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EFFECTS OF BLOOD LACTATE ON OXYGEN UPTAKE KINETICS DURING RECOVERY AFTER SPRINT IN HUMANS

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ABSTRACT: The purpose of this study was to examine the effects of blood lactate level (La) on oxygen uptake ($\dot{V}O_2$) kinetics during recovery after short-term exercise with maximal effort (sprint). Three sprints were performed on a cycle ergometer with a load of 8% of body weight at maximal rotation rate. $\dot{V}O_2$ kinetics and oxygen debt were determined after three sprint tests: one 10-s cycling sprint, five repeated 10-s cycling sprints with 6-min intervals and one 30-s cycling sprint. There was no significant difference between peak power outputs in the 10-s sprint and five sprints. There was no difference in $\dot{V}O_2$ kinetics during recovery from one sprint and during recovery after five sprints. La peaked at 5 min. The peak value of La was significantly lower in one sprint (4.41 ± 0.9 mM) than in five sprints (7.01 ± 2.2 mM). Thus, despite a difference in La, there was no difference between $\dot{V}O_2$ kinetics during recovery after one sprint and after five sprints. There was a significant difference in $\dot{V}O_2$ between the five sprints and 30-s sprint from 70 s to 320 s during recovery, but there were no significant differences in La after 5 min of recovery. There were two phases in $\dot{V}O_2$. They consisted of fast oxygen debt and slow oxygen debt. There were also no differences in slow and fast oxygen debts between the two 10-s sprints despite significant differences in blood lactate during recovery. Peak La in the five sprints was not significantly different from that in the 30-s sprint (8.68 ± 1.2 mM). However, slow oxygen debt was significantly greater in the 30-s sprint than in the five sprints. It is concluded that $\dot{V}O_2$ kinetics during recovery are not affected by an increase in blood lactate.

KEY WORDS: blood lactate, oxygen uptake kinetics, oxygen debt, cycling sprint, muscle fatigue

INTRODUCTION

Oxygen uptake ($\dot{V}O_2$) exponentially increases at the onset of moderate exercise and then rapidly decreases at the offset of exercise (fast phase) [25]. In heavy exercise above the lactate threshold (LT), $\dot{V}O_2$ has one fast phase [24,25] or two phases at the offset of exercise [6,15]. In severe exercise, $\dot{V}O_2$ has fast and slow phases at the offset of exercise [24]. The integration of slow phase used to be called lactacid oxygen debt [17,18] since it was considered that the post-exercise fate of lactate was that ~80% was taken up by the liver and resynthesized into glucose or glycogen and 20% oxidized to O_2 and CO_2 [13]. However, it has been pointed out that this name is not appropriate since an experiment using rats has shown that oxidation is the primary post-exercise fate of lactate [9].

This finding in rats has been tested by leg occlusion in moderate exercise in humans [26]. It has shown that blood lactate level increased by about 5 mM more than that without occlusion but that there was no difference in $\dot{V}O_2$ kinetics during recovery from the two exercises. This suggests that oxidation is the primary fate of lactate. However, Hermansen and Vaage [12] reported that synthesis to glycogen in skeletal muscle is the primary fate of lactate produced during exercise. Åstrand et al. [1] also reported that ~50% of the lactate formed during severe exercise is transformed to glycogen via gluconeogenesis in muscle during recovery and that lactate uptake by the liver is only 10%. For this disagreement, Nordheim and Vollestad [23] showed that in the presence of a high lactate level, muscle glycogen is resynthesized in inactive type II muscle fibres (type II), while lactate is oxidized in type I muscle fibres (type I), which are activated in moderate exercise after severe exercise. A slow phase in $\dot{V}O_2$ during recovery is not always observed at a low blood lactate level [6,15, 24,25]. Therefore, it is hypothesized that a slow phase in $\dot{V}O_2$ during recovery can be observed at blood lactate level above 5 mM.

A sprint can produce lactate. It has been reported that when a sprint for 10 s was repeated with 6-min intervals, blood lactate was accumulated without muscle fatigue [19]. Since in this case, it seemed that $\dot{V}O_2$ at the end of each interval was not changed despite progress of repetition, this sprint mode could be used to test the present hypothesis. Furthermore, as blood lactate level in a sprint for 30 s can reach almost the same level as that in repeated sprints, a sprint for 30 s could also be used as a tool to test the hypothesis.

Thus, the purpose of the present study was to examine the effects of blood lactate level on $\dot{V}O_2$ kinetics during recovery after a sprint.

MATERIALS AND METHODS

Subjects

Six healthy male undergraduate and graduate students participated in the present study. The subjects' mean age, height and body weight were 20.7 ± 1.2 (SD) yr, 178.2 ± 4.1 cm and 68.8 ± 6.8 kg, respectively. Each subject signed a statement of informed consent following a full explanation regarding the nature of the experiment. The Ethics Committee of Hokkaido University Graduate School of Education approved the present study.

Design

Each subject attended our laboratory for three tests. The time intervals between the three consecutive tests were at least 3 days, and all tests were completed within 3 weeks. Body mass (BM) was used to determine the loads of the cycling sprint. Each subject was instructed to refrain from performing intense physical exercise, drinking alcohol, and consuming caffeine products for 24 h prior to each visit.

Experimental protocol

Each subject performed three sprint tests with maximal effort on separate days. The tests included one cycling sprint for 10 s (10-s sprint), one cycling sprint for 30 s (30-s sprint), and a 10-s cycling sprint repeated five times with time intervals of 6 min (five sprints). Each subject came to the laboratory 1 hour before the start of the test. Immediately after coming to the laboratory, the subjects emptied their bladders and were weighed. Then experimental instruments were attached to the subjects before the experiment.

All sprints were performed with a resistive load (Newton units) of $0.075 \cdot BM \cdot 9.81^{-1}$ [2]. Subjects were instructed to pedal as many revolutions as possible during a cycling sprint. For all tests, subjects were in the seated position during exercise and recovery.

Cycling sprint

All exercise tests were carried out on a bicycle ergometer (POWER-MAX-V_{II}, Combi, Tokyo, Japan). The duration and resistive load were adjusted by a built-in computer. The computer also calculated peak rpm (rpm_{peak}) for a given exercise and displayed the results. Time series behaviour in rpm during each cycling sprint was recorded by an online computer at a rate of 10 Hz. Each subject's feet were strapped to the pedals to prevent them from slipping. The seat height was adjusted so that there was a slight bend in the knee joint when the foot pedal was at its lowest position. The rpm was measured by 0.1 s. Load was $0.075 \cdot BM \cdot 9.81^{-1}(F)$ [22]. Therefore, power output (PO) was calculated as follows:

Power output (W) = $rpm \cdot 6 \cdot F \cdot 0.624^{-1}$,

where 6 is the distance calculated by the built-in computer as the flywheel went into a 360-degree roll (m), and 0.624 is the value for transforming Nm units to W units ($Nm \cdot min^{-1} \cdot W^{-1}$).

Fatigue index was defined as $((PPO - PE) \cdot PPO^{-1}) \cdot 100$, where PE is the power output at the end of the sprint and PPO is peak power output [2,19].

Blood lactate concentration (La)

Blood samples (25 μL) were collected from fingertips using capillary tubes and analyzed using a lactate analyser (YSI 1500 SPORT, YSI, OH, USA) to measure blood lactate concentration. The lactate analyser was calibrated with a standard lactate solution of 5 $mmol \cdot L^{-1}$ before each test. Blood was sampled at rest (Rest), immediately after the cycling sprint, and at 5 min, 10 min, 20 min and 30 min during recovery.

Respiration gas

Data on respiration gas exchange were obtained breath-by-breath using a respiratory gas analyser (AE-280S, Minato Medical Science, Osaka, Japan). Ventilation (VE) was measured by a hot-wire flow meter, and the flow meter was calibrated with a syringe of known volume (2.0 L). O_2 and CO_2 concentrations were measured by a zirconium sensor and infrared absorption analyser, respectively. The gas analyser was calibrated by known standard gas (O_2: 15.17%, CO_2: 4.92%). Respiration gas exchange was measured continuously during rest, exercise, and recovery periods. $\dot{V}O_2$ and CO_2 output (Vco_2) were calculated for each 10-s interval.

Application of approximation equations for $\dot{V}O_2$ during recovery

The following equations were applied for $\dot{V}O_2$ kinetics during recovery. Data for 30 s immediately after the sprint were not used for the approximation because a time delay from active muscle to the lungs appears after a sprint. The goodness of fit by Eq. 1 or Eq. 2 was evaluated by the correlation coefficient.

$$\dot{V}O_2(t) = A_1 \cdot \exp(-(t-TD)/\tau_1) + \dot{V}O_{2,base}, \quad (1)$$

$$\dot{V}O_2(t) = A_1 \cdot \exp(-(t-TD)/\tau_1) + A_2 \cdot xp(-(t-TD)/\tau_2) + \dot{V}O_{2,base}, \quad (2)$$

where $\dot{V}O_2(t)$ is $\dot{V}O_2$ at time t, $\dot{V}O_{2,base}$ is baseline $\dot{V}O_2$, A_1 is amplitude of the fast phase, A_2 is amplitude of the slow phase, TD is time delay, τ_1 is time constant of the fast phase and τ_2 is time constant of the slow phase. Fast oxygen debt was calculated by $A_1 \cdot \tau_1$. Slow oxygen debt was calculated by $A_2 \cdot \tau_2$.

Statistical analysis

All data are presented as means ± SD. Correlation coefficients were used to evaluate the goodness of fit of approximation equations and the relationship between fatigue index and slow oxygen debt. The paired t-test was used to evaluate the difference in goodness of fit by Eq. 1 and Eq. 2. The paired t-test was also used to evaluate the significance of difference in $\dot{V}O_2$ kinetics during recovery among sprints. We found a significant difference in $\dot{V}O_2$ kinetics during recovery with the paired t-test. Therefore, one-way ANOVA was used to evaluate the differences in La, parameters of Eq. 2 and

O_2 debt among the three sprint modes. If the F ratio was significant, the means were compared by using Fisher's PLSD post hoc test. The level of significance was set at $p<0.05$.

RESULTS

Table 1 shows PPO and fatigue index. There were no significant differences in PPO among the three sprint modes. Fatigue index in the 30-s sprint was significantly greater than that in the other sprint modes.

Figure 1 shows blood lactate levels at rest and during recovery from the three sprint modes. La in the 10-s sprint was significantly lower than the levels in the other sprint modes at each measurement point during recovery. La in the 30-s sprint was significantly lower than that in the five sprints at 0 min during recovery. There were no significant differences between La levels in the 30-s sprint and the five sprints at 5, 10, 20, and 30 min during recovery. Peak values in La were 4.41 ± 0.9 mM in one sprint, 7.01 ± 2.2 mM in five sprints and 8.68 ± 1.2 mM in the 30-s sprint.

TABLE 1. PEAK POWER OUTPUT (PPO) AND FATIGUE INDEX IN THE THREE SPRINT MODES

	PPO	Fatigue Index
10-s sprint	757 ± 105	11.8 ± 6.5
5 sprints	774 ± 70	10.9 ± 6.1
30-s sprint	766 ± 115	43.2 ± 8.5

Note: The values are presented as mean ± SD

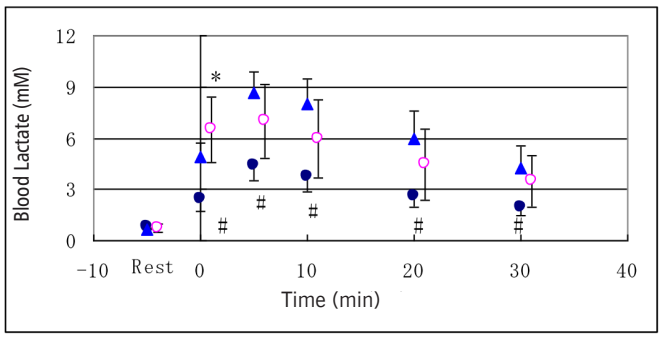

FIG. 1. BLOOD LACTATE AT REST AND DURING RECOVERY AFTER THE 10-S SPRINT (CLOSED CIRCLES), 5 SPRINTS (OPEN CIRCLES) AND 30-S SPRINT (TRIANGLES).
Note: #: significant difference between 10-s sprint and 5 sprints or 30-s sprint. *: significant difference between 5 sprints and 30-s sprint. plots of 5 sprints are shifted to avoid overlap

Figure 2 shows $\dot{V}O_2$ during recovery after the three sprint modes $\dot{V}O_2$ during recovery from the 10-s sprint increased until 30 s and then decreased. Its peak value was 1872 ± 295 ml·min^{-1}. $\dot{V}O_2$ during recovery from the five sprints increased until 30 s and then decreased. Its peak value was 1923 ± 121 ml·min^{-1}. $\dot{V}O_2$ in the 30-s sprint showed a peak value at 10 s during exercise (2253 ± 1111 ml·min^{-1}), and the decrease in $\dot{V}O_2$ during recovery was slow for 30 s and then rapid. There was no difference between the values of $\dot{V}O_2$ after the 10-s sprint and after the five sprints. $\dot{V}O_2$ was significantly higher from 70 s to 320 s of recovery after the 30-s sprint than after the five sprints. Correlation coefficients obtained by Eq. 1 were all significantly lower than those obtained by Eq. 2 (Table 2). Variations of data from the lines represented by the equations were

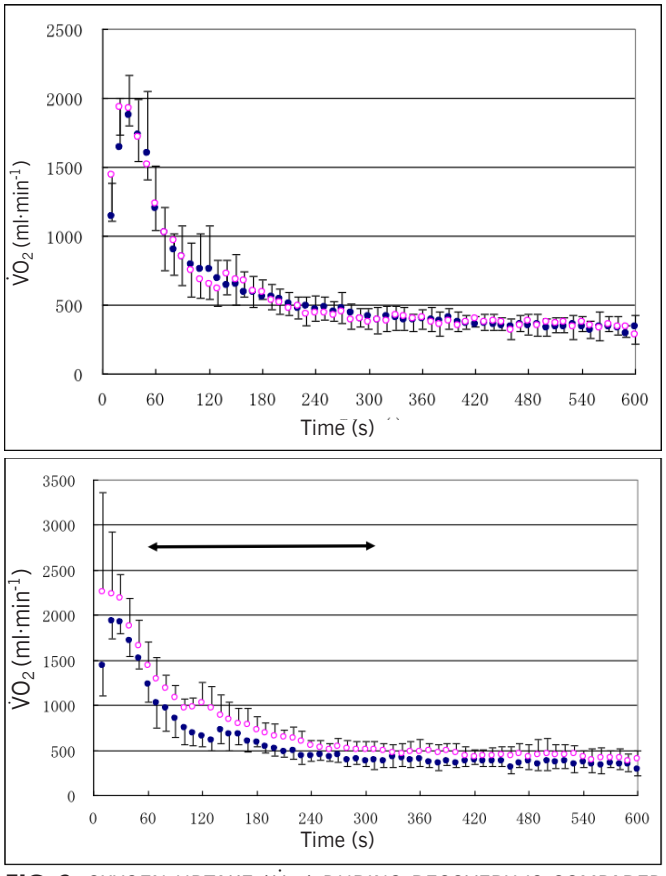

FIG. 2. OXYGEN UPTAKE ($\dot{V}O_2$) DURING RECOVERY IS COMPARED BETWEEN THE 10-S SPRINT (CLOSED CIRCLES) AND 5 SPRINTS (OPEN CIRCLES) IN THE UPPER PANEL AND BETWEEN THE 5 SPRINTS (CLOSED CIRCLES) AND 30-S SPRINT (OPEN CIRCLES) IN THE LOWER PANEL. ARROW SHOWS SIGNIFICANT DIFFERENCE IN $\dot{V}O_2$

TABLE 2. CORRELATION COEFFICIENTS IN APPROXIMATION OF OXYGEN UPTAKE DURING RECOVERY BY EQUATION 1 AND EQUATION 2

	10-s sprint		5 sprints		30-s sprint	
	Eq. 1	Eq. 2	Eq. 1	Eq. 2	Eq. 1	Eq. 2
Mean	0.940	0.960*	0.905	0.925*	0.949	0.962*
SD	0.010	0.009	0.042	0.038	0.015	0.012
Variation (%)	11.6	7.8	18.1	14.5	9.9	7.4

Note: *: significant difference between equations 1 and 2.

TABLE 3. PARAMETERS OBTAINED BY EQUATION 2, OXYGEN DEBT FOR FAST PHASE (O_2 DEBT-F) AND SLOW PHASE (O_2 DEBT-S) AND TOTAL OF SLOW AND FAST OXYGEN DEBT (O_2 DEBT-T)

		10-s sprint	5 sprints	30-s sprint
A_1	$ml \cdot min^{-1}$	1930 ± 433	1648 ± 289	1614 ± 296
τ_1	s	41.3 ± 21.3	30.4 ± 20.5	54.1 ± 26.9
A_2	$ml \cdot min^{-1}$	413 ± 148	490 ± 217	556 ± 290
τ_2	s	268 ± 85	365 ± 410	594 ± 604
TD	s	25.6 ± 3.0	26.7 ± 3.4	17.2 ± 9.3
$\dot{V}O_2$base	$ml \cdot min^{-1}$	288 ± 47	301 ± 60	288 ± 100
O_2debt-F	l	1.31 ± 0.66	0.87 ± 0.66	1.51 ± 0.82
O_2debt-S	l	1.72 ± 0.40	1.85 ± 0.47	4.13 ± 2.92 *,#
O_2debt-T	l	3.03 ± 0.59	2.72 ± 1.07	5.64 ± 3.30 *,#

Note: The values are presented as mean ± SD; *: significant difference between 10-s sprint and 30-s sprint. #: significant difference between 5 sprints and 30-s sprint

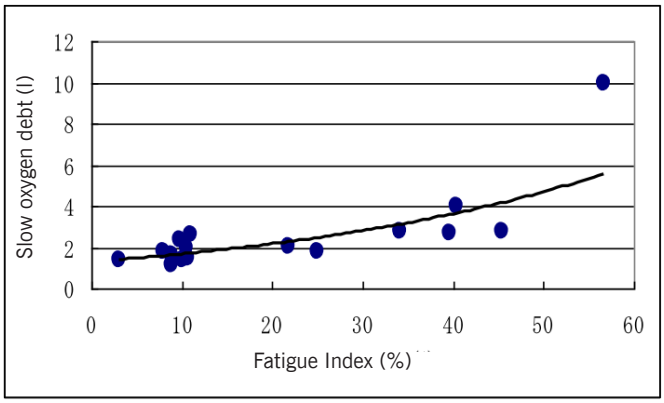

FIG. 3. RELATIONSHIP BETWEEN FATIGUE INDEX AND SLOW OXYGEN DEBT

calculated by $(1 - r^2) \cdot 100$, where r is the correlation coefficient. Eq. 2 showed a good fit. Therefore, Table 3 shows the parameters obtained by Eq. 2. Total oxygen debt and slow oxygen debt in the 30-s sprint were significantly longer than those in the other sprint modes.

Figure 3 shows the relationship between fatigue index and slow oxygen debt. There was an exponential relationship (r=0.836). The approximation equation was $1.28 \cdot \exp(0.0258 \cdot La)$.

DISCUSSION

The purpose of the present study was to examine the effect of blood lactate level on $\dot{V}O_2$ kinetics during recovery by using three sprint modes. There was no significant difference between $\dot{V}O_2$ kinetics after the 10-s sprint and that after the five sprints despite the difference in blood lactate. Despite the significant difference in $\dot{V}O_2$ kinetics during recovery, there was no significant difference in La between the five sprints and 30-s sprint. This difference in $\dot{V}O_2$ kinetics was due to the slow oxygen debt.

There are various definitions of oxygen deficit. For example, in moderate exercise, $\dot{V}O_2$ at a steady state is used to calculate the oxygen deficit as oxygen requirement level [25]. In heavy exercise, $\dot{V}O_2$ at 3 min [25], total amplitude of the fast and slow phases in $\dot{V}O_2$ [4], or amplitude for the fast phase and amplitude for the additional slow phase in $\dot{V}O_2$ [6] is regarded as the oxygen requirement level. In supramaximal exercise, the oxygen requirement level is estimated from the $\dot{V}O_2$-power output relationship obtained at submaximal exercise [3]. In this case, the relationship is assumed to be linear, but it is known that $\dot{V}O_2$ obtained in exercise above the lactate threshold [LT] is greater than $\dot{V}O_2$ estimated from the relationship obtained in exercise below LT [24,25,28,29]. Thus, the definition of oxygen deficit is still under consideration. Therefore, it is difficult to determine the significance of oxygen debt only from the oxygen deficit-debt relationship in short intensive exercise such as that in the present study.

In the present study, oxygen debt was calculated by using the time constant and amplitude of $\dot{V}O_2$ kinetics during recovery, and time delay was not used for the calculation. In the calculation of oxygen deficit as well as oxygen debt, time delay is usually taken into consideration [6]. This is true when oxygen deficit and oxygen debt are compared at the lung level. However, time delay is thought to be due to the time delay in circulation from muscle to lungs. In fact, after the sprint, there was an increasing phase, suggesting delayed appearance from the $\dot{V}O_2$ with muscle working. Therefore, we prefer to estimate oxygen debt at the muscle level rather than at the lung level.

$\dot{V}O_2$base was assumed to be constant. This assumption is affected by increases in catecholamine concentration, ventilation, heart rate and lactate uptake in inactive muscle [5,9]. However, ventilation and heart rate have little effect on oxygen debt [17]. Also, the difference between slow oxygen debt after the 30-s sprint and that after the five sprints is too great to be explained by these effects.

It was hypothesized that the slow phase in $\dot{V}O_2$ during recovery can be observed at a blood lactate level above 5 mM. In the present study, despite blood lactate level being below 5 mM after the 10-s sprint, there were two phases. This suggests that the slow phase is not associated with glycogen synthesis. Furthermore, there were no differences in the two phases between the 10-s sprint and the five sprints, although blood lactate level was increased by the five sprints. This suggests that the effect of increased blood lactate is less on slow oxygen debt.

Contents of leg oxygen debt in one knee-extension exercise have been estimated as follows [3]. Resynthesis of nucleotides and creatine phosphate (CP) accounted for less than 10% of the leg oxygen debt, and lactate elimination including resynthesis of glycogen accounted for another 25%. Most of the glycogen synthesis from lactate occurred within 10 min. There was little reloading of haemoglobin and myoglobin in one knee-extension exercise (although oxygen debt in cycling exercise includes repayment of oxygen stores as haemoglobin combined with oxygen in venous blood). It has also been shown that 2/3 of leg oxygen debt was not accounted for by restoration of these energy sources [3].

Muscle fatigue occurred in the 30-s sprint. CP has been shown to decrease from 80.7 to 36.1 mM (45%) in a 10-s sprint [8] and from 77.1 to 15.1 mM (20%) in a 30-s sprint [10]. If CP restoration occurs only in the fast phase, there should be a difference between fast oxygen debts after the 10-s and 30-s sprints. The half time of CP resynthesis has been reported to be 56.6 s [7]. This corresponds to a time constant of 82 s (half time = 0.693·time constant). It has been reported that CP resynthesis has two phases [11,21]. Therefore, slow oxygen debt may include CP resynthesis. However, the present results showed that total oxygen debt was increased by about 50%. This increase cannot be explained only by the difference in CP resynthesis.

It is thought that power output with maximal effort is performed by both type I and type II but that the participation ratio of types I and II is dependent on speed [27]. If participation of type I is associated with the fast phase and type II with the slow phase, two phases can exist in $\dot{V}O_2$ kinetics during recovery. In an experiment using cats, CP recovered more slowly in fast-twitch muscle than in slow-twitch muscle [16]. Although an electromyographic (EMG) study showed that the mean power frequency (MPF) for the vastus lateralis increased in ten repetitions of a 10-s sprint with 6-min intervals [19], MPF for the rectus femoris in a 30-s sprint decreased during the exercise [20]. Since this suggests that type I was recruited in the 30-s sprint, fast oxygen debt should have been greater in the 30-s sprint. However, the present results do not support this possibility.

$\dot{V}O_2$ during recovery from severe exercise could be affected by both blood lactate and muscle fatigue, but since muscle fatigue accompanies higher blood lactate level in constant exercise, we cannot distinguish the effects of fatigue and blood lactate on $\dot{V}O_2$ during recovery by this type of exercise. However, repetition of 10-s cycling sprints with 6-min intervals can induce an increase in blood lactate without remarkable muscle fatigue [19]. In this type of exercise, the effect of blood lactate on $\dot{V}O_2$ during recovery can be extracted without the effect of muscle fatigue. Furthermore, power output is reduced due to muscle fatigue if a cycling sprint continues for longer time [8,11,20]. In this case, blood lactate is increased, but if the repetition of a 10-s sprint is compared with this long sprint, the effect of muscle fatigue could be extracted under the condition of high blood lactate level.

Since it has been shown by irreversible thermodynamics that the glycolytic pathway is a dissipative structure [10,22], the concept has shifted from molecular biology to system biology. From this concept, it is interpreted that muscle fatigue is not induced by a molecular action (e.g. lactate) but by a change in the total muscular cellular system. A large amount of oxygen may be required for recovery from the system change due to muscle fatigue. This may be the slow oxygen debt.

CONCLUSIONS

It is concluded that $\dot{V}O_2$ kinetics during recovery are not affected by an increase in blood lactate. Instead of blood lactate, it seems that muscle fatigue is an important factor.

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