Excess CO₂ Output Response during and after Short-Term Intensive Exercise in Sprinters and Long-Distance Runners

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Abstract: The purpose of the present study was to examine the response of excess CO₂ output to short-term intensive exercise in sprinters (SPR) and long-distance runners (LDR). End-tidal CO₂ pressure (PETCO₂) increased up to about 20 s postexercise and then returned to the resting level at about 2–3 min postexercise. Thereafter, PETCO₂ remained below the resting level. V̇CO₂ excess, defined as the difference between V̇CO₂ and VO₂ was integrated from the start of exercise until PETCO₂ returned to the resting level. This integrated V̇CO₂ excess was defined as the first phase of CO₂ excess (1st CO₂ excess). The subsequent integrated V̇CO₂ excess until 10 min postexercise was defined as the second phase of CO₂ excess (2nd CO₂ excess). The ratio of 1st CO₂ excess to the lactate rise from rest to the peak value was significantly lower in SPR than in LDR, whereas 2nd CO₂ excess was significantly greater in SPR than in LDR. The decrease in PETCO₂ at 10 min postexercise was significantly larger in SPR than in LDR. The 2nd CO₂ excess was closely related to the decrease in PETCO₂. The results in the second phase suggest that the difference in the response of excess CO₂ output is derived from the difference in the respiratory chemosensitivity to lactic acid rise.


Key words: intensive exercise, lactate, CO₂ excess, end-tidal CO₂ pressure, hyperventilation.

During an incremental exercise, lactic acid starts to accumulate in the blood above a certain work rate (anaerobic threshold). Since most of the lactic acid is dissociated into lactate ions (La⁻) and hydrogen ions (H⁺) within physiological pH values in the body fluids [1], H⁺ simultaneously begin to be buffered by the bicarbonate system (H⁺ + HCO₃⁻ ⇌ H₂O + CO₂). Consequently, bicarbonate ions (HCO₃⁻) decrease in proportion to the rise in La⁻ in the arterial blood [2, 3], resulting in excess CO₂ output (CO₂ excess). CO₂ pressure in the arterial blood (PaCO₂) and end-tidal CO₂ pressure (PETCO₂), however, do not change for some time (isocapnic buffering phase) and then decrease until exhaustion (respiratory compensation).

Buffering capacity, which is measured using biopsy, was reported to be higher in sprinters than in long-distance runners [4, 5]. It was later confirmed that sprint training enhances the muscle buffering capacity [6, 7]. These findings suggest that the relative contribution of the bicarbonate system to whole buffering is reduced in sprinters, resulting in less CO₂ excess. In fact, CO₂ excess relative to the rise in blood lactate was reported to be less in sprinters than in long-distance runners [8, 9]. However, as the CO₂ excess reported included the respiratory compensation phase, respiratory CO₂ excess output was not excluded.

The duration of the isocapnic buffering phase was reported to be related to the buffering capacity [10]. It might be possible to interpret that its duration is long in subjects with a high buffering capacity such as sprinters since respiratory compensation is not greatly needed in such subjects. However, as PaCO₂ and PETCO₂ are directly determined by the control of pulmonary respiration, the duration is not thought to be...
determined only by the subject’s buffering capacity. Moreover, since the isocapnic buffering phase continues until exhaustion in the case of a rapid incremental rate [11–13] and begins at the onset of metabolic acidemia in the case of a slow incremental rate [14], it is actually difficult to define the duration of the isocapnic buffering phase.

Thus, as the effects of lactic acid and pulmonary respiration have not been clarified, the difference in the response of excess CO₂ output according to the subject’s characteristics should be examined by another method.

CO₂ kinetics have been examined in constant-load exercise as well as in incremental exercise. In heavy exercise, it is known that the concentration of HCO₃⁻ changes in reciprocal fashion to that of La⁺ in arterial blood, and that PaCO₂ first increases and then decreases to below the resting level [15]. In supramaximum exercise (intensive exercise for 60 s), it has been observed that CO₂ is not completely excessively expired during exercise but after the end of exercise, although lactic acid begins to be produced from the start of the exercise [16]. Furthermore, it was found that PETCO₂ returns to the resting level after a temporary increase during exercise and recovery and continues to decrease to below the resting level [16]. This suggests that CO₂ excess could be distinguished into lactic and respiratory phases before and after the point at which PETCO₂ returns to the resting level during recovery.

We have therefore employed a short-term intensive exercise test to examine the difference in response of excess CO₂ output during the lactic phase and respiratory phase according to a subject’s characteristics.

**METHODS**

Seven male sprinters (SPR) with a mean 100-m running time of 11.1±0.4 s and six male long-distance runners (LDR) with a mean 5000-m running time of 16.09±0.45 min participated in the present experiments. Physical characteristics of the two groups are listed in Table 1. Each subject was informed of the purpose of the study, the experimental procedure, and the risks associated with the experiments before consent was obtained. Furthermore, they were instructed to abstain from heavy training on the day before the experiment and to rest for 1 h prior to the start of the experiment.

A cycle ergometer (Powermax-VII, Combi) was used in the experiments. On the first day, each subject performed a 30-s maximal exercise test at a load (kp) corresponding to 7.5%×body mass (Wingate test) [17]. During the Wingate test, the power output (watts, W) was calculated from the imposed load and cycling rate (rpm) averaged for every 5 s. A few days later, a short-term intensive exercise test (SIET) was performed at a work load corresponding to 80% (SPR, 364±53 W; LDR, 347±37 W) of the power exerted during the last 5 s of the Wingate test. Each subject, after resting on the cycle ergometer for 5 min, performed intensive exercise until he could no longer maintain 90 rpm. After the end of exercise, each subject sat on a chair for a 30-min recovery period.

Ventilation (V̇E), O₂ uptake (V̇O₂), CO₂ output (V̇CO₂) and end-tidal CO₂ pressure (PETCO₂) were measured breath-by-breath using a respiratory gas analyzer (Aeromonitor AE-280S, Minato) throughout the SIET. These data were averaged and recorded for each 20-s period. V̇E was measured by a hot-wire flow meter that was fixed to the front of the subject’s gas mask. The flow meter was calibrated with a syringe of known volume (2.0 l). Sample gas was drawn continuously from the front of the gas mask (220 ml/min) for determination of the fractional concentrations of O₂ and CO₂ by a zirconium sensor and an infrared absorption analyzer, respectively. The gas analyzer was calibrated by known standard gases. Twenty-five microliters of blood was sampled from a finger tip using a capillary tube at rest before exercise, immediately after the end of exercise, and at 3, 6, 10, 20 and 30 min postexercise. The lactate concentration (La) in the sampled blood was determined by an automatic lactate analyzer (1500 sport, YSI). The analyzer was calibrated with a standard liquid (5 mM of lactate, YSI).

Excess CO₂ output per unit time (V̇CO₂ excess) was calculated by subtracting the V̇O₂ values from the V̇CO₂ values during the SIET [16, 18]. The V̇CO₂ excess was integrated from the start of exercise to 10 min postexercise, and this was defined as the total excess CO₂ output (CO₂ excess). The CO₂ excess was divided by body mass and ΔLa10min (the difference between...
tween La at 10 min postexercise and at rest) to obtain the ratio of CO2 excess per body mass to 1 mM rise in La (CO2 excess ratio).

As shown in Fig. 1, individual CO2 excess was separated into two phases. The first phase ranged from the start of exercise until the time when $P_{ETCO2}$ returned to the resting level. The $V_{CO2}$ excess was integrated for this period, which was defined as the first phase of CO2 excess (1st CO2 excess). The subsequent integration of the $V_{CO2}$ excess until 10 min postexercise was defined as the second phase of CO2 excess (2nd CO2 excess).

We used an unpaired $t$-test to compare variables between the two groups. The strength of the relationship between dependent and independent variables was expressed by a single correlation coefficient of Pearson. A $p$ value of 0.05 or less was considered significant. Results are expressed as mean±standard deviation.

RESULTS

Peak power and peak power per body mass during the Wingate test were significantly higher in SPR than in LDR ($p<0.05$ and $p<0.01$, respectively, Table 1).

There was no significant difference in the duration of exercise in the SIET between the two groups (SPR, 88.6±16.8 s; LDR, 81.7±14.7 s).

Figure 1 shows the changes in $V_{CO2}$ excess and $P_{ETCO2}$ in one subject during the SIET. $V_{CO2}$ excess temporarily showed a negative value after the start of exercise and then a positive value. This value peaked at 38.6±22.0 s after the end of exercise for SPR and at 31.6±21.4 s after the end of exercise for LDR. Subsequently, $V_{CO2}$ excess decreased to zero at 10.43±2.29 min postexercise for SPR and at 11.53±2.12 min postexercise for LDR. $P_{ETCO2}$ increased up to approximately 20 s after the end of exercise and then returned to its pre-exercise level at 1.91±0.43 and 3.53±1.06 min postexercise in SPR and LDR, respectively. Subsequently, $P_{ETCO2}$ was lower than the resting level over the whole recovery period. $\Delta P_{ETCO2}$ (the difference between $P_{ETCO2}$ at 10 min postexercise and at rest) was significantly ($p<0.01$) larger in SPR (−8.21±1.36 mmHg) than in LDR (−4.48±2.47 mmHg).

As shown in Fig. 2, La reached peak values at 3 min after the end of exercise and then decreased in both groups. There were no significant differences in La during recovery between the two groups. There were also no significant differences in $\Delta L_{a10min}$ between the two groups (SPR, 8.13±1.95 mM; LDR, 6.99±1.58 mM) or in $\Delta L_{apeak}$ (the difference between La at peak and at rest) between the two groups (SPR, 9.46±1.61 mM; LDR, 8.22±1.42 mM).

As shown in Table 2, CO2 excess per body mass (CO2 excess/w) and the ratio of CO2 excess/w to $\Delta L_{a10min}$ (CO2 excess ratio) were similar in the two groups. A significant correlation ($r=0.77$, $p<0.01$, Fig. 3) was found between CO2 excess/w and $\Delta L_{a10min}$ when the data from the two groups were combined.

We separated CO2 excess into 1st CO2 excess and...
2nd CO₂ excess. There was no significant difference in 1st CO₂ excess/w between the two groups (Table 2).

The values of \( \dot{V}_{\text{CO₂}} \) excess were integrated during the exercise period. The value in SPR (5.59 ± 8.54 ml/kg) was similar to that in LDR (4.99 ± 4.91 ml/kg). The integrated \( \dot{V}_{\text{CO₂}} \) excess occupied only a small part of 1st CO₂ excess. As shown in Fig. 4, 1st CO₂ excess/w was related to \( \Delta L_{\text{at peak}} \) in SPR (\( r = 0.920, p < 0.01 \)) and LDR (\( r = 0.588, p > 0.05 \)). 1st CO₂ excess/w was higher in LDR than in SPR at the same lactate concentration, except for one subject, who had the poorest record of the LDR in 5000-m running. However, even when this subject was included, the ratio of 1st CO₂ excess/w to \( \Delta L_{\text{at peak}} \) (1st CO₂ excess ratio) was significantly higher in LDR than in SPR (\( p < 0.01, *p < 0.05 \) (significant difference between SPR and LDR). **p < 0.01 (significant difference between SPR and LDR).

2nd CO₂ excess. There was no significant difference in 1st CO₂ excess/w between the two groups (Table 2). The values of \( \dot{V}_{\text{CO₂}} \) excess were integrated during the exercise period. The value in SPR (5.59 ± 8.54 ml/kg) was similar to that in LDR (4.99 ± 4.91 ml/kg). The integrated \( \dot{V}_{\text{CO₂}} \) excess occupied only a small part of 1st CO₂ excess. As shown in Fig. 4, 1st CO₂ excess/w was related to \( \Delta L_{\text{at peak}} \) in SPR (\( r = 0.920, p < 0.01 \)) and LDR (\( r = 0.588, p > 0.05 \)). 1st CO₂ excess/w was higher in LDR than in SPR at the same lactate concentration, except for one subject, who had the poorest record of the LDR in 5000-m running. However, even when this subject was included, the ratio of 1st CO₂ excess/w to \( \Delta L_{\text{at peak}} \) (1st CO₂ excess ratio) was significantly higher in LDR than in SPR (\( p < 0.01, *p < 0.05 \) (significant difference between SPR and LDR). **p < 0.01 (significant difference between SPR and LDR).

2nd CO₂ excess/w, which is the other component of CO₂ excess/w, was significantly greater in SPR than in LDR (\( p < 0.05 \), Table 2). 2nd CO₂ excess/w was significantly correlated with the \( \Delta P_{\text{ETCO₂}} \) obtained at 10 min postexercise (\( r = -0.63, p < 0.05 \)). The larger the decrease in \( P_{\text{ETCO₂}} \) was, the higher the value of 2nd CO₂ excess/w became (Fig. 5). The ratios of 2nd CO₂ excess/w to 1 mmHg of \( \Delta P_{\text{ETCO₂}} \) (2nd CO₂ excess ratio) were similar in the two groups (Table 2) provided that the ratio for one subject whose \( \Delta P_{\text{ETCO₂}} \) was approximately zero was excluded from the calculation (refer to Fig. 5).

**DISCUSSION**

In this study, the response of excess CO₂ output to short-term intensive exercise was examined in sprinters and long-distance runners. The results showed that

<table>
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<th>Group</th>
<th>SPR (n=7)</th>
<th>LDR (n=6)</th>
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<tbody>
<tr>
<td>CO₂ excess/w (ml/kg)</td>
<td>68.1 ± 17.2</td>
<td>65.7 ± 12.9</td>
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<td>CO₂ excess ratio (ml/kg/mmHg)</td>
<td>8.42 ± 1.28</td>
<td>9.49 ± 1.05</td>
</tr>
<tr>
<td>1st CO₂ excess/w (ml/kg)</td>
<td>38.6 ± 11.0</td>
<td>47.4 ± 13.7</td>
</tr>
<tr>
<td>1st CO₂ excess ratio (ml/kg/mmHg)</td>
<td>4.03 ± 0.65**</td>
<td>5.79 ± 1.27</td>
</tr>
<tr>
<td>2nd CO₂ excess/w (ml/kg)</td>
<td>29.5 ± 8.40*</td>
<td>18.2 ± 8.90</td>
</tr>
<tr>
<td>2nd CO₂ excess ratio (ml/kg/mmHg)</td>
<td>3.65 ± 1.09</td>
<td>3.52 ± 1.68</td>
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*\( p < 0.05 \) (significant difference between SPR and LDR). **\( p < 0.01 \) (significant difference between SPR and LDR).
there was a significant correlation between CO₂ excess/w and ΔLa₁₀min. The ratios of CO₂ excess/w to ΔLa₁₀min (CO₂ excess ratio) were similar in the two groups. CO₂ excess was separated into two components: 1st CO₂ excess ratio, which was significantly higher in LDR than in SPR, and 2nd CO₂ excess/w, which was significantly higher in SPR than in LDR.

In this study, we obtained the values of excess CO₂ output (V̇CO₂ excess) throughout the periods of exercise and recovery. The values of V̇CO₂ excess were integrated to give the 1st CO₂ excess from the start of exercise until the time when PETCO₂ returned to the resting level. The reason for this calculation is as follows. Not only does PETCO₂ increase during this type of exercise, as shown in the present study, but so does venous CO₂ partial pressure [19, 20]. As this CO₂ rise induces a rightward shift in the bicarbonate system (CO₂ + H₂O = H⁺ + HCO₃⁻), the level of HCO₃⁻ increases. Similarly, the buffering of lactic acid causes a leftward shift in the bicarbonate system, resulting in a decrease in HCO₃⁻. This difference between the increase and decrease in HCO₃⁻ is thought to be expressed as excess CO₂ output per unit of time (V̇CO₂ excess). Therefore, it is necessary to exclude the effect of CO₂ partial pressure (PCO₂) in order to clarify the effect of lactic acid on CO₂ excess alone. For this reason, we calculated CO₂ excess so as to counteract the upward drift by the downward drift of PCO₂ by referring to the change in PETCO₂.

The 1st CO₂ excess ratio obtained in the present study agrees closely with previously reported CO₂ excess ratios for SPR and LDR in incremental exercise [8, 9]. It is known that PETCO₂ decreases below the resting level during an incremental exercise. However, this decrease occurs in the case of a slow incremental rate but not in the case of a rapid incremental rate [11–13]. Although PETCO₂ was not shown in previous reports [8, 9], it is thought that PETCO₂ would not have drifted greatly because a moderate incremental rate (about 30 W/min) was used in these studies. Furthermore, in an incremental exercise, mixed venous CO₂ pressure (PvCO₂) increases linearly in accordance with the rise in work rate and is unrelated to the rise in lactic acid [21]. As the CO₂ output phase related to PvCO₂ is extracted independent of the CO₂ excess phase, this rise in PvCO₂ does not affect the CO₂ excess phase in an incremental exercise. Under these conditions, CO₂ excess obtained during an incremental exercise would only reflect the effect of lactic acid.

Two possible factors have been suggested to be responsible for the differences in 1st CO₂ excess ratio among subjects [8]. The first is the difference in muscle capillary density. The main buffering system in extracellular fluid is the bicarbonate system, whereas the nonbicarbonate system (protein, phosphocreatine, and so on) is thought to be a main buffering system in the muscle since the bicarbonate system in the muscle cell accounts for only 15–20% [22] of the total buffering capacity of muscle. If lactic acid in the muscle is diffused into the extracellular fluid, the amount of lactic acid buffered mainly by the bicarbonate system would be great. Muscle capillary density could be regarded as one factor influencing the lactic acid efflux. In fact, it is well-known that the muscle capillary density of long-distance runners is high [23, 24]. The other possible factor is the difference in buffering capacity. If the buffering capacity in muscle is high owing to the nonbicarbonate buffering capacity, the amount of lactic acid buffered by the nonbicarbonate system will be great. In fact, it has been reported that the muscle buffering capacity of sprinters is high compared with that of long-distance runners [4, 5].

The other component of CO₂ excess, 2nd CO₂ excess/w, was found to be significantly correlated with ΔPETCO₂ (p<0.05, Fig. 5). Since there was a significant difference in ΔPETCO₂ between the two groups (p<0.01), the difference in 2nd CO₂ excess/w between SPR and LDR is thought to depend on the difference in the decrease in PETCO₂.

It has been observed that hyperventilation induces an excessive output of CO₂ followed by a reduction in PETCO₂ and ṖVCO₂. The excessive CO₂ output decreases in two exponential manners: a fast phase (about 25% of CO₂ washed out), derived from CO₂ stores in well-perfused organs such as the lungs, and blood; and a slow phase (about 75% of CO₂ washed out), derived from less well-perfused tissue such as muscle [25, 26]. Therefore, excess CO₂ output per mmHg change in ṖCO₂ is known to be less when the duration of hyperventilation is short. Furthermore, it has been reported that this value decreases during exercise even if hyperventilation continues for a long period [26, 27]. The average value obtained in the present study (3.60 ml/kg/mmHg) agrees closely with the value obtained during long-term hyperventilation at rest [25].

The decline in PETCO₂ due to hyperventilation magnifies the difference between arterial and tissue ṖCO₂ and could promote the removal of CO₂ from working muscle into the blood. In the bicarbonate system, this CO₂ removal from the tissue is essential because the buffering capacity decreases under the condition in which CO₂ removal is limited (closed system) [22]. Thus, it seems that physiological hyperventilation occurs in order to enhance the buffering efficiency of the bicarbonate system during the 2nd CO₂ excess phase.

Ventilatory response to CO₂ has been measured by
a rebreathing technique. It has been observed that the response is higher in sprinters than in long-distance runners [28] and that the response is dulled by endurance training [29]. These results suggest that chemosensitivity to hypercapnia is high in SPR compared to that in LDR. However, since the level of $H^+$ increases in accordance with the rise in $PCO_2$ in a CO$_2$ load experiment, the sensitivity to $H^+$ and $PCO_2$ stimulus cannot be discerned [30]. If chemosensitivity to $H^+$ is different among subjects, it is considered that when the level of $H^+$ increases accompanying the rise in blood lactic acid, as occurred in the present study, the effect of $H^+$ rise on ventilation would be different. Consequently, the degree of physiological hyperventilation ($\Delta\rho_{ETCO_2}$) might have been different between SPR and LDR.

In conclusion, 1st CO$_2$ excess response to La rise was significantly higher in LDR than in SPR. On the other hand, 2nd CO$_2$ excess was significantly higher in SPR than in LDR, and this is attributed to the difference in the respiratory chemosensitivity to lactic acid rise.

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