Oxygen uptake kinetics during treadmill running in boys and men

CRAIG A. WILLIAMS,1 HELEN CARTER,2 ANDREW M. JONES,3 AND JONATHAN H. DOUST2
1University of Brighton, Chelsea School Research Centre, Eastbourne BN20 7SP; 2University of Surrey Roehampton, London SW15 3SN; and 3Exercise Physiology Group, Manchester Metropolitan University, Alsager ST7 2HL, United Kingdom

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Williams, Craig A., Helen Carter, Andrew M. Jones, and Jonathan H. Doust. Oxygen uptake kinetics during treadmill running in boys and men. J Appl Physiol 90: 1700–1706, 2001.—The purpose of this study was to compare the kinetics of the oxygen uptake (VO2) response of boys to men during treadmill running using a three-phase exponential modeling procedure. Eight boys (11–12 yr) and eight men (21–36 yr) completed an incremental treadmill test to determine lactate threshold (LT) and maximum VO2. Subsequently, the subjects exercised for 6 min at two different running speeds corresponding to 80% of VO2 at LT (moderate exercise) and 50% of the difference between VO2 at LT and maximum VO2 (heavy exercise). For moderate exercise, the time constant for the primary response was not significantly different between boys (10.2 ± 1.0 (SE) s) and men (14.7 ± 2.8 s). The gain of the primary response was significantly greater in boys than men (239.1 ± 7.5 vs. 167.7 ± 5.4 ml·kg−1·km−1; P < 0.05). For heavy exercise, the VO2 on-kinetics were significantly faster in boys than men (primary response time constant = 14.9 ± 1.1 vs. 19.0 ± 1.6 s; P < 0.05), and the primary gain was significantly greater in boys than men (209.8 ± 4.3 vs. 167.2 ± 4.6 ml·kg−1·km−1; P < 0.05). The amplitude of the VO2 slow component was significantly smaller in boys than men (19 ± 19 vs. 289 ± 40 ml/min; P < 0.05). The VO2 responses at the onset of moderate and heavy treadmill exercise are different between boys and men, with a tendency for boys to have faster on-kinetics and a greater initial increase in VO2 for a given increase in running speed.

Oxygen uptake slow component; mathematical modeling; children

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might provide some insight into the control of the primary and SC responses (8, 19).

Therefore, the aim of the present study was to compare the VO2 response of boys to men during treadmill running of moderate- and heavy-exercise intensity using the three-phase exponential modeling procedures of Barstow et al. (5). We hypothesized that the VO2 kinetic responses in children would be faster than in adults during treadmill running because of differences in oxygen transport chain dimensions. We further hypothesized that the oxygen cost of exercise would be higher in children because of a reduced anaerobic energy contribution to the total ATP yield.

METHODS

Eight boys [age: 12 ± 0.2 (SD) yr, body mass: 43.5 ± 6.8 kg] and eight men [age: 30 ± 7.3 yr, body mass: 75.0 ± 5.9 kg] volunteered to participate in the study. After the experimental procedures and the associated risks and benefits of participation were explained, written informed consent was obtained from the adult men, the boys’ parents, and the boys. The study was approved by the University of Brighton Ethics Committee. Before testing, it was ensured that all subjects were fully familiar with laboratory exercise testing procedures.

The 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. Subjects wore the same running shoes and lightweight running kit for all tests. 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.

Experimental design. The subjects were required to visit the laboratory on four occasions. The first visit was used to determine the LT and the VO2 max. During the remaining sessions, the subjects performed two to four repetitions of square-wave transitions from rest to one of two exercise intensities: 80% LT and 50% of the difference in VO2 between LT and VO2 max (50%Δ). On a given day, a subject would complete two or three transitions of the same exercise intensity. The transitions were separated by 1 h of recovery. The transitions performed on a given day were determined at random, and the study was completed within 2 wk for all subjects.

Procedures. All tests were performed on a motorized treadmill (Woodway, Cardiokinetics, Salford, UK) with the grade set at 1% (18). 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 mm H2O L⁻¹ s⁻¹) 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 analyzed for O2, CO2, and N2 concentrations by a quadrupole mass spectrometer (CaSE QP9000, Gillingham, Kent, UK), which was calibrated before each test using 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. Respiratory gas-exchange variables (VO2, CO2 production, minute ventilation) were calculated and displayed for every breath. Heart rate was recorded telemetrically throughout the exercise tests (Polar Electro Oy, Kempele, Finland).

Subjects performed incremental exercise to volitional exhaustion to determine LT and VO2 max during treadmill running. For the test, the initial running speed was 5.0–6.0 km/h for the boys and 8.0–9.0 km/h for the men. Subjects completed six to eight submaximal stages of 3-min duration, with running speed increased by 1.0 km/h between stages. At the end of each stage, the subjects supported their weight with their hands and moved their feet to the sides of the treadmill belt. Fingertip capillary blood samples (~25 µl) were collected in capillary tubes and subsequently analyzed for lactate concentration using an automated analyzer (YSI 2300, Yellow Springs Instruments). All subjects recommended running within 15–20 s. When heart rate exceeded 90% of the known or age-predicted maximum heart rate, the running speed was maintained, and the treadmill grade was increased by 1% per minute until the subject reached volitional exhaustion.

Plots of blood lactate against running speed and VO2 were provided to two independent reviewers who determined the LT as the first sudden and sustained increase in blood lactate above resting concentrations. The breath-by-breath gas exchange data collected during the incremental tests were averaged over consecutive 30-s periods. The VO2 max was defined as the average VO2 attained in the last 30 s of the tests. The running speed at VO2 max was estimated by extrapolation of the sub-LT relationship between VO2 and running speed. The running speeds calculated to require 80% of the VO2 at LT (moderate-intensity exercise) and 50%Δ (heavy-intensity exercise) were determined [equal to LT + 0.5 × (VO2 max – LT)].

Subsequently, subjects performed a series of square-wave transitions of 6-min duration at the two exercise intensities on separate days. The exercise protocol began with 2 min of standing rest with feet astride the moving treadmill belt and hands holding the treadmill guard rails. At the start of exercise, the subjects supported their body mass with their hands on the guard rails until their leg speed matched treadmill belt speed, after which they let go of the guard rails and began running. The transition from rest to exercise took 2–4 s. This rapid transition would have had a negligible effect on our kinetic analysis because this was contained within the cardiodynamic phase of the gas-exchange response to exercise. Fingertip capillary blood samples were taken immediately before and after the 6-min exercise period. The difference between the end-exercise lactate and the resting lactate concentration was expressed as a delta value (Δ[ lactate]). After a 1-h recovery period, a further blood sample was taken to ensure that blood lactate had returned to resting levels. The subjects then performed an identical square-wave transition. For the moderate-exercise trial (80% LT), the subjects performed a total of four transitions, whereas for the heavy-exercise trials (50%Δ), the subjects performed two transitions.

Data analysis. For each exercise transition, the breath-by-breath data were interpolated to give second-by-second values. The transitions for each intensity were then time aligned to the start of exercise and averaged to enhance the underlying response characteristics. Nonlinear regression techniques were used to fit the VO2 data after the onset of exercise with an exponential function. An iterative process ensured the sum of squared error was minimized. The math-
the descriptive data and the \( \dot{V}O_2 \) responses of the boys used to determine the significance of differences between pressed relative to body mass) for the two exercise intensities.

\[
\dot{V}O_2(t) = \dot{V}O_2(b) + A_c (1 - e^{t/\tau_c}) + A_p [1 - e^{(t - TD_p)/\tau_p}] + A_s [1 - e^{(t - TD_s)/\tau_s}]
\]

where \( \dot{V}O_2(b) \) is the resting baseline value; \( A_c, A_p, \) and \( A_s \) are the asymptotic amplitudes for the three exponential terms: cardiodynamic, primary, and slow components, respectively; \( \tau_c, \tau_p, \) and \( \tau_s \) are the time constants of the cardiodynamic, primary, and slow components, respectively; and \( TD_p \) and \( TD_s \) are the time delays of the primary and slow components, respectively. The phase 1 term was terminated at the start of phase 2 (i.e., at \( TD_p \)) and assigned the value for that time (\( A_c \))

\[
A_c = A_c * (1 - e^{-TD_p/\tau_c})
\]

The \( \dot{V}O_2 \) at the end of phase 1 (\( A_c \)) and the amplitude of phase 2 (\( A_p \)) were summed to calculate the amplitude at the end of the primary component (\( A_{c+p} \)). The SC at the end of exercise was calculated and is used in preference over the asymptotic value, which can lie beyond physiological limits. The gain of the primary component (\( G_{c+p}; A_{c+p}/\)running speed expressed relative to body mass) for the two exercise intensities was also calculated.

Statistical analysis. Independent sample \( t \)-tests were used to determine the significance of differences between the descriptive data and the \( \dot{V}O_2 \) responses of the boys and men. Pearson product-moment coefficients were used to assess the significance of relationships between the SC, TD, and the increase in blood lactate. Statistical significance was accepted at \( P < 0.05 \). Results are presented as means ± SE.

### Results

The \( \dot{V}O_2\max \) expressed relative to body mass in boys (52.1 ± 1.7 ml·kg\(^{-1} \cdot \text{min}^{-1} \)) was not statistically different from that in men (56.6 ± 3.0 ml·kg\(^{-1} \cdot \text{min}^{-1} \); \( t_{15} = 1.3, P = 0.22 \)). Although the \( \dot{V}O_2 \) at LT was significantly higher in the men than in the boys (42.3 ± 2.5 vs. 36.1 ± 1.4 ml·kg\(^{-1} \cdot \text{min}^{-1} \); \( t_{15} = 8.8, P < 0.001 \)), the percentage of \( \dot{V}O_2\max \) at which LT occurred was similar (74.8 ± 2.5 vs. 69.2 ± 1.7%; \( t_{15} = 1.8, P = 0.09 \)). The protocol was successful in ensuring that the subject groups exercised at the same relative intensities during both moderate (79.5 ± 0.7 and 84.2 ± 1.9% of LT for men and boys, respectively) and heavy exercise (43.3 ± 6.6 and 43.0 ± 4.7% for men and boys, respectively).

Table 1 shows the parameters from the modeling of the \( \dot{V}O_2 \) response to both exercise intensities in the boys and men. Heart rate and blood lactate concentrations are also presented. The \( \dot{V}O_2 \) data from a typical child and adult are shown in Fig. 1.

### Table 1. Parameters of the \( \dot{V}O_2 \) response during moderate and heavy exercise in boys and men

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Moderate Exercise</th>
<th>Heavy Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Boys</td>
<td>Men</td>
</tr>
<tr>
<td>Running speed, km/h</td>
<td>6.1 ± 0.6</td>
<td>9.9 ± 1.4*</td>
</tr>
<tr>
<td>BL ( \dot{V}O_2 ), ml·min</td>
<td>258.0 ± 24.4</td>
<td>418.3 ± 26.9*</td>
</tr>
<tr>
<td>( A_c ), ml·min</td>
<td>593.8 ± 46.5</td>
<td>901.3 ± 136*</td>
</tr>
<tr>
<td>( \tau_c ), s</td>
<td>5.3 ± 1.3</td>
<td>7.8 ± 3.0</td>
</tr>
<tr>
<td>( TD_p ), s</td>
<td>24.7 ± 2.6</td>
<td>26.8 ± 3.3</td>
</tr>
<tr>
<td>( A_{c+p} ), ml·min</td>
<td>1,047.3 ± 52.3</td>
<td>2,084.6 ± 138.6*</td>
</tr>
<tr>
<td>( \tau_p ), s</td>
<td>10.2 ± 1.0</td>
<td>14.7 ± 2.8</td>
</tr>
<tr>
<td>( G_{c+p} ), ml·kg(^{-1} \cdot \text{km}^{-1} )</td>
<td>239.1 ± 7.5</td>
<td>167.7 ± 5.4*</td>
</tr>
<tr>
<td>MRT, s</td>
<td>19.77 ± 2.0</td>
<td>27.16 ± 2.4*</td>
</tr>
<tr>
<td>( A_s ), ml·min</td>
<td>122.2 ± 7.2</td>
<td>115.9 ± 7.0</td>
</tr>
<tr>
<td>Relative ( A_c ) %</td>
<td>18.6 ± 18.9</td>
<td>288.5 ± 39.7*</td>
</tr>
<tr>
<td>( \tau_s ), s</td>
<td>0.9 ± 1.2</td>
<td>8.3 ± 1.0</td>
</tr>
<tr>
<td>( G_s ), ml·kg(^{-1} \cdot \text{km}^{-1} )</td>
<td>218.2 ± 24.7</td>
<td>254.3 ± 31.2</td>
</tr>
<tr>
<td>EE HR, beats/min</td>
<td>147 ± 3</td>
<td>123 ± 7*</td>
</tr>
<tr>
<td>( \Delta [\text{lactate}] ), mM</td>
<td>0.2 ± 0.2</td>
<td>-0.1 ± 0.2</td>
</tr>
</tbody>
</table>

Values are means ± SE. BL, baseline; \( \dot{V}O_2 \), \( O_2 \) uptake; \( A_c \), amplitude at end of the cardiodynamic phase; \( \tau_c \), time constant for cardiodynamic component; \( TD_p \), time delay for primary component; \( A_{c+p} \), sum of amplitude for cardiodynamic component (\( A_c \)) in Eq. 1 and amplitude at the end of the primary component (\( A_p \)); \( \tau_p \), time constant for primary component; \( G_{c+p} \), increase in \( \dot{V}O_2 \) above baseline at the end of the primary component relative to body mass and running speed; MRT, mean response time, calculated as the weighted sum of all 3 phases, and represents the time taken to attain 63% of the overall \( \dot{V}O_2 \) response; \( TD_s \), time constant of the slow component; \( A_s \), amplitude of slow component at end of exercise; relative \( A_c \) (\( A_c/A_{c+p} + A_s \)), relative contribution of slow component to net increase in \( \dot{V}O_2 \) at end exercise; \( \tau_s \), time constant of slow component; \( G_s \), increase in \( \dot{V}O_2 \) above baseline at end of exercise relative to body mass and running speed; EE HR, heart rate at end of exercise; \( \Delta [\text{lactate}] \), difference between end-exercise lactate and resting lactate concentration. \( TD_p \), \( \tau_p \), \( TD_s \), and \( \tau_s \) are from Eq. 1. Significantly different from value in boys, * \( P < 0.05 \).
As would be expected because of the differences in running speed between the two groups, $A_{c+p}$ was significantly higher in the adult group for both moderate exercise ($2,084.6 \pm 138.6$ vs. $1,047.3 \pm 52.3$ ml/min; $t_{15} = 7.0, P < 0.001$) and heavy exercise ($3,193.6 \pm 174$ vs. $1,546.5 \pm 79.5$ ml/min; $t_{15} = 8.6, P < 0.001$). However, when both the running speed and the body mass of each subject were taken into account, $G_{c+p}$ was significantly higher in boys during both moderate exercise ($239.1 \pm 7.5$ vs. $167.7 \pm 5.4$ ml·kg$^{-1}$·km$^{-1}$; $t_{15} = 3.6, P = 0.003$) and heavy exercise ($209.8 \pm 4.3$ vs. $167.2 \pm 4.6$ ml·kg$^{-1}$·km$^{-1}$; $t_{15} = 5.5, P < 0.001$). Although in the adults the $G_{c+p}$ was similar across the two intensities, in the boys the $G_{c+p}$ was significantly higher in moderate exercise compared with heavy exercise ($t_{15} = 4.4, P < 0.001$). Figure 2 shows the responses in a typical child and a typical adult subject to heavy exercise.

The $\tau_p$ tended to be faster in boys for moderate exercise ($10.2 \pm 1.0$ vs. $14.7 \pm 2.8; t_{15} = 1.5, P = 0.15$) and was significantly faster for heavy exercise ($14.9 \pm 1.1$ vs. $19.0 \pm 1.6; t_{15} = 2.2, P = 0.001$). There was a tendency for $\tau_p$ to be increased for heavy compared with moderate exercise in both boys and men, but this was not significant (see Table 1).

The three-phase model describing the $\bar{V}_O_2$ data in the boys’ heavy-exercise bouts yielded very small SCs ($18.6 \pm 18.9$ ml/min). In contrast, the adult group exhibited a substantial SC during heavy exercise ($288.5 \pm 39.7$ ml/min). The SC was found to be significantly different between the boys and the men ($t_{15} = 6.1, P < 0.01$). The SC contributed ~8% to the total end-exercise $\bar{V}_O_2$ for men compared with only 1% in total for the boys. There was no correlation found between $\Delta$[lactate] and the SC in adults ($r = 0.3, P > 0.05$) or children ($r = 0.2, P > 0.05$).

Fitting the boys’ heavy exercise data with both a two-phase and a three-phase model for heavy exercise revealed no significant differences in the size of the residuals for each fit ($t_7 = 1.66, P = 0.14$). This would suggest that the simpler two-phase model may be more appropriate where there is a negligible SC, for example, in children (22).

**DISCUSSION**

To our knowledge, this is the first study that has investigated $\bar{V}_O_2$ kinetics in children and adults during treadmill running. These data have shown that the boys differ from the men in their $\bar{V}_O_2$ response to moderate and heavy exercise during treadmill running. We observed a significantly higher $G_{c+p}$ in boys for both moderate and heavy exercise. In addition, significantly faster kinetics were observed for boys compared with men for heavy exercise. The results of this study also demonstrate a negligible SC in boys.
compared with men, supporting previous findings in cycle ergometry (1).

For moderate exercise, the $\tau_p$ tended to be faster in boys than men, but this difference was not significant. Previous investigators using moderate exercise intensities have also found that the time constants for exercise below LT were faster in younger children (5 boys and 5 girls, 7–10 yr) than teenagers (5 boys and 5 girls, 15–18 yr) but not significantly so (10). Cooper and Barstow (9) used their findings to suggest that the dynamic $\text{VO}_2$ response from rest to exercise was independent of size and age during growth. In contrast, Macek and Vavra (21) found that 10- to 11-yr-old boys had a faster increase in exercise $\text{VO}_2$ than did 20- to 22-yr-old men. Armon et al. (1) also found significantly faster time constants for work rates at 80% LT in children (12 girls and 10 boys, age 6–12 yr) compared with adults (7 men, age 27–40 yr). In their study, with the use of cycle ergometry at 80% LT, the time constant of $\text{VO}_2$ for children and adults was 26 ± 8 and 44 ± 7 s, respectively.

For heavy exercise, $\tau_p$ was significantly faster for boys than men. Our findings are supportive of those of Armon et al. (1), who found significantly faster time constants during cycling exercise at 25, 50, and 75% $\Delta$ in children compared with adults. Armon et al. suggested that the faster kinetics during high-intensity exercise could be linked to a blunted anaerobic response in the initial stages of exercise in children compared with adults. Also, the difference in the time constant has been suggested to be a function of child and adult differences in hemoglobin concentration, capillary density, mitochondrial density, and oxidative enzyme activity. In aerobic training studies in adults, the $\text{VO}_2$ kinetics have been shown to be faster after training, partly as a result of increases in mitochondrial density and capillarity (14, 25). In contrast to the reports of faster time constants in children, Hebestreit et al. (16) found no significant differences in $\tau_p$ for boys and men during cycle exercise at 50, 100, or 130% $\text{VO}_2$ peak. However, this study did not objectively determine phase I, and no exercise bouts were conducted at an intensity that induced a SC and allowed ~6 min of exercise. Therefore, the selected exercise duration was too short to fully determine the $\text{VO}_2$ kinetic differences between men and boys.

It is possible that differences in the ages of the subjects and methods of data analysis can account for the equivocal research findings relating to the time constants for moderate- and heavy-exercise intensities. In the study of Macek and Vavra (21), a single-exponential equation was used to model the entire $\text{VO}_2$ response, whereas in the study by Armon et al. (1), a linear model starting at time 0 was chosen. It has been established that the SC emerges after a discernible time delay and that accurate interpretation of the $\text{VO}_2$ response to exercise requires the primary response to be distinguished from the SC response (4).

To account for body mass and running speed differences between the two groups, the $G_{c+p}$ was calculated.

The $G_{c+p}$ in the boys were found to be significantly higher than in the men during moderate and heavy exercise. These results confirm those reported by Armon et al. (1), who found higher oxygen costs in boys compared with men at all work rates during cycle ergometry. Similarly, in a study by Hebestreit et al. (16), significant differences were found between boys’ and men’s oxygen cost at 50 and 100% $\text{VO}_2$ peak. There are a number of possible mechanisms to explain the higher oxygen cost of exercise in boys than in men. Biomechanical analyses of running have revealed that children, who have a shorter stride length, have to run at a higher stride frequency to achieve the same running speed as adults, thereby increasing the total work output at any speed (11). It has also been suggested that the smaller body mass of children elicits less of an elastic energy return. More recently, it has been shown that the $G_{c+p}$ during heavy, constant-load exercise (5) and the $\text{VO}_2$-work rate slope during ramp exercise (6) are positively related to endurance fitness and percentage of type I fibers, and it could, therefore, be speculated that the differences between boys and men may be related to differences in muscle morphology and/or motor unit recruitment patterns. Differences in muscle metabolism between children and adults, including reduced glycolytic enzyme profiles and lower ratios of glycolytic to oxidative enzymes in the skeletal muscle of children, may enable children to meet a greater proportion of the total energy demand through aerobic pathways. Alternatively, it is possible that the child and adult differences arise because of the limited ability of children to generate ATP through anaerobic metabolism. Children have been found to have a lower anaerobic performance as measured during the Wingate test (2) and other tests of anaerobic capacity (7) compared with adults. Although a number of explanations have been suggested to explain the lower lactate values in children, including an age-dependent rise in the lactate-to-pyruvate ratio (26) and a lower concentration and rate of utilization of glycogen (17, 32), the balance of evidence suggests that the glycolytic activity is not fully developed in childhood. Results from magnetic resonance spectroscopy studies have found higher intramuscular pH and lower P$_i$-to-phosphocreatine ratios in children compared with adults during exercise (20, 31).

Interestingly, the $G_{c+p}$ in boys was significantly higher during moderate exercise than during heavy exercise. It is possible that the calculated intensity domain for moderate exercise was not conducive to an economical running style, as the speed was too slow and a jogging style had to be adopted. It is possible that the faster speed during heavy exercise was, therefore, more economical and thus resulted in a lower $G_{c+p}$.

Only two studies that have investigated the $\text{VO}_2$ SC in children could be found (1, 23). Armon et al. (1) observed that, at the 50% $\Delta$ work intensity, only 11 of the 22 children demonstrated a SC. The results from the present study confirm the lack of a SC during treadmill running in boys, as previously found in cycling. The SC contributed a greater amount to the end-exercise $\text{VO}_2$ in men than boys. In contrast, Obert
et al. (23) found a SC during high-intensity cycle exercise (90% maximal aerobic power) with 12 well-trained and 11 untrained prepubertal boys. In adults, the SC was first thought to be associated with lactacidemia; however, it is generally accepted that blood lactate is not a primary determinant of the SC (4, 15, 27). In this study, the lack of a significant relationship between the SC and blood lactate values in heavy exercise suggests a poor relationship between the two variables. No correlation ($r = 0.44, P > 0.05$) between the SC and $\Delta$[lactate] was found in the study by Obert et al. (23). During heavy exercise, the $\Delta$[lactate] was significantly less in children than in the adults, amounting to an increase of only 0.6 mM. However, this was not unexpected given that the blood lactate concentration at LT is the same in both populations but lower in children after maximal exercise (12).

A current focus for research into the mechanisms responsible for the SC is the recruitment of low-efficiency type II muscle fibers during heavy exercise (5). Poole et al. (28) have demonstrated that the majority of the SC resides within the exercising muscle. In one of the few muscle biopsy studies in children, Oertel (24) reported that the proportion of type I fibers increased from 40% at birth to $\sim 60\%$ by 2 yr of age. From 2 yr onward, the relative proportions of type I and II fibers remained constant. Therefore, the available evidence suggests that maturation-related changes in muscle fiber-type distribution cannot explain differences in the amplitude of the SC between children and adults. Several recent studies have suggested that the amplitude of the $\dot{V}O_2$ SC may be reduced in running compared with cycling exercise (8, 19). The mechanisms that may be responsible for these differences include a greater involvement of the upper body during cycling, a greater intramuscular tension development during cycling, and a greater ability to store and subsequently release elastic energy during the stretch-shortening activity of running (8, 19).

In conclusion, the $\dot{V}O_2$ kinetics in treadmill running during moderate- and heavy-intensity exercise were found to be different in boys and men, with the boys exhibiting faster on-kinetics, greater primary increases in $\dot{V}O_2$, and a reduced SC. We suggest that these differences may arise because of children’s limited ability to generate ATP anaerobically, coupled with their greater ability to meet the energetic demands of exercise through aerobic pathways. Our results using treadmill running add to the limited number of studies defining children’s $\dot{V}O_2$ kinetic responses to exercise. More studies are required using a wider range of exercise-intensity domains, different test modalities, and assessment of maturity differences to define children’s $\dot{V}O_2$ kinetic responses to exercise more fully.

REFERENCES


