Longitudinal changes in the kinetic response to heavy-intensity exercise in children

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Fawkner, Samantha G., and Neil Armstrong. Longitudinal changes in the kinetic response to heavy-intensity exercise in children. J Appl Physiol 97: 460–466, 2004. First published March 19, 2004; 10.1152/japplphysiol.00784.2003.—The purpose of this study was to investigate longitudinal changes with age in the kinetic response to cycling at heavy-intensity exercise in boys and girls. Twenty-two prepubertal children (13 male, 9 female) carried out a series of exercise tests on two test occasions with a 2-yr interval. On each test occasion, the subject completed multiple transitions from baseline to exercise tests on two test occasions with a 2-yr interval. Each subject’s breath-by-breath responses were interpolated to 1-s intervals, time aligned, and averaged. The data after phase 1 were fit with 1) a double exponential model and 2) a single exponential model within a fitting window that was previously identified to exclude the slow component. There were no significant differences in the parameters of the primary component between each model. Subsequent analysis was carried out using model 2. The V̇O₂ slow component was computed as the difference between the amplitude of the primary component and the end-exercise V̇O₂ and was expressed as the percent contribution to the total change in V̇O₂. Over the 2-yr period, the primary time constant (boys 16.8 ± 5.3 and 21.7 ± 5.3 s, girls 21.1 ± 8.1 and 26.4 ± 8.4 s, first and second occasion, respectively) and the relative amplitude of the slow component (boys 9.4 ± 4.6 and 13.8 ± 5.3%, girls 10.3 ± 2.4 and 15.5 ± 2.8%, first and second occasion, respectively) significantly increased with no sex differences. The data demonstrate that children do display a slow-component response to exercise and are consistent with an age-dependent change in the muscles’ potential for O₂ utilization.

slow component; age; modeling; confidence intervals

THE OXYGEN UPTAKE (V̇O₂) kinetic responses to both moderate-intensity exercise [below the anaerobic threshold (AT)] and heavy-intensity exercise (above AT and below critical power) are well documented in adults, but data from children are relatively sparse (6, 34). After the onset of exercise, three phases in V̇O₂ dynamics have been identified. The phase 1 or cardiodynamic phase is followed by a primary and rapid exponential process (phase 2), which in the moderate domain achieves a steady state (phase 3) within ~3 min (6, 35). In the heavy-intensity domain, with adult subjects, the third phase is characterized by a delayed rise in V̇O₂ (slow component) that causes V̇O₂ to increase toward an elevated steady state (9). The mechanisms by which this slow component causes an elevation in the O₂ cost of exercise (gain) above that predicted by the subthreshold V̇O₂-work rate relationship remain to be con-
that $\dot{V}O_2$ kinetic responses to heavy-intensity exercise change over a 2-yr period in this group.

METHODS

Subjects. Twenty-two children (15 male, 9 female) were included in the study. Written, informed consent was obtained from subjects and their parents. Ethical approval was granted by the Local Research Ethics Committee. Subjects were not presently undertaking any regular exercise training. Subjects completed all tests on two occasions, separated by a 2-yr interval.

Resting measures. Subjects visited the laboratory on at least four occasions over a 2-wk period, and testing took place at approximately the same time of day for each subject. Stature was measured with a Seca 220 stadiometer (Vogel & Halke, Hamburg, Germany) and body mass was determined by use of Seca electronic scales (Vogel & Halke). Sexual maturity was assessed on the first test occasion only by using the index of pubic hair described by Tanner (30). All observations were made by the same nurse. Exercise was carried out on the same electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, the Netherlands) with the seat height, handlebar height, and crank length adapted to each child and subsequently maintained throughout that testing period. The Lode ergometer was calibrated according to the manufacturer’s recommendations and had a baseline pedaling resistance equivalent to 10 W at 70 rpm.

Measurement of peak $\dot{V}O_2$ and AT. Peak $\dot{V}O_2$ and AT were determined by a ramp test to voluntary exhaustion. During exercise, gas-exchange variables were measured and displayed online by use of an EX670 mass spectrometer and analysis suite (Morgan Medical, Rainham, UK) that was calibrated according to the manufacturer’s instructions. Expired volume was measured by a turbine flowmeter (Interface Associates) with a dead space volume of 90 ml. Volume calibration was achieved by using a handheld calibration syringe (Interface Associates) with a dead space volume of 90 ml. All observations were made by the same nurse. Exercise was carried out on the same electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, the Netherlands) with the seat height, handlebar height, and crank length adapted to each child and subsequently maintained throughout that testing period. The Lode ergometer was calibrated according to the manufacturer’s recommendations and had a baseline pedaling resistance equivalent to 10 W at 70 rpm.

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After a 3-min warm-up of unloaded pedaling, the resistance increased continuously at either 10 or 15 W/min to attain a test $\sim$8–10 min in duration. Subjects pedaled at a cadence of 70 $\pm$ 5 rpm, and the children were actively encouraged to continue until voluntary exhaustion. Maximal effort was considered to have been given if, in addition to subjective indications of intense effort (e.g., excessive hyperpnea, facial flushing, sweating, discomfort), respiratory exchange ratio reached a value $>1.0$. All subjects satisfied these criteria. Peak $\dot{V}O_2$ was taken as the highest recorded 10-s stationary average value during the maximal exercise test.

AT was determined noninvasively by the $V$-slope method as the point at which $CO_2$ production ($\dot{V}CO_2$) began to rise at a more rapid rate than $\dot{V}O_2$ (3, 7). The gas-exchange and ventilatory responses were analyzed with purpose-designed software developed by use of LabVIEW (National Instruments, Newbury, UK). The first 60 s of data after the onset of the exercise and data after the respiratory compensation point were deleted. The respiratory compensation point was recorded as the time at which ventilation ($V_{E}$) began to rise more rapidly than $\dot{V}O_2$ and was determined subjectively from a plot of $V_{E}$ dependent on $\dot{V}O_2$, with data smoothed by use of a 9-s moving average. By using the data from the selected time period only, $V$-slope was determined from a plot of $VCO_2$ dependent on $\dot{V}O_2$. After smoothing the data using a 9-s moving average, we determined the $V$-slope by systematically dissecting the $\dot{V}CO_2/\dot{V}O_2$ data and plotting linear regression lines using all data from either side of that point. The $V$-slope was recorded as the point at which the ratio of the largest standard error of the two lines and the distance from the intersection of the two lines to a single regression line drawn through the data set was minimized. The data and resulting regression lines were displayed graphically, and the experimenter visually confirmed the computer-generated selection of the inflection point.

Constant-work rate exercise tests. On subsequent visits, subjects completed a step-change exercise test that consisted of 6 min of unloaded pedaling, followed instantaneously by a work rate that, from extrapolation from the ramp response, corresponded to 40% of the difference between the $\dot{V}O_2$ at $V$-slope and peak $\dot{V}O_2$ (40% $\Delta\dot{V}O_2$) for 9 min. A pedal cadence of 70 rpm was maintained throughout. Fingertip blood samples were taken immediately after the end of exercise and assayed for blood lactate concentration by use of a whole-blood automated and self-calibrating analyzer (YSI 2300 Stat Plus, Yellow Springs Instruments, Yellow Springs, OH).

A single transition was completed on each visit, and at least three and in most cases four transitions were completed in total. This was the number of transitions required to obtain 95% confidence intervals in the primary time constant of approximately $\pm$ 5 s. The 1-s interpolated responses for each individual to each rest-to-exercise transition were time aligned to the start of exercise and averaged together to form a single data set for analysis.

Kinetic analysis. The duration of phase 1 was estimated from the averaged response profile as the time at which there was a marked inflection point in the $\dot{V}O_2$, $\dot{V}CO_2$, and $V_{E}$ response and change in end-tidal $O_2$, end-tidal $CO_2$, and respiratory exchange ratio from baseline values (35). All data before the end of phase 1 were removed from the data set.

Model 1. A double exponential model (Eq. 1) was applied to the averaged response file, and parameters and their 95% confidence intervals were estimated by least squares nonlinear regression analysis (GraphPad Prism, GraphPad Software, San Diego, CA).

\[
\Delta \dot{V}O_2 = A_1 \cdot (1 - e^{-r_{1}(t)} ) + A_2 \cdot (1 - e^{-r_{2}(t)} ) (t > \text{phase 1})
\]

where $\Delta \dot{V}O_2$ is the increase in $\dot{V}O_2$ at time $t$ above the prior control level, which was calculated as the mean $\dot{V}O_2$ from the last minute of baseline pedaling; $A_1$ and $A_2$, $r_1$ and $r_2$, and $\delta_1$ and $\delta_2$ are the amplitudes, time constants, and independent time delays of each exponential, respectively.

Model 2. Model 2 was designed to remove the possible influence of arbitrarily parametrizing the slow component on the dependent parameters of the primary component. By using a purpose-designed software program developed with LabVIEW (National Instruments, Newbury, UK), a single exponential with a delay term was fit to data after the end of phase 1 (Eq. 2). The fitting window was iteratively widened by 1-s intervals, starting from a 60-s fitting window and finishing with a fitting window that encompassed the entire data set (27).

\[
\Delta \dot{V}O_{2,0} = A_1 \cdot (1 - e^{-r_{1}(t)} ) (t < 0.540 s)
\]

where $\Delta \dot{V}O_{2,0}$ is the increase in $\dot{V}O_2$ at time $t$ above the prior control level, which was calculated as the mean $\dot{V}O_2$ from the last minute of baseline pedaling; $A_1$, $r_1$, and $\delta_1$ are the amplitude, time constant, and time delay.

The estimated time constant ($\tau_1$) for each fitting window was plotted against time. This was to allow for the beginning of the slow component to be determined through visual inspection and therefore to identify the optimal fitting window with which to estimate the parameters of the primary component. The onset of the slow component was defined as the point at which a plateau in the estimated $\tau_1$ was followed by a progressive increase in the estimated $\tau_1$ (Fig. 1). The time constant ($\tau_1$), delay term ($\delta_1$), and amplitude ($A_1$) and their 95% confidence intervals were subsequently estimated using the predetermined fitting window (GraphPad Prism, GraphPad Software, San Diego, CA).

In the case of both models 1 and 2, the confidence interval for $A_1$ was considered as a percentage of $A_1$. The amplitude of the slow component ($A_1$) was calculated as the difference in the mean of the $\dot{V}O_2$ amplitude over the last 30 s of exercise ($\Delta \dot{V}O_2\text{tot}$) and $A_1$. The
slow component was expressed in relative terms as the percentage contribution of \( A_2 \) to \( \Delta V_O_2 \) tot. The functional primary and secondary gain (G_1 and G_2, respectively) were expressed as the \( \Delta V_O_2 \) relative to the change in work rate (G_1, A1/\( \Delta W \); G_2, \( \Delta V_O_2 \) tot/\( \Delta W \)).

Statistical analyses. Peak \( V_O_2 \) was expressed relative to body mass as a ratio (ml·kg\(^{-1} \)·min\(^{-1} \)) and as a power function ratio (ml·kg\(^{-0.71} \)·min\(^{-1} \)). The b exponent represents the gradient of the log\(_e\) peak \( V_O_2 \) (l/min) – log\(_e\) body mass (kg) relationship and was derived by log-linear analysis of covariance. Repeated-measures ANOVA was performed to identify significant changes in response measures between the two models and to test for differences in the slow component after 6 and 9 min of exercise. Repeated-measures ANOVA was performed to test occasions 1 and 2, respectively. Table 3 presents the model parameters using model 2. The mean ± SD confidence intervals for \( A_1 \) and \( \tau_1 \) were 2.7 ± 1.0%, 4.2 ± 1.4 s and 2.9 ± 1.0%, 5.5 ± 2.3 s for test occasions 1 and 2, respectively. There was no significant difference in \( A_1 \) or \( \tau_1 \) between models 1 and 2 and no significant difference between the determination of the onset of the slow component between each analysis method.

Further response parameters were derived after 9 min of exercise by using the parameters estimated from model 2 and are presented in Table 3. There was a significant increase with age in all response variables, apart from \( \delta_1 \) and G_2, and there was a significant reduction in G_1 (Figs. 2 and 3). The contribution of the \( V_O_2 \) slow component to the total change in \( V_O_2 \) after 6 min was boys, 8.4 ± 7.4, 12.3 ± 5.2; and girls, 7.8 ± 3.3, 13.2 ± 4.0%, for test occasions 1 and 2, respectively. The relative magnitude of the slow component was significantly greater after 9 min of exercise than after 6 min on both test occasions. There was no sex interaction or sex difference in any of the variables. Correlation analysis revealed that there was no relationship between the slow component or the gains and peak \( V_O_2 \) or end-exercise blood lactate.

DISCUSSION

We have demonstrated that a slow component of \( V_O_2 \) is apparent in prepubertal children, contributing ~10% of the final \( V_O_2 \) after 9 min of exercise, and that the response to heavy-intensity exercise undergoes changes over the following

Table 1. Physical characteristics and peak exercise responses

<table>
<thead>
<tr>
<th></th>
<th>Test Occasion 1</th>
<th>Test Occasion 2</th>
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<tbody>
<tr>
<td></td>
<td>Boys (n = 13)</td>
<td>Girls (n = 9)</td>
</tr>
<tr>
<td>Age, yr</td>
<td>10.6±0.3‡</td>
<td>10.9±0.2</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>35.0±6.5</td>
<td>38.4±5.8</td>
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<tr>
<td>Stature, m</td>
<td>1.42±0.08</td>
<td>1.45±0.06</td>
</tr>
<tr>
<td>Peak ( V_O_2 ), l/min</td>
<td>1.70±0.27‡</td>
<td>1.47±0.21†</td>
</tr>
<tr>
<td>Peak ( V_O_2 ), ml·kg(^{-1} )·min(^{-1} )</td>
<td>49.0±5.7§</td>
<td>38.9±6.3</td>
</tr>
<tr>
<td>Peak ( V_O_2 ), ml·kg(^{-0.71} )·min(^{-1} )</td>
<td>136.5±14.1§</td>
<td>111.3±15.3</td>
</tr>
<tr>
<td>Peak lactate, mmol/l</td>
<td>6.4±1.8</td>
<td>6.6±1.9</td>
</tr>
<tr>
<td>V-slope, l/min</td>
<td>0.92±0.20</td>
<td>0.79±0.15</td>
</tr>
<tr>
<td>V-slope-( V_O_2 )%</td>
<td>53.7±6.2</td>
<td>53.3±5.0</td>
</tr>
<tr>
<td>( V_O_2 ) 40% ( \Delta, ) l/min</td>
<td>1.23±0.21</td>
<td>1.08±0.15</td>
</tr>
<tr>
<td>( \Delta W, ) W</td>
<td>76±16</td>
<td>66±10</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, number of subjects. Peak \( V_O_2 \), peak oxygen uptake; V-slope-\( V_O_2 \)%, percentage of peak \( V_O_2 \) at which V-slope occurred; \( V_O_2 \) 40% \( \Delta, \) 40% of the difference between peak \( V_O_2 \) and V-slope; \( \Delta W, \) change in exercise intensity (in watts); RM ANOVA, repeated-measures analysis of variance. Significant difference between test occasions, †P < 0.01. Significant interaction between test occasion and sex, P < 0.01. Significant within-test occasion sex differences, ‡P < 0.05, §P < 0.01.
2 yr. This therefore conflicts with previous studies that have suggested that the slow component is negligible in children but supports the view that the magnitude of the slow component is age dependent (1, 36).

Methodological considerations. This study has taken into consideration the 95% confidence intervals with which the nonlinear regression procedure was able to estimate the model parameters. The parameters derived by using the double exponential model (model 1) were not significantly different from those derived by the alternative approach (model 2). However, the wide confidence intervals identified for $\tau_2$ and $A_2$ identify the concern with applying model 1, especially with response patterns that have an inherently low signal-to-noise ratio (i.e., with children). As well, the properties of the nonlinear regression process are such that there is dependence between estimated parameters, and thus it is not advisable to force a model to define data if the physiological properties may not match the model design (such as may be the case when applying an exponential to the slow component). These concerns are illustrated by examining the 95% confidence intervals of the primary component for models 1 and 2. Confidence intervals are estimated by using the error of the entire data set, and the inclusion of “noisy” data during the slow component in model 1 inevitably resulted in wider 95% confidence intervals of the parameters of the primary response ($\tau_1$ and $A_1$). This was not the case in model 2, in which the modeling process focused on data with known exponential properties.

Why we have clearly identified a slow component in children whereas previous authors (1, 36) have not is not readily apparent but might be due to methodological issues. With a group of boys and girls (10.0 ± 2.2 yr), Armon et al. (1) concluded that a single exponential was sufficient to describe children’s Vo2 responses both above and below AT. This was despite finding a positive linear term in 73% of the children’s responses above AT and positive slopes of the regression of Vo2 between the 3rd and 6th minute of exercise in 50% of the children exercising at 50% AT (mean 0.27 ± 0.73 ml·kg⁻¹·min⁻²). To investigate whether the conflicting results could be explained by the length of the exercise periods, we analyzed the slope of the linear regression between the 3rd and 6th minute with the present data. For both the boys and girls collectively, on test occasion 1 when the mean age was similar to the children in the study of Armon et al., the slope was substantially greater than that previously reported (0.54 ± 0.38 ml·kg⁻¹·min⁻²). The slope increased to 0.70 ± 0.31 ml·kg⁻¹·min⁻² on test occasion 2.

Williams et al. (36) reported that, after 6 min of treadmill running, the slow component response in eight 11- to 12-yr-old boys contributed 0.9 ± 1.2% to the total change in Vo2 and that a single exponential model could suitably describe the responses. Our analysis revealed a substantially greater contribution in the present data after 6 min than was reported by Williams et al. and that the relative contribution of the slow component to the total change in Vo2 was significantly greater after 9 min of exercise. Although it is apparent that terminating

Table 2. Model 1 response parameters

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<th>Test Occasion 1</th>
<th>Test Occasion 2</th>
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<tr>
<td></td>
<td>Boys (n = 12)</td>
<td>Girls (n = 8)</td>
</tr>
<tr>
<td>Phase 1, s</td>
<td>*</td>
<td>16.7 ± 3.3</td>
</tr>
<tr>
<td>$\delta_1$, s</td>
<td>†</td>
<td>14.0 ± 4.0</td>
</tr>
<tr>
<td>$\tau_1$, s</td>
<td>†</td>
<td>17.1 ± 6.2</td>
</tr>
<tr>
<td>$A_1$, l/min</td>
<td>†</td>
<td>0.91 ± 0.12</td>
</tr>
<tr>
<td>$\delta_2$, s</td>
<td>†</td>
<td>127.8 ± 37.5</td>
</tr>
<tr>
<td>$\tau_2$, s</td>
<td>†</td>
<td>127.2 ± 60.0</td>
</tr>
<tr>
<td>$A_2$, l/min</td>
<td>†</td>
<td>0.10 ± 0.07</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, number of subjects. $A_1$, $A_2$, amplitude of the primary and secondary components, respectively; $\Delta$Vo2tot, end-exercise $\Delta$Vo2; $\Delta$Vo2tot, total change in $\Delta$Vo2; SC, $A_1$ expressed as a percentage of $\Delta$Vo2tot; EELactate, end-exercise lactate; $G_1$, $O_2$ cost (gain) of the primary component; $G_2$, $O_2$ cost (gain) at the end of exercise; $\Delta G$, additional $O_2$ cost attributable to the slow component ($G_2 - G_1$). Significant difference between test occasions, *P < 0.05, †P < 0.01.

Table 3. Model 2 response parameters and derived parameters

<table>
<thead>
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</tr>
<tr>
<td>$\delta_1$, s</td>
<td>†</td>
<td>13.2 ± 4.9</td>
</tr>
<tr>
<td>$\tau_1$, s</td>
<td>†</td>
<td>16.8 ± 5.3</td>
</tr>
<tr>
<td>$A_1$, l/min</td>
<td>†</td>
<td>0.88 ± 0.15</td>
</tr>
<tr>
<td>$\Delta$Vo2tot, l/min</td>
<td>†</td>
<td>1.33 ± 0.23</td>
</tr>
<tr>
<td>$A_1$, l/min</td>
<td>†</td>
<td>0.97 ± 0.19</td>
</tr>
<tr>
<td>SC, %</td>
<td>†</td>
<td>0.10 ± 0.06</td>
</tr>
<tr>
<td>EELactate, mmol/l</td>
<td>†</td>
<td>9.4 ± 4.6</td>
</tr>
<tr>
<td>$G_1$, ml·min⁻¹·W⁻¹</td>
<td>*</td>
<td>3.8 ± 1.1</td>
</tr>
<tr>
<td>$G_2$, ml·min⁻¹·W⁻¹</td>
<td>†</td>
<td>11.7 ± 1.1</td>
</tr>
<tr>
<td>$G_2$, l/min⁻¹·W⁻¹</td>
<td>†</td>
<td>17.2 ± 1.0</td>
</tr>
<tr>
<td>$G_2$, l/min⁻¹·W⁻¹</td>
<td>†</td>
<td>1.21 ± 0.57</td>
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Values are means ± SD; n, number of subjects. $A_1$, $A_2$, amplitude of the primary and secondary components, respectively; $\Delta$Vo2tot, end-exercise $\Delta$Vo2; total change in $\Delta$Vo2; SC, $A_1$ expressed as a percentage of $\Delta$Vo2tot; EELactate, end-exercise lactate; $G_1$, $O_2$ cost (gain) of the primary component; $G_2$, $O_2$ cost (gain) at the end of exercise; $\Delta G$, additional $O_2$ cost attributable to the slow component ($G_2 - G_1$). Significant difference between test occasions, *P < 0.05, †P < 0.01.
the exercise test after only 6 min excludes some development of the slow component, conflicting conclusions drawn from the present data and from previous studies cannot be explained fully by differences in the period of exercise. Other methodological variance between the studies may be influential, including the ergometer used and the imposed relative exercise intensities.

**Phase 1.** The shorter phase 1 reported presently on the first test occasion supports the work by Hebestreit et al. (14), who reported that a shorter phase 1 (15.3 ± 8.5 s) was found in 9- to 12-yr-old boys compared with men (22.5 ± 6.3 s) exercising at 50% peak V\text{\textsubscript{O2}}. Nevertheless, caution must be taken in interpreting the duration of phase 1 visually because of the limited amount of available data and the breath-by-breath noise that invariably masks any clear changes in ventilatory variables. Further investigation into the properties of phase 1 with children may prove valuable but will require more rigorous technologies and procedures in its determination than have been used previously.

**Primary component.** The higher O\text{\textsubscript{2}} cost of work during the primary component displayed by the younger children is supportive of previous studies that have identified a higher O\text{\textsubscript{2}} cost of exercise above AT in children compared with adults. Zanconato et al. (38) reported that, after 1 min of cycling at 50%Δ, the mean O\text{\textsubscript{2}} cost in 10 children (7–11 yr) was 10.9 ± 2.1 ml O\text{\textsubscript{2}}·min\textsuperscript{-1}·W\textsuperscript{-1} compared with 7.4 ± 1.2 ml O\text{\textsubscript{2}}·min\textsuperscript{-1}·W\textsuperscript{-1} in 13 adults (26–42 yr). Hebestreit et al. (14) observed that after 2 min cycling at 100% peak V\text{\textsubscript{O2}} the O\text{\textsubscript{2}} cost was 10.4 ± 1.4 and 8.3 ± 1.0 ml O\text{\textsubscript{2}}·min\textsuperscript{-1}·W\textsuperscript{-1} in children and adults, respectively. Exercising at 50%Δ the children in the study of Armon et al. (1) demonstrated a mean O\text{\textsubscript{2}} cost after 6 min of cycling of 11.47 ± 1.71 ml O\text{\textsubscript{2}}·min\textsuperscript{-1}·W\textsuperscript{-1} compared with 9.90 ± 0.71 ml O\text{\textsubscript{2}}·min\textsuperscript{-1}·W\textsuperscript{-1} in the adults. These authors noted also that after 6 min of cycling at 75%Δ the O\text{\textsubscript{2}} cost of exercise after 3 min was significantly higher in the children than the adults but that there was no significant difference by the end of the exercise period (12.56 ± 1.33 vs. 11.4 ± 1.30 ml O\text{\textsubscript{2}}·min\textsuperscript{-1}·W\textsuperscript{-1} in children and adults, respectively). Similarly, in the present study, we found a greater primary gain on the first test occasion but no difference between the two occasions in the gain at the end of the exercise period. This is also in accord with the results of Williams et al. (36), who compared boys with men, although in this latter study the V\text{\textsubscript{O2}} was reported in relation to body mass rather than power output, which confounds further interpretation.

The greater O\text{\textsubscript{2}} cost of exercise in younger subjects during the dynamic response has provided other authors with the notion either that the aerobic capacity for exercise is enhanced in children or that they have a lesser ability to generate energy anaerobically (1, 36, 38). However, if the control theories regarding the dynamics of V\text{\textsubscript{O2}} at the onset of exercise are correct, the primary response is considered to depend predominantly on the mitochondrial potential to generate the required ATP for exercise (32). We observed a faster primary time constant (τ\textsubscript{1}) response when the children were younger, which is consistent with studies that have reported a faster kinetic adjustment to exercise above AT in children compared with adults (1, 19, 20, 26, 28, 36). Our laboratory has previously reported a faster τ\textsubscript{1} in children exercising at moderate-intensity exercise compared with adults (8). Together, the data suggest that a developmental influence on the O\text{\textsubscript{2}} utilization potential, possibly a function of mitochondrial enzyme activation or intracellular concentrations of putative metabolic controllers (12, 31, 33), presides over a reduced glycolytic potential. Although there is some evidence that adolescents may have an enzyme profile supportive of a greater rate of pyruvate oxida-
tion than adults (13), there are to the authors’ knowledge no
data monitoring changes in enzyme profiles from prepuber-
cence through to adolescence or adulthood. Of significance
though is that these responses are characteristic of subjects who
differ in the fiber-type profile of the muscle. A greater \( \dot{V}O_2 \) cost
(2, 25) and faster primary component time constant (25) have
been reported in adult subjects with a high ratio of type I to
type II muscle fibers. There is presently limited evidence to
suggest that the fiber-type profile of the muscle changes during
growth and maturation. Some studies have indicated that the
proportion of type I fibers undergoes no change from childhood
through to adulthood (4, 22), whereas Lexell et al. (17) have
identified a reduction in the percentage of type I fibers in
subjects ranging from age 5 (69%) to age 15 (58%) and age 35
(45%).

There is also evidence that \( \dot{O}_2 \) delivery (muscle blood flow
per unit tissue) may decrease from the ages of 12 to 14 yr (15).
The extent to which \( \dot{O}_2 \) delivery limits the phase 2 response to
heavy-intensity exercise is clouded by contradictory literature
(5, 10, 11, 16, 18, 29). Therefore, whether these results are
indicative of a change in the fiber-type profile of the muscle
after the prepubertal period and/or the properties of those fibers
and delivery of \( \dot{O}_2 \) remains to be elicited. It should be noted,
however, that although this is the first study to report the
confidence intervals for \( \tau_1 \) with children during heavy-intensity
exercise, we do not suggest that these are suitably tight to draw
any robust conclusions.

**\( \dot{V}O_2 \) slow component.** A greater potential for \( \dot{O}_2 \) utilization or
\( \dot{O}_2 \) delivery during the primary phase in the subjects on the first
test occasion is consistent with their subsequent response
characteristics and the magnitude of the slow component of
\( \dot{V}O_2 \). Although there have been a number of possible explana-
tions for the slow component [see Gaesser and Poole (9) for a
discussion], work by Poole et al. (23) has indicated that \( \sim 86\% \)
of the additional \( \dot{V}O_2 \) originates from the exercising muscle,
and therefore it is probable that the changes observed reside
here. It is likely that this greater \( \dot{O}_2 \) cost of exercise manifested
as the slow component is likely to be due to an increase in the
phosphat cost of generating muscular force rather than the \( \dot{O}_2 \)
cost of phosphate production (27). This may result from the
recruitment of low oxidatively efficient fibers (type II) or the
progressive recruitment of a greater number of high oxidatively
efficient fibers. The smaller slow component that was demon-
strated in the younger years resulted in an elevated final \( \dot{O}_2 

\) gain that was equal on both test occasions, and this correlates
closely with the pattern of comparison between high and low
percentages of type I fibers with adults (2, 25). Hence changes
in fiber-type recruitment may have contributed to the increases
in the amplitude of the slow component with age.

It has been suggested that the primary amplitude represents
the projected \( \dot{V}O_2 \) for the exercise task, and the slow compo-
nent reflects a metabolic process that is additive (29). However,
contrary to this, the \( \dot{O}_2 \) cost by the end of the exercise period
on both test occasions was equal, as has been found previously
with adult experimental groups (2, 16, 25) and between chil-
dren and adults (1, 36). This may suggest that the phosphate
turnover required to maintain heavy-intensity exercise was in
fact independent of age and that in the older children a lesser
proportion of the required \( \dot{O}_2 \) for the given exercise intensity
was achieved in the primary phase. In this sense, the slow
component acts as a “catch-up” mechanism during which

**equilibrium in \( \dot{O}_2 \) delivery and fiber recruitment and efficiency
is achieved.**

Such a system may suggest that the higher blood lactates
achieved in the older children are indicative of generating a
lesser proportion of the required \( \dot{O}_2 \) aerobically during the
primary phase and presumably during this catch-up phase.
However, because there was no correlation between end-
exercise blood lactates and the gain of either the primary phase
or the slow component, the relationship between these re-
spose variables and blood lactate remains elusive, as has been
indicated previously with both children and adults (21, 36, 37).

We found that, consistent with the literature, the peak \( \dot{V}O_2 

\) of
children increased over the 2-yr interval, and the magnitude of
the slow component also increased. There were significant sex
differences in peak \( \dot{V}O_2 \) when scaled to body mass on each
occasion, but no sex difference in the slow component or any
relationship between peak \( \dot{V}O_2 \) and the slow component. These
results support the work of Obert et al. (21), who reported that
there was no difference in the magnitude of the slow compo-
nent (after 90% maximal aerobic power until exhaustion)
between 12 trained and 12 untrained children. Hence it seems
that a relationship between the slow component and peak \( \dot{V}O_2 

\) is tenuous in children.

Interestingly, we found no significant sex differences in any
of the response parameters to the heavy-intensity exercise. This
was surprising, because earlier work from our laboratory iden-
tified longer primary time constants and larger slow compo-
nents in girls compared with boys (6). Although the present
data indicate sex differences in these directions, it is likely that
the small sample sizes and large standard deviations of the
response variables precluded statistical significance. Classifi-
cation of sex differences in the response to heavy-intensity
exercise will require further investigation with larger sample
sizes and stringent methodological rigor.

Unfortunately, although we were able to assess pubic hair at
the first test occasion, because of the prevailing sociological
climate regarding such screening we were unable to repeat this
procedure on the subsequent occasion. The interaction between
test occasion and sex for stature suggests that some of the girls
had initiated their growth spurt by test occasion 2, but this does
not appear to have impacted on any of the measured responses
to heavy-intensity exercise, which did not evidence any sex
interaction on either occasion. It is possible that maturational
changes have an independent influence over and above chro-
nological age, but further investigation is restricted by the
present difficulty in the rigorous assessment of changes in
maturational status.

In conclusion, this is the first study that has attempted to
investigate longitudinally the influence of age on the slow
component and kinetic response to exercise in children. We
have clearly identified that a slow component does exist in both
male and female prepubertal children and that its magnitude
increases over the subsequent 2-yr period independent of peak
\( \dot{V}O_2 \). The increase in the slow component is associated with a
reduction in the relative amplitude of the primary phase and a
slowing of the primary kinetics, and the entire response profile
appears to project toward a predetermined \( \dot{O}_2 \) cost of the
relative exercise intensity independent of age. These results are
consistent with an age-dependent change in the muscles’ po-
tential for \( \dot{O}_2 \) utilization.
GRANTS

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REFERENCES


