# Blood Lactate Changes during Isocapnic Buffering in Sprinters and Long Distance Runners

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**Abstract**  This study was carried out to compare blood lactate changes in isocapnic buffering phase in an incremental exercise test between sprinters and long distance runners, and to seek the possibility for predicting aerobic or anaerobic potential from blood lactate changes in isocapnic buffering phase.  Gas exchange variables and blood lactate concentration ([lactate]) in six sprinters (SPR) and nine long distance runners (LDR) were measured during an incremental exercise test ($30 \ W \cdot min^{-1}$) up to subject's voluntary exhaustion on a cycle ergometer. Using a difference between [lactate] at lactate threshold (LT) and [lactate] at the onset of respiratory compensation phase (RCP) and the peak value of [lactate] obtained during a recovery period from the end of the exercise test, the relative increase in [lactate] during the isocapnic buffering phase ($[lactate]_{ICBP}$) was assessed. The [lactate] at LT (mean ± SD) was similar in both groups ($1.36 \pm 0.27$ for SPR vs. $1.24 \pm 0.24$ $mmol \cdot l^{-1}$ for LDR), while the [lactate] at RCP and the peak value of [lactate] were found to be significantly higher in SPR than in LDR ($3.61 \pm 0.33$ vs. $2.36 \pm 0.45$ $mmol \cdot l^{-1}$ for RCP, P<0.001, $10.18 \pm 1.53$ vs. $8.10 \pm 1.61$ $mmol \cdot l^{-1}$ for peak, P<0.05).  The $[lactate]_{ICBP}$ showed a significantly higher value in SPR ($22.5 \pm 5.9\%$, P<0.05) compared to that in LDR ($14.2 \pm 5.0\%$) as a result of a twofold greater increase of [lactate] from LT to RCP ($2.25 \pm 0.49$ for SPR vs. $1.12 \pm 0.39$ $mmol \cdot l^{-1}$ for LDR).  In addition, the $[lactate]_{ICBP}$ inversely correlated with oxygen uptake at LT ($\dot{V}O_2LT$, r=−0.582, P<0.05) and maximal oxygen uptake ($\dot{V}O_2max$, r=−0.644, P<0.01).  The results indicate that the $[lactate]_{ICBP}$ is likely to give an index for the integrated metabolic, respiratory and buffering responses at the initial stage of metabolic acidosis derived from lactate accumulation. *J Physiol Anthropol 21 (3): 143–149, 2002* http://www.jstage.jst.go.jp/en/

**Keywords:** end-tidal $CO_2$ partial pressure, lactate threshold, respiratory compensation phase, bicarbonate buffering

## Introduction

It is generally recognized that during incremental exercise the end-tidal carbon dioxide partial pressure ($P_{ET}CO_2$) at work rates above lactate threshold (LT) becomes relatively constant or increases slightly until the onset of respiratory compensation phase (RCP) in spite of increases in blood lactate concentration ([lactate]). Thereafter the $P_{ET}CO_2$ decreases progressively until the point of fatigue.  This steady phase of $P_{ET}CO_2$ from LT to RCP has been described as "isocapnic buffering" (Wasserman et al., 1981; Whipp and Ward, 1998). Wasserman et al. (1981) have pointed out that the isocapnic buffering phase is characteristically found in work rate increments of short duration (1 min or less) but not in work rate increments of long duration (4 min or more).  In addition, the patients with carotid body resection did not show the isocapnic buffering phase because of the lack of respiratory compensation for metabolic (lactic) acidosis (Wasserman et al., 1975).  It has been therefore hypothesized that the duration of the period in the isocapnic buffering phase would depend on the sensitivity of carotid bodies to metabolic acidosis of exercise (Whipp et al., 1989).

Recently, attempts have been proposed to assess aerobic and anaerobic power from several physiological variables in the basis of the isocapnic buffering phase (Oshima et al., 1997; Röcker et al., 1994).  Physiological responses during this phase would be considered as a better indication for the early adjustments to the initial stage of metabolic acidosis derived from lactate accumulation.  Röcker et al. (1994) regarded LT as a starting point of buffering and RCP as an endpoint of buffering and estimated relative functional buffering capacity from running speed at LT and that at RCP.  In

**Table 1** Physical and physiological characteristics of the subjects

| Group | Age (years) | Height (cm) | Mass (kg) | $\dot{V}O_2LT$ ($ml \cdot kg^{-1} \cdot min^{-1}$) | $\dot{V}O_2max$ ($ml \cdot kg^{-1} \cdot min^{-1}$) |
|---|---|---|---|---|---|
| LDR (n=9) | 20.2 (1.2) | 172.2 (4.3) | 58.6 (3.6) | 38.4 (3.9) | 62.2 (3.6) |
| SPR (n=6) | 19.8 (1.3) | 175.7 (4.5) | 66.4 (6.0)** | 29.3 (4.4)*** | 48.5 (1.5)*** |

Values are given as mean (± SD). $\dot{V}O_2LT$, oxygen uptake per body mass at lactate threshold (LT); $\dot{V}O_2max$, maximal oxygen uptake per body mass. **P<0.01, ***P<0.001, significantly different from LDR.

addition, they found that changes in [lactate] during the isocapnic buffering phase was different among subjects due to training status (i.e. anaerobic or aerobic training). It is therefore inferred that the difference of [lactate] changes from the LT to RCP could be induced by different physiological adaptations to exercise training. However, it is unknown as to whether the [lactate] changes during the isocapnic buffering phase reflect aerobic or anaerobic potential.

This study was carried out to compare [lactate] changes during the isocapnic buffering phase in an incremental exercise test between sprinters and long distance runners, and to seek the possibility for predicting aerobic or anaerobic potential from the [lactate] changes during the isocapnic buffering phase.

## Methods

### Subjects

Six male sprinters (SPR) and nine male long distance runners (LDR), ranging in age from 19 to 22 years, participated in this study. The SPR and LDR groups have been performing sprint- and endurance-training 6 days a week, respectively, for at least 3 years. Informed consent was obtained from all the subjects in accordance with the Policy Statement Regarding the Use of Human Subject of the American College of Sports Medicine after the possible risks and aim of the present study had been explained. Mean values of their age, body height, body mass, oxygen uptake at LT ($\dot{V}O_2LT$) and maximal oxygen uptake ($\dot{V}O_2max$) are listed in Table 1. The best athletic running records (mean ± SD) were 11.20 ± 0.45 s in 100-m in SPR, range from 10.92 to 12.00 s, 32.49 ± 0.77 min in 10,000-m in LDR, range from 31.22 to 33.48 min.

### Exercise protocol and gas exchange variables

The subjects performed an incremental exercise test on a cycle ergometer (Monark, Crescent, Sweden) in order to evaluate the changes of [lactate] as well as of end-tidal $CO_2$ fraction (ET-$CO_2$) during the isocapnic buffering phase. The work rate, after a 4-minute warm-up of pedaling without load, was progressively increased by 30 watts (W) each minute until the subject's limit of tolerance was reached. The variables related to gas exchange (minute ventilation ($\dot{V}_E$), $\dot{V}O_2$, carbon dioxide

production ($VCO_2$), ET-$CO_2$ were continuously measured breath by breath throughout the exercise test by an automatic gas analyzer (AE 280S, Minato Medical Science, Osaka, Japan). This gas analysis system is composed of a zirconia method for $O_2$, an infrared absorption analyzer for $CO_2$ and a hot-wire flowmeter for inspiration and expiration volumes. The analyzers for $O_2$ and $CO_2$ were calibrated by a known standard mixed gas ($O_2$: 15.17%, $CO_2$: 4.927% in $N_2$ balance) and the flow meter by a 2-L syringe before and after the tests.

### Blood lactate measurement

A cannula (21G) was inserted percutaneously into and placed in an antecubital vein, and blood samples for [lactate] determination were withdrawn at rest, every one minute from the 5th min to exhaustion and at the 1st and 3rd min after the end of exercise test. Immediately after blood collection, [lactate] was analyzed by an automatic lactate analyzer (2300 STAT, YSI, USA) which was calibrated by two known lactate standard solutions (5 and 15 $mmol \cdot l^{-1}$).

### Detection of LT and RCP

[lactate] and ET-$CO_2$ were plotted against the time of exercise as shown in Fig. 1. The LT was detected by the point of intersection of the two regression lines between the time of exercise and [lactate] at work rates below and above an abrupt rise of [lactate] from the resting level. Similarly, the RCP was also detected by the point of intersection of the two regression lines between time of exercise and ET-$CO_2$ at work rates below and above the onset of decrease in ET-$CO_2$. This determination of RCP was chosen since the determination of RCP using ET-$CO_2$-time course relation is more reliable in comparison with other volume-dependent variables of gas exchange ($\dot{V}_E$, $\dot{V}O_2$, $\dot{V}CO_2$) (Röcker et al., 1994). The [lactate] corresponded to the RCP was calculated from the interpolation of the relationship between the time of exercise and [lactate] at work rates above LT because in all the subjects the RCP was found at higher work rates than LT.

### Isocapnic buffering phase

According to the previous studies (Oshima et al., 1997; Röcker et al., 1994; Scheuermann and Kowalchuk, 1998;
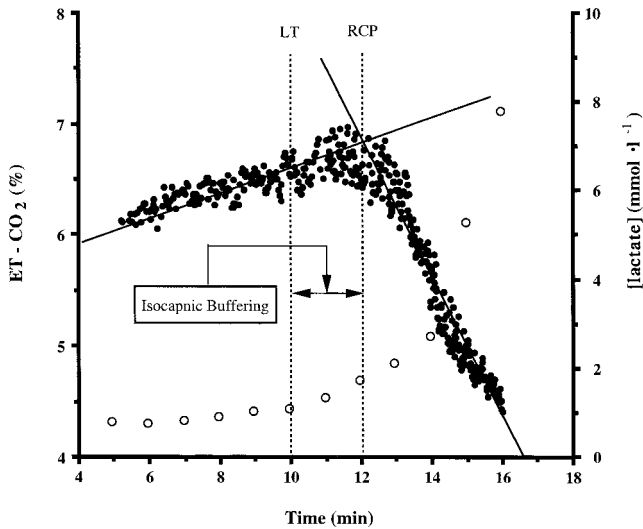
**Fig. 1**  Dynamics of blood lactate concentration ([lactate],  ) and breath-by-breath plots of end-tidal $CO_2$ fraction (ET-$CO_2$,  ) during incremental exercise for a single subject.  Two vertical dotted lines in the figure denote lactate threshold (LT) and onset of respiratory compensation phase (RCP), respectively.  See text for LT and RCP detection.

Whipp et al., 1989), the phase from LT to RCP was defined as isocapnic buffering.  Röcker et al. (1994) calculated relative functional buffering capacity from the treadmill running speeds corresponded to the LT and RCP.  Therefore, the relative increase in [lactate] during the isocapnic buffering phase ([lactate]$_{ICBP}$), expressed as a percentage of the amount of [lactate] accumulation during the isocapnic buffering phase to a total amount of [lactate] accumulation, was calculated by dividing a difference of [lactate] between LT to RCP by a peak value of [lactate] due to the exercise test.

$$[lactate]_{ICBP} (\%) = ([lactate]_{RCP} - [lactate]_{LT}) \div [lactate]_{peak} \times 100$$

where [lactate]$_{RCP}$ and [lactate]$_{LT}$ are the blood lactate concentrations at the RCP and LT, respectively, and [lactate]$_{peak}$ is the peak value of [lactate] obtained at the 1st or 3rd min after the end of the exercise test.

*Statistics*

Data were expressed as mean and standard deviation ($\pm$ SD).  An independent Student's t-test (two-tailed test) was employed to test significant differences of all physiological variables between SPR and LDR.  Linear regression analysis was made by a least-squares method and Pearson's correlation coefficient (r) was used to estimate a relationship between selected variables.  The statistical significance was accepted at P<0.05 level for all tests.

**Results**
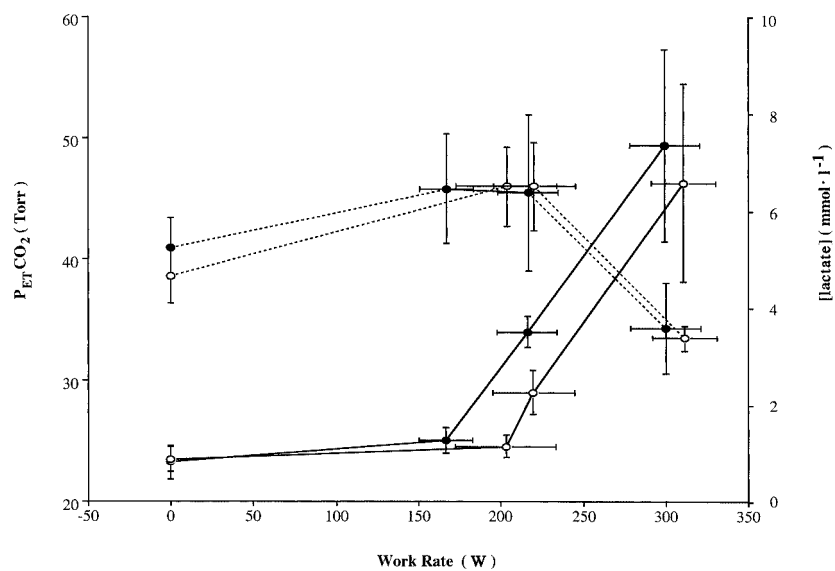
Figure 2 shows mean values ($\pm$ SD) of [lactate] and



**Fig. 2**  Changes in blood lactate concentration ([lactate], solid line) and end-tidal $CO_2$ partial pressure (P$_{ET}CO_2$, dotted line) during incremental exercise in sprinters (  ) and long distance runners (  ).  Points given in the figure are average [lactate] and P$_{ET}CO_2$ versus average work rates for the two groups corresponded to rest (work rate=0), LT, RCP and exhaustion.  Vertical and horizontal bars at each point denote $\pm$ SD.
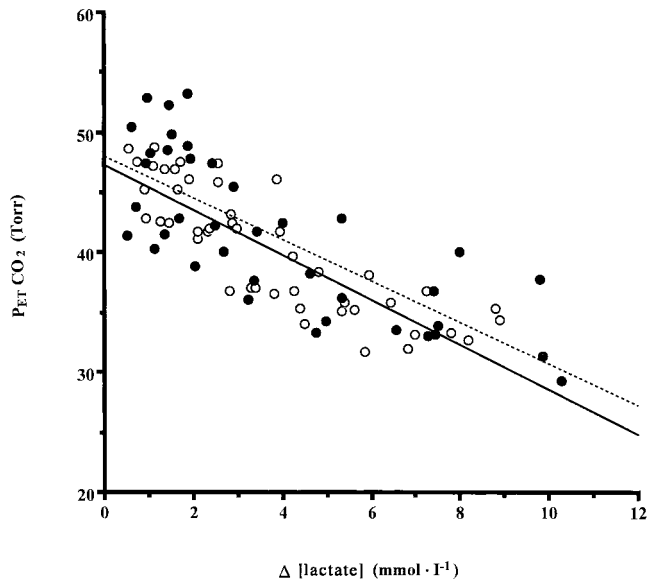
**Fig. 3** Relationships between end-tidal $CO_2$ partial pressure ($P_{ET}CO_2$) and blood lactate increase ($\Delta$[lactate]) from the resting level to work rates above lactate threshold during incremental exercise in sprinters ( ) and long distance runners ( ). Dashed line is for sprinters ($P_{ET}CO_2=48.07-1.74*\Delta$[lactate], r=−0.766, P<0.0001, n=38) and solid line for long distance runners ($P_{ET}CO_2=47.32-1.87*\Delta$[lactate], r=−0.839, P<0.0001, n=46).

$P_{ET}CO_2$ (converted from ET-$CO_2$ with ambient barometric pressure and water vapor pressure at body temperature (37°C)), for the SPR and LDR groups in terms of the absolute work rates at LT, RCP and exhaustion during the incremental test. The $P_{ET}CO_2$ was kept constant between LT and RCP for the two groups. We found a significant difference (P<0.05) in work rate at LT between SPR (167.5 ± 16.3 W) and LDR (203.7 ± 30.5 W), whereas work rates at RCP (216.8 ± 18.3 W for SPR, 220.5 ± 24.7 W for LDR) and at exhaustion (300.0 ± 21.2 W for SPR, 311.7 ± 19.5 W for LDR) were similar. All the subjects showed that the RCP appeared at higher work rates than LT.

$P_{ET}CO_2$ was plotted against [lactate] increase ($\Delta$[lactate]) from the resting level to the work rates above LT during the incremental exercise test (Fig. 3). The $P_{ET}CO_2$ was found to progressively decrease with increasing $\Delta$[lactate] in the two groups. There were no differences in slope and intercept of the regression line for the $P_{ET}CO_2$-[$\Delta$lactate] relationship between SPR ($P_{ET}CO_2=48.07-1.74*\Delta$[lactate]) and LDR ($P_{ET}CO_2=47.32-1.87*$[$\Delta$lactate]), although considerable scatter was observed for the $P_{ET}CO_2$ plots against $\Delta$[lactate] within the identical group.

As given in Table 2, the [lactate] at LT showed similar values in both groups, whereas [lactate] at RCP (P<0.001) and the peak value of [lactate] (P<0.05) were found to be significantly higher in SPR than in LDR. As a consequence, the increase of [lactate] from LT to RCP was significantly greater in SPR (2.25 ± 0.48 mmol·l$^{-1}$) than in LDR (1.12 ± 0.39 mmol·l$^{-1}$) by twofold (P<0.001). The [lactate]$_{ICBP}$ revealed a significantly higher value in SPR (22.5 ± 5.9%) than in LDR (14.2 ± 5.0%) (P<0.05) owing to a twofold larger increase of [lactate] from LT to RCP in SPR, despite the fact that SPR had approximately 2 mmol·l$^{-1}$ higher level in the peak value of [lactate] compared with LDR. In addition, the [lactate]$_{ICBP}$ inversely correlated with both $\dot{V}O_2LT$ (r=−0.582, P<0.05) and $\dot{V}O_2max$ (r=−0.644, P<0.01) (Fig. 4).

## Discussion

The higher [lactate]$_{ICBP}$ in the SPR group was a consequence of a greater increase in [lactate] between the LT and RCP than LDR, not from the difference of the peak value of [lactate] in the two groups as this was higher in SPR than LDR (Table 2). It is, therefore, possible that the greater [lactate] increase from LT to RCP would be attributable to the slower respiratory compensation for metabolic acidosis during incremental exercise. In the literature describing isocapnic buffering, it has been indicated that the duration in the period of isocapnic buffering phase varies among subjects and that the period of isocapnic buffering phase would depend on the sensitivity of carotid bodies to acute metabolic acidosis caused by severe exercise (Wasserman et al., 1981; Whipp et al., 1989). Thus it seems likely that the longer period of the isocapnic buffering phase would be due to lower sensitivity of carotid bodies to acute metabolic acidosis and vice versa. Oshima et al. (1997) reported that the range of isocapnic buffering was longer

**Table 2** Blood lactate concentrations at rest, at lactate threshold (LT), at onset of respiratory compensation phase (RCP), at recovery phase from incremental exercise (Peak) and relative increase of blood lactate during isocapnic buffering phase ([lactate]$_{ICBP}$)

| Group | Rest | LT | RCP | Peak | [lactate]$_{ICBP}$ |
|---|---|---|---|---|---|
| LDR (n=9) | 0.97 (0.27) | 1.24 (0.24) | 2.36 (0.45) | 8.10 (1.61) | 14.2 (5.0) |
| SPR (n=6) | 0.91 (0.35) | 1.36 (0.27) | 3.61 (0.33)*** | 10.18 (1.53)* | 22.5 (5.9)* |

Values are given as mean (± SD). Blood lactate concentration is in mmol·l$^{-1}$ and [lactate]$_{ICBP}$ in %. See text for definition and calculation in [lactate]$_{ICBP}$. *P<0.05, ***P<0.001; significantly different from LDR.

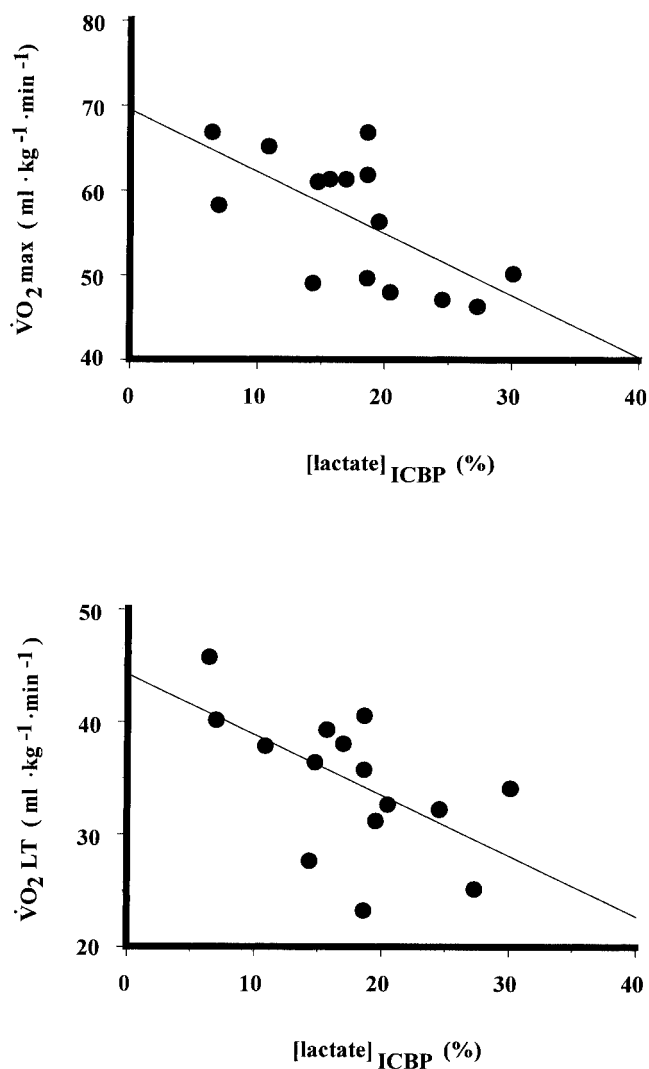**Fig. 4** Relationships between relative lactate increase during isocapnic buffering phase ([lactate]$_{ICBP}$) and maximal oxygen uptake ($\dot{V}O_2$max, upper panel, y=69.41−0.73*x, r=−0.644, P<0.01) and oxygen uptake at lactate threshold ($\dot{V}O_2$LT, lower panel, y=44.10−0.54*x, r=−0.582, P<0.05).

however, there was no difference in the change of $P_{ET}CO_2$ for a 1 mmol·l$^{-1}$ change in [lactate] increase between SPR (1.74 torr decrease) and LDR group (1.87 torr decrease). It is suggested that the magnitude in respiratory compensation for acute metabolic acidosis of exercise would be the same for SPR and LDR.

It had been reported that the [lactate] increase from LT to RCP was significantly greater in sprint-trained than in endurance-trained men (Röcker et al., 1994), which is consistent with the present data regarding [lactate] changes during the isocapnic buffering phase. The greater [lactate] increase from LT to RCP in the SPR group could be accounted for by factors other than the sensitivity of carotid bodies to metabolic acidosis. Some possible explanations for this are 1) lactate kinetic process, and 2) buffering process at the initial stage of lactate production between the two athletic groups which are examined. In this study blood samples for lactate measurements were taken from the antecubital vein. It is known that lactate produced in the leg and released into blood will be taken up and metabolized as it passes through other tissues including "inactive" arm muscle, which will have implications not only on blood lactate concentration differences between arm venous, leg femoral venous and arterial concentrations, but also on the relative "timing" of lactate concentration changes, which probably be delayed relative to instantaneous pulmonary end-tidal gas changes. Therefore, the increase of [lactate] in the venous blood would be the result of several kinetic processes like compartmental distribution, redistribution and metabolism (Gladden, 1996). Blood and muscle lactate have been reported to be lower at the same absolute and relative work rates following endurance training (Holloszy and Coyle, 1984). A differing lactate distribution and metabolism could account for the differences in the increase of [lactate] from LT to RCP during incremental exercise between SPR and LDR.

It has been shown that buffering (non-bicarbonate buffering) capacity of skeletal muscle in sprint-trained men is higher than that in endurance-trained men (Bell and Wenger, 1988; Parkhouse et al., 1985; Sahlin and Henriksson, 1984; Sharp et al., 1986). The initial increase of [lactate] without a concomitant decrease in the blood bicarbonate (Beaver et al., 1986), which would reflect the onset of a non-bicarbonate buffering mechanism within skeletal muscle, could be related to hydrogen ion release from working muscles independently of lactate ion (Barbee et al., 1983; Medbø and Sejersted, 1985). Taken together, at the initial blood lactate accumulation sprint trained-men could buffer more hydrogen ion in non-bicarbonate buffer system (presumably protein and phosphate buffers). In contrast, endurance trained-men could buffer relatively more hydrogen ion in bicarbonate buffer system due to their lower non-bicarbonate

in athletes who had higher $\dot{V}O_2$max than in athletes with lower $\dot{V}O_2$max. From these findings, it would be expected that the different sensitivities of carotid bodies to metabolic acidosis could lead to the different ranges of isocapnic buffering among athletes depending on their aerobic capacity ($\dot{V}O_2$max). Although a direct measurement on the respiratory response of carotid bodies was not carried out in this study, it is thought that the respiratory compensation for metabolic acidosis could be assessed from a relationship between [lactate] increase and $P_{ET}CO_2$ during exercise (Medbø and Sejersted, 1985). As shown in Fig. 3, it was observed to be a considerable scatter for the plots of $P_{ET}CO_2$ against Δ[lactate] even in the identical group. On average,

buffering capacity of skeletal muscle, which would lead to eliminate more excess $CO_2$ resulting from bicarbonate buffering of lactic acid (Hirakoba et al., 1992; Yano, 1987). It is therefore inferred that, at the initial increase of [lactate], the relative magnitude in respiratory compensation for regulating pH would be less in SPR than in LDR. Thus this could result in the slower appearance of RCP and/or higher [lactate] at RCP in SPR.

The [lactate]$_{ICBP}$ reported in this study was expressed as a percentage of the amount of [lactate] accumulation during the isocapnic buffering phase to a total amount of [lactate] accumulation due to the incremental exercise. From the above-mentioned argument that lactate accumulation may not reflect proton accumulation because of the differences in bicarbonate and non-bicarbonate buffering between the two groups, the [lactate]$_{ICBP}$ is thought to reflect both lactate kinetics and buffering processes during the isocapnic buffering phase. Röcker et al. (1994) suggested that the higher relative functional buffering capacity (calculated from the treadmill speeds at the LT and RCP) in sprinters, compared with endurance trained-subjects, might be considered as the higher contents of buffer-active proteins in their skeletal muscle. If the greater increase of [lactate] from LT to RCP is derived from the differences of lactate kinetics and buffering processes in a whole body, it could be expected that the higher [lactate]$_{ICBP}$ in the SPR group is associated with a relatively higher amount of anaerobic energy production to a total energy output as demonstrated by the negative relationships between the [lactate]$_{ICBP}$ and aerobic fitness ($\dot{V}O_2LT$, $\dot{V}O_2max$). However, this attempt to relate the [lactate]$_{ICBP}$ to the relative anaerobic energy production during the isocapnic buffering phase may not be consistent with the contention that [lactate] changes obtained in the venous blood probably reflects a balance between lactate production, accumulation, utilization and transport out of active muscle into blood, and uptake and utilization in other tissues. Consequently, it would be necessary to determine the attempt in this study from the kinetics in lactate, bicarbonate and pH in the artery during the isocapnic buffering phase.

In conclusion, although it is not clear from our results whether the [lactate]$_{ICBP}$ reflects anaerobic potential, aerobic potential or a combination of both, the [lactate]$_{ICBP}$ is likely to give an index for the integrated metabolic, respiratory and buffering responses at the initial stage of metabolic acidosis due to lactate accumulation. The physiological responses during the isocapnic buffering phase may provide useful information on the early adjustments to the initial stage of metabolic acidosis during exercise. However, more detailed assessments will be needed to clarify this possibility.

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