Effect of endurance training on excessive CO_2 expiration due to lactate production in exercise

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Summary. We attempted to determine the change in total excess volume of CO_2 output (CO_2 excess) due to bicarbonate buffering of lactic acid produced in exercise due to endurance training for approximately 2 months and to assess the relationship between the changes of CO_2 excess and distance-running performance. Six male endurance runners, aged 19-22 years, were subjects. Maximal oxygen uptake ($\dot{V}O_{2max}$), oxygen uptake ($\dot{V}O_2$) at anaerobic threshold (AT), CO_2 excess and blood lactate concentration were measured during incremental exercise on a cycle ergometer and 12-min exhausting running performance (12-min ERP) was also measured on the track before and after endurance training. The absolute magnitudes in the improvement due to training for CO_2 excess per body mass to the blood lactate accumulation in exercise (CO_2 excess $\cdot$ mass^{-1} : Δla^-), 12-min ERP, $\dot{V}O_2$ at AT (AT-$\dot{V}O_2$) and $\dot{V}O_{2max}$ on average were 0.8 ml $\cdot$ kg^{-1} $\cdot$ mmol^{-1} $\cdot$ l^{-1}, 97.8 m, 4.4 ml $\cdot$ kg^{-1} $\cdot$ min^{-1} and 7.3 ml $\cdot$ kg^{-1} $\cdot$ min^{-1}, respectively. The percentage change in CO_2 excess $\cdot$ mass^{-1} : Δla^- (15.7%) was almost same as those of $\dot{V}O_{2max}$ (13.7%) and AT-$\dot{V}O_2$ (13.2%). It was found to be a high correlation between the absolute amount of change in CO_2 excess $\cdot$ mass^{-1} : Δla^- and the absolute amount of change in AT-$\dot{V}O_2$ ($r=0.94$, $P<0.01$). Furthermore, the absolute amount of change in CO_2 excess $\cdot$ mass^{-1} : Δla^-, as well as that in AT-$\dot{V}O_2$ ($r=0.92$, $P<0.01$), was significantly related to the absolute amount of change in 12-min ERP ($r=0.81$, $P<0.05$). It was concluded that a large CO_2 excess $\cdot$ mass^{-1} : Δla^- of endurance runners could be an important factor for success in performance related to comparatively intense endurance exercise such as 3,000-4,000 m races.

Key words: CO_2 excess - Blood lactate - Hydrogen ion - Endurance training - Distance-running performance

Introduction

At higher exercise intensities accompanied by blood lactate accumulation (Δla^-), It is widely accepted that excess CO_2 is expired due to bicarbonate buffering of the hydrogen ion (H^+) dissociated from lactic acid produced in working skeletal muscles (Wasserman et al. 1973, 1981; Sutton and Jones 1979). The difference between the rate of actual CO_2 production ($\dot{V}CO_2$) and aerobically produced $\dot{V}CO_2$ is defined as the excess $\dot{V}CO_2$ at a given exercise intensity above the anaerobic threshold (AT) and reflects the excess CO_2 output due to bicarbonate buffering of lactic acid (Beaver and Wasserman 1991; Yano 1987). The integral of the excess $\dot{V}CO_2$ (expired $\dot{V}CO_2$ minus aerobically produced $\dot{V}CO_2$) from the AT to the end of a progressive incremental exercise is defined as CO_2 excess (Yano et al. 1984; Yano 1987) or VCO_2buf (total excess volume of CO_2 due to bicarbonate buffering of lactic acid; Beaver and Wasserman 1991). It has been pointed out that CO_2 excess unit of body mass to Δla^- during exercise (CO_2 excess $\cdot$ mass^{-1} : Δla^-) depends on the rates of buffering in bicarbonate and nonbicarbonate systems for lactic acidosis (Yano 1987), and that this is higher in endurance runners than in sprinters (Yano 1987) and untrained men (Hirakoba et al.1987, 1990).

The aim of this study was to determine the change in CO_2 excess $\cdot$ mass^{-1} : Δla^- due to endurance training and to assess the relationship between the changes in CO_2 excess $\cdot$ mass^{-1} : Δla^- and distance-running performence.

Methods

Subjects. Six male long distance runners aged 19-22 years, who had trained relatively little, were subjects. They gave their informed consent to participate after having the procedures and potential risks of the experiments explained. The individual and mean values of their physical characteristics, maximal oxygen uptake ($\dot{V}O_{2max}$), and oxygen uptake ($\dot{V}O_2$) at AT (AT-$\dot{V}O_2$) are given in Table 1. Endurance training consisted of interval and paced running. The mean training running intensity was at approximately 64.1 (SD2.5)% of the individual 12-min running-to-exhaustion speed obtained in the pre-test. The average daily running distance and training period were 11.9 km (range 9.8-14.2 km) and 62 days (range 25-102 days), respectively.

Table 1.

60.7

Experimental protocol. The experimental protocols used for exercise testing before and after endurance training were as follows: the subjects reported to the laboratory 1 h before testing and were asked to rest in a sitting position for about 0.5 h. Each subject performed a 1-min incremental exercise test on a cycle ergometer (Monark-Cresent AB, Varburg, Sweden). The cycle test was started by pedalling unloaded for 4 min and thereafter the intensity was increased by 30 W every minute until the subject reached exhaustion. The pedal rate, paced by a metronome, was kept at 60 rpm throughout the test. In addition, each subject carried out a 12-min exhausting track run twice a week after the cycle exercise test to measure the distance covered in this 12-min exhausting running performance (12-min ERP). The best performance was used for data analysis.

Gas exchange variables of ventilation ($\dot{V}_E$), VO_2 and $\dot{V}CO_2$ were measured continuously throughout the incremental exercise test. The O_2 and CO_2 concentrations in the expired gas were analysed by automatic gas analyser (Aerobics Processor 391, San-ei Inst., Tokyo, Japan). This analyser was calibrated by two standard gases before and after measurements.

Blood samples from a superficial dorsal hand vein were collected at rest, every minute during the phase of incrementally increasing exercise and at 1 min after exhaustion. After deproteinization, the venous blood samples were analysed by an enzymatic method to determine blood lactate concentrations (Hadjivassiliou and Pieder 1968).

The AT was determined by the gas exchange parameters ($\dot{V}_E$, $\dot{V}_E/\dot{V}O_2$ and $\dot{V}_E/\dot{V}CO_2$) and blood lactate concentrations (Wasserman et al. 1973; Davis et al. 1979) using the following criteria:

1. The point of departure from a linear relationship in V_E.
2. An increase of ventilatory equivalent for O_2 without an increase in the ventilatory equivalent for CO_2.
3. The point of abrupt increase from a resting level in the blood lactate concentration.

As illustrated in Fig. 1, CO_2 excess was calculated according to the method of Yano (1987) and Hirakoba et al. (1990). A regression line was determined for each individual for the relationship between $\dot{V}CO_2$ and $\dot{V}O_2$ at exercise intensities below the AT during incremental exercise ($\dot{V}CO_2 = a \cdot \dot{V}O_2 - b$). Excess $\dot{V}CO_2$ was assessed as the difference between the measured $\dot{V}CO_2$ ($\dot{V}CO_{2,measured}$) and predicted $\dot{V}CO_2$ ($\dot{V}CO_{2,predicted}$; from an extrapolation of the regression line of the $\dot{V}CO_2$-$\dot{V}O_2$ relationship) at exercise intensities above the AT. The integral with respect to time of excess $\dot{V}CO_2$ from the AT to the end of exercise has been abbreviated as CO_2 excess [CO_2 excess = $\int$ excess $VCO_2 = \int (\dot{V}CO_{2,measured} - VCO_{2,predicted})$ with respect to time] (shaded area in Fig. 1).

Fig. 1.

Statistics. Statistical comparisons of possible differences in the variables as a result of endurance training were made by the paired Student's t-test. The Pearson correlation technique was used to evaluate the correlation coefficients. For all statistical tests, the 95% level of confidence was accepted for significance.

Results

As shown in Table 1, there was little variation in indivisual running distance per day (from 9.8 to 14.2 km), but the training period varied considerably, ranging from 25 to 102 days. In particular, subject NU, whose endurance training was interrupted by a sport's injury, could train for 25 days only.

The magnitudes of the improvements between pre- and posttests for $\dot{V}O_{2max}$, AT-$\dot{V}O_2$ and 12-min ERP were 7.3 ml·kg^{-1}·min^{-1}, 4.4 ml·kg^{-1}·min^{-1} and 98 m, respectively (Table 2). The $\dot{V}O_{2max}$ increased significantly by 13.7% with training ($P<0.01$), but the differences between the pre- and postvalues for AT-$\dot{V}O_2$ and 12-min ERP were not significant because subject NU, with his shorter training period, showed no changes.

Table 2

Blood lactate accumulation due to incremental exercise (Δla^-, the difference between blood lactate concentration at 1 min after exhaustion and that at rest) decreased significantly by approximately 2.0 mmol · l^{-1} ($P<0.05$) from 10.4 mmol · l^{-1} to 8.4 mmol · l^{-1} as a result of training. Therefore, CO_2 excess (ml) and CO_2 excess per unit of mass (ml·kg^{-1}) tended to be lower in the posttraining compared with pretraining because CO_2 excess increased or decreased depending on blood lactate concentrations (Table 2). On the other hand, CO_2 excess · mass^{-1} : Δla^- showed a tendency to be higher at posttraining (6.55) than at pretraining (5.75). The percentage change in CO_2 excess · mass^{-1}: Δla^- (15.7%) was almost the same as those of $\dot{V}O_{2max}$ and AT-$\dot{V}O_2$ (Fig. 2). CO_2 excess · mass^{-1} : Δla^- as well as AT-$\dot{V}O_2$ and 12-min ERP in subject NU decreased slightly after training .

Fig. 2

Figure 3 shows the relationships of CO_2 excess · mass^{-1} : Δla^{-1} change with changes in $\dot{V}O_{2max}$ and AT-$\dot{V}O_2$. There was no significant correlation between the changes in CO_2 excess · mass^{-1} : Δla^- and $\dot{V}O_{2max}$ ($r=0.24$, NS). However, the change in CO_2 excess · mass^{-1} : Δla^- was significantly correlated to the absolute amount of change in AT-$\dot{V}O_2$

Fig. 3

(r=0.94, P<0.01). Also the change in 12-min ERP correlated significantly with the changes in CO_2 excess · mass^{-1} : Δla^- (r=0.81, P<0.05) and AT-$\dot{V}O_2$ (r=0.92, P<0.01), while there was no significant correlation between the change in Vo_{2max} and the change in 12-min ERP (r=0.62, NS) as shown in Fig. 4.

4.

Discussion

As exercise intensity becomes greater, the energy required to continue exercise shifts from aerobic to anaerobic sources. At exercise intensities above the AT anaerobic metabolism results in an accumulation of muscle and blood lactate. Lactic acid produced in working muscles is almost completely dissociated into H^+ and lactate within a range of physiological pH, which contributes to the metabolic acidosis. The greater part of H^+ dissociated from lactic acid is buffered by the bicarbonate and nonbicarbonate buffer systems to maintain the homeostasis of the body. Since Beaver et al. (1986) have indicated that the bicarbonate buffer system is the major buffering mechanism in the cells and in extracellular fluid ($H^+ + HCO_3^- \rightarrow H_2O + CO_2\uparrow$), it would appear that CO_2 is expired in excess of that from aerobic metabolism at exercise intensities which are accompanied by Δla^-. Consequently, actual $\dot{V}CO_2$ above the AT is derived from aerobic metabolism (produced $\dot{V}CO_2$) and bicarbonate buffering of lactic acid (excess $\dot{V}CO_2$; Beaver and Wasserman 1991; Hirakoba et al. 1987, 1990; Yano et al. 1984; Yano 1987). When CO_2 excess (integral of excess $\dot{V}CO_2$) is compared among individuals or, in an individual before and after training, it is necessary to estimate CO_2 excess $\cdot$ mass^{-1} : Δla^-, because CO_2 excess increases or decreases depending on the fluctuations of blood lactate concentrations (Beaver and Wasserman 1991; Hirakoba et al. 1990; Yano 1987).

The results of the present study show that CO_2 excess $\cdot$ mass^{-1}: Δla^- was higher in five of six subjects after endurance training, despite the fact that Δla^- in posttraining (8.4 mmol $\cdot$ l^{-1}) was significantly lower than pretraining (10.4 mmol $\cdot$ l^{-1}). The percentage increase in CO_2 excess $\cdot$ mass^{-1} : Δla^- (15.8%) was about the same as $\dot{V}O_{2max}$ (13.7%) and AT-$\dot{V}O_2$ (13.2%). In previous studies (Yano 1987; Hirakoba et al. 1990), it has been shown that CO_2 excess $\cdot$ mass^{-1} : Δla^- of endurance runners was significantly higher than in sprinters and untrained men.

These studies and the present one would suggest that endurance trained men may generate more CO_2 excess at the same blood lactate concentrations as compared with non-endurance trained and untrained men.

There are two possible explanations for this: firstly, the increase in CO_2 excess $\cdot$ mass^{-1} : Δla^- may be due to the decrease of buffering in the nonbicarbonate system or, secondly, the increase of buffering in the bicarbonate system (Yano 1987). If an endurance runner has less non-HCO_3^- buffering capacity in his muscle, the contribution of the bicarbonate system to total body buffering action will be enhanced. Parkhouse et al. (1985) reported that there was a significant relationship between muscle buffering capacity and carnosine concentrations, and that marathon runners with low carnosine concentrations had lower muscle buffering capacity than sprinters with high carnosine concentrations. In other words, the higher CO_2 excess $\cdot$ mass^{-1} : Δla^- would seem to imply that the contribution of proteins to total buffering in skeletal muscle (nonbicarbonate system) is lower in endurance runners than in sprinters. However, it has been reported that there are no differences in muscle buffering capacity (Parkhause et al. 1985) and blood buffering capacity (Sharp et al. 1983) between endurance trained and untrained men. It is, therefore, unlikely that body buffering capacity could be changed by endurance training for only 2 months. The increase in CO_2 excess $\cdot$ mass^{-1} : Δla^- after endurance training may be accounted for by a factor other than body buffering capacity which accompanies endurance training.

Another possible mechanism for the increased CO_2 excess $\cdot$ mass^{-1} : Δla^- is the increase of CO_2 efflux from muscles to blood. It has been indicated that exercising muscle is the major source of CO_2 excess output because H^+ from lactic acid must be buffered at the site of its formation (i.e. intramuscular HCO_3^-; Beaver and Wasserman 1991; Kowalchuk et al. 1988). In addition, Hultman and Sahlin (1980) have demonstrated that during isometric contractions accompanied by the occlusion of the

local circulation within the muscle, the CO_2 formed by the bicarbonate buffering of lactic acid could not escape from the muscle. It seems likely that CO_2 efflux from the muscle is accelerated due to the improvement of the peripheral circulation within exercising muscle. It is therefore inferred that the rate of CO_2 efflux could be enhanced in posttraining by an improvement in the peripheral circulation because it is well known that the muscle capillary density is increased after endurance training (Andersen 1975; Sjogaard 1984). Thus, this would result in a greater CO_2 efflux per unit increase in lactate concentration, which would lead to the increase in CO_2 excess $\cdot$ mass^{-1} : Δla^-.

As shown in Fig. 3, the change in CO_2 excess $\cdot$ mass^{-1} : Δla^- correlated significantly with the change in AT-$\dot{V}O_2$. This result suggested that CO_2 excess $\cdot$ mass^{-1} : Δla^- may have been an important determinant for endurance capacity, which is supported by the facts that the absolute amount of change in CO_2 excess $\cdot$ mass^{-1} : Δla^-, as well as that in AT-$\dot{V}O_2$, was significantly related to the absolute amount of change in 12-min ERP (Fig. 4). The high CO_2 excess $\cdot$ mass^{-1} : Δla^- would have been expected to be associated with less H^+ accumulation (greater H^+ uptake) in muscles at the same amount of lactate production, because the decrease of CO_2 efflux from muscles could reduce H^+ uptake in muscles as described by Hultman and Sahlin (1980).

Since it is known that proton accumulation within working muscles may inhibit muscle contractions both directly and indirectly via several mechanisms (Hultman and Sahlin 1980; Mainwood and Renaud 1984; Trivedi and Danforth 1966), an enhanced proton uptake could improve muscle performance (Bouissou et al. 1988; Jones et al. 1977; Wilkes et al. 1983).

Although the present study suggested that a high CO_2 excess $\cdot$ mass^{-1} : Δla^- and high AT-$\dot{V}O_2$ in endurance runners may be important factors for success in performance of comparatively intense endurance exercise (e.g. 3,000-4,000 m races lasting 10-12 min), further studies should be

carried out to assess the relationships between body buffering capacity, CO_2 excess and endurance performance.

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Fig. 1. Diapram of carbon dioxide production ($\dot{V}CO_2$) kinetics during incremental exercise. Shaded area represents CO_2 excess, which is the integral of the difference (excess $\dot{V}CO_2$) between the measured ($\dot{V}CO_{2,measured}$) and predicted ($\dot{V}CO_{2,predicted}$) values [from an extrapolation of the regression line of $\dot{V}CO_2$ - oxygen uptake ($\dot{V}O_2$) relationship; $\dot{V}CO_2 = a \cdot \dot{V}O_2 - b$] from a point at anaerobic threshold to the end of exercise [CO_2 excess = $\int$ excess $\dot{V}CO_2 = \int (\dot{V}CO_{2,measured} - VCO_{2,predicted})$]

Fig. 2. Percentage changes after endurance training for physiological variables and 12-min exhausting running performance (12-min ERP). $\dot{V}O_{2max}$, maximal oxygen uptake; AT-$\dot{V}O_2$, oxygen uptake at anaerobic threshold; Δla^-, blood lactate accumulation

Fig. 3. Relationships between the absolute amount of change in CO_2 excess $\cdot$ mass^{-1} : ΔLa^- and the absolute amount of changes in $\dot{V}O_{2max}$ and AT-$\dot{V}O_2$. For definitions see Fig. 2

Fig. 4. Relationships between the absolute amount of change in 12-min ERP and the absolute amount of change in $\dot{V}O_{2max}$, AT-$\dot{V}O_2$ and CO_2 excess $\cdot$ mass^{-1} : Δla^-. For definitions see Fig. 2

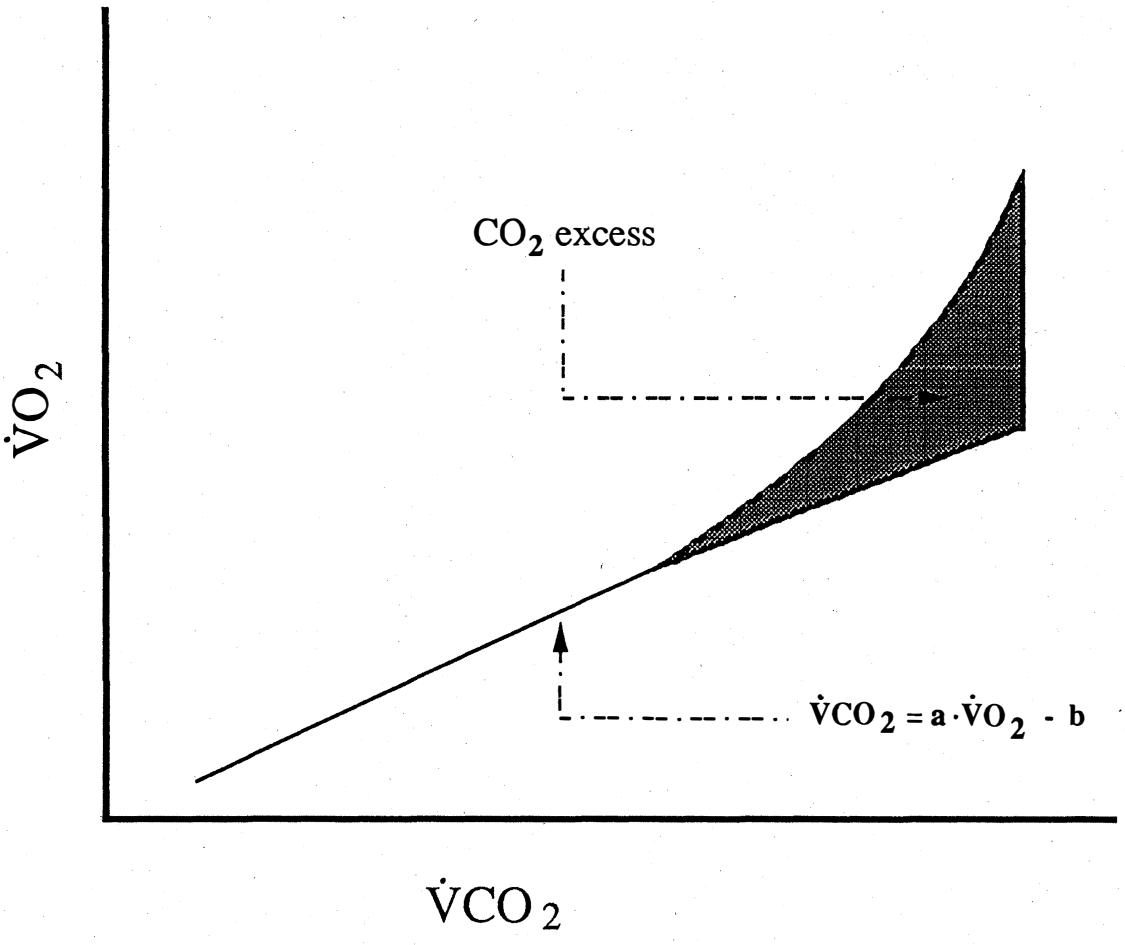

Figure 1.

A: Mass
B: $\dot{V}O_2$ max
C: AT-$\dot{V}O_2$
D: CO_2 excess
E: CO_2 excess • mass $^{-1}$
F: CO_2 excess • mass^{-1} : Δla^-
G: Δ la^-
H: 12-min ERP

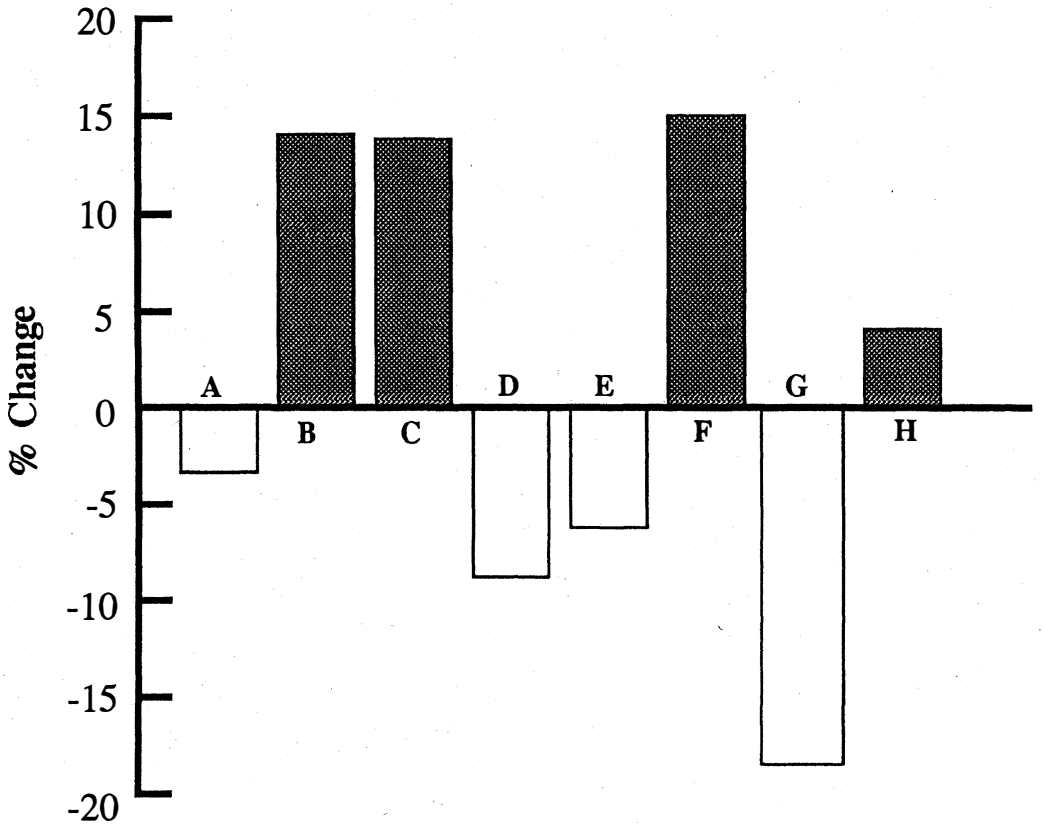

Figure 2.

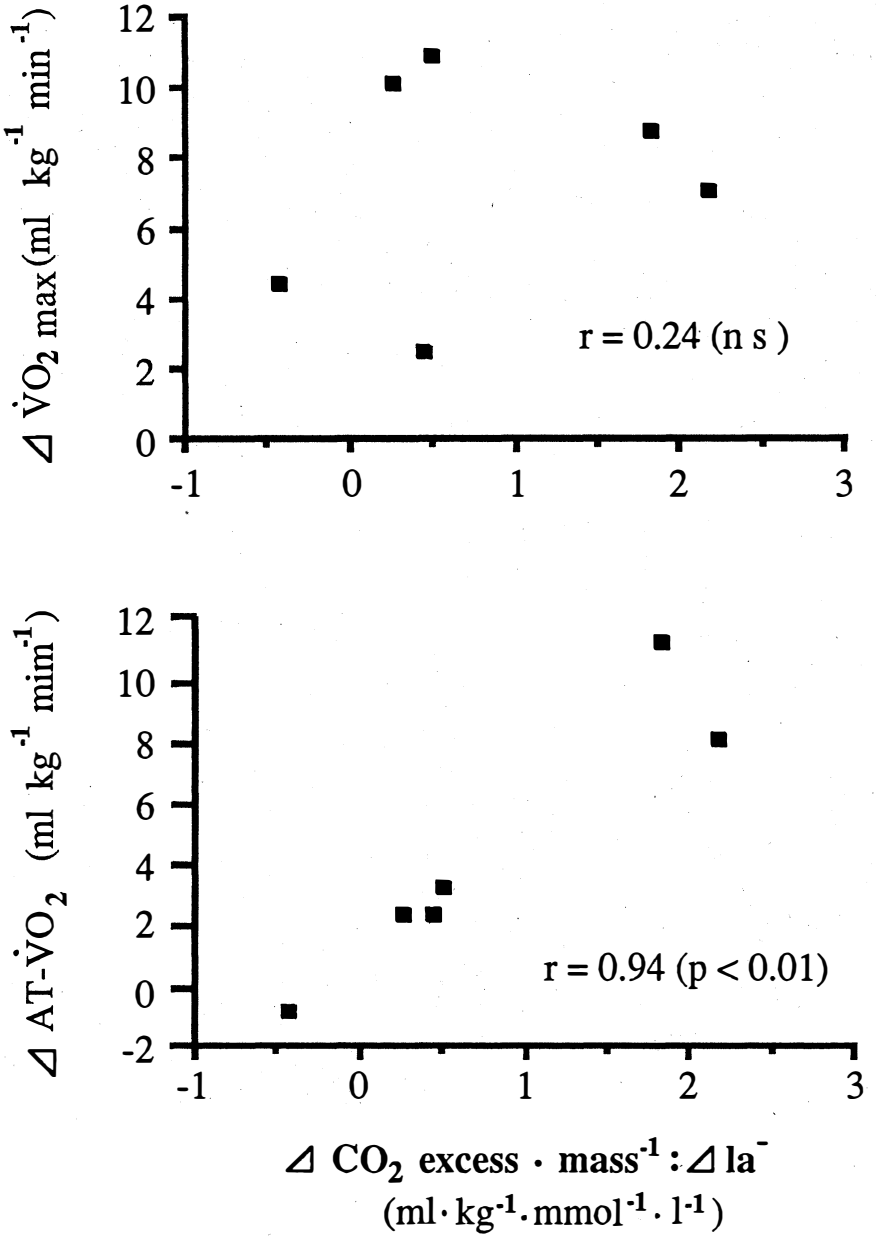
Δ V̇O2 max (ml kg-1 min-1)
12
10
8
6
4
2
0
-1
0
1
2
3
r = 0.24 (n s)
Δ AT-V̇O2 (ml kg-1 mim-1)
12
10
8
6
4
2
0
-2
-1
0
1
2
3
r = 0.94 (p < 0.01)
Δ CO2 excess · mass-1 : Δ la-
(ml · kg-1 · mmol-1 · l-1)

Figure 3.

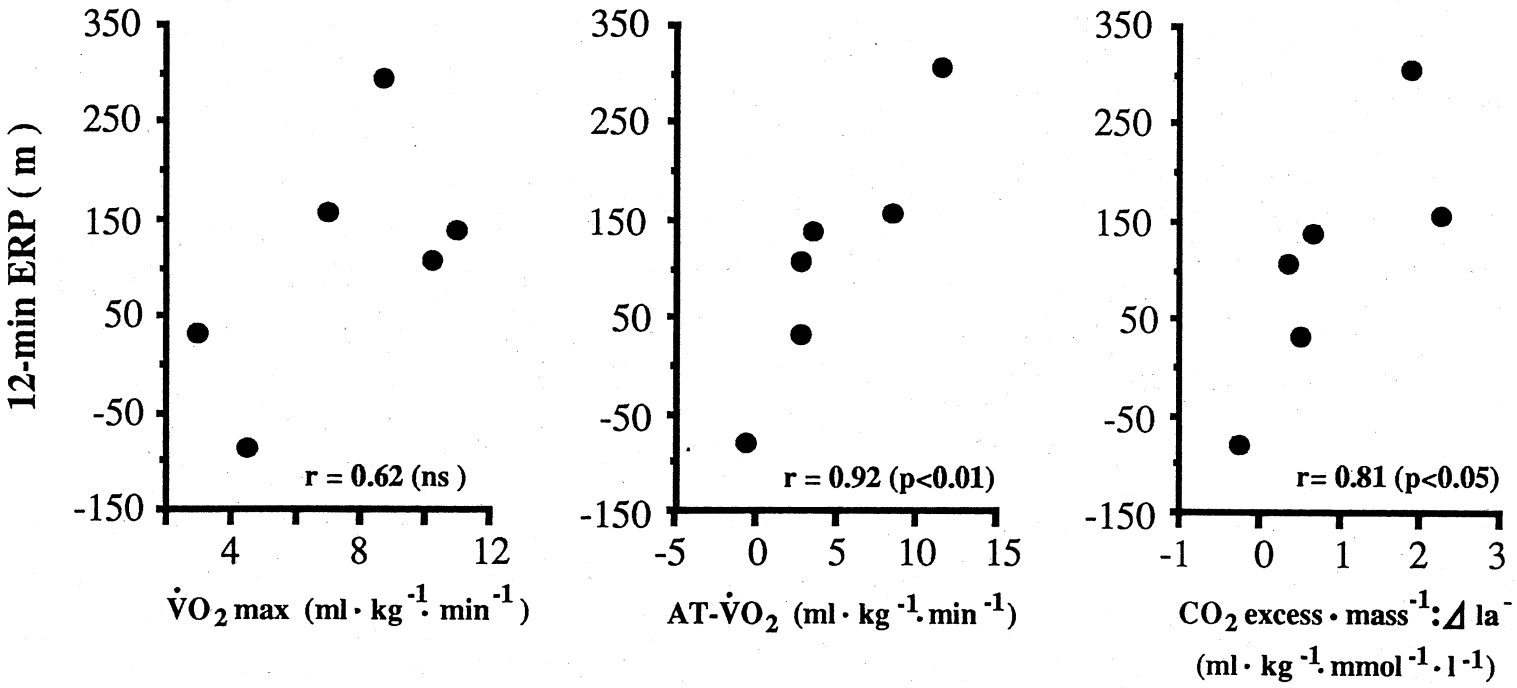
12-min ERP (m)
350
250
150
50
-50
-150
4
8
12
r = 0.62 (ns)
V̇O2 max (ml · kg-1 · min-1)
-5
0
5
10
15
r = 0.92 (p<0.01)
AT-V̇O2 (ml · kg-1 · min-1)
-1
0
1
2
3
r= 0.81 (p<0.05)
CO2 excess · mass-1 : Δ la-
(ml · kg-1 · mmol-1 · l-1)

Figure 4.

Table 1. Individual and mean values of physical characteristics, maximal oxygen uptake ($\dot{V}O_{2\,max}$), oxygen uptake at anaerobic threshold (AT-$\dot{V}O_2$), running distance and training period of subjects

Subject	Age (years)	Height (cm)	Mass (kg)	$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	AT-$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	Running distance ($km \cdot day^{-1}$)	Training period (days)
YK	20	173	63.4	54.1	34.2	9.8	31
KO	19	172	58.6	56.7	37.9	12.6	90
T I	19	167	63.0	48.7	31.3	14.2	53
TY	19	171	57.7	61.3	42.8	12.9	102
AH	19	169	52.9	51.1	31.0	11.5	63
NU	22	174	59.7	55.2	37.2	10.7	25
Mean	19.6	171.0	59.2	54.2	35.7	11.9	60.7
SD	1.1	2.4	3.5	4.0	4.1	1.5	28.2

For definitions see Fig. 2

Table 2. Changes of physiological variables and 12-min ERP after endurance training

Variable	Before		After		Δ	
	mean	SD	mean	SD	mean	SD
Mass (kg)	59.2	3.5	57.4	2.9 *	-1.8	1.2
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	54.5	4.0	61.8	3.0 **	7.3	3.1
AT-$\dot{V}O_2$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	35.7	4.1	40.1	3.8	4.4	4.1
Δ la^- ($mmol \cdot l^{-1}$)	10.4	1.9	8.4	0.9*	-2.0	1.3
CO_2 excess (ml)	3469	591	3140	578	-329	509
CO_2 excess $\cdot$ $mass^{-1}$ ($ml \cdot kg^{-1}$)	58.5	9.5	54.5	8.7	-4.0	9.1
CO_2 excess $\cdot$ $mass^{-1}$: Δ la^- ($ml \cdot kg^{-1} \cdot mmol^{-1} \cdot l^{-1}$)	5.75	1.01	6.55	1.01	0.8	0.9
12-min ERP (m)	3587	179	3685	133	98	118

Δ is the absolute amount of change as a result of endurance training. Δ la^- is the difference between blood lactate at rest and that at 1 min after exhaustion during incremental exercise.

* $p < 0.05$, ** $p < 0.01$, significantly different from before training.

For other definitions see Fig. 2