.48 (SD 0.72) mg · g^{-1} wet tissue (−3.2 %) in the IRT group and from 4.47 (SD 0.75) to 4.24 (SD 0.80) mg · g^{-1} wet tissue (−5.1%) in the RRT group, but these changes were not significant (Table 1). In contrast, the CS activity was significantly decreased from 5.27 (SD 1.45) to 4.49 (SD 1.48) μmol · min^{-1} · g^{-1} wet tissue (−14.8%) in the IRT group and from 5.33 (SD 2.09) to 4.85 (SD 1.87) μmol · min^{-1} · g^{-1} wet tissue (−9.0%) in the RRT group after the 8-week RT ($P < 0.05$, Table 1).

Table 1 Maximal voluntary force, and muscle endurance performance, myoglobin concentration ([Mb]) and citrate synthase (*CS*) activity before (*pre*) and after (*post*) the 8 weeks of resistance training. *1 RM* One repetition maximal force, *IRT* interval resistance training, *RRT* repetition resistance training. Ratio of torque decrement is the relative change of torque during 50 repetitive contractions

	n		1 RM (kg)		Isometric force (Nm)		Ratio of torque decrement (%)		[Mb] (mg · g^{-1} wet tissue)		CS activity (μmol · min^{-1} · g^{-1} wet tissue)	
			Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
IRT	6	Pre	52.9	6.8	252.9	49.0	62.4	6.9	4.63	0.63	5.27	1.45
		Post	71.3	6.3**	301.8	53.5*	51.2	6.4*	4.48	0.72	4.49	1.48*
RRT	5	Pre	59.0	8.2	255.7	60.2	68.9	4.6	4.47	0.75	5.33	2.09
		Post	84.5	12.4**	348.5	78.3**	67.2	6.6	4.24	0.80	4.85	1.87*

Asterisks represent significant differences from pre-training, *$P < 0.05$, **$P < 0.01$

Table 2 Muscle fibre composition and muscle fibre cross-sectional area (*CSA*) before, (*pre*) and after (*post*) 8 weeks of resistance training. Definitions as in Table 1

	n		% Type I fibres (%)		% Type IIa fibres (%)		% Type IIb fibres (%)		Type I CSA (μm^2)		Type IIa CSA (μm^2)		Type IIb CSA (μm^2)	
			Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
IRT	6	Pre	32.0	11.4	44.9	4.1	23.1	11.4	4461.8	1261.1	4685.8	1404.7	4189.2	1439.9
		Post	37.2	9.7	42.8	8.8	20.1	8.0	5063.1	1143.2*	5551.0	1132.9*	4838.2	1282.2*
RRT	5	Pre	30.4	20.3	51.2	8.8	18.3	11.5	5596.3	837.6	4945.0	511.5	3990.2	951.0
		Post	32.0	4.6	48.5	5.5	19.6	4.3	6063.5	812.6*	5698.9	628.2*	4459.9	1149.4*

Asterisks represent significant differences from pre training, $*P < 0.05$

The muscle fibre compositions did not show any significant changes following either type of RT, and there was no significant difference in the fibre compositions between the groups (Table 2). The mean muscle fibre CSA increased by approximately 16.1% in response to the 8-week RT. The type I fibre CSA was enlarged by 13.5% in the IRT group and by 8.3% in the RRT group. The type IIa fibre CSA also increased, by 18.5% in the IRT subjects and by 15.2% in the RRT subjects. The type IIb fibre CSA increased by 15.5% in the IRT group and by 11.8% in RRT compared to the pre-training values. The IRT tended to produce greater gains in the type I fibre and type IIb fibre CSA compared to RRT.

Discussion

There have been numerous studies regarding the physiological adaptation of muscles to RT (see Kraemer et al. 1996). However, no information has been available about the adaptation of Mb to RT in humans. In our recent study of the [Mb] during compensatory hypertrophy in rats (Masuda et al. 1997), we have observed that the [Mb] of the plantaris muscle was maintained at the control level during the compensatory hypertrophy. These results, however, cannot be directly applied to human muscle since a discrepancy has been observed between the Mb adaptations to exercise training in humans (Jansson and Sylvén 1981; Jansson et al. 1982, 1983; Möller and Brandt 1982; Svedenhag et al. 1983; Sylvén et al. (1984) and in animals (Hickson 1981; Hickson and Rosenkoetter 1981; Harms and Hickson 1983; Beyer and Fattore 1984; Hickson et al. 1984); the [Mb] of animal skeletal muscle was increased by endurance training but that in humans was not. Therefore, the effects of RT on the [Mb] in humans have not yet been described. Since changes in muscle strength, hypertrophy, capillarization and enzyme activities induced by RT have been shown to vary depending on the protocol of RT used (Kraemer and Fleck 1988), the adaptation of Mb in human skeletal muscle must also be examined using different protocols.

In the present study, however, the [Mb] after 8-week RT did not change and did not differ as a result of the two RT protocols used. This phenomenon could be interpreted as showing that the Mb protein synthesis was enhanced by both RT protocols which would not have supported our hypothesis that the [Mb] would show specific adaptation according to the protocol used, since we have previously observed greater muscle hypertrophy and a greater increase in C/F ratio in an IRT group compared to an RRT group (Choi et al. 1998a, b). Therefore, the question arises as to why this Mb upregulation was not dependent on the RT protocol but was actually similar in both types of RT. Several factors, which are not related to the training protocols, should be considered regarding the upregulation of Mb synthesis by RT.

The adaptation of Mb may not show changes parallel to those of other oxidative mechanisms as a consequence of RT. In this study the [Mb] was maintained, but the CS activities in both the IRT and RRT groups decreased as a response to RT. The decrease in CS activity could be interpreted as a dilution effect by muscle hypertrophy and the mitochondria enzymes may not have been stimulated sufficiently by RT. Similar results have been obtained regarding other mitochondrial enzyme activities and mitochondrial volume density (MacDougall et al. 1979; Tesch 1988). With regard to capillarization, in a similar protocol a greater increase in C/F ratio has been observed in an IRT group compared to an RRT group (Choi et al. 1998a, b), although the IRT group showed greater muscle hypertrophy than the RRT group. However, the changes in C/F ratio did not correspond to those in muscle fibre CSA in both groups but actually C/F ratio showed less change than the muscle fibre CSA (Choi et al. 1998a). In this case, because the capillary domain area must increase in the hypertrophied muscle, the capacity for oxygen supply of capillary to active muscle should be diminished. Therefore, it could be speculated that the Mb adaptation as a result of RT compensated for the transport of oxygen from capillary to mitochondria, and to enhance the oxygen flux into hypertrophied muscle cells. Furthermore, Mb, mitochondrial enzyme activity and capillaries are regulated by different mechanisms and influences.

The Mb may not influence whole body aerobic capacity in RT. There have been several studies in which chronic adaptations to RT have occurred of whole body

aerobic capacity and the cardiovascular system (Hickson et al. 1980; Docherty et al. 1987; Murray et al. 1989; Petersen et al. 1989; Stone et al. 1991). The circuit-type of intense exercise (similar to IRT) has been demonstrated to induce an increase in $\dot{V}O_{2\,max}$ (Docherty et al. 1987; Petersen et al. 1989), whereas high-intensity RT (similar to RRT) has not increased $\dot{V}O_{2\,max}$ (Hickson et al. 1980). Stone et al. (1991) have shown that cardiac output of power-lifters increased but not of sedentary subjects during RT exercise. In addition, bodybuilders have been shown to have a significantly greater cardiac output and stroke volume but not heart rate, compared to power-lifters (Murray et al. 1989). Thus, the differences in whole body aerobic capacity ($\dot{V}O_{2\,max}$) brought about by the RT protocol should be affected by the differences in adaptations of the *central* determinants according to the protocol employed.

The hypoxic condition of muscle has been considered to be one of the factors enhancing Mb synthesis. High-altitude natives have been found to have higher [Mb] than sea-level natives (Reynafarje 1962; Wittenberg 1970), and endurance training in a hypoxic simulators has been shown to cause an increase in the [Mb] in humans (Terrados et al. 1990). On the other hand, recent work has shown that lower mitochondria volume density and lower mitochondria enzyme activities occurred in altitude-born natives compared with sea-level controls, which implied that chronic exposure to hypoxia did not always increase mitochondria capacity (Kayser et al. 1991, 1996).

During intense RT exercise with a load of 50% maximal voluntary contraction or higher, it has been found that peripheral occlusion occurs in a contracting muscle and oxygen supply through the capillaries to an active muscle will be prevented at every contraction because of the high internal pressure of the contracting muscle (Zwarts and Arendt-Nielsen 1988). Under these conditions, the contracting muscle has to use stored oxygen that is bound to Mb during the contractions, and it could be difficult for the muscle to use further oxygen to produce adenosine triphosphate. Therefore, the Mb synthesis found in the present study may have been brought about by the hypoxic condition of muscle cells during the forceful muscle contraction.

A morphometric change in muscle could be considered as another major factor, which might cause an increase in Mb content. Hypertrophy in muscle fibre creates a greater diffusion distance from the capillary to the centre of the muscle cell. Hypoxia and a lack of stored oxygen in hypertrophied muscle cells can occur because the [Mb] would decrease in these muscle cells if the Mb content remained unchanged. Therefore, Mb synthesis may have been enhanced as a result of the change in the size of the muscle cells. Thus, this adaptation of Mb may have depended on a morphometric change in the muscle cells and not on the protocol of training.

The effect of muscle fibre transformation on [Mb] may have been quite small in the present study. It has been accepted that more Mb is present in type I than in type II fibres (Jansson and Sylvén 1983; Nemeth and Lowry 1983; Kunishige et al. 1996). Thus, it has been speculated that if the changes in muscle fibre composition (increase in percentage of type I fibres) induced by the present RT were marked and if the changes depended on the training protocol, the effects of muscle fibre composition would be reflected in [Mb]. In the present study, however, there were no changes in the muscle fibre compositions after 8 weeks of either IRT or RRT. Previous studies have demonstrated a muscle fibre transformation from type IIb to type IIa fibres as a result of RT (Hather et al. 1991; Staron et al. 1994). Muscle fibre transformation from type IIa to type I fibres due to RT has seldom been reported. The discrepancy in the results regarding composition changes in the muscle fibres between this and previous studies could be due to the shorter RT period and lesser frequency of training sessions in this study compared to other studies. An influence of muscle fibre transformation would thus not be involved in the [Mb] changes.

In conclusion, the 8 weeks of RT using different protocols (those of power-lifters and those of bodybuilders) did not cause a significant change in [Mb] of human skeletal muscle, even though hypertrophy of the muscle fibres and a decrease in CS activity occurred. This Mb adaptation was not specific in response to the different RT protocols. Therefore, the capacity for storing oxygen, facilitating of oxygen diffusion and oxygen transport from capillaries to mitochondria that are mediated by Mb was not altered even in the hypertrophied muscles in our subjects.

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