

Influence of muscle fiber type and pedal frequency on oxygen uptake kinetics of heavy exercise

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Barstow, Thomas J., Andrew M. Jones, Paul H. Nguyen, and Richard Casaburi. Influence of muscle fiber type and pedal frequency on oxygen uptake kinetics of heavy exercise. *J. Appl. Physiol.* 81(4): 1642–1650, 1996.—We tested the hypothesis that the amplitude of the additional slow component of $\dot{V}O_2$ during heavy exercise is correlated with the percentage of type II (fast-twitch) fibers in the contracting muscles. Ten subjects performed transitions to a work rate calculated to require a $\dot{V}O_2$ equal to 50% between the estimated lactate (Lac) threshold and maximal $\dot{V}O_2$ (50% Δ). Nine subjects consented to a muscle biopsy of the vastus lateralis. To enhance the influence of differences in fiber type among subjects, transitions were made while subjects were pedaling at 45, 60, 75, and 90 rpm in different trials. Baseline $\dot{V}O_2$ was designed to be similar at the different pedal rates by adjusting baseline work rate while the absolute increase in work rate above the baseline was the same. The $\dot{V}O_2$ response after the onset of exercise was described by a three-exponential model. The relative magnitude of the slow component at the end of 8-min exercise was significantly negatively correlated with %type I fibers at every pedal rate ($r = 0.64$ to 0.83 , $P < 0.05$ – 0.01). Furthermore, the gain of the fast component for $\dot{V}O_2$ (as $\text{ml} \cdot \text{min}^{-1} \cdot \text{W}^{-1}$) was positively correlated with the %type I fibers across pedal rates ($r = 0.69$ – 0.83). Increase in pedal rate was associated with decreased relative stress of the exercise but did not affect the relationships between %fiber type and $\dot{V}O_2$ parameters. The relative contribution of the slow component was also significantly negatively correlated with maximal $\dot{V}O_2$ ($r = -0.65$), whereas the gain for the fast component was positively associated ($r = 0.68$ – 0.71 across rpm). The amplitude of the slow component was significantly correlated with net end-exercise Lac at all four pedal rates ($r = 0.64$ – 0.84), but Lac was not correlated with %type I ($P > 0.05$). We conclude that fiber type distribution significantly affects both the fast and slow components of $\dot{V}O_2$ during heavy exercise and that fiber type and fitness may have both codependent and independent influences on the metabolic and gas-exchange responses to heavy exercise.

energetics; muscle fiber type; pedal frequency; slow component of oxygen uptake

AFTER THE ONSET OF EXERCISE of moderate intensity [below the threshold for accumulation of blood lactate during incremental exercise, i.e., lactate threshold (LT)], pulmonary oxygen uptake ($\dot{V}O_2$) rises monoexponentially until steady-state $\dot{V}O_2$ is achieved, usually within 2–3 min in healthy volunteers. However, during heavy exercise that engenders a significant lactic acidosis ($>LT$), pulmonary $\dot{V}O_2$ does not achieve an early steady state but continues to rise for several minutes until either a delayed steady state is achieved, or exercise is terminated, or exhaustion ensues (6, 34, 35). This slow additional rise in $\dot{V}O_2$ projects above, rather than

toward, the $\dot{V}O_2$ requirement as predicted from exercise below LT.

The physiological mechanism(s) for the $\dot{V}O_2$ slow component remains obscure. A number of suggestions have been put forward to account for the phenomenon, including the level of circulating catecholamines, an additional oxygen cost of high rates of ventilation, and increasing muscle and body temperature (12, 34). However, measurement of tissue oxygenation with near-infrared spectroscopy (7), direct measurement of femoral vein oxyhemoglobin saturation (39), and measurement of leg $\dot{V}O_2$ during heavy exercise (33) all suggest that the predominant portion of the slow component of $\dot{V}O_2$ associated with heavy exercise originates in the exercising limbs. The close correlation between the magnitude of the $\dot{V}O_2$ slow component and the change in blood lactate concentration during above LT exercise (35) led to speculation that the $\dot{V}O_2$ slow component might be related to the catabolism of lactate as exercise substrate or to the use of lactate in glycogenesis (12, 34). However, neither infusion of lactate into working dog gastrocnemius (32) nor elevation of blood lactate concentration by infusion of epinephrine in humans (21) affected the $\dot{V}O_2$ response to exercise.

Human skeletal muscle is composed of two main fiber types, type I (slow twitch) and type II (fast twitch) (11). It is known that the type II fiber is less efficient energetically, i.e., the high-energy phosphate produced per oxygen molecule consumed (P/O) is less than the type I fiber (17, 26). In elite cyclists, individuals with higher proportions of type I fibers in the vastus lateralis muscle generate higher power outputs for the same $\dot{V}O_2$ (16, 24). Electromyographic studies (27) and analysis of glycogen content in the various fiber pools from muscle biopsy specimens (40) demonstrate that type II fibers are active in the work rate domain associated with the $\dot{V}O_2$ slow component. Thus the consensus in current thinking suggests a mechanistic link between the energetics of contraction of type II fibers and the $\dot{V}O_2$ slow component (31).

This study was designed to test the hypothesis that the $\dot{V}O_2$ slow component is related to the recruitment of type II muscle fibers (Fig. 1). We recognized that there is as yet no reliable method for quantifying the instantaneous contribution of different fiber types to muscle recruitment and to the resulting overall metabolic responses measured either in the blood or at the mouth. Therefore, we utilized two indirect approaches to test the hypothesis. In the first approach, the $\dot{V}O_2$ kinetics during heavy exercise were correlated with the muscle fiber type distribution, determined from muscle biopsy, in subjects selected on the basis of their activity levels and/or sports specialization. In the second approach,

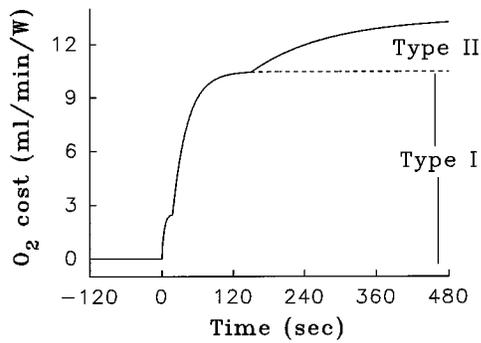


Fig. 1. Schematic of *hypothesis 1*, showing predicted influence of type II (fast-twitch) muscle fibers on whole body O₂ uptake ($\dot{V}O_2$) response during heavy exercise. Note that influence is hypothesized to be reflected exclusively in additional slow component of $\dot{V}O_2$, with primary fast $\dot{V}O_2$ response representing respiration of type I (slow-twitch) fibers.

the physiological responses of the subjects to heavy exercise for a range of pedal frequencies [45–90 revolutions/min (rpm)] and resulting range of muscle tensions in the same metabolic rate domain were examined. Available evidence suggests that, for the same external power output, type II motor units may be recruited preferentially at low pedal frequencies (40–50 rpm), i.e., when muscle tension is high (2), and at high pedal rates (90+ rpm) when contraction velocity is high (37). Thus we hoped to enhance any effect of type II fiber recruitment on the $\dot{V}O_2$ kinetics of heavy exercise by manipulation of pedal frequency. Our second hypothesis was that extremes in pedal frequency (e.g., 45 and 90 rpm) would show greater discrimination of any influence of muscle fiber type on $\dot{V}O_2$ kinetics during heavy exercise.

METHODS

Subjects. Ten subjects (9 male, 1 female) gave written consent to participate in this study after all procedures and the possible risks and benefits of participation were explained. The experimental protocol and consent form were reviewed and approved by the Human Subjects Committee of Harbor-UCLA Medical Center. Physical characteristics of the subjects are given in Table 1.

Protocol. Subjects visited the Pulmonary and Exercise Physiology Laboratories at Harbor-UCLA Medical Center on

four occasions within a 2-wk period. Exercise testing took place at the same time of day (± 2 h) for each subject. The seat height and handlebar position on the cycle ergometer were recorded on the first visit and replicated on subsequent visits. Subjects were instructed to avoid the consumption of food, alcohol, and caffeine in the 4 h preceding each test and to avoid strenuous exercise in the 24 h preceding test sessions.

The first visit was used to familiarize subjects with the procedures for exercise testing and for determination of estimated LT, peak $\dot{V}O_2$ ($\dot{V}O_{2peak}$), and the metabolic cost of unloaded cycling at each of the pedal frequencies used in this study (45, 60, 75, and 90 rpm). Exercise was performed on an electronically braked cycle ergometer (Quinton Corival model 844). Calibration of the actual power output for a given setting at each of the test pedal frequencies was performed before the beginning of the study by using a cycle ergometer calibrator designed in our laboratory by Dr. Andrew Huszczuk. The calibrator consisted of a lever arm that was attached to the shaft of an electric motor. In turn, the other end of the motor shaft was connected to the crankshaft of the cycle ergometer. The lever arm rested on a load cell. The instantaneous torque required to match the torque produced by the cycle ergometer crankshaft was measured at several work rate settings for each rpm and converted to power output in watts. The resulting calibration curves were used to correct the work rates at each pedal frequency. Respiratory gas exchange and heart rate (HR) were measured on a breath-by-breath basis, as described below.

Subjects first performed unloaded cycling for 4 min each at pedaling frequencies of 45, 75, and 90 rpm. After a brief rest, subjects then performed 4 min of unloaded cycling at 60 rpm, followed by a progressively increasing work rate (ramp) test to volitional fatigue with the pedaling rate maintained at 60 rpm. Data were averaged over 10-s periods and plotted. $\dot{V}O_{2peak}$ was defined as the highest 10-s value seen during exercise, whereas the LT was estimated from gas-exchange responses as the $\dot{V}O_2$ above which there was hyperventilation with respect to $\dot{V}O_2$ but not to CO production ($\dot{V}CO_2$) (41). The work rate calculated to require a $\dot{V}O_2$ half way between the LT and $\dot{V}O_{2peak}$ [$50\% \Delta$, equal to $LT + 0.5 * (\dot{V}O_{2peak} - LT)$] for exercise at 60 rpm was determined.

On each of two subsequent days, subjects performed two square-wave transitions at different pedal frequencies from equivalent metabolic rates to a work rate predicted to require $50\% \Delta \dot{V}O_2$ for each pedal frequency. For each day, bouts were separated by at least 45 min, and the second bout did not commence until HR and blood lactate levels had returned to resting levels. The order in which the four exercise bouts at different pedal frequencies were performed was randomly

Table 1. *Subject characteristics*

Subject	Body Mass, kg	$\dot{V}O_{2max}$, l/min	$\dot{V}O_{2max}$, ml·kg ⁻¹ ·min ⁻¹	LT, l/min	Fiber Type, %			Physical Activity Patterns
					I	Ia	Iib	
1	73	3.90	53.4	2.42	50	47	3	GFT
2	73	2.80	38.5	1.53	44	45	11	Sedentary
3	70	2.96	42.3	1.35	28	72	0	100-m sprinter
4	68	3.35	49.3	1.70	52	45	3	Karate
5	86	3.94	45.8	1.70				Wrestling
6	66	3.30	50.4	1.55	64	36	0	GFT
7	69	2.78	40.5	1.35	18	80	2	Sedentary
8	61	2.95	48.4	1.50	56	42	2	Aerobics instructor
9	79	4.02	50.9	1.93	41	58	1	GFT
10	63	3.97	63.0	2.15	67	28	5	Marathon runner
Mean \pm SD	70.7 \pm 7.5	3.40 \pm 0.52	48.2 \pm 7.1	1.72 \pm 0.35	47 \pm 16	50 \pm 17	3 \pm 3	

$\dot{V}O_{2max}$, maximum O₂ uptake; LT, lactate threshold, estimated here from gas exchange responses; GFT, general fitness training.

assigned. Respiratory gas exchange and HR were measured breath by breath during a 2-min period of quiet breathing while the subjects were seated on the ergometer, a 4-min period of baseline cycling, 8 min of exercise at the 50%Δ work rate, and 8 min of recovery at the specific baseline conditions for that pedal frequency. The minimum possible baseline work rate for pedaling at 90 rpm was unloaded cycling. Baseline work rates for the three slower pedal rates (75, 60, and 45 rpm) were calculated based on the measured $\dot{V}O_2$ during the last 2 min of the unloaded cycling period for each pedal frequency, to which 1 W was added for each additional 10 ml of desired $\dot{V}O_2$. In this way, the resulting baseline work rate for each pedal frequency would theoretically require the same $\dot{V}O_2$ as the $\dot{V}O_2$ measured during unloaded cycling at 90 rpm (see Table 2). The exercise work rate was initially calculated for 60 rpm so that the predicted steady-state $\dot{V}O_2$ would require a $\dot{V}O_2$ equal to 50%Δ from the ramp exercise test. The same Δwork rate (i.e., exercise work rate – baseline work rate) was then added to the baseline work rate for each of the other pedal frequencies to produce the exercise work rates for those conditions (see Table 2). This design was deemed appropriate because pilot work in five subjects demonstrated that the estimated LT and $\dot{V}O_{2peak}$ were not significantly different across pedal frequencies.

Arterialized capillary blood was obtained from a fingertip at rest, at the end of the 4-min baseline exercise, at the end of the exercise period, and each minute for the first 3 min of recovery. These samples were immediately analyzed for whole blood lactate concentration (YSI STAT 2300, Yellow Springs, OH). During the last 30 s of exercise, subjects were asked to rate their perceived exertion using a modified Borg scale (1 = nothing at all; 10 = very, very heavy).

Measurement of pulmonary gas exchange. Pulmonary gas exchange ($\dot{V}O_2$ and $\dot{V}CO_2$ output), minute ventilation, HR, and other related respiratory variables were measured breath by breath with a computer-based system, as previously described (4). Alveolar estimates of $\dot{V}O_2$ and $\dot{V}CO_2$, which appreciably reduce the breath-to-breath noise and enhance the underlying response characteristics, were calculated off-line for each breath.

Muscle biopsy. Muscle biopsies were obtained from the left vastus lateralis by using the needle biopsy technique of

Bergstrom (9). Care was taken to obtain the biopsies from approximately the same depth for each subject (2 cm below the skin). Muscle samples were mounted in embedding medium and frozen in isopentane previously cooled to its freezing point in liquid nitrogen. The embedded samples were stored at –80°C until further analysis. Serial cross sections (8–10 μM thick) were cut in a cryostat maintained at –20°C. The sections for myofibrillar adenosinetriphosphatase histochemistry were preincubated at pH values of 4.6 and 9.4. According to the lability to the acid and alkaline preincubation, the fibers were classified as either type I, IIA, or IIB (11). For each subject, 500–900 fibers were analyzed, and each fiber type was expressed as a percentage of the total number counted.

Data analysis. For each exercise transition at each pedal frequency, the breath-by-breath data were interpolated to give values second by second and were time aligned to the start of exercise. The time course of alveolar $\dot{V}O_2$ after the onset of exercise was described in terms of an exponential function that was fit to the data with the use of nonlinear regression techniques in which minimizing the sum of squared error was the criterion for convergence. The mathematical model for the alveolar $\dot{V}O_2$ response consisted of three exponential terms, each representing one phase of the response (Fig. 2). The first exponential term started with the onset of exercise (*time* = 0), whereas the other terms began after independent time delays

$$\begin{aligned} \dot{V}O_2(t) = & \dot{V}O_2(b) + A_0*(1 - e^{-t/\tau_0}) \text{ Phase 1 (initial component)} \\ & + A_1*(1 - e^{-(t-TD_1)/\tau_1}) \text{ Phase 2 (primary component)} \quad (I) \\ & + A_2*(1 - e^{-(t-TD_2)/\tau_2}) \text{ Phase 3 (slow component)} \end{aligned}$$

where $\dot{V}O_2(b)$ is the unloaded cycling 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)

$$A'_0 = A_0*(1 - e^{-TD_1/\tau_0})$$

Table 2. Work rate, RPE, net lactate, and parameters of $\dot{V}O_2$ response as functions of pedal rate

	rpm			
	45	60	75	90
BL $\dot{V}O_2$	1.06 ± 0.22	1.03 ± 0.30	1.00 ± 0.24	1.07 ± 0.18
BL WR	50.3 ± 14.5	45.5 ± 16.1	35.7 ± 13.3	16.0 ± 0.0
ΔEE $\dot{V}O_2$	1.89 ± 0.48 ^{a,b}	1.92 ± 0.44 ^{a,b}	1.75 ± 0.40 ^a	1.53 ± 0.37
ΔWR	124.5 ± 28.3	133.5 ± 29.8	128.9 ± 28.8	126.2 ± 28.6
RPE	7.3 ± 1.1 ^a	7.1 ± 1.4 ^a	6.3 ± 1.8 ^d	5.4 ± 1.9
Δlactate	3.7 ± 2.0 ^{a,c}	3.9 ± 1.3 ^{a,c}	3.2 ± 1.3	2.7 ± 1.4
A'_0	0.62 ± 0.24	0.63 ± 0.25	0.58 ± 0.31	0.41 ± 0.21
TD_1	24.8 ± 5.9	25.0 ± 5.6	24.6 ± 8.5	23.2 ± 7.0
A_1	1.49 ± 0.41 ^a	1.54 ± 0.42 ^a	1.47 ± 0.41 ^a	1.20 ± 0.38
G_1	11.9 ± 1.1 ^a	11.5 ± 1.5 ^a	11.4 ± 2.2 ^a	9.4 ± 1.3
τ_1	21.5 ± 8.5	27.7 ± 9.6	32.0 ± 22.0	23.7 ± 8.2
TD_2	118.7 ± 47.3	140.1 ± 55.1	146.9 ± 64.4	120.8 ± 56.9
A_2	0.39 ± 0.22 ^c	0.37 ± 0.21	0.28 ± 0.22	0.32 ± 0.18
τ_2	265 ± 553	178 ± 191	6.7 × 10 ⁵ ± 2.1 × 10 ⁵	1.3 × 10 ⁵ ± 3.1 × 10 ⁵
$A_2/(A_1 + A_2)$	0.21 ± 0.10	0.20 ± 0.11	0.16 ± 0.13	0.22 ± 0.12

Values are means ± SD. rpm, Revolutions/min; BL, baseline; ΔEE $\dot{V}O_2$, increase above baseline in O₂ uptake ($\dot{V}O_2$) at end exercise. WR, work rate; RPE, relative perceived exertion. TD_1 , τ_1 , TD_2 and τ_2 are from Eq. 1; A'_0 , value of initial component at end of *phase 1*; A_1 , sum of A_1 in Eq. 1 and A'_0 (equal to amplitude of primary component); A_2 , value of slow component at end of exercise; $A_2/(A_1 + A_2)$, relative contribution of slow component to net increase in $\dot{V}O_2$ at end-exercise. ^a $P < 0.01$, compared with 90 rpm; ^b $P < 0.01$, compared with 75 rpm; ^c $P < 0.05$, compared with 75 rpm; ^d $P < 0.05$, compared with 90 rpm.

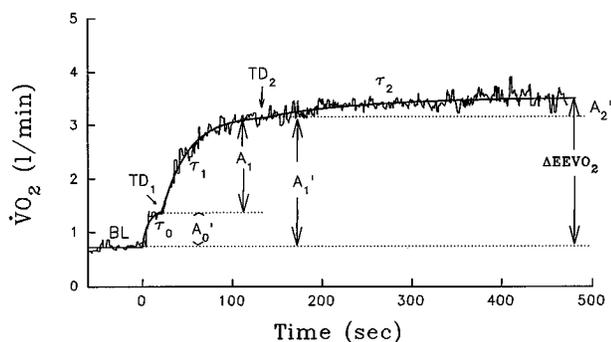


Fig. 2. Schematic showing 3-exponential model used to describe response of $\dot{V}O_2$ during exercise. Parameters correspond to those defined in Eq. 1. $\Delta EE\dot{V}O_2$, increase above baseline of $\dot{V}O_2$ at end exercise; BL, baseline.

The physiologically relevant amplitude of the fast primary exponential component during *phase 2* (A_1') was defined as the sum of $A_0' + A_1$. Because of concerns regarding the validity of using the extrapolated asymptotic value for the slow exponential component (A_2) for comparisons, we used the value of the slow exponential function at the end of exercise, defined as A_2' . In addition, to facilitate comparison across the subjects and different absolute work rates, the gain of the primary response ($G_1 = A_1'/\Delta\text{work rate}$) and end-exercise response [total gain (GT) = $(A_1' + A_2')/\Delta\text{work rate}$] were calculated.

Recovery kinetics for $\dot{V}O_2$ were analyzed with a similar function to Eq. 1, except that after *phase 1* both the primary and slow exponential terms shared the same time delay (TD_1), equivalent to the duration of *phase 1* in recovery. All of the gas exchange responses during recovery exhibited a *phase 1* portion of the response.

Statistical analysis. The effects of pedal frequency on parameters of the $\dot{V}O_2$ gas-exchange response were tested using one-way analysis of variance with repeated measures (pedal frequency) for each parameter. Individual significant differences were then examined post hoc using the Newman-Keuls test. Linear correlation was used to assess the relationships between $\dot{V}O_2$ parameters and end-exercise lactate or %type I fibers; significance was determined from the correlation coefficient. For all tests, significance was declared when $P < 0.05$. Dispersions about the mean are expressed \pm SD unless otherwise specified.

RESULTS

Peak exercise and fiber type. Maximal $\dot{V}O_2$ ($\dot{V}O_{2\max}$) was 3.40 ± 0.52 l/min (48.2 ± 7.1 ml·kg⁻¹·min⁻¹), whereas the estimated LT was 1.72 ± 0.35 l/min, or $\sim 50\%$ $\dot{V}O_{2\max}$ (Table 1). One subject (*subject 5*) did not consent to the muscle biopsy procedure. The mean %type I (slow-twitch) fibers in the rest of the subject group ($n = 9$) averaged $47 \pm 16\%$. As desired, there was a wide range of %type I fibers among subjects (18–67%, Table 1). Percentage of type IIb was low ($3 \pm 3\%$).

Effects of pedal frequency. An example of the $\dot{V}O_2$ response to the two extreme pedal rates (45 and 90 rpm) for one subject is shown in Fig. 3. Characteristics of the exercise protocol across the four pedal frequencies for all subjects are shown in Table 2. As intended, baseline $\dot{V}O_2$ was not significantly different for the four conditions, but the work rate required to produce that $\dot{V}O_2$ progressively increased at the slower pedal frequencies. The initial asymptote for $\dot{V}O_2$ for the 60 rpm study

(as baseline + A_1' in Table 2) represented $50.5 \pm 7.9\%$ Δ, demonstrating that our protocol produced the desired increase in oxidative metabolism. The net end-exercise $\dot{V}O_2$ progressively and significantly fell with increasing rpm; this trend in $\dot{V}O_2$ was accompanied by significantly lower net blood lactate levels and relative perceived exertion at the higher rpm conditions (Table 2).

Important parameters of the $\dot{V}O_2$ kinetic response as a function of pedal frequency are given in Table 2. Neither *phase 1* amplitude (A_0') nor duration (TD_1) was significantly affected by changing pedal rate. The amplitude of the predominant, fast component of $\dot{V}O_2$ in *phase 2*, expressed either as liters per minute (A_1') or as gain ($G_1 = A_1'/\Delta\text{work rate}$) was significantly less for the 90-rpm condition compared with the other rpm studies, but the time constant (τ_1) was not consistently affected. Characteristics of the slow component for $\dot{V}O_2$, as the time of onset (TD_2), amplitude at end-exercise (A_2'), time constant (τ_2), or relative contribution to the overall rise in $\dot{V}O_2$ [$A_2'/(A_1' + A_2')$] were generally not significantly affected by pedal frequency. The one exception was that A_2' at 45 rpm (0.39 ± 0.22 l/min) was significantly greater than that at 75 rpm (0.32 ± 0.18 l/min, $P < 0.05$).

The parameters for the response of $\dot{V}O_2$ during recovery at each pedal rate are given in Table 3. As with the responses during exercise, only the amplitude of the primary response (A_1') varied significantly with pedal rate, decreasing as pedal rate was increased. Furthermore, there was no significant difference between each parameter for the recovery response and its corresponding value for the response during exercise at each pedal rate. In other words, there was symmetry between the exercise and recovery $\dot{V}O_2$ responses at each pedal rate.

Influence of fiber type. There was no significant influence of fiber type on the GT for any of the pedaling rates (r of -0.41 to -0.32 , $P > 0.05$). The relationship between the %type I fibers and four of the relevant parameters of the $\dot{V}O_2$ response for each of the four pedal rates are shown in Fig. 4, and the correlations for each pedal frequency are given in Table 4. The amplitude of the slow component, as absolute liters per minute (A_2'), was not significantly correlated with %type I fibers at any pedal frequency, but when ex-

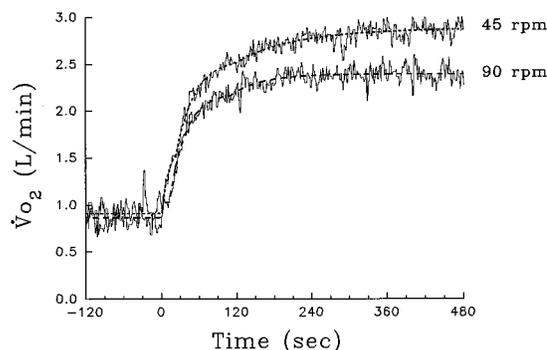


Fig. 3. Example of $\dot{V}O_2$ response for 1 subject performing transitions while pedaling at 2 pedal rate extremes (45 and 90 rpm). Note similar baseline but reduced $\dot{V}O_2$ response at 90 rpm.

Table 3. Parameters of $\dot{V}O_2$ response during recovery

	rpm			
	45	60	75	90
A'_0	0.44 ± 0.26	0.53 ± 0.23	0.53 ± 0.31	0.36 ± 0.17
TD_1	22.2 ± 3.2	22.4 ± 5.0	24.2 ± 5.2	22.2 ± 2.8
A'_1	$1.62 \pm 0.52^*$	$1.56 \pm 0.40^*$	$1.52 \pm 0.53^*$	1.19 ± 0.36
τ_1	21.4 ± 6.2	26.6 ± 9.2	24.3 ± 7.0	23.8 ± 8.0
A_2	0.27 ± 0.16	0.30 ± 0.29	0.18 ± 0.12	0.37 ± 0.27
τ_2	$-3.9 \times 10^7 \pm 1.2 \times 10^8$	$2.4 \times 10^5 \pm 7.1 \times 10^5$	$-1.0 \times 10^7 \pm 5.1 \times 10^7$	$-1.8 \times 10^7 \pm 7.1 \times 10^7$
End Rec $\dot{V}O_2$	1.06 ± 0.26	1.12 ± 0.30	1.06 ± 0.25	1.01 ± 0.22

Values are means \pm SD. End Rec $\dot{V}O_2$, $\dot{V}O_2$ at end of recovery (8 min). * $P < 0.05$, compared with 90 rpm.

pressed as a relative contribution to the overall increase in $\dot{V}O_2$ ($A'_2/A'_1 + A_2$), the slow component was significantly inversely related to the %type I fibers. In other words, the more type I fibers, the smaller was the relative size of the slow component. There was no significant relationship between %type I fibers and the time constant of the fast primary exponential portion of the response in phase 2 (τ_1). However, there was a strong correlation between the gain of the fast component (G_1) and %type I fibers across all pedal frequencies tested. Finally, there was no significant interaction between pedal rate and %type I fibers for any of the parameters shown in Fig. 4 (i.e., slopes were not significantly different).

Role of blood lactate levels. Both the absolute (A_2) and relative amplitudes ($A_2/A'_1 + A_2$) of the slow component were also significantly correlated with the net increase in blood lactate ($r = 0.64-0.84$) (Table 4), but neither the gain nor the time constant for the fast component

was significantly related. Δ Lactate was not related to %type I fibers for any of the pedal frequencies.

Influence of fitness. We also examined the potential influence of fitness or conditioning (as $\dot{V}O_{2max}$ in $ml \cdot kg^{-1} \cdot ml^{-1}$) on various parameters of the $\dot{V}O_2$ response. The only parameters with significant correlations with $\dot{V}O_{2max}$ were the relative amplitude of A'_2 at 90 rpm and the gain for the fast component (G_1) at both 45 and 90 rpm. Both $\dot{V}O_{2max}$ and the estimated LT (in $ml \cdot kg^{-1} \cdot ml^{-1}$) were also significantly correlated with the %type I fibers ($r = 0.74$ and 0.67 , respectively) (Fig. 5). There was no significant relationship between LT as a percentage of $\dot{V}O_{2max}$ and %type I fiber types ($r = 0.06$).

DISCUSSION

These results are consistent with our first hypothesis that the relative contribution of the slow component to

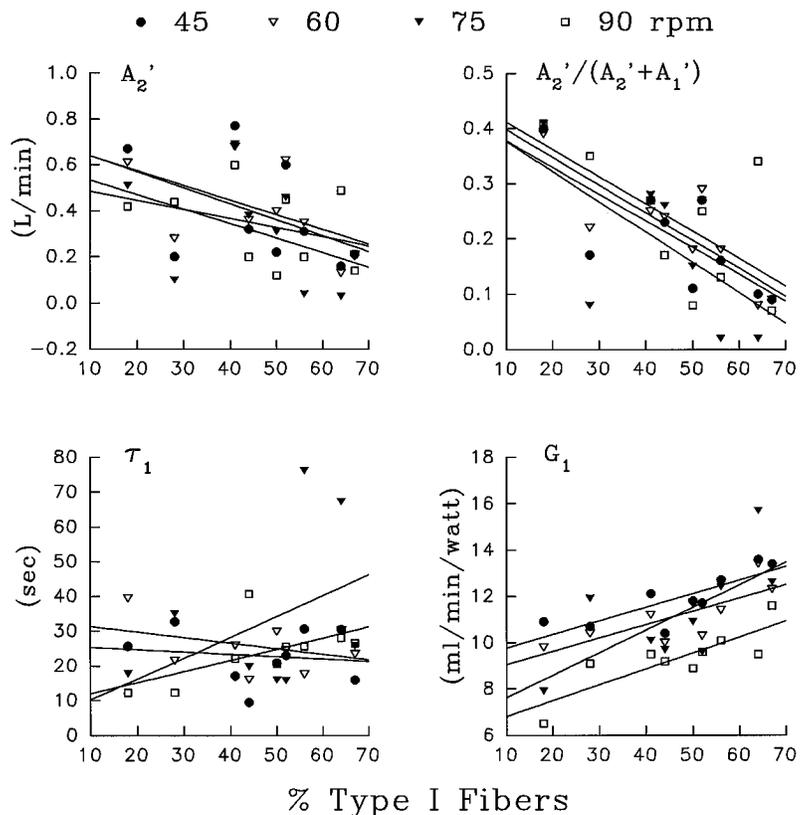


Fig. 4. Absolute (A_2) and relative [$A_2/(A'_1 + A_2)$] amplitude of slow component, time constant for the primary component (τ_1), and gain of the primary $\dot{V}O_2$ component (G_1 ; as A'_1/Δ work rate) as functions of %type I fibers in contracting muscles for each pedal frequency. See Table 4 for individual linear correlation coefficients and significance.

Table 4. Linear correlation coefficients between exercise characteristics, %fiber type, and parameters of $\dot{V}O_2$ kinetics

	rpm			
	45	60	75	90
vs. $\Delta Lactate$				
A'_2	0.76	0.84	0.72	0.64
$A'_2/(A_1 + A_2)$	0.72	0.65	0.67	0.46
G_1	-0.43	-0.54	-0.65	-0.45
τ_1	0.30	-0.23	-0.30	-0.06
vs. %Type I fibers				
A'_2	-0.48	-0.53	-0.44	-0.36
$A'_2/(A_1 + A_2)$	-0.76	-0.83	-0.65	-0.64
G_1	0.82	0.78	0.69	0.83
τ_1	-0.14	-0.36	0.41	0.59
vs. $\dot{V}O_{2max}$				
A'_2	-0.14	-0.18	-0.09	-0.35
$A'_2/(A_1 + A_2)$	-0.50	-0.52	-0.35	-0.65
G_1	0.68	0.48	0.31	0.71
τ_1	-0.22	-0.05	0.07	0.01

$P < 0.05$ for $r \geq 0.621$; $P < 0.025$ for $r \geq 0.707$; $P < 0.01$ for $r \geq 0.789$; $P < 0.005$ for $r \geq 0.834$.

the overall $\dot{V}O_2$ response during heavy exercise would be related to the %type II fibers of the contracting muscles. However, inconsistent with our second hypothesis, the influence of fiber type on $\dot{V}O_2$ kinetics was not enhanced at the slow (45 rpm) or fast (90 rpm) pedal

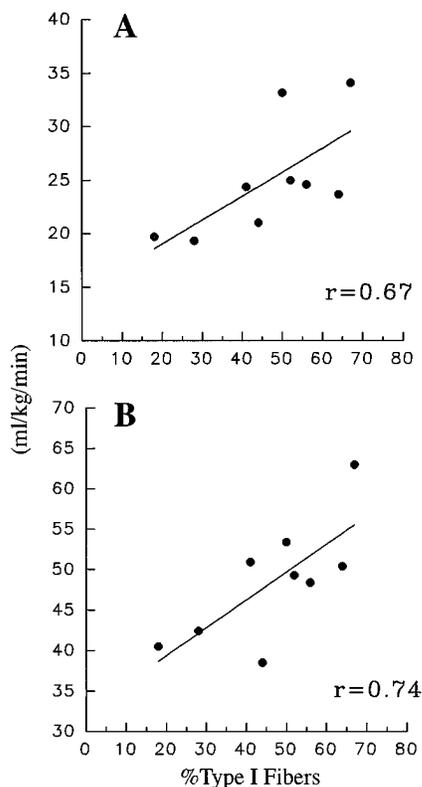


Fig. 5. Estimated lactate threshold (A) and an index of aerobic fitness (B; maximal $\dot{V}O_2$), both expressed in $ml \cdot kg^{-1} \cdot min^{-1}$, as functions of %type I fibers in contracting muscles. Both relationships are significant, $P < 0.05$.

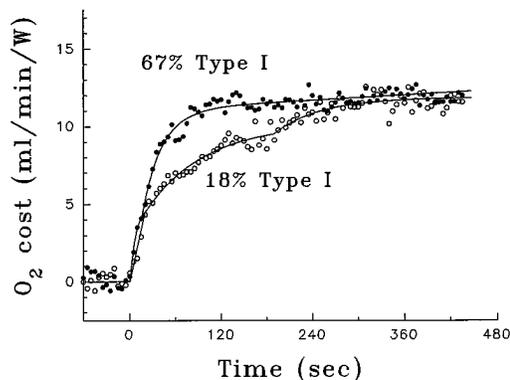


Fig. 6. Summary of observed relationship between muscle fiber type and kinetics of $\dot{V}O_2$ (normalized as O_2 cost) during heavy exercise, shown at 60 rpm for 2 subjects. ●, Subject 10, who had highest %type I fibers (67%); ○, subject 7 with lowest %type I fibers (18%). Compare with hypothesized response in Fig. 1. Note that, unlike the hypothesized relationship, fiber type affects both the primary fast component and the slow, additional component of $\dot{V}O_2$ response. However, by 8 min of exercise, effect of fiber type was no longer discernable.

frequencies but generally was observed equally well (similar correlation coefficients) at all pedal frequencies. Surprisingly, the muscle fiber type distribution also influenced the amplitude of the primary component of the $\dot{V}O_2$ response (A_1).

These results can be summarized in Fig. 6, where the $\dot{V}O_2$ responses at 60 rpm for the two subjects with the greatest and least %type I fibers are shown. In contrast to our hypothesis that only the slow component of the $\dot{V}O_2$ response would be influenced by fiber type (Fig. 1), this influence is expressed in the magnitude of both the fast and slow components. A similar effect of a greater amplitude for the fast component and reduced contribution of the slow component of $\dot{V}O_2$ during heavy exercise can be observed in the data that we have reported for two other conditions, i.e., in adults after endurance exercise training (12) and in children compared with adults (3). The finding of significant correlations between %type I fibers and $\dot{V}O_{2max}$ (Fig. 5) and between $\dot{V}O_{2max}$ and the primary gain G_1 at the slowest and fastest frequencies (Table 4) raises the possibility that differences in aerobic conditioning are a partial explanation for these findings.

It is presently unclear what the independent and dependent influences of muscle fiber type and overall fitness are in determining the metabolic and gas-exchange responses to heavy exercise. Cross-sectional data suggest a positive association between %type I fibers, oxidative capacity, and overall conditioning in adults (14). In a heterogeneous group of volunteers, LT was significantly correlated with %type I fibers, similar to the present findings, whereas both the LT and $\dot{V}O_{2max}$ were significantly related to the respiratory capacity of the contracting muscles (25). Also in one study of well-trained cyclists, LT as a percentage of $\dot{V}O_{2max}$ was also found to be significantly correlated with %type I fibers (15). However, the training response differs somewhat from the representation in Fig. 6, in that the reduction in the slow component after training is greater than the increase in the fast component, so that

the steady-state or end-exercise $\dot{V}O_2$ is lower (12). Conditioning, fiber type distribution, and metabolic responses to exercise are not always codependent, however. The oxidative capacity of the contracting muscle(s) increases with endurance training but with little or no appreciable change in %type I fibers, even when the intensity of the exercise is very heavy (36). In otherwise homogeneous, well-trained cyclists (24), $\dot{V}O_{2\max}$ and LT do not correlate with %type I fibers, yet both gross efficiency (total work/total $\dot{V}O_2$) and net efficiency (Δ work rate/ $\Delta\dot{V}O_2$) are positively correlated with %type I fibers (16, 24). Similarly, a reduced energetic cost of treadmill running with greater %type I fibers has been reported (10). At first glance, our data would appear to be in conflict with these observations, because we saw no significant correlation between the G_T for $\dot{V}O_2$ with fiber type. However, our data describe the responses over the first 8 min only of heavy exercise, whereas the protocols of Coyle et al. (16) either consisted of three 5-min stages of progressive and continuous exercise or of 1 h of maximally sustained work (24). It is possible that the $\dot{V}O_2$ measured at 8 min in our study did not represent an asymptotic value, especially for those responses in which the slow component was more dominant (i.e., in those subjects with greater %type II fibers). In this case, the $\dot{V}O_2$ at 8 min might not reflect differences in $\dot{V}O_2$ that might occur later into exercise.

The data regarding fiber type distribution in children are understandably sparse. In one study of leg muscle (vastus lateralis) in children aged 6 yr, Bell et al. (8) found an average of 59% type I fibers, similar to the distribution for our active adults (subjects 6 and 10 in Table 1) and that reported for endurance-trained athletes (14). Unfortunately, oxidative capacity was not determined in the study of Bell et al. (8). In both children and conditioned adults, the greater $\dot{V}O_2$ early into heavy exercise with less of a slow component is associated with less lactate accumulation (12, 19, 29). This suggests that both of these groups have reduced dependency on anaerobic (glycolytic) energy production during the first minutes of heavy exercise. However, appearance or accumulation of lactate in the blood per se does not necessarily represent recruitment of type II fibers. In the present study, as in that of Dudley et al. (18), there was no correlation between end-exercise lactate levels and %type I fibers. Dudley et al. did find significant correlation between %type II fibers and ammonia levels, which may be a marker of enhanced activity of another pathway for "anaerobic" energy production, the purine nucleotide cycle. This cycle is appreciably active only in type II fibers (28). These results clearly show that fiber type distribution and measures of aerobic conditioning ($\dot{V}O_{2\max}$, LT, muscle oxidative capacity) have both dependent and independent influence on the energetics of heavy exercise.

The potential underlying mechanisms for the slow component of $\dot{V}O_2$ during heavy exercise have recently been discussed at length (31, 43) but remain to be identified with certainty. One point to be remembered when considering possible mechanisms is the delayed

onset of the slow component (2–3 min into exercise) (4, 30). Although the slow component is correlated with blood lactate levels (34, 35), estimates of the rate and energy cost of hepatic glycogen resynthesis from lactate suggest that this pathway could only contribute minimally to the additional $\dot{V}O_2$ (6, 43). Elevated lactate levels per se, whether produced by lactate infusion (32) or by infusion of epinephrine (21), do not increase either isolated-muscle (32) or whole body (21) $\dot{V}O_2$. Proposed extracontracting muscle sources of the additional $\dot{V}O_2$, including increased work of breathing (1), may contribute some metabolism to the slow component, but measurement of leg $\dot{V}O_2$ (33) suggests that most of the $\dot{V}O_2$ comes from the contracting limbs.

Several features of the energetics of type II fibers compared with those of type I fibers make their nomination as the source of the additional $\dot{V}O_2$ seen during heavy exercise reasonable. The time constant for rise in $\dot{V}O_2$ of mouse extensor digitorum longus muscle (virtually all type IIa and IIb fibers) is longer (138 s) than that for soleus muscle (predominantly type I and IIa) (36 s) (17) but is similar to the median time constants seen for the $\dot{V}O_2$ slow component in humans, when that response is appropriately exponential in nature (4). In vitro, type II fibers produce greater heat (50–600% more) and consume more oxygen for the same tension development (17, 22, 42). Calcium pump activity, which is adenosinetriphosphatase dependent, is five- to tenfold greater in type II fibers (22, 42), as is actinomyosin turnover (17). Finally, isolated mitochondria from type II fibers exhibit an 18% lower P/O ratio (44), which would predict a greater $\dot{V}O_2$ for any given ATP resynthesis rate. This difference in P/O ratio may be due to a greater relative reliance on the α -glycerophosphate shuttle over the malate-aspartate shuttle in type II muscle (38).

However, one of the unexpected observations in the present study was that the amplitude of the primary component for $\dot{V}O_2$ (A'_1) was significantly related to fiber type (Fig. 6). This finding has profound significance for interpreting the energetics of heavy exercise. Fundamental to this discussion is whether, at the onset of exercise, A'_1 represents the true initial oxygen cost of the exercise, which then becomes modified over time or, rather, that the delayed steady state represents the real initial oxygen cost that existed from the beginning, and the early responses are attenuated in subjects with increased %type II fibers. The time of onset of the slow component (2–3 min) is much longer than can be attributed to circulatory transit times (5), suggesting that the process(es) responsible for this additional $\dot{V}O_2$ is not a fundamental component of the initial response to exercise. Thus Fig. 6 implies that at the onset of exercise, A'_1 represents the initial target amplitude for the rise in $\dot{V}O_2$. It is presently unclear how differences in fiber type would affect this amplitude. As noted above, type I fibers are more efficient energetically. Thus, for the same ATP turnover rate, one might predict a lower $\dot{V}O_2$ amplitude, not a greater one, for subjects with more type I fibers, as implied by the data of Coyle et al. (16) for long-term exercise. The lower $\dot{V}O_2$

with greater %type II fibers could reflect an initial underestimation in these subjects of the number of motor units necessary to sustain the power output for more than a couple of minutes. The resulting impending fatigue would lead to recruitment of more fibers (slow component), but in this case predominantly type II, with a greater oxygen cost. In this scheme, a subject with greater %type I fibers might better anticipate the number of motor units required to sustain the work rate; in this case, the initial rise in $\dot{V}O_2$ would more closely approximate the "steady-state" $\dot{V}O_2$ necessary to maintain the power output for an extended period of time. Alternatively, if the true oxygen cost of the exercise from the very onset was defined by the eventual steady-state level of $\dot{V}O_2$, then the lower $\dot{V}O_2$ amplitude seen with more type II fibers in the present study could be a consequence of some time-dependent impairment in the ability to increase $\dot{V}O_2$. However, one would expect this to affect the kinetics (time constant) more than the amplitude of the response. Our previous finding of a linear A_1 /work rate relationship (6) suggests that this latter explanation may not be the case. Further work is necessary to delineate these fundamental issues regarding the energetics of exercise.

Another important finding from the present data relevant to this discussion is the symmetry of the kinetic responses of $\dot{V}O_2$ between exercise and recovery. Thus the relative contribution of the slow component to the overall $\dot{V}O_2$ response is retained during recovery, irrespective of the pedal rate. These data suggest that the primary and slow components represent distinct metabolic processes that retain their distinction in recovery. These data differ from those of Paterson and Whipp (30), who found a faster time constant and greater amplitude for the primary component (τ_1), and less of a contribution of the slow component, in recovery from exercise of comparable relative work intensity as in this study. It is currently unclear whether these differences are methodological; exercise in the study of Paterson and Whipp lasted 6 min compared with 8 min in our study, and the analysis used to differentiate the fast and slow components of $\dot{V}O_2$ was also different.

The increase in oxygen cost of unloaded cycling with increasing pedal rate is commonly seen (20, 23). However, the decrease in net end-exercise $\dot{V}O_2$, lactate, and relative perceived exertion at higher pedal rates in the present study suggest that the relative metabolic stress of the exercise was less, despite the Δ work rate being similar. Reduction in the slope of $\Delta\dot{V}O_2/\Delta$ work rate with increasing pedal rates has been seen by some (13, 23) but not all (20) investigators. In the present study, this reduction in $\Delta\dot{V}O_2$ at higher pedal rates was quantitatively similar to the increase in $\dot{V}O_2$ during unloaded cycling; this would predict a similar total (gross) $\dot{V}O_2$ for the same absolute work rate across pedal rates. This similar gross $\dot{V}O_2$ was seen by Ahlquist et al. (2) at 50 and 100 rpm during heavy exercise and is implied in the results of Hagberg et al. (23) up to at least some unspecified rpm. However, both Gaesser and Brooks (20) and Coast and Welch (13) found gross $\dot{V}O_2$ to be increased for the same absolute work rate as pedal

rate increased. The reason for this discrepancy is not readily apparent. One likely mechanism that would reduce $\Delta\dot{V}O_2$ as pedal frequency increases would be a greater contribution of recoil of elastic energy to the overall energetic cost of muscle contraction at higher pedal rates (10). Finally, contrary to our second hypothesis that recruitment of type II motor units would be augmented at slower or faster pedal rates, fiber type distribution did not affect this response, either as net end-exercise $\dot{V}O_2$ or as the fast or slow components (similar slopes in Fig. 4).

In conclusion, fiber type distribution was found to significantly affect the characteristics of the $\dot{V}O_2$ response during heavy exercise. Not only was the slow component of $\dot{V}O_2$ negatively correlated with %type I fibers but the relative contribution of the fast component was positively correlated. The relative contributions of the slow and fast components to the overall $\dot{V}O_2$ response were not affected by pedal rate. The correlation between $\dot{V}O_{2\max}$ and the %type I fibers on the one hand, and the relative contributions of the fast and slow components of $\dot{V}O_2$ at the extremes of pedal frequency on the other, suggest there may be both independent and codependent features of relative fitness (as $\dot{V}O_{2\max}$, ml·kg⁻¹·ml⁻¹) and fiber type distribution on the kinetic responses of $\dot{V}O_2$ to heavy exercise.

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