# Effect of training status on oxygen uptake response to exercise onset

林, 直亨
九州大学健康科学センター

吉田, 敬義
大阪大学健康体育部

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— ORIGINAL —

# Effect of training status on oxygen uptake response to exercise onset

Naoyuki HAYASHI[1)]* and Takayoshi YOSHIDA[2)]

## Abstract

We examined the effect of training on oxygen uptake ($\dot{V}O_2$) kinetic response to the exercise onset, using cross sectional model. Twenty male subjects ($\dot{V}O_2$max range: 41 - 60 mL/min/kg) performed six-minute cycle ergometer exercise at a work rate corresponding to 80% of the ventilatory threshold after three minutes of unloaded cycling. The responses in $\dot{V}O_2$, heart rate (HR), cardiac output ($\dot{Q}$), and deoxygenated Hb+Mb change (DeoxyHb) were determined from the data averaged from four repetitions, fitted with a mono-exponential model. The response time was assessed as the sum of delay and time constant (MRT), which represents the time to reach 63% of the steady state response. The $\dot{V}O_2$ MRT (phase 2) significantly correlated with the HR MRT (r= 0.55) and $\dot{Q}$ MRT (r= 0.57). The DeoxyHb MRT did not correlate with the $\dot{V}O_2$ MRT in all subjects (r= 0.17), but correlated with the $\dot{V}O_2$ MRT in each swimmer and untrained group (r= 0.96, n= 6; r=0.73, n=7, respectively). It was suggested that responses of central circulation and local oxygen utilization affect individual $\dot{V}O_2$ response in various training status.

***Key words***: kinetic response, central circulation, oxygen utilization, physical training, near infrared spectroscopy

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## Introduction

Maintaining homeostasis, cardiorespiratory system is activated at the physical exercise onset to supply oxygenated blood to the active muscles according to their oxygen demand. The oxygen uptake ($\dot{V}O_2$) kinetics response to the exercise onset has been determined to describe the nature of aerobic metabolism during non-steady state (e.g., review[1)]). Daily life includes non-steady state as well as steady state. Thus, it is important to assess kinetic response as to evaluate regulation of physical ability. It is well known that the $\dot{V}O_2$ response, which is calculated from exponential fitting, has wide variety among individuals, and that trained subjects has shorter response time than sedentary subjects. However, it has not been fully elucidated what factors influence the individual response. The candidates varying the $\dot{V}O_2$ kinetics below the intensity corresponding to individual ventilatory threshold (Tvent) can be divided into dynamic factors measured during non-steady state, and non-dynamic factors measured during resting and steady state. The former factors are determined by the responses of oxygen transportation to the active muscle[2,3,4,5)], and oxygen utilization in the active muscle.[6,7,8,9,10,11)] The latter factors are the baseline and amplitude in the $\dot{V}O_2$ and heart rate (HR), which affect the $\dot{V}O_2$ kinetics as well.[12,13,14,15, 16,17)]

Physical training is the other candidate related to the wide variation of the responses. Many studies

1) Institute of Health Science, Kyushu University 11, Kasuga 816-8580, Japan
2) School of Health and Sport Sciences, Osaka University
* Correspondence to: Institute of Health Science,Kyushu University,6-1 Kasuga-Koen,Kasuga,Fukuoka 816-8580,Japan
Tel/Fax: +81-92-583-7848 E-mail: naohayashi@ish.kyushu-u.ac.jp

indicated faster oxygen uptake response in trained than in the untrained subjects.[11,12,14,17,18,19,20,21,22] However the effective factor is not determined yet because of issues related to the non-dynamic factors. In longitudinal studies, loading the same relative intensity, such as a work rate corresponding to 80% Tvent, the increase in amplitude of $\dot{V}O_2$ brought by training results in lengthening response time. Using the same absolute work intensity, the decrease in amplitude of HR brought by training results in shortening response time. Thus, it is unclear, from longitudinal studies just observing the relationship between $\dot{V}O_2$ response and training, whether a physical training alters the dynamic factors or non-dynamic factors, and consequently affects the $\dot{V}O_2$ response.

Cross-sectional studies also reported that the $\dot{V}O_2$ response is shorter in trained subjects. Correlations in $\dot{V}O_2$max with the half time[22] and the time to reach 75% of the steady state $\dot{V}O_2$[17] were shown. The cross-sectional study can examine or neglect the effect of baseline and gain. However, no cross-sectional study considered the effect of the non-dynamic factors.

It seems clear that a subject with a higher $\dot{V}O_2$max or physically trained shows faster $\dot{V}O_2$ kinetics. When the change of $\dot{V}O_2$ response is correlated with given a factor, it is concluded that the factor modulates the individual $\dot{V}O_2$ response. The aim of this study was to examine what factor(s) explains the individual $\dot{V}O_2$ kinetics response in subjects ranging from untrained to trained in some sporting events. We used the near infrared spectroscopy to estimate the local metabolic effect.

## Materials and Methods

Twenty male volunteers participated in the present study. Their mean ± SD (range) in age, height, body mass and $\dot{V}O_2$ max were 20.4 ± 2.2 (18-26) yrs, 174.5 ± 4.2 (167-182) cm, 65.7 ± 4.2 (58-76) kg, and 50.7 ± 6.1 (40.9-59.8) mL/min/kg, respectively. Six swimmers, a boxer, a cyclist, three American Football players, two Shorinji Kempo (a type of kung-fu) athletes, and seven active but untrained men were included. This study was approved by the local ethics committee. and all procedures conformed to the Declaration of Helsinki.The volunteers were fully informed of any risk and discomfort associated with the experiments before giving their written consent to participate in the study.

Subjects exercised on an electrically braked cycle ergometer (Model 232C, Combi, Japan). The exercise protocol consisted of an abrupt work increase for a 6-min period at a work rate corresponding to 80% of the individual Tvent after an initial 3-min period of unloaded pedaling. The Tvent was determined as the work rate at which minute ventilation and carbon dioxide production increased not straight as a function of oxygen uptake during 20 W/min ramp fashion incremental exercise. The work was continued to exhaustion of each subject. The maximal oxygen uptake ($\dot{V}O_2$max) was determined by the data for 20 s moving average. In the step function work, the work rate was increased without telling its starting to the subject. Subjects maintained a constant pedaling rate of 60 rpm during cycling. Each subject repeated each trial four times on different days. The $\dot{V}O_2$max per body mass was also calculated ($\dot{V}O_2$max/wt).

Ventilatory flow was measured with a hot wire flow meter (RM-300, Minato Medical Sciences, Japan). Fractional concentrations of $O_2$ and $CO_2$ were analyzed with a mass spectrometer (WSMR-1400, Westron, Japan). This system was calibrated with a 2 L syringe, fresh air and a precision gas. The time delay for gas flow to the analysers was compensated for gas exchange calculation. Cardiac output was determined continuously by the impedance method[23]. Two-band electrodes were placed on the neck and chest. The changes in impedance and electrocardiogram were stored on a hard disk at a sampling frequency of 250 Hz. The beat-by-beat stroke volume change was calculated by the method of Kubicek et al.[23] and was applied to ensemble averaging.[24] HR was calculated from the R-R interval data. The product of HR and stroke volume was linearly interpolated every second. The HR, $\dot{Q}$, $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ were averaged for four trials in each subject.

The changes in sum of deoxygenated Hb and Mb (DeoxyHb) were measured with a NIRO-500 (Hamamatsu Photonics, Japan). This equipment uses four laser lights at wavelengths of 775, 825, 850 and 904 nm. Near infrared lights with a 100 ns pulse width from pulse laser diodes were applied to muscle tissue at 1.9 kHz. The photo-multiplier detected the light through the tissue by photon-counting methods. The probes for diode and detector were fixed to a black rubber plate. The distance between the diode and probe was 4.5 cm. The center between the diode and probe was on the right vastus lateralis 10 cm from the knee joint. The NIRO-500 calculated the change in DeoxyHb from changes in the light absorption according to the modified Beer-Lambert's law. The oxygenated and deoxygenated (Hb+Mb), and cytochrome changes were calculated from four simultaneous equations. This method for calculation was previously described.[25] The

data were calculated every second by NIRO-500 and stored onto a hard disk. NIRS measurements do not provide an absolute value of DeoxyHb because the path length through the tissue is uncertain. Therefore, the change in DeoxyHb is represented as μmol/L/cm, where the unit of cm represents the path length.

The non-linear least squares fitting was applied to HR, $\dot{Q}$, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$, and DeoxyHb data from the second minute of unloaded pedaling to sixth minute after the increase by using a computerized regression algorithm;

$$f(t) = Baseline + A\{ 1 - \exp [ - (t - TD) / \tau ]\},$$

where f(t) represents the HR, $\dot{Q}$, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$, and DeoxyHb at the time t; A is the predicted steady-state increase from unloaded pedaling at infinite time (amplitude); and TD and τ are the time delay and time constant, respectively. Mean response time (MRT; sum of time constant and delay), which represents the time to reach 63 % (1-1/e) of the gain, was used to estimate the response of those variables. $\dot{V}O_2$ during the first 15 s just after increase of exercise was excluded from the calculation to obtain the phase-2 $\dot{V}O_2$ response.[27] The baselines and gains in $\dot{Q}$ and Deoxy-(Hb+Mb) were not represented because each time course has been non-dimentionalized with the resting value equal to zero and the steady-state value equal to 100%.

A linear regression analysis was used to detect the correlations between $\dot{V}O_2$ MRT and the other variables. Significance level was set at $p<0.05$.

## Results

The fastest and the slowest $\dot{V}O_2$ responses to the abrupt work increase in subjects are shown in Fig. 1. The fastest MRT in $\dot{V}O_2$ was 29 s in a trained Shorinji Kempo athlete with 47 mL/min/kg of $\dot{V}O_2$max, and the slowest was 47 s in an untrained subject with 41 mL/min/kg. The mean MRT in the $\dot{V}O_2$, $\dot{Q}$, HR, $\dot{V}CO_2$, $\dot{V}_E$ and DeoxyHb are shown in Tab. 1.

No significant correlation between $\dot{V}O_2$ MRT and $\dot{V}O_2$max/wt was found (Tab. 1). In addition, t-test did not show significant difference between the $\dot{V}O_2$ MRT in the subject with more than 50 mL/min/kg (n=11) and the subjects with less than 50 mL/min/kg (n=9). The $\dot{V}O_2$max and $\dot{V}O_2$max/wt significantly correlated with the HR MRT and $\dot{V}_E$ MRT (r= -0.43, -0.60 vs. $\dot{V}O_2$max; r= -0.51, -0.44 vs. $\dot{V}O_2$max/wt, respectively). The relationship between the HR MRT and $\dot{V}O_2$max/wt is shown in Fig. 2. The higher $\dot{V}O_2$max had the faster HR and $\dot{V}_E$ responses. The $\dot{V}O_2$max/wt did not correlate with the MRTs in the $\dot{Q}$, $\dot{V}CO_2$ and DeoxyHb (r= -0.20, -0.21, -0.34, respectively). Also, the $\dot{V}O_2$max/wt did not correlate with the baseline and amplitude of the $\dot{V}O_2$ and HR (baseline r= 0.20, -0.22; amplitude r= 0.36, 0.02, respectively). The Tvent correlated with the $\dot{V}O_2$ amplitude (r=0.68), but not with the HR amplitude (r=0.34).

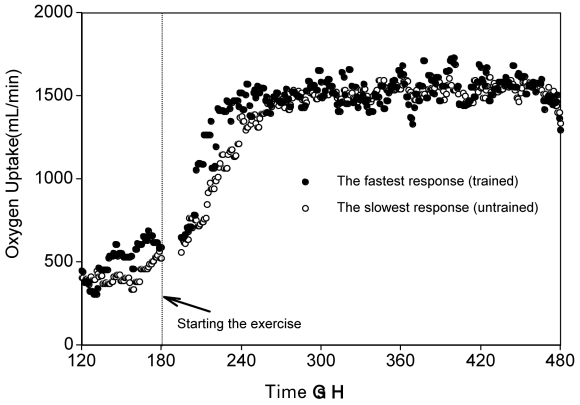


Fig. 1 The fastest and slowest $\dot{V}O_2$ response at the exercise onset in an athlete (MRT=29s) and in an untrained subject (MRT=47s)

Table 1 Mean response time (MRT) in the response variables, and correlation with $\dot{V}O_2$ MRT

| MRT | $\dot{V}O_2$ | $\dot{Q}$ | HR | $\dot{V}CO_2$ | $\dot{V}_E$ | DeoxyHb |
|---|---|---|---|---|---|---|
| Mean±SD (s) | 35.7 ± 4.5 | 27.5 ± 10.9 | 27.9 ± 11.1 | 56.4 ± 9.1 | 62.3 ± 13.5 | 15.2 ± 6.2 |
| R vs. $\dot{V}O_2$max | -0.33 | -0.20 | -0.51* | -0.21 | -0.44* | 0.34 |
| R vs. $\dot{V}O_2$ MRT | N.A. | 0.57* | 0.55* | 0.87* | 0.67* | 0.17 |

*: $p<0.05$, n=20

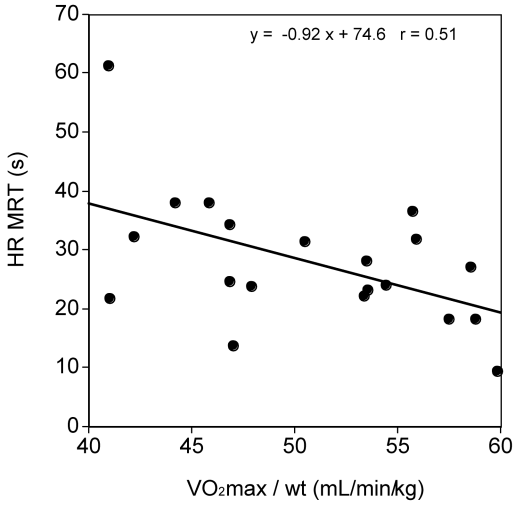


Fig. 2 The relationship between the $\dot{V}O_2$max/wt and HR MRT. Significant negative correlation was obtained (r=—0.51, $p<0.05$).

The $\dot{V}O_2$ MRT significantly and positively correlated with the $\dot{Q}$, HR, $\dot{V}CO_2$, and $\dot{V}_E$ MRTs (r= 0.57, 0.55, 0.87, 0.67, respectively) but not with the DeoxyHb

MRT (r=0.17). The relationship between the $\dot{V}O_2$ MRT and HR MRT is shown in Fig. 3.

The relationships between the MRT in each variable, and baseline and amplitude of each variable are shown in Tab. 2. The amplitude in HR significantly correlated with the HR MRT (r=0.49, Fig. 4). Amplitudes in the other variables did not significantly correlate with the MRT in any variable. The baseline had no effect on any variable of MRT. Also, the baseline and amplitude in the HR did not correlate with the $\dot{V}O_2$ MRT (r= -0.04, -0.14, respectively).

The regression analysis was applied to each of swimmer (n=6) and untrained (n=7) group. There was significant correlation between the $\dot{V}O_2$max/wt and HR MRT (r=-0.75 in swimmer, r=-0.81 in untrained). While the untrained group showed a significant correlation between the $\dot{V}O_2$max/wt and $\dot{V}_E$ MRT (r=-0.81), it was not significant in the swimmer group (r=-0.13). There was a significant correlation between the $\dot{V}O_2$ MRT and DeoxyHb in the swimmer (r=0.96) and the untrained (r=0.73) groups whereas no correlation was found in all subjects as indicated above (r=0.17). The exponential fitting for the DeoxyHb time course in a swimmer is shown in Fig. 5. The group mean of DeoxyHb MRT was 15.2±5.2 s.

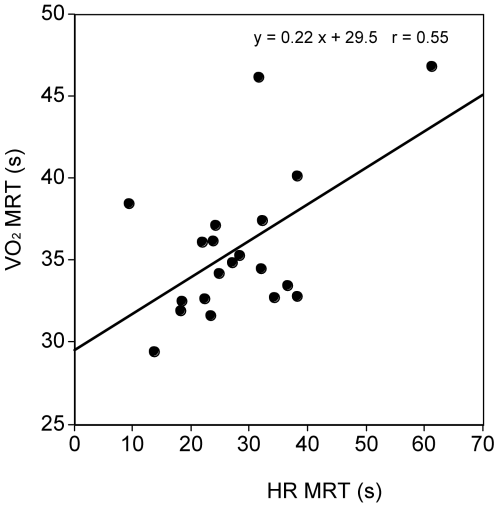


Fig. 3 The relationship between the HR MRT and $\dot{V}O_2$ MRT. Significant positive correlation was obtained (r=0.55, p<0.05).

Table 2 Relationship between mean response time, and baseline and ampliatude in each reriablc

| Variable | $\dot{V}O_2$ | HR | $\dot{V}CO_2$ | $\dot{V}_E$ |
|---|---|---|---|---|
| Baseline | 453 ± 40 | 72 ± 7 | 424 ± 45 | 16 ± 2 |
| Gain | 1183 ±244 | 47 ±11 | 1223 ±278 | 31 ± 9 |
| R in MRT vs. Baseline | -0.17 | 0.01 | -0.22 | -0.30 |
| R in MRT vs. Amplitude | -0.32 | 0.49* | -0.42 | -0.08 |

*: p<0.05, n=20

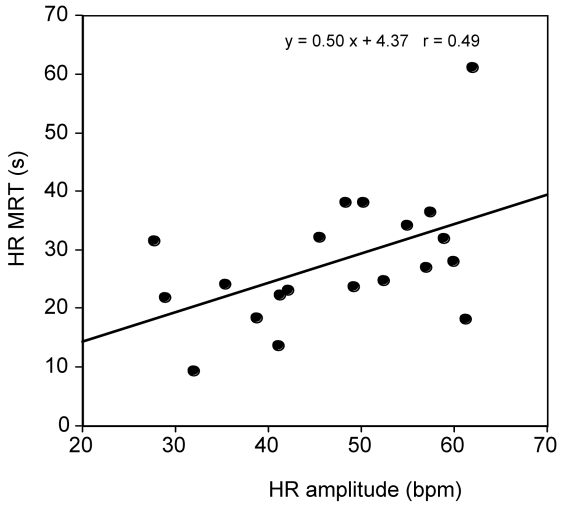


Fig. 4 The relationship between the HR amplitude and HR MRT. Significant positive correlation was obtained (r=0.49, p<0.05).

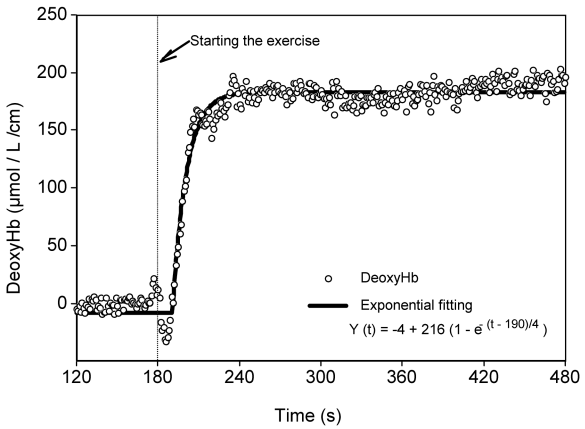


Fig. 5 The time course of the DeoxyHb change from the baseline unloaded pedaling to the work increase. The solid line was calculated from a mono-exponential model.

## Discussions

The present study showed that the $\dot{V}O_2$ MRT correlated with the HR, $\dot{Q}$, $\dot{V}CO_2$, and $\dot{V}_E$ MRTs. Considered the cause and effect relationship among these variables, the correlation indicated that the HR response affects the $\dot{V}O_2$ response through the $\dot{Q}$ response. The correlation between the HR MRT and HR amplitude indicates that the non-dynamic factors influence response. No correlation between the $\dot{V}O_2$max and $\dot{V}O_2$ MRT is partly explained by the effect of non-dynamic factors and different adaptation of local oxygen utilization in heterogeneous subjects. This is supported by the finding that all subjects did not showed significant correlation between the $\dot{V}O_2$ MRT

and DeoxyHb MRT, whereas significant correlation was found in either swimmer or untrained group.

**Effect of $\dot{V}O_2$max on response at the exercise onset**

A number of studies revealed that trained subjects having higher $\dot{V}O_2$max show a faster $\dot{V}O_2$ response than in the untrained, calculated from half time[19,20,21,22], time to reach 3/4 of steady state response[17,21], time constant[12,14,18,28] and mean response time [29] to step function exercise, and response to a sinusoidal function work.[26] These studies indicated that physical training shortens the $\dot{V}O_2$ response time. This faster response can be explained by an adaptation in local factors[19, 26] and oxygen utilization.[21] Subjects in the present study had various training status. The effect of aerobic capacity on the response must relate to the variation in the response time.

The result did not show the relationship between$\dot{V}O_2$max and $\dot{V}O_2$ MRT. Fukuoka et al.[26] also observed no significant relationship between the $\dot{V}O_2$max and $\dot{V}O_2$ dynamic response to sinusoidal work in either distance runners or American Football player subjects. The present result is consistent with their findings. These findings indicate that increase of $\dot{V}O_2$max does not generally shorten the $\dot{V}O_2$ response time. There are four possibilities explaining the non-significant relationship. First, although the training increases the $\dot{V}O_2$max, the adaptation does not necessarily improve the factors affecting the $\dot{V}O_2$ response, as previously speculated.[26] Second, there were negative effect on the relationship between the $\dot{V}O_2$max and $\dot{V}O_2$ response, that is non-dynamic factors as found in the present study such as the baseline and amplitude in the $\dot{V}O_2$ and HR. Zhang et al.[17] reported a slower time to reach 75% of the steady-state $\dot{V}O_2$ in the response to a higher work rate. Cooper et al.[12] reported a significant relationship between the HR half time and the HR baseline. Hughson and Morrissey[30] showed that the exercise onset from a high baseline induces a sluggish response in the $\dot{V}O_2$ and HR. Whipp and Wasserman[16] demonstrated that a high work rate increases the $\dot{V}O_2$ half time. Grucza et al.[14] reported the HR time constant from rest to 50 W was less than rest to 94 W. These previous studies indicate the slower $\dot{V}O_2$ and HR response to either higher work rate or higher baseline. Third, the previous research[26] explained that no correlation resulted from the upper limit of the $\dot{V}O_2$ response in distance runners and combined training in football players. However, in the present study, the upper limit could not explain the result since the $\dot{V}O_2$max of the subject was not so high. Fourth, the present and the previous[26] studies used heterogeneous subjects. The nature of adaptation is not different among sports, as mentioned below.

The HR MRT correlated with the $\dot{V}O_2$max. This indicates that an increase in the $\dot{V}O_2$max or physical training improves the response of HR to the exercise onset. However, there was no significant relationship between the $\dot{Q}$ MRT and $\dot{V}O_2$max. We speculate the difference in relationships between the $\dot{V}O_2$max and HR MRT, and the $\dot{V}O_2$max and $\dot{Q}$ MRT results from the different adaptation in vagal and sympathetic nervous activities. The autonomic nervous system increases heart rate at the exercise onset by fast vagal withdrawal.[31] Additional increase in heart rate and cardiac output is slow because the response of sympathetic nervous activity is slower than vagal withdrawal[32]. It is plausible that vagal withdrawal response to the exercise onset is faster in trained than in untrained subject, but the sympathetic increase response to exercise is not or slightly improved by training.

In the present study, the $\dot{V}O_2$max did not correlate with the baseline and gain of the $\dot{V}O_2$ and HR. While the effect of the baseline and gain on the $\dot{V}O_2$ response was revealed, this effect does not result from improvement of the $\dot{V}O_2$max. It is possible that the effect of the HR amplitude on the HR MRT counterbalances the effect of $\dot{V}O_2$max on HR MRT. As mentioned above, physical training increases the $\dot{V}O_2$max and concomitantly improves the HR response. On the other hand, training increases the Tvent, resulting in the non-dynamic factor increasing the HR amplitude, which delays the heart rate kinetics. Thus, only when the effect of training on the HR response overrides the effect of HR amplitude, the HR response is accelerated.

We summarized the effect of training on shortening the $\dot{V}O_2$ response time in Fig. 6. In the present study, we implied the effect of vagal activity change on the $\dot{V}O_2$ response through the HR and $\dot{Q}$ responses by training. We also suggested the effect of the balance of the amplitude increase and the improvement in heart rate response by vagal activity. However, training does not generally improve the $\dot{V}O_2$ response since the effect of the baseline and amplitude were revealed. The $\dot{V}O_2$ MRT variation among individuals can cause the variation in the $\dot{V}CO_2$ MRT, as a result of aerobic metabolism.

**Dynamic factors affecting individual $\dot{V}O_2$ response**

The correlation between the $\dot{V}O_2$ MRT, and $\dot{Q}$ and HR MRTs in the present results and the previous

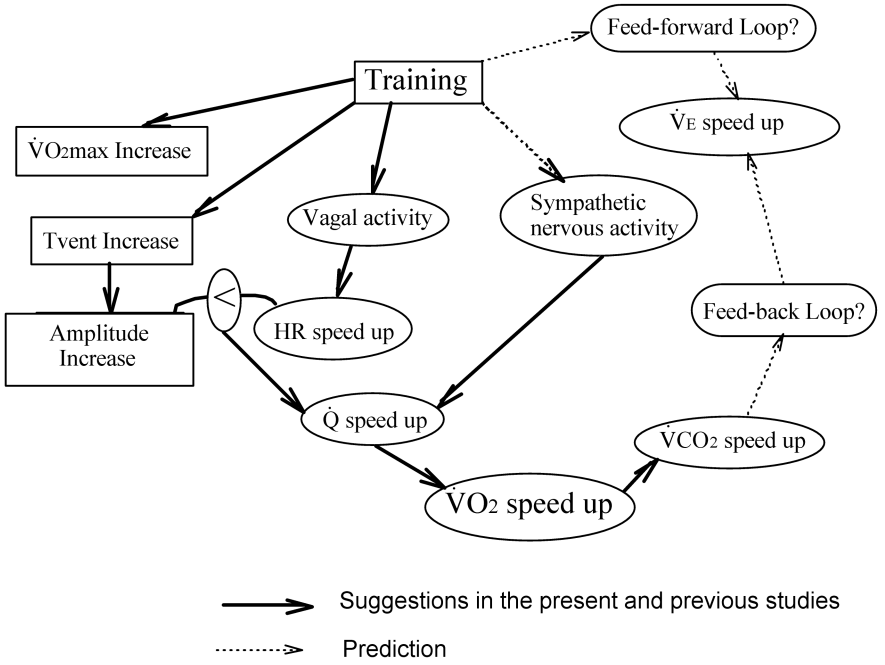


Fig. 6 Schematic model of effect of training status on individual $\dot{V}O_2$ response to the exercise onset is shown. Factors reported in previous studies and suggested in the present study are included. Squares mean non-dynamic factors, and ovals mean dynamic factors.

findings, which suggested the central circulation as a determinant of $\dot{V}O_2$ response, confirms that response of central circulation affects individual $\dot{V}O_2$ kinetics. Previous studies, which suggested dynamic factors maffecting $\dot{V}O_2$ response, used acute manipulations such as higher amplitude of work intensity[14,27], higher baseline before work increase[17,30], prior-exercise input[11], hypoxia[4], supine posture[3], decreased muscle temperature[10], lower body negative pressure[3], and altered diffusive capacity in active muscle.[2] These anipulations generate changes in responses of independent variables. When a given manipulation changes the $\dot{V}O_2$ kinetics, it was concluded that the variable affects the $\dot{V}O_2$ kinetics. Local metabolism [10,11],central circulation[4,30] and peripheral circulation[5,31] have been suggested as factors altering the $\dot{V}O_2$ kinetics. These factors can affect the long-term change in $\dot{V}O_2$ kinetics and explain individual $\dot{V}O_2$ response. The present study did not use an acute manipulation such as used in the previous study. We, alternatively, looked at a variation of training status among individual as an experimental manipulation.

**Effect of baseline and amplitude on response**

Previous studies did not elucidate the effect of baseline and amplitude on individual variety in $\dot{V}O_2$ response. We observed the positive correlation in the HR amplitude and HR MRT. This observation is consistent with the previous study.[14] As mentioned above, the previous studies indicated higher baseline and amplitude induces the slower response. The effect of amplitude was indirectly observed in some previous researches that reported the correlation between the $\dot{V}O_2$ kinetics and blood lactate.[19,28] These findings also can be interpreted as a consequence of the decreased relative work intensity by training. Thus we need to keep the effect of amplitude and baseline in mind when we use the $\dot{V}O_2$ response to evaluate and compare the training effect.

**$\dot{V}O_2$ response and oxygen utilization in active muscle**

Some studies suggested that an adaptation of oxygen utilization in active muscle shorten the $\dot{V}O_2$ response time after training. It was reported that runners had a faster $\dot{V}O_2$ response in cycle ergometer work, while a swimmer had a faster response in arm cranking work.[19] A runner with a higher $\dot{V}O_2$max than American Football players and control subjects were reported to have a faster response to the sinusoidal work change.[26] However, no study examined the peripheral factor's role for the individual response among various training status.

We determined the local oxygen metabolism response with a non-invasive method using the NIRS as reported in a previous report.[33] The near infrared light through the skeletal muscle is absorbed by Hb and

myoglobin (Mb).[25,34,35] The NIRS method evaluates the sum of changes in Hb and Mb. Consequently, increase in the DeoxyHb indicates the extraction of oxygen from haemoglobin and myoglobin in the muscle tissue.

The DeoxyHb MRT did not correlate with the $\dot{V}O_2$ MRT in all subjects. This must be explained by different adaptations in local response. It is natural that different training induces different local adaptationsspeculated by Fukuoka et al.[26] In the present study, subjects were trained in different sporting events, so the local metabolism response must be different among subjects.

On the other hand, we found a significant positive correlation betweenthe DeoxyHb and $\dot{V}O_2$ MRT in either swimmer or untrained group. This is consistent with the previous observation in sedentary subjects.[33] It is plausible that faster oxygen utilization in the active muscle explains the faster $\dot{V}O_2$ MRT. The positive correlation between the $\dot{V}O_2$ MRT and DeoxyHb MRT indicates a faster oxygen utilization, i.e., $(Ca\text{-}Cv)O_2$ increase, in the active muscle accelerates the $\dot{V}O_2$ response. The present findings suggest that the difference in the sporting event results in different relationship between $\dot{V}O_2$ MRT and DeoxyHb MRT in subjects.

In summary, we examine what factors induce variation of oxygen uptake kinetics response among individuals. Considering the known cause and effect relationship, it was suggested that the variation in heart rate response explain the variation of oxygen uptake response through a cardiac output response. The maximal oxygen uptake did not related to the $\dot{V}O_2$ MRT. The heart rate response is altered by the amplitude of the heart rate and $\dot{V}O_2$max. The $\dot{V}O_2$max did not alter the $\dot{V}O_2$ response because non-dynamic factors could change the response. The DeoxyHb response affects the $\dot{V}O_2$ response. It was suggested that responses of central circulation and local oxygen utilization affects individual $\dot{V}O_2$ response in various training status.

## References


1) Koga S, Barstow TJ, Shiojiri T, Fukuba Y, Kondo N, Fukuoka Y, Shibasaki M, and Poole DC (2993): Pulmonary and muscle oxygen uptake kinetics at the onset of exercise, In Nose H, Mack GW, Imaizumi K (eds), Exercise, Nutrition, and Environmental Stress vol. 3. Cooper Publishing, MI, USA, pp. 63-84.

2) Hayashi N, Ishihara M, Tanaka A, Yoshida T (1999): Impeding $O_2$ unloading in muscle delays oxygen uptake response to exercise onset in humans. Am J Physiol 277 (Regulatory Integrative Comp Physiol 46): R1274-R1281.

3) Hughson RL, Cochrane JE, Butler GC (1993): Faster $O_2$ uptake kinetics at onset of supine exercise with than without lower body negative pressure. J Appl Physiol 75: 1962-1967.

4) Hughson RL, Kowalchuk JM (1995): Kinetics of oxygen uptake for submaximal exercise in hyperoxia, normoxia, and hypoxia. Can J Appl Physiol 20: 198-210.

5) Shoemaker JK, Hodge L, Hughson RL (1994): Cardiorespiratory kinetics and femoral artery blood velocity during dynamic knee extension exercise. J Appl Physiol 77: 2625-2632.

6) Grassi B, Poole DC, Richardson RS, Knight DR, Erickson BK, Wagner PD (1996): Muscle $O_2$ uptake kinetics in humans: implications for metabolic control. J Appl Physiol 80: 988-998.

7) Grassi B, Gladden B, Samaja M, Stary CM, Hogan MC (1998): Faster adjustment of O2 delivery does not affect VO2 on-kinetics in isolated in situ canine muscle. J Appl Physiol 85: 1394-1403.

8) Poole DC, Richardson RS (1997): Determinants of oxygen uptake: Implications for exercise testing. Sports Med 24: 308-320.

9) Sahlin K, Ren JM, Broberg S (1988): Oxygen deficit at the onset of submaximal exercise is not due to a delayed oxygen transport. Acta Physiol Scand 134: 175-180.

10) Shiojiri T, Shibasaki M, Aoki K, Kondo N, Koga S (1997): Effect of reduced muscle temperature on the oxygen uptake kinetics at the start of exercise. Acta Physiol Scand 159: 327-333.

11) Yoshida T, Kamiya J, Hishimoto, K (1995): Are oxygen uptake kinetics at the onset of exercise speeded up by local metabolic status in active muscle? Eur J Appl Physiol 70: 482-486.

12) Cooper DM., Berry C, Lamarra N, Wasserman K (1985): Kinetics of oxygen uptake and heart rate at onset of exercise in children. J Appl Physiol 59: 211-217.

13) Di Prampero PE, Davies CTM, Cerretelli P, Margaria R (1970): An analysis of $O_2$ debt contracted in submaximal exercise. J Appl Physiol 29: 547-551.

14) Grucza R, Nakazono Y, Miyamoto Y (1989): Cardiorespiratory response to absolute and relative work intensity in untrained men. Eur J Appl Physiol 59: 59-67.

myoglobin (Mb).[25,34,35] The NIRS method evaluates the sum of changes in Hb and Mb. Consequently, increase in the DeoxyHb indicates the extraction of oxygen from haemoglobin and myoglobin in the muscle tissue.

The DeoxyHb MRT did not correlate with the $\dot{V}O_2$ MRT in all subjects. This must be explained by different adaptations in local response. It is natural that different training induces different local adaptationsspeculated by Fukuoka et al.[26] In the present study, subjects were trained in different sporting events, so the local metabolism response must be different among subjects.

On the other hand, we found a significant positive correlation betweenthe DeoxyHb and $\dot{V}O_2$ MRT in either swimmer or untrained group. This is consistent with the previous observation in sedentary subjects.[33] It is plausible that faster oxygen utilization in the active muscle explains the faster $\dot{V}O_2$ MRT. The positive correlation between the $\dot{V}O_2$ MRT and DeoxyHb MRT indicates a faster oxygen utilization, i.e., $(Ca\text{-}Cv)O_2$ increase, in the active muscle accelerates the $\dot{V}O_2$ response. The present findings suggest that the difference in the sporting event results in different relationship between $\dot{V}O_2$ MRT and DeoxyHb MRT in subjects.

In summary, we examine what factors induce variation of oxygen uptake kinetics response among individuals. Considering the known cause and effect relationship, it was suggested that the variation in heart rate response explain the variation of oxygen uptake response through a cardiac output response. The maximal oxygen uptake did not related to the $\dot{V}O_2$ MRT. The heart rate response is altered by the amplitude of the heart rate and $\dot{V}O_2$max. The $\dot{V}O_2$max did not alter the $\dot{V}O_2$ response because non-dynamic factors could change the response. The DeoxyHb response affects the $\dot{V}O_2$ response. It was suggested that responses of central circulation and local oxygen utilization affects individual $\dot{V}O_2$ response in various training status.

## References


1) Koga S, Barstow TJ, Shiojiri T, Fukuba Y, Kondo N, Fukuoka Y, Shibasaki M, and Poole DC (2993): Pulmonary and muscle oxygen uptake kinetics at the onset of exercise, In Nose H, Mack GW, Imaizumi K (eds), Exercise, Nutrition, and Environmental Stress vol. 3. Cooper Publishing, MI, USA, pp. 63-84.

2) Hayashi N, Ishihara M, Tanaka A, Yoshida T (1999): Impeding $O_2$ unloading in muscle delays oxygen uptake response to exercise onset in humans. Am J Physiol 277 (Regulatory Integrative Comp Physiol 46): R1274-R1281.

3) Hughson RL, Cochrane JE, Butler GC (1993): Faster $O_2$ uptake kinetics at onset of supine exercise with than without lower body negative pressure. J Appl Physiol 75: 1962-1967.

4) Hughson RL, Kowalchuk JM (1995): Kinetics of oxygen uptake for submaximal exercise in hyperoxia, normoxia, and hypoxia. Can J Appl Physiol 20: 198-210.

5) Shoemaker JK, Hodge L, Hughson RL (1994): Cardiorespiratory kinetics and femoral artery blood velocity during dynamic knee extension exercise. J Appl Physiol 77: 2625-2632.

6) Grassi B, Poole DC, Richardson RS, Knight DR, Erickson BK, Wagner PD (1996): Muscle $O_2$ uptake kinetics in humans: implications for metabolic control. J Appl Physiol 80: 988-998.

7) Grassi B, Gladden B, Samaja M, Stary CM, Hogan MC (1998): Faster adjustment of O2 delivery does not affect VO2 on-kinetics in isolated in situ canine muscle. J Appl Physiol 85: 1394-1403.

8) Poole DC, Richardson RS (1997): Determinants of oxygen uptake: Implications for exercise testing. Sports Med 24: 308-320.

9) Sahlin K, Ren JM, Broberg S (1988): Oxygen deficit at the onset of submaximal exercise is not due to a delayed oxygen transport. Acta Physiol Scand 134: 175-180.

10) Shiojiri T, Shibasaki M, Aoki K, Kondo N, Koga S (1997): Effect of reduced muscle temperature on the oxygen uptake kinetics at the start of exercise. Acta Physiol Scand 159: 327-333.

11) Yoshida T, Kamiya J, Hishimoto, K (1995): Are oxygen uptake kinetics at the onset of exercise speeded up by local metabolic status in active muscle? Eur J Appl Physiol 70: 482-486.

12) Cooper DM., Berry C, Lamarra N, Wasserman K (1985): Kinetics of oxygen uptake and heart rate at onset of exercise in children. J Appl Physiol 59: 211-217.

13) Di Prampero PE, Davies CTM, Cerretelli P, Margaria R (1970): An analysis of $O_2$ debt contracted in submaximal exercise. J Appl Physiol 29: 547-551.

14) Grucza R, Nakazono Y, Miyamoto Y (1989): Cardiorespiratory response to absolute and relative work intensity in untrained men. Eur J Appl Physiol 59: 59-67.