Effect of endurance training on oxygen uptake kinetics during treadmill running

HELEN CARTER,[1] ANDREW M. JONES,[2] THOMAS J. BARSTOW,[3] MARK BURNLEY,[4] CRAIG WILLIAMS,[4] AND JONATHAN H. DOUST[1]
[1]*University of Surrey Roehampton, London SW15 3SN;* [2]*Exercise Physiology Group, Manchester Metropolitan University, Alsager ST7 2HL, United Kingdom;* [3]*Department of Kinesiology, Kansas State University, Manhattan, Kansas 66506-0302; and* [4]*Chelsea School Research Centre, University of Brighton, Eastbourne BN20 7SP, United Kingdom*

Carter, Helen, Andrew M. Jones, Thomas J. Barstow, Mark Burnley, Craig Williams, and Jonathan H. Doust. Effect of endurance training on oxygen uptake kinetics during treadmill running. *J Appl Physiol* 89: 1744–1752, 2000.—The purpose of this study was to examine the effect of endurance training on oxygen uptake ($\dot{V}o_2$) kinetics during moderate [below the lactate threshold (LT)] and heavy (above LT) treadmill running. Twenty-three healthy physical education students undertook 6 wk of endurance training that involved continuous and interval running training 3–5 days per week for 20–30 min per session. Before and after the training program, the subjects performed an incremental treadmill test to exhaustion for determination of the LT and the $\dot{V}o_{2\,max}$ and a series of 6-min square-wave transitions from rest to running speeds calculated to require 80% of the LT and 50% of the difference between LT and maximal $\dot{V}o_2$. The training program caused small (3–4%) but significant increases in LT and maximal $\dot{V}o_2$ ($P < 0.05$). The $\dot{V}o_2$ kinetics for moderate exercise were not significantly affected by training. For heavy exercise, the time constant and amplitude of the fast component were not significantly affected by training, but the amplitude of the $\dot{V}o_2$ slow component was significantly reduced from 321 ± 32 to 217 ± 23 ml/min ($P < 0.05$). The reduction in the slow component was not significantly correlated to the reduction in blood lactate concentration ($r = 0.39$). Although the reduction in the slow component was significantly related to the reduction in minute ventilation ($r = 0.46$; $P < 0.05$), it was calculated that only 9–14% of the slow component could be attributed to the change in minute ventilation. We conclude that the $\dot{V}o_2$ slow component during treadmill running can be attenuated with a short-term program of endurance running training.

oxygen uptake slow component; lactate threshold; gas exchange; modeling

CARDIORESPIRATORY ADAPTATIONS to a period of endurance training have been well documented. Changes in maximal oxygen uptake ($\dot{V}o_2$; $\dot{V}o_{2\,max}$; Refs. 17, 43), exercise economy (9, 27), and the blood lactate response to exercise (16, 47, 51) have all been previously reported. The response characteristics of pulmonary $\dot{V}o_2$ to step changes in work rate have been comprehensively de-scribed for cycle exercise (4, 7) and may be altered by a period of endurance training (3, 13, 57).

For moderate constant-load exercise below the lactate threshold (LT), after the initial cardiodynamic phase, $\dot{V}o_2$ rises monoexponentially until a steady state is reached, usually within 2–3 min in healthy subjects. Heavy constant-load exercise above the LT, however, results in $\dot{V}o_2$ kinetics that are considerably more complex. For constant-load exercise above the LT, $\dot{V}o_2$ may reach a delayed steady state that is higher than the $\dot{V}o_2$ requirement estimated by extrapolating the relationship between $\dot{V}o_2$ and work rate for moderate exercise (5, 55) or may rise continuously until $\dot{V}o_{2\,max}$ is attained and/or exercise is terminated (39). The physiological mechanism(s) responsible for this slow-component rise in $\dot{V}o_2$ with time during supra-LT exercise remains to be determined but appears to reside in the exercising muscle (38) and to be related to the temporal profile of changes in blood lactate (39, 55).

Although few studies have examined the $\dot{V}o_2$ slow component during treadmill running, it appears that its magnitude is lower in running than in cycling (10, 31). However, in the previous studies that have assessed $\dot{V}o_2$ kinetics in running, the magnitude of the slow component has been determined rather simplistically by calculating the increase in $\dot{V}o_2$ between 3 and 6 min of exercise (28, 31) or between 3 min of exercise and the time at which exhaustion was reached (8, 10). It has been shown that the $\dot{V}o_2$ response to a square-wave heavy exercise challenge is better described by using three exponential terms, with the three terms describing the cardiodynamic phase (phase I), the fast component (phase II), and the $\dot{V}o_2$ slow component (phase III), respectively (7).

The $\dot{V}o_2$ slow component has been suggested to be an important determinant of exercise tolerance in both patient populations (54) and athletic groups (18). After a period of endurance training, the steady-state $\dot{V}o_2$ during moderate-intensity, constant-load cycle ergometry is unchanged (16, 21, 22), although the phase II

Address for reprint requests and other correspondence: H. Carter, Univ. of Surrey Roehampton, West Hill, London SW15 3SN, UK (E-mail: helen.carter@roehampton.ac.uk).

kinetics may be speeded (21, 22, 35). In contrast, after training, the $\dot{V}O_2$ slow component during heavy cycle exercise is attenuated (3, 13, 40, 57). Recent studies (10, 31) have suggested that differences in muscle contraction modes between running and cycling exercise may result in differences in $\dot{V}O_2$ kinetics. It is not known whether endurance running training can affect $\dot{V}O_2$ kinetics in running.

Therefore, the purpose of the present study was to comprehensively characterize the $\dot{V}O_2$ response to constant-speed moderate- and heavy-intensity treadmill running and to test the hypothesis that a period of endurance running training will alter the $\dot{V}O_2$ kinetics, in particular the amplitude of the slow component.

METHODS

Subjects. Twenty-three subjects (14 men; age 22 ± 3 yr, height 1.73 ± 0.06 m, body mass 70.3 ± 9.1 kg; means $\pm$ SD) volunteered to take part in this study. The subjects were young, healthy physical education students who were recreationally active in sport but not specifically trained for endurance running. Subjects were fully familiar with the laboratory environment and were habituated to treadmill running before the study commenced. The subjects gave written, informed consent after the experimental procedures and the associated risks and benefits of participation were explained. The procedures used in this study were approved by the Chelsea School Ethics Committee, University of Brighton. The subjects were all fully familiar with laboratory exercise testing procedures, having previously participated in other similar studies.

Subjects were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 3 h postprandial, and to avoid strenuous exercise in the 48 h preceding a test session. For each subject, tests took place at the same time of day (± 2 h) to minimize the effects of diurnal biological variation on the results.

Procedures. All exercise tests were performed on a motorized treadmill (Woodway, Cardiokinetics, Salford, UK), with the grade set at 1% (29). During the exercise tests, pulmonary gas exchange was determined breath by breath. Subjects breathed through a low-dead space (90 ml), low-resistance (0.65 cmH$_2$O$\cdot$l$^{-1}\cdot$s^{-1} at 8 l/s) mouthpiece and turbine assembly. Gases were continuously drawn from the mouthpiece through a 2-m capillary line of small bore (0.5 mm) at a rate of 60 ml/min and were analyzed for O_2, CO_2, and N_2 concentrations by a quadrupole mass spectrometer (CaSE QP9000, Gillingham, Kent, UK), which was calibrated before each test by use of gases of known concentration. Expiratory volumes were determined by using a turbine volume transducer (Interface Associates). The volume and concentration signals were integrated by computer, after analog-to-digital conversion, with account taken of the gas transit delay through the capillary line. Respiratory gas exchange variables [$\dot{V}O_2$, carbon dioxide production, and minute ventilation ($\dot{V}E$)] were calculated and displayed for every breath. The $\dot{V}O_2$ values were not treated with an alveolar correction algorithm and therefore represent values measured at the mouth. Heart rate was recorded telemetrically throughout the exercise tests (Polar Electro Oy, Kempele, Finland).

On the first visit to the laboratory, the subjects performed an incremental treadmill test to volitional exhaustion for the determination of LT and $\dot{V}O_{2\,max}$. The initial treadmill speed was between 6.0 and 7.0 km/h for the women and between 8.0 and 9.0 km/h for the men. The subjects completed 6–8 sub-

maximal stages of 4-min duration, with running speed increased by 1.0 km/h between stages (30). At the end of each stage, the subjects grasped the handrails and moved their feet astride the treadmill belt. A fingertip capillary blood sample (~ 25 µl) was collected into a capillary tube for subsequent analysis of blood lactate concentration ([lactate]) using an automated lactate analyzer (YSI 2300, Yellow Springs, OH). The subjects recommenced running within 10–15 s. When heart rate exceeded 90% of the known or age-predicted maximum heart rate, the running speed was increased by 1.0 km/h per minute until the subject reached volitional exhaustion.

Plots of blood lactate against running speed and $\dot{V}O_2$ were provided to two independent reviewers, who determined the LT as the first sustained increase in blood lactate above baseline levels. The breath-by-breath gas exchange data collected during these tests were averaged over consecutive 30-s periods. The $\dot{V}O_{2\,max}$ was defined as the average $\dot{V}O_2$ attained in the last 30 s of the tests. Attainment of $\dot{V}O_{2\,max}$ was confirmed by a high incidence of a plateau phenomenon in $\dot{V}O_2$ (96%), respiratory exchange ratio values above 1.10 (78%), and heart rates within 5 beats/min of age-predicted maximum (92%). In all subjects, at least two of the three criteria were met. The running speed at $\dot{V}O_{2\,max}$ was estimated by extrapolation of the sub-LT relationship between $\dot{V}O_2$ and running speed. Individual regression equations were calculated using the $\dot{V}O_2$-running speed relationship for all exercise stages below the LT. The $\dot{V}O_{2\,max}$ was then entered into the equation given, providing an estimate of the running speed at $\dot{V}O_{2\,max}$ (v-$\dot{V}O_{2\,max}$, where v is velocity). The individual regression equations were then used to calculate the running speeds corresponding to 80% of the $\dot{V}O_2$ at LT and 50% of the difference between the $\dot{V}O_2$ at LT and $\dot{V}O_{2\,max}$ (50% Δ).

Subsequently, subjects performed a series of square-wave transitions at the two exercise intensities on separate days. The subjects completed three transitions to the moderate-intensity running speed or two transitions to the heavy-intensity running speed. The days on which the moderate- and heavy-intensity exercise bouts were performed were presented to the subjects in random order. The exercise protocol started with 2 min of standing rest, with the subject's feet astride the moving treadmill belt and hands holding the guard rails. Subjects commenced running by supporting their body mass with their hands on the guard rails until leg speed matched treadmill belt speed, after which they let go of the handrails and commenced running. This transition from rest to exercise took 3–5 s. The exercise continued for 6 min. At the end of the 6-min period, the subjects grasped the guard rails and moved their feet astride the treadmill belt. Fingertip capillary blood samples were taken immediately before and immediately after exercise. The difference between the end-exercise lactate and the resting lactate was expressed as a delta value (Δ[lactate]). For the moderate-intensity running speed condition, the subjects completed three identical transitions on the same day, with at least 30 min of rest between the trials. For the heavy-intensity running speed condition, the subjects completed two identical transitions on the same day, with at least 60 min of rest between the trials. Before the second transition, a capillary blood sample was taken to ensure that lactate concentration had returned to resting levels. These procedures were replicated at the end of the training period. In addition, a randomly chosen subgroup of 10 subjects (5 men) also performed transitions to the recalculated 50% Δ running speeds derived from the post-training incremental test.

Training. After completion of the initial testing battery, the subjects began a 6-wk program of endurance training. Table 1 describes the training undertaken and shows the increases in training duration and frequency over the 6-wk period. For the continuous sessions, the LT was used to regulate the training intensity because this should provide a high-quality aerobic training stimulus without the accumulation of lactate that might compromise training duration. It has been shown that this type of training significantly im-

Nonlinear regression techniques were used to fit the time course of the $\dot{V}O_2$ response after the onset of exercise with an exponential function. An iterative process was used to minimize the sum of squared error. The empirical model consisted of two (moderate exercise) or three (heavy exercise) exponential terms, each representing one phase of the response (see Ref. 6). The first exponential term started with the onset of exercise (time = 0), whereas the other terms began after independent time delays

$$\dot{V}O_2(t) = \dot{V}O_2(b) + A_0 \cdot (1 - e^{-t/\tau_0}) \quad \textit{Phase 1 (initial component)}$$
$$+ A_1 \cdot (1 - e^{-(t - TD_1)/\tau_1}) \quad \textit{Phase 2 (primary component)} \quad (1)$$
$$+ A_2 \cdot (1 - e^{-(t - TD_2)/\tau_2}) \quad \textit{Phase 3 (slow component)}$$

proves the LT (33). Although the average intensity of the continuous and interval training sessions was similar, the interval efforts were designed to be appreciably above the LT to invoke lactate accumulation. In a previous study, this training program significantly increased the running speed at LT, the running speed at the maximal lactate steady state, and the $\dot{V}O_{2\,max}$ in 16 subjects of similar initial fitness status to the subjects of the present study (11).

To quantify the training undertaken, all subjects were issued telemetric heart rate monitors and instructed to collect and download the heart rate data for each training session completed. For the continuous training sessions, subjects exercised at the heart rate measured at the LT (± 5 beats/min) in the pretraining incremental test. The interval training sessions involved a 5-min warm-up jog, then 10 efforts of 2-min duration (at a heart rate of ~ 10 beats/min above the heart rate at LT) separated by a 2-min jog recovery (at a heart rate of ~ 10 beats/min below the heart rate at LT), and finally a 5-min jog. Mean running speeds across the group were 10.4 ± 1.9 km/h ($72.6 \pm 6.0\%$ $\dot{V}O_{2\,max}$ or $\sim 67\%$ v-$\dot{V}O_{2\,max}$) for the continuous session and 11.9 ± 2.2 km/h ($76.9 \pm 4.3\%$ $\dot{V}O_{2\,max}$, or $\sim 77\%$ v-$\dot{V}O_{2\,max}$) for the interval session. Heart rate data from individual training sessions were checked on a weekly basis to ensure that subjects exercised at the prescribed heart rates. All subjects completed training diaries listing all physical activity performed over the 6-wk study period so that training compliance could be ascertained. In addition, subjects recorded their diet before the pretraining laboratory visits and replicated this diet for their return visits to the laboratory at the end of the training period.

Data analysis. For each exercise transition, the breath-by-breath data were interpolated to give second-by-second values and were time aligned to the start of exercise. The transitions for each intensity were then averaged to enhance the underlying response characteristics.

where $\dot{V}O_2(t)$ is the $\dot{V}O_2$ at a given time; $\dot{V}O_2(b)$ is the resting baseline value; A_0, A_1, and A_2 are the asymptotic amplitudes for the exponential terms; τ_0, τ_1, and τ_2 are the time constants; and TD_1 and TD_2 are the time delays. The *phase 1* term was terminated at the start of *phase 2* (i.e., at TD_1) and assigned the value for that time (A_0')

$$A_0' = A_0 \cdot (1 - e^{-TD_1/\tau_0})$$

The $\dot{V}O_2$ at the end of *phase 1* (A_0') and the amplitude of *phase 2* (A_1) were summed to calculate the amplitude of *phase 2* (A_1'). The amplitude of the slow component was determined as the increase in $\dot{V}O_2$ from TD_2 to the end of exercise (defined A_2'), rather than from the asymptotic value (A_2), which lies beyond physiological limits (6). The slow component was also described by using the difference in $\dot{V}O_2$ between 3 and 6 min of exercise (using the average $\dot{V}O_2$ values between 2.75 and 3.00 and between 5.75 and 6.00 min). The gain of the phase II exponential response (A_1'/Δrunning speed expressed relative to body mass) for the two exercise intensities was also calculated.

Statistical analysis. Paired *t*-tests were used to determine the significance of any differences in the measured variables before vs. after training. Statistical significance was set at the 5% level. A repeated-measures ANOVA (Wilks lambda) was used to identify differences in the subgroup of subjects. Pearson product-moment correlation coefficients were used to examine the significance of relationships between the slow component and changes in blood lactate and $\dot{V}E$. The results are presented as means $\pm$ SE.

RESULTS

Table 2 shows the effect of training on the physiological variables that were measured during the incremental treadmill test. There were significant increases in $\dot{V}O_{2\,max}$ ($t_{22} = -4.22$, $P < 0.001$), the $\dot{V}O_2$ at the LT ($t_{22} = -3.78$, $P < 0.001$), and a significant reduction in maximal heart rate ($t_{22} = 3.36$, $P = 0.03$).

For moderate exercise, which caused no increase in blood lactate above baseline, the kinetics of the $\dot{V}O_2$ response were fit with two exponential terms. The $\dot{V}O_2$ kinetics for moderate exercise, including the amplitudes, time constants, and time delays, were unchanged with training (Table 3). For heavy exercise, which caused a significant increase in blood lactate above baseline, the kinetics of the $\dot{V}O_2$ response were fit

Table 1. *Progression of training duration and frequency through 6 wk of training*

Week	No. of sessions	Duration	Type
1	2	20 min	Continuous
	1	30 min	Interval
2	3	23 min	Continuous
	1	30 min	Interval
3	3	26 min	Continuous
	2	30 min	Interval
4, 5, and *6*	3	30 min	Continuous
	2	30 min	Interval

Table 2. *Subject group description and index of aerobic fitness pre- and posttraining*

	Pretraining	Posttraining
Mass, kg	70.3 ± 1.9	70.2 ± 2.0
$\dot{V}O_{2\,max}$, ml·kg^{-1}·min^{-1}	54.9 ± 2.1	$56.4 \pm 2.1^*$
RS-$\dot{V}O_{2\,max}$, km/h	15.5 ± 0.6	16.6 ± 0.6
HR_{max}, beats/min	199 ± 2.0	$195 \pm 2^*$
RS-LT, km/h	10.4 ± 0.4	$11.4 \pm 0.4^*$
$\dot{V}O_2$ at LT, l/min	2.77 ± 0.1	$2.89 \pm 0.1^*$
HR at LT, beats/min	159 ± 3	$165 \pm 2^*$

Values are means $\pm$ SE. HR, heart rate. RS-$\dot{V}O_{2\,max}$ was calculated as the running speed (RS) at maximal oxygen uptake ($\dot{V}O_{2\,max}$) extrapolated from the subthreshold RS-$\dot{V}O_2$ relationship. RS-LT, running speed at lactate threshold (LT). *Significant differences pre to post.

with three exponential terms. Endurance training caused a significant reduction in the blood lactate accumulation ($t_{22} = 3.11$, $P = 0.005$) but did not affect the time delays or the time constants of the exponential model or the amplitude of phase II (Table 3). It was of interest that the time constant for phase II was unchanged with training ($t_{22} = 0.49$, $P = 0.63$). However, it should be noted that our subjects were relatively fit at the time of recruitment to the study. When the data of six subjects (2 men, 4 women) who had the lowest fitness on recruitment to the study were examined ($\dot{V}O_{2\,max}$ of 40.2 ± 1.2 ml·kg^{-1}·min^{-1}), τ_1 for heavy exercise was reduced from 31.5 ± 1.0 to 19.5 ± 1.5 s by the training period ($t_5 = 2.92$, $P = 0.033$).

The amplitude of the $\dot{V}O_2$ slow component was significantly reduced with training both in absolute terms (Fig. 1, $t_{22} = 3.53$, $P = 0.002$) and when expressed as a proportion of the total $\dot{V}O_2$ response to the exercise (Table 3, $t_{22} = 3.57$, $P = 0.002$). All subjects demonstrated an attenuated slow component with training, although the magnitude of the reduction varied between individuals (range 8–308 ml/min). This reduction in the slow component resulted in a significantly reduced end-exercise $\dot{V}O_2$ ($t_{22} = 4.90$, $P = 0.004$). Neither the increase in $\dot{V}O_{2\,max}$ nor the increase in LT was correlated with the decrease in the amplitude of $\dot{V}O_2$ slow component ($r = 0.03$ and $r = 0.04$, respectively).

There was no significant improvement in running economy with training in the subjects. The average steady-state $\dot{V}O_2$ at 80% LT (calculated from the three transitions of 6-min duration that our subjects performed at this exercise intensity) was not significantly different before and after training. Furthermore, there was no consistent or significant reduction in $\dot{V}O_2$ during the submaximal stages of the incremental test.

In the 10 subjects who performed a bout of heavy exercise at 50%Δ recalculated after training, despite increases in the running speed utilized and the amplitude of phase II ($t_9 = -2.7$, $P = 0.02$), the slow component amplitude was similar compared with the pretraining 50%Δ condition (Table 4, $t_9 = 1.37$, $P = 0.20$). Data from a typical subject can be seen in Fig. 2.

Before training, there was a strong correlation between the $\dot{V}O_2$ slow component and the increase in blood lactate above baseline ($r = 0.69$; $P < 0.001$). However, after training, the strength of this association diminished ($r = 0.45$; $P = 0.03$). The relationship between the reduction in blood lactate accumulation and the reduction in the slow component with training was not significant ($r = 0.39$, $P = 0.65$). Similarly, there was a strong relationship between the $\dot{V}O_2$ slow component and the increase in $\dot{V}E$ over the same time frame before training ($r = 0.70$; $P < 0.01$), which diminished after training ($r = 0.20$; $P = 0.86$). There

Table 3. *Parameters of oxygen uptake response during moderate and heavy exercise before and after a period of endurance training*

Parameter	Moderate		Heavy	
	Pretraining	Posttraining	Pretraining	Posttraining
Running speed, km/h	7.8 ± 0.3	7.8 ± 0.3	13 ± 0.5	13 ± 0.5
A_0', ml/min	792 ± 70.4	771 ± 63.7	$1,152 \pm 88.4$	$1,150 \pm 71.6$
τ_0, s	4.0 ± 0.9	4.9 ± 1.2	7.0 ± 1.2	5.8 ± 1.0
TD_1, s	22.3 ± 1.8	23.3 ± 1.7	17.8 ± 1.0	18.4 ± 0.7
A_1', ml/min	$1,615 \pm 96.4$	$1,604 \pm 103$	$2,730 \pm 124.3$	$2,677 \pm 117$
τ_1, s	16.3 ± 1.1	13.9 ± 1.4	19.4 ± 1.2	18.8 ± 4.9
Gain, ml·kg^{-1}·km^{-1}	177 ± 6.5	175 ± 6.5	180 ± 3.2	177 ± 2.5
MRT, s	21.3 ± 0.9	21.7 ± 1.8	58.2 ± 1.8	$47.4 \pm 1.8^*$
TD_2, s			114.1 ± 6.0	114.3 ± 5.1
A_2', ml/min			321.4 ± 32	$217 \pm 23^*$
Relative A_2', %			10.2 ± 1.6	$7.3 \pm 0.7^*$
τ_2, s			243.8 ± 13.0	247.3 ± 13.3
EE $\dot{V}O_2$, ml/min			$3,052 \pm 144$	$2,894 \pm 131^*$
$\Delta\dot{V}E$, l/min			17.5 ± 1.8	$12.2 \pm 1.6^*$
EE HR, beats/min	128 ± 3	129 ± 3	173 ± 2	$170 \pm 2^*$
Δ[lactate], mM	0.0 ± 0.1	0.0 ± 0.1	2.7 ± 0.2	$2.1 \pm 0.2^*$

Values are means $\pm$ SE. BL, baseline; TD and TD_2, time delays; τ_0, τ_1, and τ_2, time constants from *Eq. 1*; A_0', amplitude at end of phase 1; A_1', sum of A_1 in *Eq. 1* and A_0' (equal to amplitude of fast primary component); A_2', value of slow component at end of exercise; Relative A_2' ($A_2'/A_1' + A_2'$), relative contribution of slow component to net increase in $\dot{V}O_2$ at end exercise (EE); EE $\dot{V}O_2$, increase in $\dot{V}O_2$ above baseline at end of exercise; MRT, mean response time, the weighted sum of all three phases, yielding a value for the time taken to reach 63% of the EE $\dot{V}O_2$; Gain, A_1'/Δ running speed expressed relative to body mass; $\Delta\dot{V}E$, change in minute ventilation; Δ[lactate], change in lactate concentration. *Significantly different after training, $P < 0.05$ (exact P values given in text).

Fig. 1. Example of oxygen uptake ($\dot{V}O_2$) response of 1 subject at 50%Δ (50% of the difference between $\dot{V}O_2$ at lactate threshold and $\dot{V}O_{2\,max}$), before ($\bullet$) and after ($\circ$) the training intervention. Note the reduced A_2' post-training.
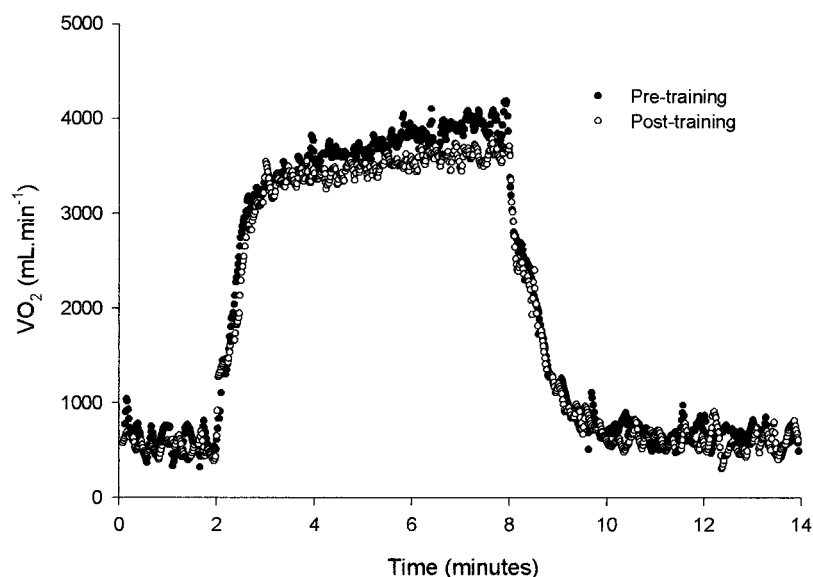
was a significant relationship between the reduction in $\dot{V}E$ and the reduction in the slow component with training ($r = 0.46$; $P = 0.03$).

DISCUSSION

The principal finding of this study was that a 6-wk period of endurance training caused an attenuation of the slow component in running of $\sim$35%, i.e., from 321 to 217 ml/min, on average. This is of interest in that our subjects were young, healthy subjects who were actively engaged in sports and were of a relatively high fitness at entry into the study. These results suggest that the $\dot{V}O_2$ slow component can be reduced by a short period of endurance training despite relatively small changes in traditional measures of aerobic fitness such as $\dot{V}O_{2\,max}$ and LT.

The endurance training program that our subjects undertook was successful in causing a significant improvement in the $\dot{V}O_2$ at LT ($\sim$4%) and $\dot{V}O_{2\,max}$ ($\sim$3%), although these improvements were less than those reported in other training studies (16, 23, 53). Our

subjects recorded their training in diaries over the course of the study and also used heart rate monitors to guide their training. From the heart rate records, subjects adhered to their prescribed training intensities, and, from diary records, the training compliance of the subjects was 87 ± 1.7%. The mean pretraining $\dot{V}O_{2\,max}$ of $\sim$55 ml$\cdot$kg$^{-1}\cdot$min^{-1} (in a mixed-gender group) highlights the fact that our subjects were already of good aerobic fitness on recruitment to the study. The rather small increases in $\dot{V}O_{2\,max}$ should therefore not be considered surprising.

In our study, there were no significant relationships between τ_1 and $\dot{V}O_{2\,max}$ for moderate or heavy exercise either before or after training. Our results differ from those of Powers et al. (41), who reported that, in subjects of similar training status to subjects of the present study, $\dot{V}O_2$ kinetics were faster in trained subjects with the higher $\dot{V}O_{2\,max}$ values. Our results also contrast with the study of Chilibeck et al. (14), which showed significantly faster $\dot{V}O_2$ kinetics in subjects

Table 4. *Parameters of oxygen uptake response during heavy exercise in the subgroup before and after training*

Parameter	Pretraining	Posttraining	Posttraining "New"
Running speed, km/h	12.7 ± 0.9	12.7 ± 0.9	13.6 ± 0.9
A_0, ml/min	$1,112 \pm 145$	$1,110 \pm 102$	$1,090 \pm 94.8$
τ_0, s	4.2 ± 1.2	4.8 ± 0.7	3.4 ± 1.1
TD_1, s	17.7 ± 1.3	18.8 ± 0.6	16.7 ± 0.9
A_1', ml/min	$2,684.7 \pm 218$	$2,663.9 \pm 208$	$2,825.7 \pm 223$*
τ_1, s	17.5 ± 1.2	18.4 ± 1.3	18.5 ± 1.3
Gain, ml$\cdot$kg$^{-1}\cdot$km^{-1}	172.9 ± 5.3	172.9 ± 4.2	170.5 ± 4.9
MRT, s	57.6 ± 2.6	49.7 ± 2.3*	53.4 ± 1.2*
TD_2, s	111.4 ± 9.2	115.3 ± 8.2	117.1 ± 5.9
A_2', ml/min	314.2 ± 45.1	213.2 ± 30.1*	249.4 ± 34.5
Relative A_2', %	10.3 ± 1.2	7.2 ± 0.9*	7.9 ± 0.7
τ_2, s	245.6 ± 22.9	269.5 ± 19.6	286.7 ± 46.3
EE $\dot{V}O_2$, ml/min	$2,998.9 \pm 243$	$2,877.1 \pm 228$	$3,288.1 \pm 235$
$\Delta\dot{V}E$, l/min	20.3 ± 2.3	10.3 ± 2.3*	13.4 ± 2.2
EE HR, beats/min	176 ± 3	172 ± 3*	178 ± 3
Δ[lactate], mM	3.5 ± 0.4	3.3 ± 0.3	4.3 ± 0.4

Values are means $\pm$ SE. Posttraining "new" refers to the data from the recalculated 50% Δ trial. *Significantly different from pretraining value, $P < 0.05$ (exact P values given in text).
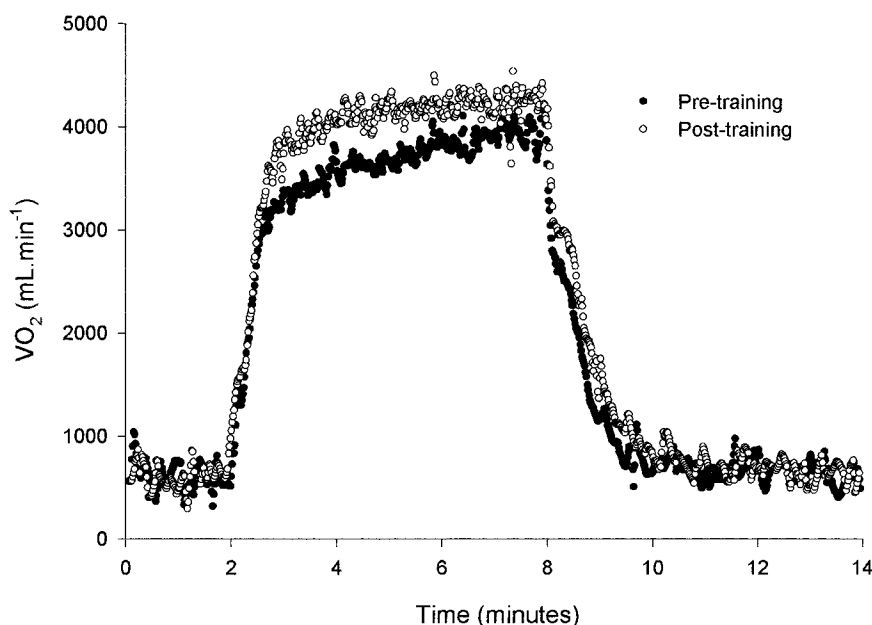
Fig. 2. Example of $\dot{V}O_2$ response of 1 subject from the subgroup. Pretraining (●) 50% $\Delta\dot{V}O_2$ and the recalculated 50% Δ posttraining (○) are shown. Note the larger A'_1 due to the higher absolute running speed but similar A'_2.

with higher $\dot{V}O_{2\,max}$ values in 16 young subjects. However, their subjects were of generally lower aerobic fitness and showed greater heterogeneity in $\dot{V}O_{2\,max}$ (25–59 ml·kg^{-1}·min^{-1}) than our subjects.

The training program had no effect on the kinetics of the $\dot{V}O_2$ response to moderate exercise or on phase II during heavy exercise. These results are in contrast to other studies that have shown a speeding of $\dot{V}O_2$ kinetics with training for exercise intensities up to ~70% $\dot{V}O_{2\,max}$ (21, 22, 58). The relatively high aerobic fitness of our subjects at the start of the training study may provide an explanation for this. Indeed, in the six subjects with the lowest initial $\dot{V}O_{2\,max}$ values (~40 ml·kg^{-1}·min^{-1}), we found that τ_1 was significantly decreased (i.e., the adjustment of $\dot{V}O_2$ was speeded) by training for heavy exercise (by 12 s) but not for moderate exercise. The faster kinetics of phase II in our low-fit subjects may be related to improved oxygen delivery to muscle or to increases in muscle mitochondrial density with training, which would improve the sensitivity of respiratory control (20).

Another explanation for the apparent discrepancy between the present study and previous literature is that earlier studies (21, 22, 41) did not attempt to partition out any effect of the possible development of the $\dot{V}O_2$ slow component on the total $\dot{V}O_2$ response but simply calculated the half-time of the response to the steady state. It is not clear in these studies whether the subjects were exercising above or below their LT. When the mean response time is considered in the present study, it can be seen that, whereas the overall $\dot{V}O_2$ kinetic response for moderate exercise was unchanged at ~22 s, the mean response time was significantly decreased for heavy exercise (from ~58 to 47 s; $t_{22} = 4.2$, $P < 0.001$; see Table 3). However, the latter results from the reduction of the slow component and not from any speeding of phase II.

Our results that the time constant of phase II does not differ significantly above and below the LT (Table 3) supports the findings of some (5, 7, 12), but not all (34), previous studies using cycle exercise. To consider whether the lack of difference in τ_1 between moderate and heavy exercise might be a consequence of the fitness of the subjects, we looked at the unfit subgroup. Interestingly, this subgroup had a considerably slower τ_1 in heavy exercise (~31 s) than in moderate exercise (~17 s) pretraining. The period of endurance training speeded the phase II response in heavy exercise (~20 s) but not in moderate exercise (~13 s), bringing τ_1 near to the values of the other 17 subjects and to the whole-group mean (~19 s). This would suggest that a period of endurance training speeds suprathreshold kinetics in less fit individuals, causing τ_1 to reduce toward subthreshold values. Despite the training improvement, the heavy exercise τ_1 was still slower than in moderate exercise in this group. This tends to support the work of Paterson and Whipp (34), who suggest slower kinetics during suprathreshold work. However, further specific studies of treadmill running are clearly required before firm conclusions can be drawn.

The amplitude of phase II was linearly related to exercise intensity because the gain (A'_1/running speed expressed relative to body mass) was not significantly different between the 80% LT and the 50% Δ conditions before ($t_{22} = 0.65$, $P = 0.67$) or after training ($t_{22} = 0.44$, $P = 0.20$). This is in accordance with previous work (7, 12, 34) and confirms that the slow component is a phenomenon of delayed onset that causes $\dot{V}O_2$ to rise above (rather than toward) the predicted steady-state value.

Despite the relatively small increases in LT and $\dot{V}O_{2\,max}$ caused by the training program, there was a substantial and significant reduction in the slow component for the same running speed posttraining. This

was true even when the six less fit subjects were removed from the analysis and when men and women were analyzed separately. The reduction of the $\dot{V}O_2$ slow component has significant implications for exercise tolerance in athletic, sedentary, and patient populations. For the competitive athlete, the ability to run at a faster running speed for a given metabolic stress (i.e., same $\dot{V}O_2$ slow component) may result in performance enhancement. This is exemplified by the data from the subgroup within the present study (Table 4). In addition to a reduced slow component at the same absolute exercise intensity, these subjects were able to run at a faster speed while eliciting a slow component of similar magnitude ($\sim$250–300 ml/min) to that of pretraining heavy exercise. For patients with cardiac and/or pulmonary disorders, even very low exercise intensities, such as slow walking, represent a severe metabolic stress (50). Endurance training will increase the range of intensities over which the $\dot{V}O_2$ slow component would not be expected to develop. This would improve the functional ability of these individuals irrespective of any change in LT or $\dot{V}O_{2\,max}$ (36).

This study is the first to investigate the effect of training on $\dot{V}O_2$ kinetics by using mathematical modeling. Previous studies (13, 57) have defined the slow component as the increase in $\dot{V}O_2$ between 3 min and the end of exercise ($\Delta\dot{V}O_2$). The slow component first becomes evident at about 2 min into exercise ($\sim$114 $\pm$ 6.0 s; see Table 3 and Ref. 6). Therefore, defining the slow component as an increase in $\dot{V}O_2$ above the value at 3 min of exercise will significantly underestimate the magnitude of the slow component. Indeed, calculating the $\Delta\dot{V}O_2$ in the present study showed a reduction in the slow component of $\sim$60 ml/min with training (193 $\pm$ 22 vs. 130 $\pm$ 15 ml/min, t_{22} = 3.18, P = 0.004). This more simplistic characterization of the slow component might also explain why some studies have reported that the slow component is trivially small during treadmill running (8, 10).

Our results are similar to those of Casaburi et al. (13) and Womack et al. (57), who showed that the $\dot{V}O_2$ slow component could be significantly reduced after 8 wk and 6 wk, respectively, of cycle ergometer training. In agreement with Casaburi et al. (13), we found that the reduction in the slow component was related to the reduction in the $\Delta\dot{V}E$ and the Δ[lactate], although the latter relationship did not attain significance in our study. We did not assess the relationship between the reduction in the $\dot{V}O_2$ slow component and changes in other putative mediators of the slow component such as body temperature and catecholamine concentrations. However, previous studies have shown that infusion of epinephrine during exercise has no effect on the exercise $\dot{V}O_2$ despite causing significant increases in plasma epinephrine and lactate (19, 37). Furthermore, Casaburi et al. found no significant relationship between the changes in catecholamines and the changes in the slow component that occurred with endurance training. In the same study, there was no significant relationship between changes in rectal temperature and changes in the slow component with training (13).

It was of interest that the strong relationship between the Δ[lactate] and the slow component found before training (r = 0.69) was reduced considerably after training (r = 0.45). Furthermore, the changes in lactate and the slow component with training were not significantly related. Several previous cross-sectional studies have shown a close relationship between the magnitude of the blood lactate increase and the slow component for cycle exercise (39, 42, 55). It has been suggested that lactic acidosis is important in the maintenance of muscle P_{O_2} during heavy exercise (48), and that the metabolic cost of glycogenesis from lactate during exercise above the LT may contribute to the development of the slow component (13). The proportion of the lactate produced during exercise the fate of which is gluconeogenesis as opposed to oxidation is not certain, but it can be calculated that the additional oxygen cost of lactate catabolism is not sufficient to make a significant contribution to the slow component (54). In two recent studies, a weaker relationship between lactate and the slow component was reported in runners (r = 0.36; Ref. 31) and triathletes (r = 0.12; Ref. 9) during treadmill exercise. In these studies, the slow component was significantly greater for cycling than for running for the same elevated blood lactate concentrations (10, 31). These results support the suggestion that the relationship between lactate increase and the slow component is coincidental rather than causal. This interpretation is supported by studies that show an unchanged $\dot{V}O_2$ when *1*) blood lactate is increased through infusion of epinephrine during exercise (19, 37), *2*) exercise blood lactate is lowered by the inhibition of carbonic anhydrase with acetazolamide (45), and *3*) L-(+)-lactate is infused into the arterial blood supply of dogs who were exercised using electrical stimulation (37). In addition, the time at which the $\dot{V}O_2$ slow component emerges ($\sim$80–140 s) is much later than the time at which lactate appears in the femoral vein (42).

The $\dot{V}O_2$ slow component was significantly related to the change in $\dot{V}E$ over the same period of time before (r = 0.70) but not after (r = 0.20) training. The reduction in $\dot{V}E$ for the same running speed after training was also significantly related to the reduction in the slow component at that running speed. This, of course, does not necessarily indicate that the increase in $\dot{V}E$ over time during heavy exercise is an important mediator of the slow component, because a reduced $\dot{V}O_2$ with training is likely to require a reduced $\dot{V}E$. The lowering of blood lactate concentrations caused by training would reduce the ventilatory load consequent to a reduced bicarbonate buffering of the lactic acidosis (13). Using the estimates of Aaron et al. (1) that the oxygen cost of $\dot{V}E$ ranges from 1.79 ml O_2 per liter for ventilatory rates of 63–79 l/min to 2.85 ml O_2 per liter for ventilatory rates of 117–147 l/min, the reduction in the $\dot{V}E$ we observed with training (Table 3) could account for only 9–14% of the reduction in the slow component. These results are consistent both with pre-

vious studies showing that $\dot{V}E$ contributes minimally ($\leq$20%) to the slow component (28, 57) and with the study of Poole et al. (38), which demonstrated that ~86% of the $\dot{V}O_2$ slow component could be accounted for by processes inherent to the exercising limbs.

One possibility for the significant reduction in the slow component posttraining is an alteration in the motor unit recruitment pattern. Both electromyographic (46) and glycogen depletion studies (49) indicate that type II muscle fibers are recruited at exercise intensities associated with the slow component. It is known that the type II muscle fiber is less efficient than the type I muscle fiber and that the ratio of phosphate produced to oxygen molecule consumed (P-O) is ~18% lower in the isolated type II fiber due, in part, to a greater reliance on the α-glycerophosphate shuttle over the malate-aspartate shuttle (15, 44, 52, 56). This would predict a greater $\dot{V}O_2$ for any given rate of ATP resynthesis. Barstow et al. (6) showed that the contribution of the slow component to the total $\dot{V}O_2$ response to 8 min of heavy exercise was significantly positively related to the proportion of type II fibers in the vastus lateralis. Endurance training is known to result in a significant enhancement in the mitochondrial density and the capillarity of type I and type II muscle fibers (2, 24). Interconversion of fiber types may also be possible (type IIb $\rightarrow$ IIa $\rightarrow$ I) (2, 26), although this has not been demonstrated in short-term human training studies. Although we cannot be certain of the extent to which our training program required the recruitment of type II muscle fibers, changes such as an increased muscle mitochondrial content and improved perfusion in type I muscle fibers might result in the recruitment of fewer type II motor units for the same exercise intensity after training.

In conclusion, we have shown that an endurance training program that produced small but significant increases in $\dot{V}O_{2\,max}$ and LT did not affect the $\dot{V}O_2$ response to moderate exercise or the phase II response during heavy exercise. However, the training led to a significant reduction in the $\dot{V}O_2$ slow component at the same absolute running speed. Although we did not measure performance directly, our results demonstrating a slow component of similar magnitude when individuals exercised at higher running speeds after training suggests improved exercise tolerance. We speculate that the reduced slow component may be linked to the recruitment of fewer low-efficiency type II muscle fibers for the same exercise intensity after training.

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