

Decrease of O₂ deficit is a potential factor in increased time to exhaustion after specific endurance training

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Demarle, Alexandre P., Jean J. Slawinski, Laurent P. Laffite, Valéry G. Bocquet, Jean P. Koralsztein, and Veronique L. Billat. Decrease of O₂ deficit is a potential factor in increased time to exhaustion after specific endurance training. *J Appl Physiol* 90: 947–953, 2001.—The main purpose of this study was to investigate the effects of an 8-wk severe interval training program on the parameters of oxygen uptake kinetics, such as the oxygen deficit and the slow component, and their potential consequences on the time until exhaustion in a severe run performed at the same absolute velocity before and after training. Six endurance-trained runners performed, on a 400-m synthetic track, an incremental test and an all-out test, at 93% of the velocity at maximal oxygen consumption, to assess the time until exhaustion. These tests were carried out before and after 8 wk of a severe interval training program, which was composed of two sessions of interval training at 93% of the velocity at maximal oxygen consumption and three recovery sessions of continuous training at 60–70% of the velocity at maximal oxygen consumption per week. Neither the oxygen deficit nor the slow component were correlated with the time until exhaustion ($r = -0.300$, $P = 0.24$, $n = 18$ vs. $r = -0.420$, $P = 0.09$, $n = 18$, respectively). After training, the oxygen deficit significantly decreased ($P = 0.02$), and the slow component did not change ($P = 0.44$). Only three subjects greatly improved their time until exhaustion (by 10, 24, and 101%). The changes of oxygen deficit were significantly correlated with the changes of time until exhaustion ($r = -0.911$, $P = 0.01$, $n = 6$). It was concluded that the decrease of oxygen deficit was a potential factor for the increase of time until exhaustion in a severe run performed after a specific endurance-training program.

running; oxygen uptake kinetics; fatigue; interval training

OXYGEN UPTAKE ($\dot{V}O_2$) response to a submaximal constant-load exercise is dependent on exercise intensity, which can be divided into several domains (19, 28, 36). When a supralactate threshold constant-load exercise is performed, four phases of $\dot{V}O_2$ kinetics can be identified (2, 19, 28, 36): *phase 1*, called the early component, is mainly attributed to the increase of pulmonary blood flow and is usually completed 15–20 s after the onset of exercise; *phase 2*, called the fast component,

corresponds to the decrease of venous content in oxygen and the further increase of pulmonary blood flow; *phase 3*, called the slow component, for which the origins are unclear, is superimposed 80–200 s after the onset of exercise on the fast component and elevates the oxygen consumption above, rather than toward, that predicted from the sublactate threshold $\dot{V}O_{2\text{-work}}$ rate relationship (3, 19, 28); *phase 4*, called the steady state of oxygen consumption ($\dot{V}O_{2\text{ss}}$), is delayed from 3 to 6 min on account of the slow-component phenomenon (34).

It is commonly reported that the parameters of $\dot{V}O_2$ kinetics may be modified after a short-term endurance training program (11, 18, 20, 21, 35, 37). When the same absolute work rate is taken into account before and after training, the time constant of the $\dot{V}O_2$ response (τ), defined as the time required to attain 63% of the $\dot{V}O_{2\text{ss}}$, may be diminished (20, 21, 37). It may, therefore, result in a smaller oxygen deficit, which is equal to $\tau \times \dot{V}O_{2\text{ss}}$ (32), reflecting a lesser anaerobic contribution at the onset of exercise (20). Such adaptation to training is thought to be important. For example, Poole and Richardson (28) have suggested that the decrease of oxygen deficit may be conducive to the increase of time until exhaustion, especially in a supralactate threshold constant-load exercise. Furthermore, for the same absolute work rate, the slow component is generally reduced after 6–8 wk of an endurance-training program (11, 18, 35). Such adaptation to training may also be important. Indeed, Poole et al. (27) have suggested that the only way to improve the work tolerance in patients who perform a supralactate threshold constant-load exercise is to lower the $\dot{V}O_2$ by decreasing the excess $\dot{V}O_2$ associated with the slow component. Nevertheless, the efficacy of such a strategy to improve the work tolerance in patients or sedentary or endurance-trained subjects remains to be firmly established.

Therefore, the special purpose of this study was to investigate the effects of an 8-wk severe interval training program on the parameters of $\dot{V}O_2$ kinetics, such as

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the oxygen deficit and the slow component, and their potential consequences on the time until exhaustion in a severe run performed at the same absolute velocity before and after training. It was hypothesized that 1) the oxygen deficit and the slow component could be reduced after a specific endurance training program, and 2) these adaptations could be, in part, responsible for the improvement of time until exhaustion at a given supralactate threshold velocity.

METHODS

Subjects. Six endurance-trained subjects volunteered to participate in this study. These subjects were specialized in middle- and long-distance running. Their mean ($\pm$ SE) age, height, and weight were, respectively, 27.0 ± 2.1 yr, 174.2 ± 1.2 cm, and 68.5 ± 2.2 kg. Before participation, all subjects were informed of the risks and stress associated with the training program and gave written voluntary informed consent in accordance with the guidelines of the University of Lille II.

Experimental design. Before and after training, the subjects performed 1) an incremental test to determine the maximal oxygen uptake ($\dot{V}O_{2 \max \text{ inc}}$), the velocity associated with the achievement of $\dot{V}O_{2 \max \text{ inc}}$ ($v\dot{V}O_{2 \max \text{ inc}}$), the velocity at the lactate threshold (vLT), the median velocity between vLT and $v\dot{V}O_{2 \max \text{ inc}}$ ($v\Delta 50$) and the running economy (RE); and 2) an all-out test (at pretraining $v\Delta 50$) to determine the time until exhaustion ($T_{\max}$). After training, the subjects also completed an additional all-out test (at the posttraining $v\Delta 50$). The tests were performed by a given subject at the same time of day in a climate-controlled environment. All training and test sessions were completed on a 400-m covered synthetic track. Throughout the tests, the subjects adopted the required velocity, thanks to an audiovisual system. This system included guide marks set at 25-m intervals along the track (inside the first lane), and audio signals determining the speed needed to cover 25-m intervals. The velocity of locomotion was strictly controlled throughout the tests with photoelectric cells (Brower Timing Systems, Salt Lake City, UT).

Procedures. Throughout the tests, the respiratory and pulmonary gas-exchange variables were measured using a breath-by-breath portable gas analyzer (Cosmed K4b², Rome, Italy), which was calibrated before each test according to the manufacturer's instructions (21, 23). Breath-by-breath data were later reduced to 30-s stationary averages (Data Management Software, Cosmed). Fingertip capillary blood samples were collected into a capillary tube and were analyzed for lactate concentration using a Doctor Lange (Berlin, Germany). This lactate analyzer was calibrated before the tests with several solutions of known lactate concentrations.

The subjects first performed an incremental test (3-min stages) to determine $\dot{V}O_{2 \max \text{ inc}}$, $v\dot{V}O_{2 \max \text{ inc}}$, vLT , $v\Delta 50$, and RE. The initial velocity was set at the average velocity maintained over 3,000 m, which has been described as being close to $v\dot{V}O_{2 \max \text{ inc}}$ (4), ~ 6 km/h, for exhaustion to occur for each subject within 20 min. The velocity increments between the stages were set at 1 km/h. All stages were followed by a 30-s period of rest. During this period, a fingertip capillary blood sample was collected. In addition, other fingertip capillary blood samples were collected before the test and immediately and 3 min after the test. Each subject was encouraged to give a maximum effort. $\dot{V}O_{2 \max \text{ inc}}$ was defined as the highest 30-s $\dot{V}O_2$ value reached in this incremental test. $v\dot{V}O_{2 \max \text{ inc}}$ was defined as the minimal velocity at which $\dot{V}O_{2 \max \text{ inc}}$ occurred

(7). When $v\dot{V}O_{2 \max \text{ inc}}$ was maintained for one-half rather than for all of the last stage, it was then considered as the median velocity maintained during the last two stages (25). vLT was defined as the velocity for which an increase in lactate concentration corresponding to 1 mmol/l occurs between 3.5 and 5 mmol/l (1). vLT was determined by two independent reviewers. The $v\Delta 50$ was defined as the median velocity between $v\dot{V}O_{2 \max \text{ inc}}$ and vLT . The $v\Delta 50$ has been described as being a velocity for which the slow component of $\dot{V}O_2$ may lead the $\dot{V}O_2$ to its maximum ($\dot{V}O_{2 \max \text{ inc}}$) (10, 15). The running economy was defined as the rate of $\dot{V}O_2$ for a given submaximal work rate (12). In this study, the rate of $\dot{V}O_2$ was averaged between the 2nd and the 3rd min of the stage run at 13 km/h ($<vLT$) and was taken as reference for the running economy.

The subjects subsequently performed an all-out test (at the pre- or posttraining $v\Delta 50$) to determine the time until exhaustion. After a 15-min period of warm-up at 60% $v\dot{V}O_{2 \max \text{ inc}}$ followed by a 5-min period of rest, the subjects were instructed to run at the required velocity within a 5-s period of transition until they were unable to sustain the fixed velocity. Each subject was encouraged to give maximum effort. A fingertip capillary blood sample was collected before the test and immediately and 3 min after the test.

Training program. Before participation, the subjects were already well trained in endurance. They generally performed a continuous training, 3–5 times/wk, consisting of 45–60 min at an exercise intensity (60–70% $v\dot{V}O_{2 \max \text{ inc}}$) below the lactate threshold. The subjects completed an 8-wk severe interval training program including two sessions of interval training and three sessions of continuous training per week. The training program was elaborated, according to recent studies (6, 15, 31), by taking into account an individualized exercise intensity ($v\Delta 50$) and an individualized exercise duration (25–50% of the time until exhaustion at $v\Delta 50$) for each runner. The sessions of interval training consisted of ($n_{\max} - 2$) or ($n_{\max} - 1$) repetitions, including a severe run at $v\Delta 50$, during 50% $T_{\max}$ at $v\Delta 50$, and a recovery run at 50% $v\dot{V}O_{2 \max \text{ inc}}$ during 25% $T_{\max}$ at $v\Delta 50$. The value of $n_{\max}$ was defined as the individual number of repetitions achieved by a given subject when the exhaustion occurs. The $n_{\max}$ was recorded during the first and the eighth sessions of interval training; thus, if the training intensity remained unchanged, the training volume was adjusted to the progress achieved by the subjects and consequently could be increased through the 8 wk of training. For example, a subject who was able to perform four repetitions during the first session of interval training would be able then to perform five or six repetitions during the eighth session of interval training. The sessions of continuous training were run at 60–70% $v\dot{V}O_{2 \max \text{ inc}}$ for 45–60 min. All sessions were controlled by a professional trainer to ensure that these instructions were respected.

$\dot{V}O_2$ kinetics. The breath-by-breath $\dot{V}O_2$ data were reduced to 5-s stationary averages. These data were then smoothed, using a three-step average filter, to reduce the noise so as to enhance the underlying characteristics (Data Management Software, Cosmed). These data were finally fitted to three distinct models (3, 32, 33) by use of an iterative nonlinear regression on Sigma Plot software (SPSS, Chicago, IL): a single-exponential model comprising a delayed linear component (Eq. 1) and two double-exponential models, the first comprising two exponential terms that start at a common time delay from the onset of exercise (Eq. 2) and the second comprising two exponential terms that start at two distinct time delays from the onset of exercise (Eq. 3). The Fisher test, which was performed with the Sigma Plot software, was used

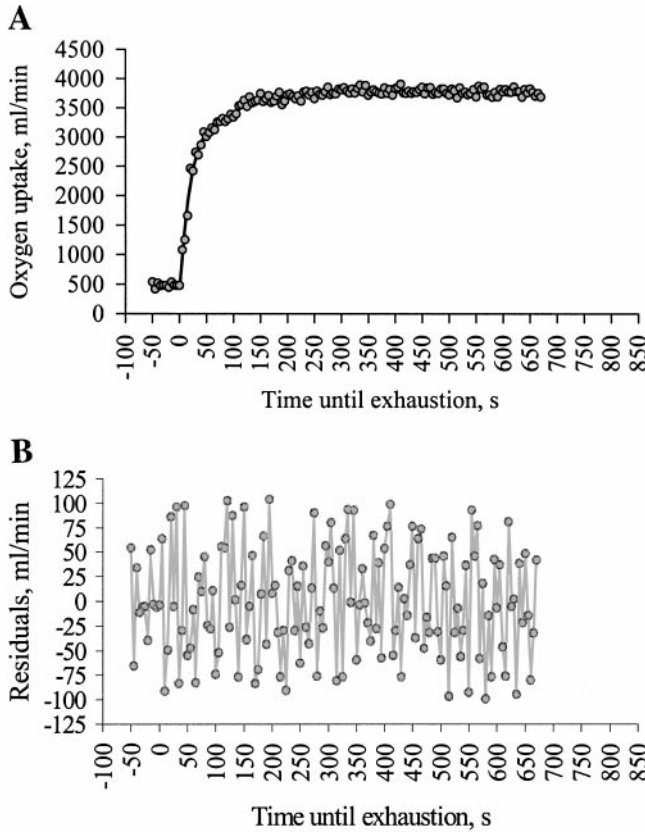


Fig. 1. A: fitting of oxygen uptake response in 1 subject before training. B: residuals corresponding to the fitting of oxygen uptake response illustrated in A.

to choose the model whose fit was associated with the highest F value

$$\dot{V}O_2(t) = A_0 + A_1 \times \{1 - e[-(t - TD_1)/\tau_1]\} \times u_1 + [p \times (t - TD_2)] \times u_2 \quad (1)$$

where $u_1 = 0$ when $t < TD_1$, $u_1 = 1$ when $t \geq TD_1$, $u_2 = 0$ when $t < TD_2$, $u_2 = 1$ when $t \geq TD_2$, A_0 is the baseline value (ml/min), A_1 is the asymptotic amplitude for the exponential term (ml/min), τ_1 is the time constant(s), TD_1 is the time delay from the onset of exercise(s), p is the slope of the linear term, and TD_2 is the time delay from the onset of exercise(s).

$$\dot{V}O_2(t) = A_0 + A_1 \times \{1 - e[-(t - TD_1)/\tau_1]\} \times u_1 + A_2 \times \{1 - e[-(t - TD_1)/\tau_2]\} \times u_1 \quad (2)$$

where A_2 is the asymptotic amplitude for the exponential terms (ml/min), τ_2 is the time constant.

$$\dot{V}O_2(t) = A_0 + A_1 \times \{1 - e[-(t - TD_1)/\tau_1]\} \times u_1 + A_2 \times \{1 - e[-(t - TD_2)/\tau_2]\} \times u_2 \quad (3)$$

Oxygen deficit. When an exercise is performed at work rates associated with the heavy and severe exercise intensity domains, two components of $\dot{V}O_2$ generally appear after two distinct time delays (TD_1 for the fast component, TD_2 for the slow component). The slow component is superimposed, 80–200 s after the onset of exercise, on the fast component and elevates the oxygen consumption above, rather than toward, that predicted from the sublactate threshold $\dot{V}O_2$ -work rate relationship (3, 19, 28). As suggested by Whipp and Ozyener (32), the slow component may represent an “excess oxygen

uptake,” whereas the fast component may represent an “expected oxygen uptake.” Thus it may be considered that only the area between the fast component response curve and the fast component asymptote corresponds to the oxygen deficit

$$DO_2 = (A_1 \times TD_1) + (A_1 \times \tau_1) \quad (4)$$

where DO_2 is the oxygen deficit (ml), A_1 is the (fast component) asymptotic amplitude (ml/s), τ_1 is the (fast component) time constant(s), and TD_1 is the (fast component) time delay from the onset of exercise(s).

Oxygen consumed. The aerobic component of the total energy requirement for the all-out tests was computed by integrating the area under the curve $\dot{V}O_2$ -time until exhaustion (32). For example, considering a double-exponential model (Eq. 3)

$$\dot{V}O_2 = \int_{TD_1}^{T_{\max}} A_1 \times \{1 - e[-(t - TD_1)/\tau_1]\} dt + \int_{TD_2}^{T_{\max}} A_2 \times \{1 - e[-(t - TD_2)/\tau_2]\} dt \quad (5)$$

i.e., total oxygen consumed = expected oxygen consumed + excess oxygen consumed, where $\dot{V}O_2$ is the oxygen consumed (ml), and $T_{\max}$ is the time(s) until exhaustion.

Time to attain and time sustained at $\dot{V}O_{2\max}$. When an exercise is performed at work rates associated with the severe exercise intensity domain, the slow component may lead to the attainment of $\dot{V}O_{2\max \text{ inc}}$ (19, 28). According to T. J.

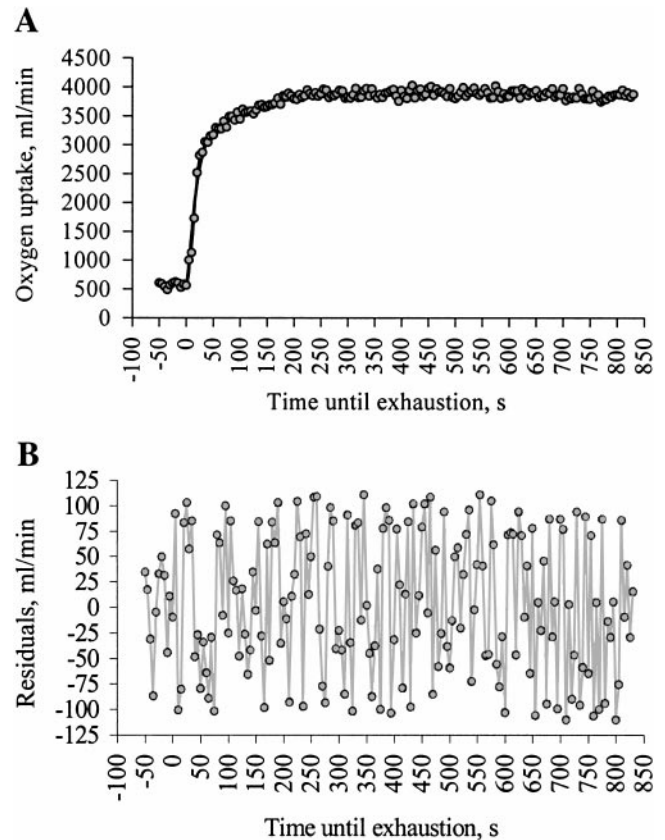


Fig. 2. A: fitting of oxygen uptake response in 1 subject after training at the same absolute velocity. B: residuals corresponding to the fitting of oxygen uptake response illustrated in A.

Table 1. Training effects on the indexes of aerobic fitness

Indexes	Before Training	After Training	P Values
Body mass, kg	68.5 ± 2.2	68.5 ± 2.2	NS
$\dot{V}O_{2\max \text{ inc}}$, ml·min ⁻¹ ·kg ⁻¹	61.2 ± 2.7	61.6 ± 2.2	NS
$\dot{V}O_{2\max \text{ inc}}$, ml/min	4,177 ± 151	4,207 ± 121	NS
$v\dot{V}O_{2\max \text{ inc}}$, km/h	18.2 ± 0.3	18.7 ± 0.4	≤0.01
vLT , km/h	15.7 ± 0.4	16.1 ± 0.4	NS
$v\Delta 50$, km/h	17.0 ± 0.4	17.4 ± 0.4	≤0.01
RE at 13 km/h, ml·min ⁻¹ ·kg ⁻¹	44.2 ± 1.5	42.6 ± 1.2	0.02

Values are means ± SE; $n = 6$. NS, not significant; $\dot{V}O_{2\max \text{ inc}}$, incremental test of maximal oxygen uptake ($\dot{V}O_{2\max}$); $v\dot{V}O_{2\max \text{ inc}}$, velocity at $\dot{V}O_{2\max \text{ inc}}$; vLT , velocity at lactate threshold; $v\Delta 50$, median velocity between vLT and $v\dot{V}O_{2\max \text{ inc}}$; RE, running economy.

Barstow (personal communication), it was considered that a given subject was able to attain $\dot{V}O_{2\max}$ when the sum of A_0 , A_1 , and A_2 was ≥98% of $\dot{V}O_{2\max \text{ inc}}$, admitting an error of 2% in the determination of $\dot{V}O_{2\max \text{ inc}}$. Then, when a double-exponential model (Eq. 3) is considered

$$TD \dot{V}O_{2\max \text{ inc}} = TD_2 - \tau_2 \times \ln \{1 - [(0.98 \times \dot{V}O_{2\max \text{ inc}} - A_0 - A_1)/A_2]\} \quad (6)$$

where $\dot{V}O_{2\max \text{ inc}}$ is ml/min, $TD \dot{V}O_{2\max \text{ inc}}$ is the time to attain $\dot{V}O_{2\max \text{ inc}}$ in (s), τ_2 is the (slow component) time constant(s), and TD_2 is the (slow component) time delay from the onset of exercise(s) and

$$T_{\max} \dot{V}O_{2\max \text{ inc}} = (T_{\max} - TD \dot{V}O_{2\max \text{ inc}}) \quad (7)$$

where $T_{\max} \dot{V}O_{2\max \text{ inc}}$ is the time sustained at $\dot{V}O_{2\max \text{ inc}}$ (s), and $T_{\max}$ is the time(s) until exhaustion.

Test-to-test reproducibility of $\dot{V}O_2$ kinetics parameters. To determine the confidence intervals over which the parameters of $\dot{V}O_2$ kinetics were accurate, a voluntary subject completed 1) three 6-min tests at vLT and 2) three 6-min tests at $v\Delta 50$. The $\dot{V}O_2$ data were fitted to two distinct models: 1) a single-exponential model (derived from Eq. 1) and 2) a double-exponential model (Eq. 3).

Statistics. One-way analysis of variance with repeated measures and paired t -test were used for data analysis. Simple and multiple correlations were used for correlation analysis. The level of significance was set at 5% ($P \leq 0.05$). All results are presented as means ± SE.

RESULTS

Test-to-test reproducibility of $\dot{V}O_2$ kinetics parameters. The mean values (± SD) of A_0 , TD_1 , τ_1 , A_1 and Do_2 , obtained in the tests at vLT (16 km/h), were equal to 530 ± 16 ml/min, 7.3 ± 0.5 s, 27.1 ± 1.5 s, $3,125 \pm 24$ ml/min, and $1,791 \pm 67$ ml, and the coefficients of variation were equal to 3.0, 6.8, 5.5, 0.8, and 3.8%. The mean values (± SD) of A_0 , TD_1 , τ_1 , A_1 , TD_2 , τ_2 , A_2 , and Do_2 obtained in the tests at $v\Delta 50$ (17 km/h) were equal to 567 ± 20 ml/min, 8.9 ± 0.4 s, 19.8 ± 0.9 s, $3,351 \pm 45$ ml/min, 84.3 ± 4.1 s, 83.7 ± 7.1 s, 357 ± 34 ml/min, and $1,606 \pm 26$ ml, and the coefficients of variation were equal to 3.6, 4.5, 4.6, 1.3, 4.8, 8.4, 9.7, and 1.6%.

Fitting of $\dot{V}O_2$ responses. The $\dot{V}O_2$ responses were fitted to a double-exponential model (Eq. 3), which provided the best fits among the three models used (Figs. 1 and 2).

Relationships between the parameters of $\dot{V}O_2$ kinetics and the time until exhaustion. Neither the oxygen deficit nor A_2 were correlated with the time until exhaustion ($r = -0.300$, $P = 0.24$, $n = 18$; $r = -0.420$, $P = 0.09$, $n = 18$, respectively).

Training program parameters. The sessions of interval training consisted of several repetitions, including a severe run at 17.0 ± 0.4 km/h for 294 ± 20 s and a recovery run at 9.1 ± 0.1 km/h for 147 ± 10 s. If the training intensity did not change throughout the training period, the training volume was significantly increased from 3.1 ± 0.3 to 4.3 ± 0.3 repetitions ($P = 0.01$) on account of the progress achieved by the subjects. The sessions of continuous training were run at 11.8 ± 0.2 km/h for 45–60 min.

Training effects on the indexes of aerobic fitness. Eight weeks of severe interval training program significantly improved 1) $v\dot{V}O_{2\max \text{ inc}}$ without change of $\dot{V}O_{2\max \text{ inc}}$, on account of the significant decrease of RE and 2) $v\Delta 50$ (Table 1).

Training effects on the time until exhaustion. When the same absolute velocity was taken into account before and after training, only three subjects greatly improved their time until exhaustion (by 65, 159, and 449 s, or 10, 24, and 101%, respectively) (Fig. 3).

Training effects on the parameters of $\dot{V}O_2$ kinetics. When the same absolute velocity was taken into account before and after training, the oxygen deficit was significantly decreased on account of the significant decrease of τ_1 . A_2 did not change. Before training, three subjects were able to attain their $\dot{V}O_{2\max \text{ inc}}$. However, after training, for the same absolute velocity, only one subject among these three subjects was able to attain $\dot{V}O_{2\max \text{ inc}}$ (Table 2).

Relationships between the changes of $\dot{V}O_2$ kinetics parameters and the changes of time until exhaustion. The changes of oxygen deficit were significantly correlated with the changes of time until exhaustion ($r = -0.911$, $P = 0.01$, $n = 6$; Fig. 3). Furthermore, the two subjects who, contrary to before training, were not able to attain their $\dot{V}O_{2\max \text{ inc}}$ after training improved their time until exhaustion.

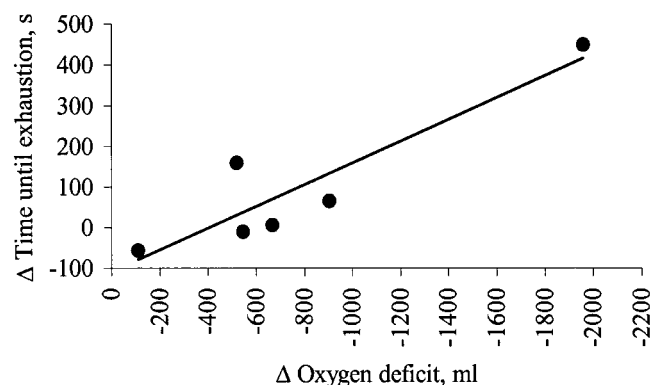


Fig. 3. Correlation between the training-induced changes (Δ) of oxygen deficit and those of time until exhaustion ($r = -0.911$, $P = 0.01$, $n = 6$).

Table 2. Training effects on the time until exhaustion and the parameters of $\dot{V}O_2$ kinetics

Parameters	Before Training	After Training, Same Absolute Velocity	After Training, Same Relative Velocity	P Values
Velocity, km/h	17.0 ± 0.4	17.0 ± 0.4	17.4 ± 0.4*	0.01
Time to exhaustion, s	588 ± 41	690 ± 60	505 ± 25*	0.03
A ₀ , ml/min	699 ± 87	710 ± 88	703 ± 79	NS
TD ₁ , s	17.2 ± 1.2	17.1 ± 0.3	18.3 ± 1.5	NS
τ ₁ , s	25.9 ± 1.2	14.2 ± 1.2*	13.8 ± 1.7*	≤0.01
A ₁ , ml/min	3,112 ± 205	2,832 ± 38	2,851 ± 112	NS
TD ₂ , s	149.8 ± 25.2	131.4 ± 13.2	103.0 ± 15.1	NS
τ ₂ , s	31.8 ± 7.3	33.2 ± 13.0	61.4 ± 18.2	NS
A ₂ , ml/min	334 ± 63	275 ± 78	461 ± 107	NS
[A ₁ /(A ₁ + A ₂)], %	90.1 ± 2.1	91.4 ± 2.3	86.4 ± 2.9	NS
[A ₂ /(A ₁ + A ₂)], %	9.9 ± 2.1	8.6 ± 2.3	13.6 ± 2.9	NS
(A ₀ + A ₁ + A ₂), ml/min	4,146 ± 258	3,818 ± 71	4,015 ± 199	NS
DO ₂ , ml	2,269 ± 264	1,486 ± 70*	1,524 ± 62*	≤0.01
Expected $\dot{V}O_2^a$, ml	27,761 ± 1999	31,208 ± 3147	22,454 ± 1361	0.04
Excess $\dot{V}O_2^b$, ml	2,257 ± 550	2,261 ± 664	2,607 ± 591	NS
Total $\dot{V}O_2^c$, ml	30,019 ± 2057	33,470 ± 3145	25,061 ± 1349	≤0.05
Time to $\dot{V}O_{2max inc}$, s	151 ± 28†	184 ± 00‡	132 ± 36§	
Time at $\dot{V}O_{2max inc}$, s	400 ± 38†	391 ± 00‡	411 ± 65§	
Blood lactate, mmol/l	11.1 ± 0.7	12.0 ± 0.9	12.1 ± 1.3	NS

Values are means ± SE; $n = 6$; † $n = 3$; ‡ $n = 1$; § $n = 2$. A₀, baseline value; A₁ and A₂, asymptotic amplitudes for the exponential terms; τ₁ and τ₂, time constants; TD₁ and TD₂, time delays from the onset of exercise; DO₂, oxygen deficit. ^a Oxygen consumed computed from the area under the fast component; ^b Oxygen consumed computed from the area under the slow component; ^c Oxygen consumed computed from the areas under the two components. * $P \leq 0.05$ when compared with before training.

DISCUSSION

The main findings of this study are as follows. 1) Neither the oxygen deficit nor the slow component (A₂) are related to the time until exhaustion in a severe run. 2) Significant adaptations and performance improvements, which are not represented by the $\dot{V}O_{2max}$, can occur in well-trained subjects after a specific endurance training program. When the same absolute velocity is taken into account before and after training, the oxygen deficit decreases after 8 wk of a severe interval training program. The slow component (A₂) remains, however, unchanged. Three subjects, in a population comprising six subjects, improved their time until exhaustion. 3) The decrease of oxygen deficit is related to the increase of time until exhaustion in a severe run performed after a specific endurance training program.

Previous studies have shown a positive relationship between the accumulated oxygen deficit and the time to fatigue at the velocity at $\dot{V}O_{2max}$, thus demonstrating that the anaerobic contribution is not negligible in such exercise (17, 30). Nevertheless, no study, to our knowledge, has investigated the relationship between the oxygen deficit and the time to fatigue at submaximal work rates. Although the determination of oxygen deficit at supralactate threshold work rates remains a subject for discussion on account of the slow-component phenomenon (32), our study shows that the oxygen deficit is not related to the time until exhaustion in a severe run, thus disproving the hypothesis that a low oxygen deficit may be associated with a great work tolerance.

Using the same absolute work rate before and after training, early studies have shown that $\dot{V}O_2$ increases more rapidly toward its steady state in the trained state compared with the untrained state, with a half-

time that can be reduced by 18–25% (20, 21). Recent studies have also shown that the time constant, defined as the time required to attain 63% of the $\dot{V}O_{2ss}$, can be reduced by 27–57% (26, 37). Correspondingly, in our study, the time constant (τ₁) is reduced by 46%. Hagberg et al. (20) have shown that the heart rate, like the $\dot{V}O_2$, increases more rapidly toward its steady state in the trained state compared with the untrained state, with a half-time that can be reduced by 50–58%. Accordingly, Yoshida et al. (37) have recently shown that the heart rate time constant can be reduced by 49%. Because the heart rate response is speeded and, hypothetically, the stroke volume is increased (16), it is speculated that oxygen delivery to the active muscles can be improved at the onset of exercise (20, 26, 37). The early attenuation of phosphocreatine depletion and blood lactate accumulation that can be seen after training may provide an alternative argument, demonstrating thus that oxygen utilization by the active muscles can also be improved at the onset of exercise (13, 14, 26, 37). Nevertheless, the decrease of A₁ may also provide a valid argument for the decrease of τ₁ (3).

Using the same absolute work rate before and after training, Hagberg et al. (20) have shown that the decrease of half-time of $\dot{V}O_2$ response by 25%, without change of $\dot{V}O_{2ss}$, leads to the decrease of oxygen deficit by 21%. Correspondingly, in our study, the oxygen deficit is reduced by 34% on account of the decreases of both τ₁ and A₁. Karlsson et al. (24) have shown that the decrease of muscle high-energy phosphate (phosphocreatine and ATP) concentrations is less marked after an endurance training program. Furthermore, blood lactate accumulation, which is an indicator of anaerobic glycolysis functioning, is reduced at the same absolute work rate. It may, therefore, lower the intracellu-

lar perturbation at the onset of exercise. Such adaptation to training is thought to be important. For example, Poole and Richardson (28) have suggested that the decrease of oxygen deficit may lead to the increase of time until exhaustion, especially in a supralactate threshold constant-load exercise. Accordingly, our study shows that the decrease of oxygen deficit is a potential factor for the increase of time until exhaustion in a severe run performed after a specific endurance training program, with the other factors remaining, however, to be elucidated.

The link between the slow component and the work tolerance remains unclear (19, 27). Although the attainment of $\dot{V}O_{2\max}$ and the progressive increase of blood lactate may lower the work tolerance (29), Billat et al. (9) have recently shown that the slow component, defined as the increase of $\dot{V}O_2$ between the 3rd min and the end of exercise (34), is not related to the time to fatigue in a severe running or cycling exercise. Correspondingly, in our study, the slow component (A_2) is not related to the time until exhaustion in a severe run, disproving thus the hypothesis that a slow component of small amplitude may be associated with a great work tolerance.

For the same absolute work rate before and after training, the slow component, as defined by Whipp and Wasserman in 1972 (34), may be reduced by 50–65% after 6–8 wk of an endurance training program (11, 18, 35). Nevertheless, the mechanisms responsible for such adaptation to training are not firmly established. Studies have shown that the slow component arises predominantly from the exercising limbs (27). It is likely that the slow component is mainly due to the glycolytic-twitch fibers' recruitment at supralactate threshold work rates, with the fast-twitch fibers being less energetically efficient than the slow-twitch fibers (19, 27). It is thus possible, if not probable, that the adaptations to training in the motor-unit recruitment pattern and in the fast- and slow-twitch fibers' mitochondrial content may account for the slow-component attenuation (19, 27, 35). Our study shows, however, that, in well-trained subjects, the slow component (A_2) does not change after a specific endurance training program, which, hypothetically, allows the fast- and slow-twitch fiber's recruitment. It raises the difficulty of investigating such adaptation to training in well-trained subjects, who, moreover, perform a supralactate threshold running exercise in which the slow component is generally small, if not nonexistent (5, 9).

Poole et al. (27) have suggested that the only way to improve the work tolerance in cardiac- and ventilatory-limited patients is to lower the $\dot{V}O_2$ by removing or decreasing the excess $\dot{V}O_2$ associated with the slow component. Such adaptation to training may be obtained by improving the lactate threshold or the critical power (18, 29). Nevertheless, the efficacy of such a strategy to improve work tolerance in patients or sedentary or endurance-trained subjects remains to be firmly established. In our study, two subjects who, contrary to before training, were not able to attain their $\dot{V}O_{2\max}$ after training, improved their time until

exhaustion. It is likely that, by increasing their critical velocity and, consequently, decreasing their slow component (A_2), they improved their work tolerance. Indeed, the critical power represents the upper limit for which the $\dot{V}O_2$, blood lactate, and blood pH can be stabilized. On the other hand, an exercise performed above the critical power is characterized by a steadily increasing $\dot{V}O_2$ and blood lactate, a decreasing blood pH, and consequently, an imminent fatigue (29).

To conclude, this study shows that significant adaptations and performance improvements, which cannot be assessed by a single incremental test, can occur in well-trained subjects after a specific endurance training program. It also shows that, for the same absolute supralactate threshold work rate before and after training, significant adaptations concerning the $\dot{V}O_2$ kinetics may lead to performance improvements in well-trained subjects.

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