Chapter 8
Effects of Low Volume Aerobic Training on Muscle Desaturation During Exercise in Elderly Subjects

Shun Takagi, Ryotaro Kime, Norio Murase, Masatsugu Niwayama, Takuya Osada, and Toshihito Katsumura

Abstract Aging enhances muscle desaturation responses due to reduced O_2 supply. Even though aerobic training enhances muscle desaturation responses in young subjects, it is unclear whether the same is true in elderly subjects. Ten elderly women (age: 62 ± 4 years) participated in 12-weeks of cycling exercise training. Training consisted of 30 min cycling exercise at the lactate threshold. The subjects exercised 15 ± 6 sessions during training. Before and after endurance training, the subjects performed ramp cycling exercise. Muscle O_2 saturation (SmO_2) was measured at the vastus lateralis by near infrared spectroscopy during the exercise. There were no significant differences in SmO_2 between before and after training. Nevertheless, changes in peak pulmonary O_2 uptake were significantly negatively related to changes in SmO_2 ($r = -0.67$, $p < 0.05$) after training. Muscle desaturation was not enhanced by low volume aerobic training in this study, possibly because the training volume was too low. However, our findings suggest that aerobic training may potentially enhance muscle desaturation at peak exercise in elderly subjects.

Keywords Aging • Cycling training • Muscle oxygen saturation • Near infrared spectroscopy • Peak aerobic capacity

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1 Introduction

Muscle desaturation responses during whole-body exercise can be measured by near infrared spatial resolved spectroscopy (NIR_{SRS}), which has been widely utilized in many previous studies [1, 2]. However, few previous studies have evaluated the effects of exercise training on muscle desaturation responses using NIR_{SRS} during cycling exercise. A previous study found that aerobic exercise training enhances muscle desaturation during incremental cycling exercise in young subjects [1]. In another study, muscle desaturation responses were found to be enhanced in elderly subjects because muscle blood flow (i.e. O_2 supply to exercising muscle) was reduced due to aging [2]. In view of that, it is unclear whether aerobic exercise training enhances muscle desaturation responses in elderly subjects. The aim of this study was to examine the effects of aerobic exercise training on muscle O_2 dynamics in elderly subjects.

2 Methods

2.1 Subjects

Untrained elderly women ($n = 10$; age: 65 ± 2 years; height: 158.6 ± 8.0 cm; weight: 62.1 ± 11.9 kg, mean ± SD) participated in the study. This study protocol was approved by the institutional ethics committee, and was conducted in accordance with the Declaration of Helsinki. Two subjects were taking a statin, and one subject was taking an angiotensin II receptor antagonist and a calcium channel blocker. All subjects were informed of the purpose and nature of the study and written informed consent was obtained.

2.2 Experimental Design

The subjects performed 12-weeks of cycling exercise training for 30 min at the individual's estimated lactate threshold (LT). Estimated LT was determined as previous studies had reported [3, 4]. Training frequency was set at two exercise sessions/week for 12 weeks.

Before and after exercise training, the subjects performed 10 or 15 W/min ramp cycling exercise until exhaustion (Strength Ergo 8, Fukuda-Denshi, Japan). Pulmonary O_2 uptake (VO_2) was monitored continuously during the experiments to determine peak VO_2 by using an automated gas analysis system (AE300S, Minato Medical Science, Japan).

Muscle O_2 saturation (SmO_2) and relative changes from rest in oxygenated hemoglobin concentration (ΔOxy-Hb), deoxygenated hemoglobin concentration

(ΔDeoxy-Hb), and total hemoglobin concentration (ΔTotal-Hb) were measured at vastus lateralis (VL) in the left leg by NIR_{SRS} (Astem Co., Japan). The probe consisted of one light source and two photodiode detectors, and the distances between light source and detector were 20 and 30 mm, respectively. The data sampling rate was 1 Hz. The obtained signals were defined as the values averaged over the last 10 s. Changes in SmO_2 were calculated as SmO_2 at peak exercise before training subtracted from SmO_2 at peak exercise after training. Although fat layer thickness affects NIR_{SRS} data because of light scattering, Niwayama et al. have recently reported that the effects of fat layer thickness can be corrected in relative changes in Hb and SmO_2 [5]. The corrected relative changes in Hb were obtained by dividing the measured values by the normalized optical path length for muscle (S_{muscle}; when the fat layer thickness is zero), and the value of S_{muscle} can be calculated by only fat layer thickness. For the calculations of SmO_2, the measurements using NIR_{SRS} can be corrected by using the appropriate curve plotting the spatial slope of light intensity and absorption coefficient of the muscle for fat layer thickness. In this study, we measured fat layer thickness at each measurement site in VL muscles with an ultrasound device (LogiQ3, GE-Yokokawa Medical Systems, Japan). Then, we calculated the muscle O_2 dynamics with correction for light scattering effects. The specifications of correction for the influence of fat layer thickness have been fully described [5]. Even though an upper limit of fat layer thickness was designated as 10 mm to correct for the effects in this study, fat layer thickness was within ~10 mm at each measurement site in all subjects.

2.3 Statistics

All data are given as means ± standard deviation (SD). To compare changes in NIRS variables during exercise between groups, a 2-way repeated-measures analysis of variance was used with training and power output as factors. Where appropriate, the Bonferroni post hoc test was conducted. Because one subject could not exercise at more than 59 W before training, repeated measures between groups were limited to rest, 20, 30, 40, and 50 W compared as a function of power output. Differences in NIR_{SRS} and cardiorespiratory variables at peak exercise were compared between groups using paired *t* tests. Pearson's correlation coefficient was employed to determine the relationship between variables. For all statistical analyses, significance was accepted at $p < 0.05$.

3 Results

Even though training frequency was set at two exercise sessions/week for 12 weeks, unfortunately, the subjects exercised 15 ± 6 sessions during 12 weeks training as their schedules permitted. Estimated LT was significantly increased after training (before: 12.8 ± 3.1 ml/kg/min, after: 14.0 ± 2.1 ml/kg/min, $p < 0.05$), while peak VO_2 was not significantly increased (before: 20.1 ± 6.0 ml/kg/min, after: 21.1 ± 4.1 ml/kg/min, $p = 0.28$). Similarly, workload at estimated LT was significantly improved after training than before (before: 54 ± 15 W, after: 62 ± 16 W, $p < 0.05$), even though peak workload was not significantly increased (before: 98 ± 30 W, after: 101 ± 30 W, $p = 0.44$). Fat layer thickness was not significantly altered after training than before (7.41 ± 2.67 vs. 7.14 ± 2.67 mm, $p = 0.29$).

During submaximal exercise, there were no significant training × power output interactions for SmO_2 ($p = 0.82$), ΔOxy-Hb ($p = 0.46$), ΔDeoxy-Hb ($p = 0.23$), or ΔTotal-Hb ($p = 0.29$) between before and after training in all subjects. Moreover, no significant main effect for training was observed in SmO_2 ($p = 0.75$), ΔOxy-Hb ($p = 0.61$), ΔDeoxy-Hb ($p = 0.48$), or ΔTotal-Hb ($p = 0.50$). Also at peak exercise, no significant difference was found in SmO_2 ($p = 0.90$), ΔOxy-Hb ($p = 0.26$), ΔDeoxy-Hb ($p = 0.20$), or ΔTotal-Hb ($p = 0.17$) (Fig. 8.1).

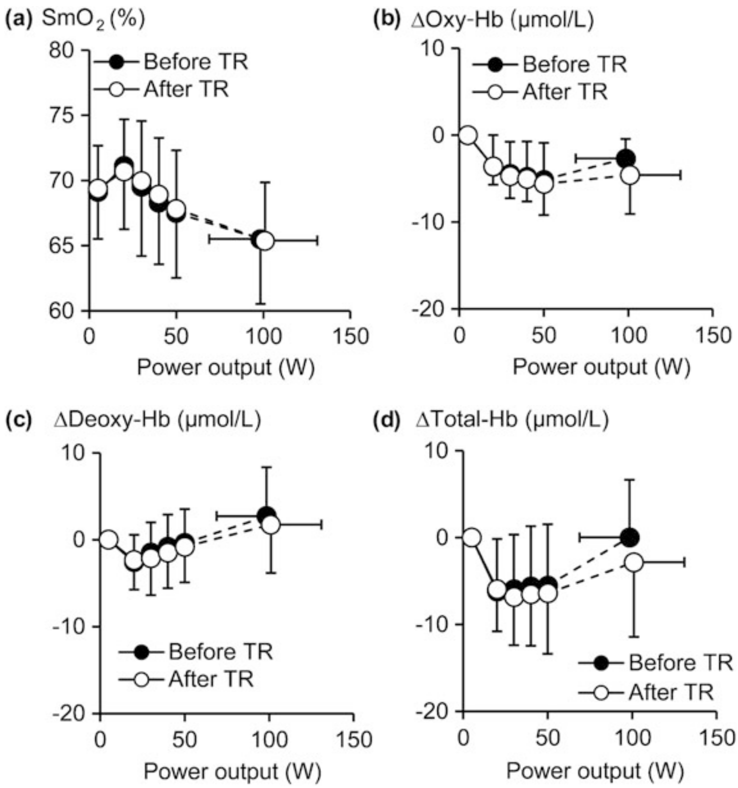

Fig. 8.1 Change in muscle O_2 saturation (SmO_2: **a**), oxygenated hemoglobin (oxy-Hb: **b**), deoxygenated hemoglobin (deoxy-Hb: **c**), and total hemoglobin (total-Hb: **d**) responses in vastus lateralis muscles during ramp cycling exercise before (*closed circles*) and after (*open circles*) exercise training

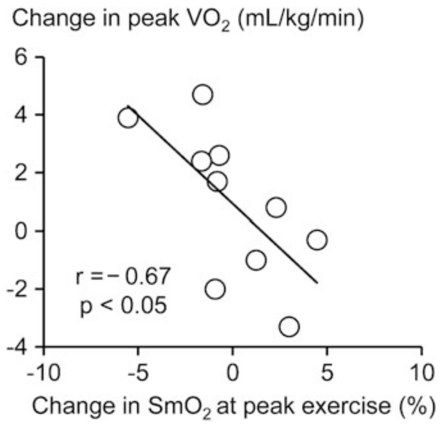

Fig. 8.2 Relationship between change in muscle O_2 saturation at peak exercise (values at after training minus values at before training) and improvement of peak VO_2 after aerobic exercise training

Improvement of peak VO_2 by exercise training was significantly positively related to the number of sessions of training ($r = 0.66$, $p < 0.05$) and negatively related to changes in SmO_2 ($r = -0.67$, $p < 0.05$) (Fig. 8.2). In addition, changes in ΔOxy-Hb ($r = -0.76$, $p < 0.05$) and ΔTotal-Hb ($r = -0.66$, $p < 0.05$) were significantly negatively associated with improvement of peak VO_2. Similarly, changes in ΔOxy-Hb ($r = -0.84$, $p < 0.01$) and ΔTotal-Hb ($r = -0.73$, $p < 0.05$) were significantly negatively associated with the number of sessions of training. However, ΔDeoxy-Hb was not significantly related to peak VO_2 ($r = 0.05$, $p = 0.88$) or training frequency ($r = 0.09$, $p = 0.80$).

4 Discussion

In the present study, muscle desaturation during submaximal and peak exercise was not significantly changed by aerobic training. One possible interpretation may be that training volume was too low when compared to previous studies [1, 6]. Some previous studies demonstrated that peak VO_2 was significantly related to muscle desaturation responses in cross-sectional observation [7, 8]. In fact, in this study, peak VO_2 was not significantly increased by exercise training, and the number of training sessions was positively related to changes in peak VO_2 after training. These results suggest that no significant difference in muscle desaturation were observed in this study due to low volume exercise training. Another possibility to explain the lack of significant difference in muscle desaturation after aerobic training may be the subjects' characteristics. Previous studies reported that aerobic training enhanced muscle desaturation in healthy young subjects [1] and heart disease patients [6]. However, to our knowledge, there have been no published reports on effects of aerobic training on muscle deoxygenation responses in elderly subjects. This area warrants further investigation.

We also observed a significant negative relationship between improvement of peak VO_2 and changes in SmO_2 at peak exercise after exercise training, even though muscle desaturation was not largely enhanced after training in all elderly subjects. In addition, the number of training sessions was significantly positively related to improvement of peak VO_2 and negatively related to changes in SmO_2 at peak exercise. These findings lead us to speculate that aerobic training may potentially enhance muscle desaturation responses at peak exercise in elderly subjects.

Remarkably, there were significant relationships between enhancement of peak VO_2 and decreases in ΔOxy-Hb or ΔTotal-Hb after exercise training, while changes in ΔDeoxy-Hb were not significantly associated with improvement of peak VO_2. ΔOxy-Hb is an indicator of the balance between O_2 supply and O_2 utilization, and ΔTotal-Hb is indicator of blood volume. Additionally, muscle O_2 supply is also affected by mechanical stress [9]. In fact, in this study, increased peak workload by training was also significantly negatively related to changes in ΔOxy-Hb ($r = -0.63$, $p < 0.05$) or ΔTotal-Hb ($r = -0.64$, $p < 0.05$) by training. These data suggest that the change in muscle desaturation at peak exercise after low volume exercise training may have been mainly due to reduced O_2 supply, secondary to increased mechanical stress, such as intramuscular pressure.

In summary, muscle desaturation was not enhanced by low volume aerobic training in this study, possibly because the training volume was too low. However, there were significant relationships between change in SmO_2, improvement of peak VO_2 and the number of sessions. These results suggest that aerobic training may potentially enhance muscle desaturation responses at peak exercise in elderly subjects. However, in this study, the change in muscle desaturation at peak exercise may have been mainly due to reduced O_2 supply, secondary to increased mechanical stress, such as intramuscular pressure.

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References

1. Kime R, Niwayama M, Fujioka M et al (2010) Unchanged muscle deoxygenation heterogeneity during bicycle exercise after 6 weeks of endurance training. Adv Exp Med Biol 662:353–358
2. Takagi S, Kime R, Murase N et al (2013) Aging affects spatial distribution of leg muscle oxygen saturation during ramp cycling exercise. Adv Exp Med Biol 789:157–162
3. Beaver WL, Wasserman K, Whipp BJ (1986) A new method for detecting anaerobic threshold by gas exchange. J Appl Physiol 60(6):2020–2027
4. Wasserman K, Whipp BJ, Koyl SN (1973) Anaerobic threshold and respiratory gas exchange during exercise. J Appl Physiol 35(2):236–243

5. Niwayama M, Suzuki H, Yamashita T et al (2012) Error factors in oxygenation measurement using continuous wave and spatially resolved near-infrared spectroscopy. J Jpn Coll Angiol 52:211–215
6. Mezzani A, Grassi B, Jones AM et al (2013) Speeding of pulmonary VO2 on-kinetics by light-to-moderate-intensity aerobic exercise training in chronic heart failure: clinical and pathophysiological correlates. Int J Cardiol 167(5):2189–2195
7. Kime R, Osada T, Shiroishi K et al (2006) Muscle oxygenation heterogeneity in a single muscle at rest and during bicycle exercise. Jpn J Phys Fit Sports Med 55(Suppl):S19–S22
8. Takagi S, Murase N, Kime R et al (2014) Skeletal muscle deoxygenation abnormalities in early post myocardial infarction. Med Sci Sports Exerc 46(11):2062–2069 doi:10.1249/MSS.0000000000000334
9. Saltin B, Radegran G, Koskolou MD et al (1998) Skeletal muscle blood flow in humans and its regulation during exercise. Acta Physiol Scand 162:421–436