J. Phys. Ther. Sci.
23: 349–355, 2011

Effect of Muscle Strength Training and Muscle Endurance Training on Muscle Deoxygenation Level and Endurance Performance

KEITA UCHIYAMA, RPT, MS[1,2], HIROICHI MIAKI, RPT, PhD[3], SHIGERU TERADA, RPT[1,2], MASAHIRO HOSO, MD, PhD[3]

[1]*Department of Rehabilitation, Kanazawa Red Cross Hospital: 2–251 Minma, Kanazawa, Ishikawa 921-8161, Japan. TEL: +81 76-242-8131, FAX: +81 76-243-7552, E-mail: reha@kanazawa-rc-hosp.jp*
[2]*Doctoral Course of Rehabilitation Science, Division of Health Sciences, Graduate School of Medical Science, Kanazawa University*
[3]*Division of Health Sciences, Graduate School of Medical Science, Kanazawa University*

Abstract. [Purpose] The purpose of this study was to compare the effects of muscle strength training and muscle endurance training on muscle deoxygenation level and endurance performance. [Subjects and Methods] Nineteen healthy young men were randomly assigned to a muscle strength training (STR: n = 6) group, muscle endurance training (END: n = 6) group, or a control (CON: n = 7) group. The training intensity for STR was 60°/sec × 10 repetitions × 5 sets/day and that for END was 240°/sec × 50% fatigue repetitions × 2 sets/day, 3 days/week, for 6 weeks. All subjects performed cardiopulmonary exercise testing (CPX) to measure maximum oxygen uptake, exercise time and muscle deoxygenation level of vastus lateralis, and underwent muscle strength and muscle endurance measurements pre- and post-training. [Results] In the STR group, muscle strength tended to increase, while muscle endurance significantly increased in the END group. Muscle deoxygenation level was significantly increased in both training groups. Maximum oxygen uptake did not change; however, in the END group alone, exercise time was significantly prolonged. [Conclusion] These results suggest that muscle endurance training is more effective at increasing endurance performance than muscle strength training.
Key words: Muscle endurance training, Muscle deoxygenation level, Exercise time

(This article was submitted Oct. 15, 2010, and was accepted Nov. 5, 2010)

INTRODUCTION

It has been shown that muscle oxidative capacity, measured by skeletal muscle mitochondrion content and oxidative enzyme activity, is decreased with hypoactivity owing to cardiac disease, respiratory disorder, and ageing[1–3]. As a consequence, endurance performance, e.g. maximum oxygen uptake (peak VO_2) and exercise time, declines.

On the other hand, it has been reported that exercise training increases skeletal muscle mitochondrion content and oxidative enzyme activity[4], increasing oxygen extraction from arterioles and capillaries in muscle. Consequently, endurance performance improves with an increase in aerobic adenosine triphosphate production and a decrease in lactic acid production in muscle and blood during exercise[5]. However, it is currently unclear which training is the most effective at increasing oxygen extraction.

Recently, near-infrared spectroscopy (NIRS) has been used to evaluate oxygen extraction of skeletal muscle. NIRS can monitor the tissue oxygenation of the region of interest, non-invasively and continuously, through different absorptions of near-infrared light in the wavelength region of 700–1000 nm by oxygenated hemoglobin/myoglobin (oxy-Hb/Mb) and deoxygenated hemoglobin/myoglobin (deoxy-Hb/Mb)[6–7]. Since this technique was introduced by Jobsis in 1977[8], who monitored the changes in the absorption of light in cats and humans, it has been widely applied in various clinical situations and experiments, which have confirmed its validity and reliability[9,10]. In 1992, Chance et al. monitored the skeletal muscle oxygenation during exercise[11]. Many researchers have used this technique to evaluate the oxygenation in brain and muscle during dynamic exercise, such as incremental and constant work rate exercise. However, there is limited evidence for the effects of exercise training on muscle oxygenation responses during exercise[6], and furthermore, to our knowledge, there is no evidence based on comparisons of the effects among two or more different training groups.

In this study, we hypothesized that muscle endurance training could improve muscle deoxygenation level and endurance performance, but muscle strength training could not. The purpose of this study was to compare the effects of muscle strength training (STR) and muscle endurance

training (END) using muscle deoxygenation level, measured by NIRS, and endurance performance (peak VO_2 and exercise time).

SUBJECTS AND METHODS

Subjects

Nineteen young men (age = 22.9 ± 2.3 yr) who gave their written informed consent participated in this study. All subjects were healthy with no known orthopedic or cardiorespiratory disease. Participants were randomly assigned to a muscle strength training (STR: n = 6) group, a muscle endurance training (END: n = 6) group, or a control group (CON: n = 7). Their physical characteristics and fitness habits are shown in Table 1. All subjects answered our questionnaires about activity in daily life and were instructed to continue with their activities as usual until their training was completed. Subjects were also given instructions to report to us when they performed other physical exercise. This study was approved by Kanazawa University Ethics Committee.

Methods

All subjects performed incremental exercise using an upright electromagnetically braked cycle ergometer (Rehcor 500P, Lode B.V, Netherlands) to determine muscle deoxygenation level, peak VO_2, and exercise time before and after training. The height of the saddle was set to flex the knee joint of each subject at 40° at the bottom dead center of the pedal. After a warm-up exercise of 3 min at 10 W, incremental exercise began, which increased progressively by 20 W every minute. Subjects were instructed to maintain a speed of 60 revolutions per minute (rpm) during the exercise. The incremental exercise was terminated when subjects could not maintain 50 rpm. Exercise time was determined from the start to the finish of the incremental exercise. Heart rate was monitored continuously with an electrocardiogram (DynaScope DS-2202, Fukuda Denshi, Japan) during the exercise. Blood pressure was also measured using a mercury sphygmomanometer at 1 min intervals.

Expired gas analysis (AE-300S, Minato Medical Science Co., Ltd., Japan) was used to measure oxygen uptake (VO_2) and carbon dioxide output (VCO_2) during incremental exercise. This equipment can continuously monitor VO_2 and VCO_2 using a breath-by-breath method under the control of a microcomputer, which integrated a gas analyzer (O_2 and CO_2 concentration meter) and a respiratory flow meter. Gas meter calibration was performed using standardized gas of known concentration before each test. The collected VO_2 data was averaged every 8 sec and peak VO_2 was defined as the average VO_2 value during the last 30 sec of the test. All subjects were instructed not to take caffeine, alcohol, or to smoke for one day before the test. In addition, they were prohibited from eating and drinking (only water was permitted) for 2 hours before the test on the test day.

Muscle deoxygenation level was assessed using a NIRS device (OM-220, Shimadzu Co., Japan). This device is composed of a main computer and a probe, and the probe has a light source and two detectors. The light source emits near-infrared light at two different wavelengths (760 nm and 830 nm) in turn, which penetrates the biological tissue with scattering and absorption and then reaches the detectors. This device can evaluate the hemoglobin concentration in biological tissue by a spatially resolved method based on diffusion theory. The distances between the emitter and two detectors are 2.5 cm and 4.0 cm, respectively. It has been shown that the depth of penetration of the light can be calculated by the distances between emitter and detector[12]. In this device, the depth is considered to be 20–30 mm.

The probe was placed on the VL muscle of the non-dominant leg, which is the agonist in cycling[13], 5 finger-breadths proximally from patella and on the outside of the thigh. To prevent misalignment of the probe during the exercise and irruption of light from outside, the probe was fixed on the site of interest using black packing tape and Velcro.

Generally, a spectral photometer can quantify the concentration of materials on the basis of the Beer-Lambert Law when a specimen is transparent and dilute, and the path length and extinction coefficient of the materials are known. However, it is impossible to measure the path length of the light in biological tissue because it is a scattering media. Therefore, it is difficult to evaluate the absolute value of tissue oxygenation[6–7]. In this study, therefore, muscle deoxygenation level was normalized by the femoral artery occlusion method[14] before incremental exercise. A tourniquet was twisted around the proximal femur vertically, then each subject was subjected to pressure at 300 mmHg for about 7 min until their changes in oxy-Hb/Mb and deoxy-Hb/Mb (Δoxy-Hb/Mb and Δdeoxy-Hb/Mb, respectively) became constant. Because deoxy-Hb/Mb can be regarded as being essentially insensitive to change in blood volume during occlusion and exercise[15], we employed this to indicate muscle deoxygenation level.

Muscle deoxygenation level was calculated by the following equation:

Muscle deoxygenation level (%) = ΔEx/ΔOc (see Fig. 1),

where ΔEx is the change in Δdeoxy-Hb/Mb during incremental exercise from start to maximum value and ΔOc is the change in Δdeoxy-Hb/Mb during occlusion from start to maximum value. The signal was recorded at a sampling frequency of 1 Hz and averaged every 10 sec.

Knee extensor strength and endurance of the non-dominant leg were measured using an isokinetic dynamometer (Cybex Norm, Cybex International Inc., USA) 3–7 days after CPX. Subjects were seated and securely strapped at the pelvis, trunk, and thigh. The axis of the dynamometer was aligned at the pivot of the knee joint. First, muscle strength measurement was performed for 3 trials at an angular velocity of 60°/s[16]. Ten minutes later, muscle endurance measurement was performed for 60 trials at 240°/s[16].

Peak torque obtained from muscle strength measurement was divided by body weight. Muscle endurance was determined by the endurance ratio, which was computed by dividing the total work from the second half of the

Table 1. Physical characteristics of subjects pre-training

		CON (n = 7)	STR (n = 6)	END (n = 6)
Age (years)		21.7 ± 1.9	23.0 ± 2.0	24.2 ± 2.6
Height (m)		1.70 ± 0.07	1.76 ± 0.05	1.76 ± 0.04
Body weight (kg)		59.3 ± 9.8	64.9 ± 9.3	64.4 ± 9.8
Body mass index (kg/m^2)		20.3 ± 1.8	21.0 ± 2.1	20.7 ± 2.9
Body fat percentage (%)		15.8 ± 2.6	17.5 ± 3.6	18.0 ± 5.3
Muscle strength (N•m/kg)		2.8 ± 0.4	2.7 ± 0.7	3.0 ± 0.3
Endurance ratio (%)		53.5 ± 6.1	51.8 ± 6.7	50.2 ± 6.2
Deoxygenation level (%)		47.5 ± 25.8	38.2 ± 19.2	47.7 ± 18.2
Peak VO_2 (ml/min/kg)		39.1 ± 8.2	36.8 ± 6.1	36.1 ± 5.6
Exercise time (sec)		581.1 ± 160.6	584.0 ± 74.9	591.5 ± 54.6
Fitness habit	No	5	5	3
		2	1	3
	Yes	basket ball, footsal	kyuudou	running, softball

Values are expressed as mean ± SD. Differences in age (F = 2.05, p = 0.16), height (F = 2.41, p = 0.12), body weight (F = 0.67, p = 0.53), body mass index (F = 0.14, p = 0.87), body fat percentage (F = 0.52, p = 0.60), muscle strength (F = 0.39, p = 0.68), endurance ratio (F = 0.45, p = 0.65), muscle deoxygenation level (F = 0.38, p = 0.69), peak VO_2 (F = 0.34, p = 0.72), and exercise time (F = 0.02, p = 0.99) among the groups were examined by one-way ANOVA. Fitness habit was analyzed by the chi-square test. CON: control group, STR: muscle strength training group, END: muscle endurance training group.

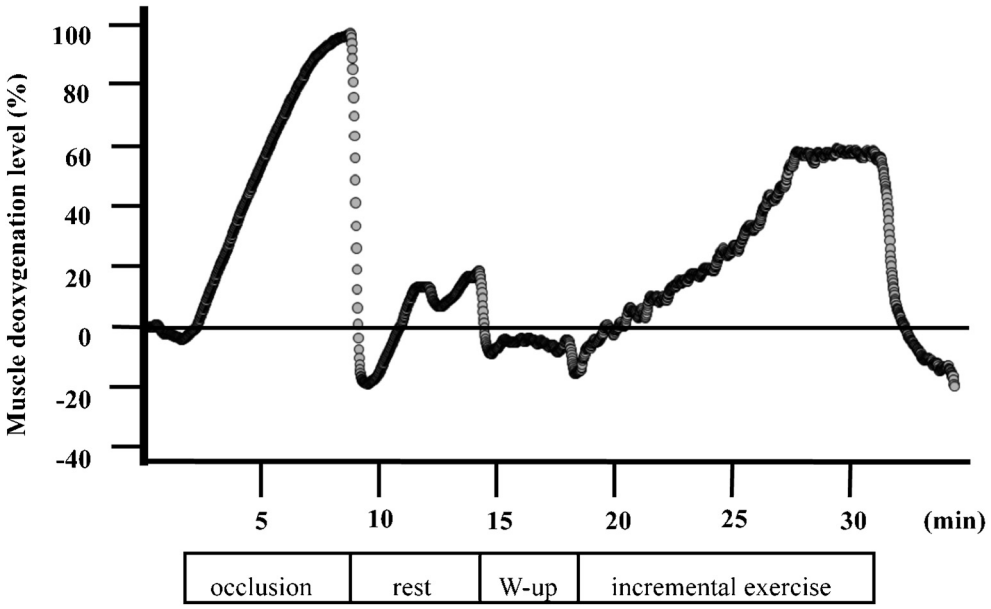

Fig.1. A typical example of the kinetics of Δdeoxy-Hb/Mb of vastus lateralis (VL) muscle during occlusion and incremental exercise. W-up: warm up. Muscle deoxygenation level was calculated by dividing the changes in Δdeoxy-Hb/Mb during incremental exercise by changes during occlusion.

repetitions performed by the total work of the first half, then multiplying the result by 100. Fifty percent of fatigue repetitions was also computed and employed on muscle endurance training (see below); this was the number of repetitions performed before 50% of maximum work in the first 3 repetitions was reached.

Subjects were supervised and given verbal encouragement by investigators during both measurements. After the training period, the same measurements were performed by the same investigators.

Training was initiated 7 days after the pre-training measurements and consisted of either 18 sessions of STR or END 3 days/week, for 6 weeks, with each training session separated by 1–2 days of rest. Subjects were prohibited from performing training for 3 consecutive days and training sessions were spaced by more than 3 days. Training was performed using Cybex Norm. Subjects were supervised and given encouragement by investigators during training sessions.

Each STR session consisted of 5 sets of 10 repetitions of

Table 2. The results of two-way ANOVA for muscle deoxygenation level, peak VO_2, and exercise time

Muscle deoxygenation level Source	SS	df	MS	F value	p value
Time	1397.59	1	1397.59	10.07	0.006**
Group	692.89	2	346.45	0.48	0.63
Time*group	314.95	2	157.48	1.14	0.35
Error	2220.02	16	138.76		
Total	4625.45	21			
Peak VO_2 Source	SS	df	MS	F value	p value
Time	33.61	1	33.61	2.38	0.14
Group	19.56	2	9.78	0.10	0.90
Time*group	23.20	2	11.60	0.82	0.46
Error	225.55	16	14.10		
Total	303.92	21			
Exercise time Source	SS	df	MS	F value	p value
Time	10600.24	1	10600.24	6.87	0.02*
Group	6574.06	2	3287.03	0.17	0.84
Time*group	3284.17	2	1642.08	1.06	0.37
Error	24689.10	16	1543.07		
Total	45147.57	21			

SS: sum of squares, df: degrees of freedom, MS: mean square. **: $p<0.01$, *: $p<0.05$

knee extension at an angular velocity of 60°/s. Each set was separated by 1 min recovery time.

Each END session consisted of 2 sets of 50% fatigue repetitions of knee extension at 240°/s, separated by 1 min recovery time. Subjects performed muscle endurance measurement every 2 weeks during the training period to readjust the number of repetitions.

Subjects of the CON group were instructed to continue their normal daily living for 6 weeks.

Statistical analysis was performed using SPSS for Windows 11.0 J (SPSS Inc., 1989–2001). A repeated two-way ANOVA was used to analyze the interactions of muscle strength, muscle endurance, muscle deoxygenation level, peak VO_2, and exercise time between training group and training period. When an interaction was not found, differences in each parameter between pre- and post-training were compared by the paired t-test. Differences between the STR and END groups in terms of change ratio of each parameter were compared by the unpaired t-test. A p value less than 5% was considered statistically significant.

RESULTS

Subjects' physical characteristics and each parameter of pre-training are shown in Table 1. There was no significant difference in the physical characteristics and each parameter among the 3 groups. These results suggest that subjects of each group had similar physical function and endurance performance. All subjects continued with their normal daily living activities during the training period. The numbers of training sessions were 15.7 ± 2.6 sessions (mean ± SD, 13–17 sessions) in the STR group and 16.3 ± 2.6 in the END group (14–18 sessions). No significant difference between the training groups was found in the number of training sessions ($p = 0.50$, unpaired t-test).

Table 2 shows the results of two-way ANOVA on muscle deoxygenation level, peak VO_2, and exercise time. A main effect of time was found for muscle deoxygenation level and exercise time. However, there was no interaction between group and time for any of the parameters. No effect was found on peak VO_2.

Table 3 shows changes in parameters at pre- and post-training in each group.

In the STR group, muscle strength tended to increase ($p = 0.09$) after 6 weeks of STR training but muscle endurance did not, while muscle endurance significantly increased after 6 weeks of END training but muscle strength did not. Muscle deoxygenation level was significantly increased in both training groups. Maximum oxygen uptake did not change in either training group; however, in the END group alone, exercise time was significantly prolonged.

There were no significant changes in any parameter of the CON group.

DISCUSSION

In this study, we compared the effects of muscle strength training (STR) and muscle endurance training (END) on muscle deoxygenation level measured by NIRS, and endurance performance (i.e. peak VO_2 and exercise time). The major results of the present study are as follows: 1) according to the training specificity, STR tended to increase muscle strength ($p = 0.09$) but did not change muscle endurance, while END significantly increased muscle

Table 3. The results of each parameter pre- and post-training

	Muscle strength (N•m/kg)		Endurance ratio (%)	
	pre	post	pre	post
CON	2.8 ± 0.4	2.9 ± 0.4	53.5 ± 6.1	51.7 ± 7.9
STR	2.7 ± 0.7	3.1 ± 0.6	51.8 ± 6.7	51.6 ± 4.2
END	3.0 ± 0.3	3.0 ± 0.2	50.2 ± 6.2	57.1 ± 8.1**
	Muscle deoxygenation level (%)		Peak VO_2 (ml/min/kg)	
	pre	post	pre	post
CON	47.5 ± 25.8	51.8 ± 22.2	39.1 ± 8.2	39.3 ± 9.2
STR	38.2 ± 19.2	53.2 ± 18.3*	36.8 ± 6.1	38.2 ± 4.3
END	47.7 ± 18.2	64.9 ± 17.9*	36.1 ± 5.6	40.1 ± 9.0
	Exercise time (sec)			
	pre	post		
CON	581.1 ± 160.6	589.3 ± 122.2		
STR	584.0 ± 74.9	626.3 ± 58.8		
END	591.5 ± 54.6	641.5 ± 71.3*		

Values are expressed as mean ± SD. CON: control group, STR: muscle strength training group, END: muscle endurance training group. **: $p<0.01$, *: $p<0.05$

endurance but did not change muscle strength; 2) muscle deoxygenation level during incremental exercise was significantly increased by STR and END, and there was no difference between the two training groups in its change ratio; 3) maximum oxygen uptake did not change in the STR and END groups, but exercise time was significantly prolonged in the END group.

In the present study, STR tended to increase muscle strength ($p = 0.09$), but muscle endurance did not change; END significantly increased muscle endurance but muscle strength did not change. Thus, we can say that both training methods employed in this study had an effect on skeletal muscle.

Limited studies have reported the effect of training on local muscle oxygenation measured by NIRS[17–20]. Furthermore, most of these studies used oxy-Hb/Mb to evaluate the local muscle oxygenation during exercise[17–18] or the recovery time after occlusion[19]. In this study, we used deoxy-Hb/Mb to evaluate muscle deoxygenation level because this signal is regarded as being essentially insensitive to change in blood volume[15]. McKay et al.[20] also used change in deoxygenated hemoglobin/myoglobin (Δ[HHb]) to evaluate local muscle oxygenation of VL muscle for a similar reason. They examined the change in time constants for pulmonary oxygen uptake (τVop2) and muscle oxygenation (τΔ[HHb]) during moderate constant loading exercise after 8 sessions of high-intensity interval training (8–12 × 1 min intervals at 120% maximal oxygen uptake separated by 1 min of rest) or 8 sessions of low-intensity endurance training (90–120 min at 65% maximal oxygen uptake) performed by 12 subjects. They observed a significant reduction in τVop2 in both training groups, but τΔ[HHb] did not change in either groups. In the present study, we observed a significant increase in muscle deoxygenation level. One of the reasons why we observed an improvement in muscle deoxygenation level is that we conducted about twice the number of training sessions as McKay et al. (16.0 ± 1.6; mean ± SD vs. 8 training sessions, respectively). In addition, subjects in our training groups performed training focused on the quadriceps femoris muscle using Cybex. Thus, we assume that the training effect on VL muscle in this study was larger than that in the experiment of McKay et al. using a cycle ergometer. Thus, we consider that training performed by the STR and END groups in this study increased muscle deoxygenation level.

Neary et al.[18] hypothesized that muscle deoxygenation observed in their subjects, who demonstrated significant deoxygenation, was related to a number of peripheral factors including a increased capillarization, mitochondrion density, and activity of oxidative enzymes, all of which likely augmented the arterial - venous oxygen content difference as a result of training. As for the adaptation of mitochondria to whole endurance training (i.e. cycling or running), Holloszy[21] reported that whole endurance training improved the capacities of pyruvate oxidation and oxidative enzymes, such as cytochrome c oxidase, which doubled in soleus muscle of rats, and also that the run time to exhaustion was prolonged (29 ± 3 min vs. 186 ± 18 min, control rats vs. trained rats, respectively). Burrelle and Hochachka4 also reported that citrate synthase and cytochrome c oxidase were significantly activated by 4 weeks of treadmill running. However, there is little evidence of the adaptation of mitochondrion content and oxidative enzyme activation to muscle endurance training (i.e. focused on the local muscle).

We hypothesized that only END improved muscle deoxygenation level because it has been reported that resistance training usually reduces mitochondrion content and oxidative enzyme capacity[22,23]. However, both STR and END resulted in significant and similar improvement in

muscle deoxygenation level. Recently, Tang et al.[24] showed that mitochondrial enzymes, such as citrate synthase, were activated by resistance training. Thus, their report indicates that resistance training can improve the skeletal muscle oxidative capacity. We did not evaluate the skeletal muscle oxidative capacity in the present study, but it is possible that our training protocol also increased mitochondrion content and oxidative enzyme capacity. Accordingly, muscle deoxygenation level would have increased with enhanced oxygen extraction by skeletal muscle.

Many studies have reported that whole endurance training improves peak VO_2[25–26] because of the training adaptations to oxygen delivery capacity, such as structural adaptation (i.e. capillary density) and functional adaptation (i.e. vascular dilation)[27], and oxygen extraction (i.e. arterial - venous oxygen content difference)[5]. The results for resistance training are conflicting, showing that it can increase peak VO_2[28] and that it cannot[25]. The present study demonstrated similarly increasing muscle deoxygenation levels in the STR group and the END group. However, peak VO_2 did not change, contrary to our hypothesis. Several studies have reported that the adaptations of mitochondria and capillaries to exercise training appear to be more pronounced in the regions and muscle fibers recruited during training[27,29]. Subjects in this study trained unilateral femoral muscles. Therefore, we consider that the adaptations of mitochondria to STR and END in this study were limited in the femoral region. Thus, the training provided in this study appears to have been insufficient to induce a significant increase in peak VO_2 as seen in whole endurance training.

Although exercise time did not change in the STR group, it was significantly prolonged in the END group. Jobrias et al.[30] examined the cellular energetic and structural adaptations of elderly VL muscle to exercise training. Their experiment resulted in a rise in muscle oxidative capacity with both endurance training and resistance training (57% and 31%, respectively) and a decline in glycolytic ATP supply (56%) with endurance training. Their results describe the disassociation of our results; i.e. only END induces a decline in glycolytic ATP production (i.e. rise in aerobic ATP production), delaying the accumulation of lactic acid during exercise, and prolonging exercise time during incremental exercise.

Generally, peak VO_2 is considered the index of maximal exercise capacity. The results of this study suggest that muscle endurance training is more effective at extending exercise time than muscle strength training. Thus, muscle endurance training may increase submaximal exercise capacity safely because it imposes less strain on the cardiopulmonary system than muscle strength training. It may be possible to use these results to benefit patients with cardiorespiratory disease and the elderly, so further investigations are needed.

In conclusion, we compared the effects of muscle strength training and muscle endurance training on muscle deoxygenation level by NIRS and endurance performance (peak VO_2 and exercise time). Muscle deoxygenation level was increased in the STR group and the END group and the change ratios in these groups did not differ. These training regimes appeared insufficient to induce a significant increase in peak VO_2; however, in the END group, exercise time during incremental exercise was significantly prolonged. Thus, it was indicated that muscle endurance training is more effective at increasing endurance performance than muscle strength training.

ACKNOWLEDGEMENT

We are grateful to T. Nakagawa, M. Hoso, M. Yokogawa, A. Ootsubo, T. Kusudo, N. Hashimoto, as well as to all subjects who were involved in this study.

REFERENCES

1) Hambrecht R, Fiehn E, Yu J, et al.: Effects of endurance training on mitochondrial ultrastructure and fiber type distribution in skeletal muscle of patients with stable chronic heart failure. J Am Coll Cardiol, 1997, 29: 1067–1073.
2) Maltais F, LeBlanc P, Whittom F, et al.: Oxidative enzyme activities of the vastus lateralis muscle and the function status in patients with COPD. Thorax, 2000, 55: 848–853.
3) Conley KE, Jubias SA, Esselman PC: Oxidative capacity and ageing in human muscle. J Physiol, 2000, 526: 203–210.
4) Burrelle Y, Hochachka PW: Endurance training induces muscle-specific changes in mitochondrial function in skinned muscle fibers. J Appl Physiol, 2002, 92: 2429–2438.
5) Holloszy JO, Coyle EF: Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. J Appl Physiol, 1984, 56: 831–838.
6) Bhambhani YN: Muscle oxygenation trends during dynamic exercise measured by near infrared spectroscopy. Can J Appl Physiol, 2004, 29: 504–523.
7) Pereira MI, Gomes PS, Bhambhani YN: A brief review of the use of near infrared spectroscopy with particular interest in resistance training. Sports Med, 2007, 37: 615–624.
8) Jobsis FF: Noninvasive, infrared monitoring of cerebral and myocardial oxygen sufficiency and circulatory parameters. Science, 1977, 198: 1264–1267.
9) Mancini DM, Bolinger L, Li H, et al.: Validation of near-infrared spectroscopy in humans. J Appl Physiol, 1994, 77: 2740–2747.
10) Belardinelli R, Barstow TJ, Porszasz J, et al.: Skeletal muscle oxygenation during constant work rate exercise. Med Sci Sports Exerc, 1995, 27: 512–519.
11) Chance B, Dait MT, Zhang C, et al.: Recovery from exercise-induced desaturation in the quadriceps muscle of elite competitive rowers. Am J Physiol, 1992, 262: 766–775.
12) Delpy DT, Cope M, van der Zee P, et al.: Estimation of optical pathlength through tissue from direct time of flight measurement. Phys Med Biol, 1988, 33: 1433–1442.
13) Ericson MO, Nisell R, Arborelius UP, et al.: Muscular activity during ergometer cycling. Scand J Rehabil Med, 1985, 17: 53–61.
14) Higuchi H, Hamaoka T, Sako T, et al.: Oxygenation in vastus lateralis and lateral head of gastrocnemius during treadmill walking and running in humans. Eur J Appl Physiol, 2002, 887: 343–349.
15) DeLorey DS, Kowalchuk JM, Paterson DH: Relationship between pulmonary O_2 uptake kinetics and muscle deoxygenation during moderate-intensity exercise. J Appl Physiol, 2003, 95: 113–120.
16) Kannus P, Alosa D, Cook L, et al.: Effect of one-legged exercise on the strength, power and endurance of the contralateral leg. A randomized, controlled study using isometric and concentric isokinetic training. Eur J Appl Physiol Occup Physiol, 1992, 64: 117–126.
17) Costes F, Prieur F, Feasson L, et al.: Influence of training on NIRS muscle oxygen saturation during submaximal exercise. Med Sci Sports Exerc, 2001, 33: 1484–1489.
18) Neary JP, McKenzie DC, Bhambhani YN: Effects of short-term endurance training on muscle deoxygenation trends using NIRS. Med Sci Sports Exerc, 2002, 34: 1725–1732.
19) Matsumura M, Ueda C, Shiroishi K, et al.: Low-volume muscular endurance and strength training during 3-week forearm immobilization was effective in preventing functional deterioration. Dyn Med, 2008, 7: 1–8.
20) McKay BR, Paterson DH, Kowalchuk JK: Effects of short-term high-

intensity interval training vs. continuous training on O_2 uptake kinetics, muscle deoxygenation, and exercise performance. J Appl Physiol, 2009, 107: 128–138.
21) Holloszy JO: Biomedical adaptations in muscle. Effects of exercise on mitochondrial oxygen uptake and respiratory enzyme activity in skeletal muscle. J Biol Chem, 1967, 242: 2278–2282.
22) Luthi JM, Howald H, Claassen H, et al.: Structural changes in skeletal muscle tissue with heavy-resistance exercise. Int J Sports Med, 1986, 7: 123–127.
23) Chilibeck PD, Syrotuik DG, Bell GJ: The effect of strength training on estimates of mitochondrial density and distribution throughout muscle fibres. Eur J Appl Physiol, 1999, 80: 604–609.
24) Tang JE, Hartman JW, Phillips SM: Increased muscle oxidative potential following resistance training induced fibre hypertrophy in young men. Appl Physiol Nutr Metab, 2006, 31: 495–501.
25) McCarthy JP, Agre JC, Graf BK, et al.: Compatibility of adaptive responses with combining strength and endurance training. Med Sci Sports Exerc, 1995, 27: 429–436.
26) Glowacki SP, Martin SE, Maurer A, et al.: Effects of resistance, endurance, and concurrent exercise on training outcomes in men. Med Sci Sports Exerc, 2004, 36: 2119–2127.
27) Laughlin MH, Roseguini B: Mechanisms for exercise training-induced increases in skeletal muscle blood flow capacity: Differences with interval sprint training versus aerobic endurance training. J Physiol Pharm, 2008, 59, Suppl 7: 71–88.
28) Hickson RC, Rosenkoetter MA, Brown MM: Strength training effects on aerobic power and short-term endurance. Med Sci Sports Exerc, 1980, 12: 336–339.
29) Gollnick PD, Armstrong RB, Saubert CW, et al.: Enzyme activity and fiber composition in skeletal muscle of untrained and trained men. J Appl Physiol, 1972, 33: 312–319.
30) Jubrias SA, Esselman PC, Price LB, et al.: Large energetic adaptations of elderly muscle to resistance and endurance training. J Appl Physiol, 2001, 90: 1663–1670.