Effects of Exercise Mode on the Oxygen Uptake Kinetic Response to Severe-Intensity Exercise in Prepubertal Children

Fabiana A. Machado, Luiz G. A. Guglielmo, Camila C. Greco, and Benedito S. Denadai

The objective of this study was to verify the effect of the exercise mode on slow component of VO$_2$ (VO$_2$SC) in children aged 11–12 years during severe-intensity exercise. After determination of the lactate threshold (LT) and peak VO$_2$ (VO$_2$peak) in both cycling (CE) and running exercise (TR), fourteen active boys completed a series of “square-wave” transitions of 6-min duration at 75% $\Delta$ [75% $\Delta = LT + 0.75 \times (VO_2peak-LT)]$ to determine the VO$_2$ kinetics. The VO$_2$SC was significantly higher in CE ($180.5 \pm 155.8$ ml $\cdot$ min$^{-1}$) than in TR ($113.0 \pm 84.2$ ml $\cdot$ min$^{-1}$). We can conclude that, although a VO$_2$SC does indeed develop during TR in children, its magnitude is considerably lower than in CE during severe-intensity exercise.

The pulmonary oxygen uptake response (VO$_2$) from rest to exercise transition is characterized by temporal phases. The initial phase I response (the cardio-dynamic phase) lasts approximately 20 s and has a small contribution on the entire response. It represents the increase in VO$_2$ attributed to an increase in pulmonary blood flow and changes in gas stores (35). The subsequent exponential phase II represents the majority of the response and is potentially dictated by active muscle oxygen consumption (VO$_2$m; 4, 20, 29). Following this fast adaptive response, VO$_2$ levels off at intensities below lactate threshold (LT). However, at intensities above LT, VO$_2$ either attains a delayed steady state or continues to increase slowly until the end of exercise (VO$_2$ slow component—VO$_2$SC; 34). Many studies have described a close coupling between VO$_2$ and VO$_2$m response to exercise (24,26) and that even a small alteration of VO$_2$ over time (occurrence of VO$_2$SC) has its origin imposed by VO$_2$m (26). Relying on these evidences, the measurement of VO$_2$ response has been frequently used to assess exercise bioenergetics in several pathological and physiological conditions (10,27).

The physiological determinants of VO$_2$SC remain poorly understood. Among the possible mechanisms that can explain the VO$_2$SC are altered substrate utilization, modifications of fiber type recruitment, increased muscle temperature, and/or lactic acidosis. Since VO$_2$SC can reduce exercise tolerance by increasing the metabolic rate, experimental designs which investigate the underlying mechanism
of VO₂SC is of significance to our understanding of exercise energetics and the limitations to human performance (31).

Few studies have attempted to assess the existence or magnitude of the VO₂SC in children (1,18,36). Despite the varying exercise intensities imposed between these studies, data obtained in this population are conflicting regarding the existence of VO₂SC. Armon et al. (1) conducted the first study that sought to investigate the VO₂SC in children. In this study, seven adults and six children (6–12 years) completed 6 min of exercise on a cycle ergometer at various exercise intensities (25%Δ, 50%Δ and 75%Δ; i.e.; 25%, 50% and 75% of the difference between LT and VO₂peak, respectively). Sixteen children also completed 6 min at 50%Δ only. Armon et al. (1) observed that, at the 50%Δ work intensity, only 11 of the 22 children demonstrated a VO₂SC (linear regression slope of VO₂ as a function of time from 3 to 6 min). Moreover, at both 75% and 50%Δ, the VO₂SC were higher in adults (1.76 ± 0.63 and 1.27 ± 0.50 mL · kg · min⁻¹) than in children (0.20 ± 0.42 and 0.27 ± 0.73 mL · kg · min⁻¹). Similarly, Williams et al. (36) verified that the magnitude of VO₂SC and the relative contribution to the total amplitude were significantly greater in men than boys (boys, 18.6 ± 18.9 mL · min⁻¹ and 0.9 ± 1.2%; men, 115.9 ± 7.0 mL · min⁻¹ and 8.3 ± 1.0%, respectively) during treadmill running at heavy-intensity exercise (50%Δ). However, Fawkner and Armstrong (18) using exercise mode (cycle ergometer) and exercise intensity (40%Δ) similar to Armon et al. (1), clearly verified in 10.6 year-old boys, the existence of the VO₂SC (100 ± 60 ml · min⁻¹; 9.4 ± 4.6%).

Different methods to determine the VO₂SC used by Armon et al. (1; linear regression slope of VO₂ as a function of time from 3 to 6 min) and Fawkner and Armstrong (18; exponential model) could, at least in part, justify these contradictory findings. Confirming this possibility, Machado et al. (23) verified that the VO₂SC values in children during treadmill running at severe-intensity exercise (75%Δ) were dependent on the method of analysis (exponential model vs. ΔVO₂ between 6th and 3rd minute of exercise). However, the method (exponential model) and chronological age analyzed by Williams et al. (36) and Fawkner and Armstrong (18) were similar, which allows us to hypothesize that the exercise mode can influence the VO₂SC in children. In fact, in adults, the VO₂SC is higher in cycling than in running, independent of the aerobic training status (8,13,21). However, considering that: a) the O₂ delivery to active muscles is an important factor which can explain the different metabolic responses between running and cycling (15,16), and; b) the higher muscles’ potential of prepubertal children for oxygen utilization and/or efficiency with which oxygen is delivered to active muscles (18), it is possible to hypothesize that the effect of exercise mode on the VO₂SC is lesser in children. Therefore, the objective of this study was to verify the effect of the exercise mode on the oxygen uptake kinetic response to severe-intensity exercise in prepubertal children.

Material and Methods

Participants

Twenty 11–12 year-old healthy active boys, sexual maturation stages 1 and 2, determined using pubic hair index developed by Tanner (32), were included in this
study. Participants were not presently undertaking any regular exercise training. Written, informed consent was obtained from subjects and their parents, and ethical approval was granted by the Local Research Ethics Committee.

Experimental Design

The participants were required to visit the laboratory on five occasions within a period of two weeks. First visit consisted of familiarization with the ergometers (cycle ergometer and treadmill) and measurement of basic anthropometric variables: height, body mass, and skinfolds (tricipital and subescapular) for determination of percent body fat (22). In the second and third visits the subjects performed an incremental test to determine LT and VO₂peak for both running and cycling exercises. These two incremental tests were performed 2 days apart and in a randomized order. During the remaining two sessions, performed also in a randomized order, the participants performed 2 repetitions of square-wave transitions from rest to exercise intensity corresponded to 75%Δ during 6 min for both treadmill and cycle exercise. The transitions were separated by 1 hr of recovery (11). All tests were performed at the same time of day (± 2 hr) in a climate-controlled (21–22 °C) laboratory.

Procedures

All running tests were performed on a motorized treadmill (INBRAMED Super ATL, Porto Alegre, Brazil) with the gradient set at 1%. Cycle tests were conducted on a mechanically braked cycle ergometer (Monark 834 E, Stockholm, Sweden). Pedal frequency was maintained constant at 70 rpm for all cycle tests. Throughout the tests, the respiratory and pulmonary gas-exchange variables were measured using a breath-by-breath portable gas analyzer (Cosmed K4b², Rome, Italy). Before each test, the O₂ and CO₂ analysis systems were calibrated using ambient air and a gas of known O₂ and CO₂ concentrations according to the manufacturer’s instructions, while the K4b² turbine flowmeter was calibrated using a 3-l syringe (Cosmed K4b², Rome, Italy). During the exercise tests, pulmonary gas exchange was determined breath by breath. Heart rate (HR) was also monitored throughout the tests (Polar, Kempele, Finland). Earlobe capillary blood samples (25 μl) were collected into a glass tube and were analyzed for lactate concentration using an automated analyzer (YSI 2300, Ohio, USA).

Subjects performed incremental exercises (3-min stages) to volitional exhaustion to determine LT and VO₂peak during both treadmill (TR) and cycle ergometer (CE) tests. For the TR test, the initial running speed was 5 km.h⁻¹. The velocity increments between the stages were set at 1 km ∙ h⁻¹. All stages were followed by a 30-s period of rest. During this period, an earlobe capillary blood sample was collected. For the CE, the test began at 30 W, with increases of 30 W in power output. At the end of each stage an earlobe capillary blood sample was collected. Each participant was encouraged to give a maximal effort. 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 > 1.00. All participants attempted these criteria. The VO₂peak was defined as the highest 15 s VO₂ value reached during the incremental test. Plots of blood [lactate] against running speed or
power output and VO$_2$ were provided to two independent reviewers, who determined LT as the first sudden and sustained increase in blood lactate above resting concentrations. The running speeds and power outputs calculated to require 75%Δ were determine as:

\[
75%\Delta = \text{LT} + 0.75 \times (\text{VO}_2\text{peak} - \text{LT}) (1)
\]

The participants subsequently performed 2 repetitions of square-wave transitions of 6-min duration at 75%Δ for both CE and TR. After a 10 min warm-up at 50% VO$_2$max followed by 5 min of rest, the participants were instructed to perform the required intensity. At the start of cycling exercise, the participants pedaled against zero resistance, until a pedal cadence of 70 rpm was reached, and at that point, the preselected work rate was imposed and timing began. For running trials, the participants supported their body mass with their hands on the guard rails until leg speed matched treadmill belt speed, after which they let go of the guardrails and began running. For both exercise modes, the transition from rest to exercise took < 5 s. Blood samples (25 μL) were collected from the ear lobe immediately before (baseline) and after (final) the 6-min period of exercise. The difference between the final [lactate] and the baseline [lactate] was calculated (Δ [lactate]). After a 1-hr recovery period, the participants performed an identical square-wave transition using the mode of exercise as for the first test (11).

**Analysis of VO$_2$ Kinetics**

For each exercise transition, the breath-by-breath data were interpolated to give second-by-second values. The transitions for each exercise mode were then time aligned to the start of exercise and averaged to enhance the underlying response characteristics. Nonlinear regression techniques were used to fit VO$_2$ data after the onset of exercise with an exponential function. An iterative process ensured the sum of squared error was minimized (MatLab, version 6.5). The mathematical model consisted of three exponential terms, each representing one phase of the response (3,5). On the basis of previous literature (3), the model was constrained to aid in identification of the key parameters. The first exponential term started with the onset of exercise (time zero), whereas the other terms began after independent time delays.

\[
\text{VO}_2(t) = \text{VO}_2b + A_0 \times (1 - e^{-t/\tau_0}) \text{ (Phase 1 – cardio dynamic component)} \\
+ A_1 \times (1 - e^{-(t-TD_1)/\tau_1}) \text{ (Phase 2—primary component)} \quad (2) \\
+ A_2 \times (1 - e^{-(t-TD_2)/\tau_2}) \text{ (Phase 3 – slow component)}
\]

Where: VO$_2$ (t) is oxygen uptake at time t; VO$_2b$ is the VO$_2$ at rest; 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$_1$ e TD$_2$ are the time delays. The phase 1 term was terminated at the start of phase 2 (i.e., at TD1) and was assigned the value for that time (A'$_0$).

\[
A'_{0} = A_0 \times (1 - e^{-TD_1/\tau_0}) \quad (3)
\]
VO₂ at the end of phase 1 (A'₀) and the amplitude of phase 2 (A₁) were summed to calculate the amplitude of phase 2 (A'₁). The amplitude of the VO₂SC was determined as the increase in VO₂ from TD₂ to the end of exercise (defined A'₂), rather than from the asymptotic value (A₂), which may project beyond the value at 6 min (end-exercise). The slow component was also calculated in relative values.

\[ A'_2 \text{ relative} = \frac{A'_2}{A_1 + A'_2} \times 100 \] (4)

**Statistical Analysis**

Subjects with a 95% confidence interval for \( \tau_1 \) larger than \( \pm 7 \) s were excluded from further analysis. Data are presented as mean ± SD. Normality of the distribution was checked by the Shapiro-Wilk’s W test. The effects of exercise mode on submaximal and maximal data obtained during incremental test were analyzed using Paired \( t \) test. The Wilcoxon test was used to compare data obtained during constant work rate tests. The correlations were made using Pearson’s Product Moment correlation coefficients. Significance was set at \( p \leq .05 \).

**Results**

Out of the 20 participants recruited, 14 children satisfied the time constant 95% confidence interval criterion. The descriptive characteristics of these 14 subjects’ were: age (11.43 ± 0.43 years), body mass (41.8 ± 10.5 Kg), stature (148.5 ± 6.6 cm) and body fat (18.7 ± 7.8%).

Table 1 presents mean ± SD values of VO₂peak, peak blood lactate (Lacpeak), maximal heart rate (HRmax), maximal speed or power at VO₂peak (IVO₂peak), respiratory exchange ratio (R) and pulmonary ventilation (VE) obtained during incremental tests for both CE and TR. The VO₂peak and HRmax were significantly higher in TR than CE (\( p < .05 \)). There was no significant difference in Lacpeak, R and VE (\( p > .05 \)).

**Table 1** Mean ± SD Values of VO₂peak, Peak Blood Lactate (Lacpeak), Maximal Heart Rate (HRmax), Maximal Speed or Power at VO₂peak (IVO₂peak), Respiratory Exchange Ratio (R) and Pulmonary Ventilation (VE) Obtained During Incremental Tests for Both Cycle Ergometer (CE) and Treadmill (TR). \( N = 14 \)

<table>
<thead>
<tr>
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<th>TR</th>
<th>CE</th>
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<tbody>
<tr>
<td>VO₂peak (mL · kg · min⁻¹)</td>
<td>45.2 ± 5.3</td>
<td>42.5 ± 7.1*</td>
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<tr>
<td>Lacpeak (mM)</td>
<td>3.7 ± 2.4</td>
<td>4.7 ± 2.2</td>
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<tr>
<td>HRmax (bpm)</td>
<td>197.0 ± 8.0</td>
<td>189.4 ± 8.1*</td>
</tr>
<tr>
<td>IVO₂peak</td>
<td>10.5 ± 1.2a</td>
<td>117.1 ± 18.1b</td>
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<tr>
<td>R</td>
<td>1.05 ± 0.07</td>
<td>1.06 ± 0.10</td>
</tr>
<tr>
<td>VE (L · min⁻¹)</td>
<td>73.0 ± 11.9</td>
<td>71.5 ± 15.3</td>
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</table>

*\( p < .05 \) in relation to TR; \(^a\) units are km/h; \(^b\) units are Watts
Table 2 presents mean ± SD values of VO$_2$, %VO$_2$peak, intensity, HR and %HR$_{\text{max}}$ corresponding to LT obtained during the incremental tests for both CE and TR. The VO$_2$, %VO$_2$peak, HR and %HR$_{\text{max}}$ corresponding to LT were significantly higher in TR than CE ($p < .05$).

Table 3 presents mean ± SD values of model parameters using exponential model with three terms and Δ [La] for both CE and TR. The mean ± SD confidence intervals for $\tau_1$ and $A_1$ were 5 ± 1 s and 5 ± 2% respectively for TR, and 5 ± 2 s and 6 ± 1% respectively for CE. The values of $A_0$, $A'_1$ and VO$_{\text{final}}$ were significantly higher in TR than CE. In other way, the values of TD$_2$, VO$_{\text{SC}}$ ($A'_2$ and %A$'_2$) and Δ [La] were significantly higher in CE than TR.

Table 2  Mean ± SD Values of VO$_2$, %VO$_2$peak, IVO$_2$ peak, HR and %HR$_{\text{max}}$ Corresponding to Lactate Threshold Obtained During the Incremental Tests for Both Cycle Ergometer (CE) and Treadmill (TR). $N = 14$

<table>
<thead>
<tr>
<th>Parameters</th>
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<th>BE</th>
</tr>
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<tbody>
<tr>
<td>VO$_2$ (mL · kg · min$^{-1}$)</td>
<td>30.2 ± 8.9</td>
<td>23.9 ± 6.6*</td>
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<tr>
<td>%VO$_2$peak</td>
<td>66.8 ± 16.4</td>
<td>56.2 ± 15.4*</td>
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<tr>
<td>IVO$_2$ peak</td>
<td>6.40 ± 1.3a</td>
<td>49.2 ± 14.9b</td>
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<tr>
<td>HR (bpm)</td>
<td>146.6 ± 16.9</td>
<td>133.2 ± 13.6*</td>
</tr>
<tr>
<td>%HR$_{\text{max}}$</td>
<td>74.5 ± 8.9</td>
<td>70.3 ± 6.6*</td>
</tr>
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</table>

* $p < .05$ in relation to TR; a units are km/h; b units are Watts.

Table 3  Mean ± SD Values of Model Parameters Using Exponential Model With Three Terms and Δ [La] for Both Cycle Ergometer (CE) and Treadmill (TR). $N = 14$

<table>
<thead>
<tr>
<th>Parameters</th>
<th>TR</th>
<th>CE</th>
</tr>
</thead>
<tbody>
<tr>
<td>$A'_0$ (mL · min$^{-1}$)</td>
<td>254.5 ± 172.1</td>
<td>198.0 ± 162.5*</td>
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<tr>
<td>TD$_1$ (s)</td>
<td>15.1 ± 4.6</td>
<td>15.5 ± 6.4</td>
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<tr>
<td>$A'_1$ (ml/min)</td>
<td>1186.6 ± 323.8</td>
<td>1044.4 ± 209.9*</td>
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<tr>
<td>$\tau_1$ (s)</td>
<td>20.1 ± 3.4</td>
<td>21.5 ± 7.7</td>
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<tr>
<td>TD$_2$ (s)</td>
<td>104.7 ± 39.0</td>
<td>147.3 ± 44.3*</td>
</tr>
<tr>
<td>$\tau_2$ (s)</td>
<td>181.9 ± 129.5</td>
<td>199.4 ± 105.8</td>
</tr>
<tr>
<td>$A'_2$ (mL · min$^{-1}$)</td>
<td>113.0 ± 84.2</td>
<td>180.5 ± 155.8*</td>
</tr>
<tr>
<td>%A$'_2$</td>
<td>8.1 ± 5.2</td>
<td>13.2 ± 10.2*</td>
</tr>
<tr>
<td>VO$_{\text{final}}$ (mL · min$^{-1}$)</td>
<td>1299. ± 376.8</td>
<td>1223.0 ± 271.9*</td>
</tr>
<tr>
<td>Δ [La] (mM)</td>
<td>1.2 ± 0.9</td>
<td>3.0 ± 1.2*</td>
</tr>
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</table>

* $p < .05$ in relation to TR; $A'_0$, VO$_2$ at the end of phase 1; $A'_1$, sum of $A'_0$ and the amplitude of phase 2 ($A_1$); $A'_2$ e % $A'_2$, amplitude of the slow component expressed as absolute and relative values, respectively; VO$_{\text{final}}$, total change in VO$_2$; Δ[La], difference between the final (6 min) and the baseline (o min) blood lactate concentration. Note, $\tau_1$ e $\tau_2$, the time constants; TD$_1$ e TD$_2$, time delays; $A'_0$, VO$_2$ at the end of phase 1; $A'_1$, sum of $A'_0$ and the amplitude of phase 2 ($A_1$); $A'_2$ e % $A'_2$, amplitude of the slow component expressed as absolute and relative values, respectively; VO$_{\text{final}}$, total change in VO$_2$; Δ[La], difference between the final (6 min) and the baseline (o min) blood lactate concentration. *$p < .05$ in relation to TR.
Figure 1 shows the VO₂ kinetic response for one representative participant exercising at 75%Δ for both CE and TR. The correlation level between Δ [La] and VO₂SC (A’₂ and %A’₂) were statistically significant in TR (r = .81; r = .57), but not in CE (r = .04; r = .03), respectively.

Discussion

To our knowledge, this is the first study that has investigated the effect of the exercise mode on the oxygen uptake kinetic response to severe-intensity exercise in prepubertal children. The main find of this study was that children with similar age and maturational level present greater VO₂SC in CE than TR. This was in accordance with previous studies conducted in adults with different aerobic training status (8,13,21).

As expected, and consistent with previous studies involving children, both VO₂peak and LT (in L ∙ min⁻¹ and as a percentage of VO₂peak) were higher for TR than for CE (2,8). Excepting participants who are not specifically cycle trained, the influence of exercise mode on the VO₂peak and LT is also verified in adults (12). In this way, the mechanisms underpinning this difference seems not be dependent of the chronological age, and may be similar to those responsible for the difference in the magnitude of the VO₂SC between exercise modes, as discussed below.

Since the amplitude and pattern of the VO₂ kinetic response differs according to the exercise intensity domain (19), the effect of the exercise mode on the VO₂SC requires that participants are exercising at the same exercise intensity relative to the domain demarcators LT (moderate to heavy intensity) and critical power/velocity (heavy to severe intensity). Above critical power/velocity (CP / CV), in the severe-intensity domain, VO₂ increase progressively in a biexponential fashion, and the slow component causes the eventual attainment of VO₂peak. However, assessment of the threshold of severe-intensity exercise (CP / CV), is especially demanding in terms of both subject effort and testing time. Therefore, we choose to normalize the exercise intensity with reference to both the LT and VO₂peak determined for the two exercise modes (i.e., 75%Δ). This approach seems to be preferable to normalizing the exercise intensity by VO₂peak alone, since the latter can lead to differences in metabolic and perceptual stress, depending on the proximity of the exercise intensity to the LT. Anyway, as in our study the exercise intensities (75%Δ) obtained during both TR (92.6% VO₂peak; 90.1% IVO₂peak) and CE (89.1% VO₂peak; 87.5% IVO₂peak) were higher than CP reported in prepubertal boys during CE (81.8 ± 3.7% VO₂peak; 17) and running (85.5 ± 3.8% IVO₂peak; 6), 75%Δ will be probably above CP in the majority of children.

In CE, our results are in accordance with Fawkner and Armstrong (18) that, utilizing a double exponential model, clearly verified in 10.6 year-old boys the existence of the VO₂SC (100 ± 6 mL ∙ min⁻¹; 9.4 ± 4.6%) during heavy-intensity exercise (40%Δ). However, Armon et al. (1) observed that, at the 50%Δ work intensity, only 11 of the 22 children demonstrated a VO₂SC (linear regression slope of VO₂ as a function of time from 3 to 6 min). Thus, it is possible to hypothesize that the disagreement among studies performed in children age 10–12 years in cycling, can be explained, at least in part, by the different mathematical models used to analyze VO₂SC.
Figure 1 — Pulmonary oxygen uptake (VO$_2$) response for one representative participant exercising at 75% ∆ for both treadmill and cycle ergometer. The continuous line represents the fitted three exponential function, with the resulting residuals displayed below. The dotted line represents the predicted O$_2$ cost of the exercise and demonstrates the magnitude of the additional O$_2$ cost due to the slow component.
In the other hand, Williams et al. (36) and Fawkner and Armstrong (18) obtained values of VO2SC during running (18.6 ± 18.9 ml ∙ min⁻¹ and 0.9 ± 1.2%) and cycling (100 ± 60 ml ∙ min⁻¹ e 9.4 ± 4.6%) respectively, that were apparently different, although the intensities (40–50%Δ) and method of analysis (exponential model) were similar. Therefore, it would be possible to hypothesize that the exercise mode can also influence the VO2SC in children. In fact, we observed that the values of VO2SC (absolute and relative) obtained in TR were significantly smaller than in CE. Since this behavior was verified in adults with different aerobic training status (sedentary, cyclists and triathletes; 8, 13, 21), the mechanisms which determine higher VO2SC in cycling, seem not be dependent on chronological age and aerobic training status.

Although systemic factors such as increased cardio-respiratory work and hormonal changes may contribute to the VO2SC, there is convincing evidence that the major contribution originates from the exercising muscle (26). The excess VO2, due VO2SC, corresponds to a reduced efficiency (work accomplished/energy expended), is likely to be due to an increase in the phosphate cost of generating muscular force rather than the O2 cost of phosphate production (28). This scenario suggests that VO2SC is linked to intramuscular causes, as altered substrate utilization, modifications of fiber type recruitment, increased muscle temperature, and/or lactic acidosis.

Thus, the reasons of the greater VO2SC in CE compared with TR may be related to the biomechanical differences between exercise modes. Even using similar muscle groups, delta efficiency (25%) for cycling is much lower than for running (45%; 6). Its contractions with longer phases of isometric characteristics (25) somehow may influence, through the decrease of muscular pump action, venous return and muscular blood flow during exercise. Besides, cycling involves high levels of intramuscular tension throughout the pedal revolution (15), which could lead to occlusion of vessels (16) that would impede blood flow and oxygen delivery to the muscles. Differently, eccentric muscle action may have two important consequences for the oxygen cost of running. Firstly, the metabolic cost of eccentric exercise is substantially lower than that of comparable concentric exercise (33). Secondly, the stretch-shortening cycle in running allows for storage of elastic energy during the eccentric phase and its subsequent release during the concentric phase of the action. In addition, running has periods of low force production, when the body is airborne, which should facilitate blood flow and oxygen delivery. Therefore, these different types of muscle contraction can increase the recruitment of fibers with a lower oxidative capacity (type II) and/or to promote the progressive recruitment of fibers with a greater oxidative capacity, during cycling compared with running when exercising at the same relative intensity (i.e., 75%Δ).

Several studies have reported a close relationship between blood lactate accumulation and both the temporal profile and the magnitude of the VO2SC (14,30). Indeed, we verified that the Δ[La] was significantly higher in CE than TR, which could explain the greater VO2SC in CE. However, there was significantly correlation between Δ[La] and VO2SC only in TR. Differently, Billat et al. (8) found that the magnitude of the VO2SC and the level of blood lactate accumulation were correlated for cycling (r = .66) but not for running (r = .12). Moreover, the Δ[La] obtained during TR and CE were apparently lower than found by Carter et al. (13)
in adults (4.0 × 5.3 mM, respectively) at the same exercise intensity (75%Δ), although the children presented VO₂SC in both exercise modes. Taken together with our results, these findings indicate that the link between end blood lactate and VO₂SC can be coincidental rather than causal.

In conclusion, this is the first study that has compared the effect of the exercise mode on the oxygen uptake kinetic response to severe-intensity exercise in prepubertal children. We have clearly identified that, although a VO₂SC does indeed develop during TR in children, its magnitude is considerably lower than in CE during severe-intensity exercise. Thus, the mechanisms which determine higher VO₂SC in CE, seem not be influenced by age-dependent change in the muscles’ potential for O₂ utilization.

References


